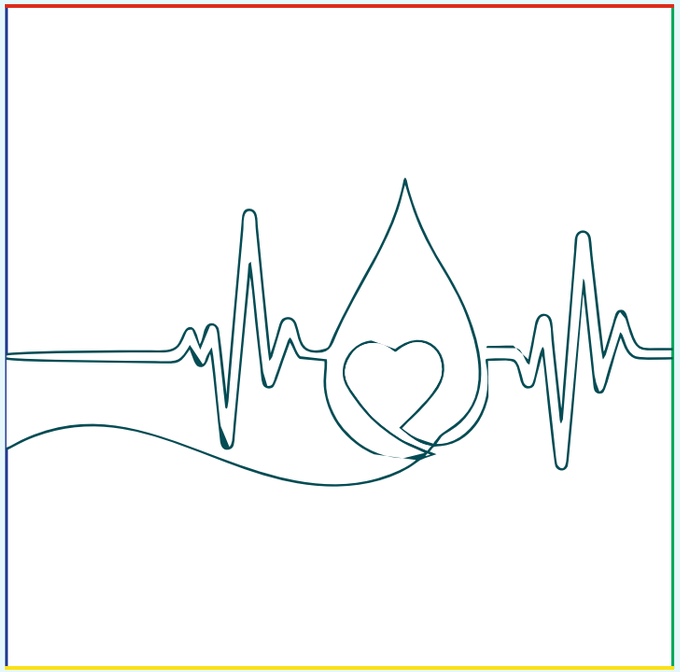
STANDARD TREATMENT GUIDELINES

Emergency Medicine



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STANDARD TREATMENT GUIDELINES

for

Emergency Medicine

Foreword

At the outset, I appreciate the work done by the respective thematic teams and coordination done by the DME. The Standard Treatment Guidelines (STG) were prepared and published in 2021 in the thick of the Covid pandemic. On the last page of these volumes the road map was mentioned. The few points are mentioned here for the recall.

"The Department of Health has been taking a systematic approach of creating and enabling multiple initiatives with a focus on prevention along with improving health care services. Health care service delivery is one of the most important services and is always seen as a barometer to assess the Governance. While it is important to develop infrastructure, an essential prerequisite is to develop systems and processes to bring in standardization in management of patient care. ……The foundation is laid and we take up the challenge to work on the unfinished agenda."

It was mentioned in the road map to have institutional mechanism to ensure updation of Standard Treatment Guidelines. The next step that was suggested was to do analysis of Karunya Arogya Suraksha Padhati (KASP) and standard treatment guidelines to work on developing a Balance Score Card to give information regarding compliance from the Hospitals and to build a "feedback loop" to improve. These initiatives remained at concept level on the last page! But following detailed discussions with Dr Vishwanathan, Director Medical Education, some of the foundational things were prioritized and given an impetus to take it to finality. In this journey, many committed doctors from various Medical Colleges of respective specialties participated. The previous coordination team members and experts were also consulted and they also participated in discussions and these Standard Treatment Guidelines are prepared.

The standard treatment guidelines will be made available in the Kerala Health portal (health.kerala.gov.in). This will enable the resource book availability not only to people within the state but to all in the country and outside our borders as well. I am confident that it will be used by students and practicing doctors. We request inputs based on the research from the Specialists and Experts. The teams shall continue to update and make any required changes in the STG by doing periodic updates.

The most important thing we all need to internalize is to have a shared vision and

work as a team to reach to a state of 'excellence'. If we take a look at the preparation of the Directorate Medical Education Management Information System, documents of each Medical Colleges, it provides information regarding 'what we are, what we do and what we aspire to do', pandemic preparedness, AMR accreditation and many more such initiatives taken on scale, which are all outcomes of collective TEAM work. This has laid a foundation for involving all the stakeholders including undergraduate and postgraduate students. This should encourage the teams in Medical Colleges to believe in themselves and build future initiatives on such a sound platform.

I express my sincere thanks to Dr Vishwanathan for his patience and bearing with relentless follow ups! I also take this opportunity to thank each and every team and their members and everyone from Directorate Medical Education and Medical Colleges who supported these initiatives.

I would like to express my sincere gratitude to all those who have contributed to publish these Standard Treatment Guidelines.

I wish all the success to DME team to make Kerala MCH as a premier knowledge hub in Medical Science.

Dr Rajan Khobragade IAS

Additional Chief Secretary
Health & Family Welfare and
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Govt of Kerala.

Message

Patient care today demands evidence-based, standardized, and contextually relevant clinical practice. In this regard, the publication of the **Second Edition of the Standard Treatment Guidelines** marks an important step forward in strengthening the quality, consistency, and accountability of healthcare delivery in Kerala.

The first edition laid a strong foundation for uniform clinical practice across specialties and super specialties. Since then, advances in medical knowledge, evolving treatment modalities, and the growing need for periodic updating have made it essential to revisit and refine these guidelines. The present edition reflects this commitment to continuous improvement and clinical excellence.

I am pleased to note that subject experts from various disciplines of Government Medical Colleges, private institutions and professional bodies have contributed as resource persons in the preparation of these guidelines. Their academic expertise, practical insight, and dedicated involvement have greatly enriched this edition. I deeply appreciate the sincere efforts of all the conveners, contributors, and coordinators whose collective commitment and teamwork made this publication possible.

These guidelines will serve as a valuable reference for clinicians, teachers, trainees, and healthcare institutions, helping to promote evidence-based decision-making and improve patient outcomes. I am confident that this edition will further support standardization of care and contribute to the advancement of medical education and clinical practice in the State.

I congratulate everyone involved in this commendable effort and commend this publication to all healthcare professionals.

Dr. K. V. Viswanathan
Director of Medical Education
Government of Kerala

Committee Members for Standard Treatment Guidelines
Emergency Medicine

Contents

Section I	
Emergency Department Organization & Triage	11
Scope	12
Introduction	12
Objectives	13
Principles of Emergency Triage	13
Emergency Department Zones	14
Time Targets for Assessment	15
Initial Assessment - ABCDE Approach	15
Time-Sensitive Emergencies	18
Initial Investigations in Critically Ill Patients	18
Documentation	19
Referral and transfer policy	19
Infection Prevention and Safety	19
Staffing Recommendations	19
Algorithms	20
Section II	
Initial Resuscitation & Critical Care Pathways	22
Scope	23
Introduction	23
Recognition of Critical Illness	24
Initial Resuscitation Principles	25
Airway Management	25
Oxygen Therapy	27
Shock Recognition	28
Fluid Resuscitation	29
Vasopressor Use	29
Cardiac Arrest Recognition	30

Mechanical Ventilation	30
Altered Mental Status	31
Interfacility Transfer of Critically Ill Patients	31
Documentation	32
Algorithms	33
Key Performance Indicators	36
References	36
Section III	
Cardiovascular Emergencies	37
Scope	38
Introduction	38
Acute Coronary Syndrome	39
STEMI Pathway	40
NSTEACS	42
Hypertensive Emergency	43
Arrhythmias	43
Acute Heart Failure	44
Cardiogenic Shock	45
Pulmonary Embolism	46
Aortic Dissection	46
Monitoring	47
Disposition	47
Algorithms	48
Quality Indicators	53
References	53
Section IV	
Neurological Emergencies	54
Scope	55
Introduction	55
Acute Ischemic Stroke	56

Intracranial Hemorrhage	60
Seizure & Status Epilepticus	60
Altered Sensorium	61
Meningitis / Encephalitis	62
Head Injury	63
Raised Intracranial Pressure	63
Monitoring	64
Disposition	64
Algorithms	65
Quality Indicators	70
References	70
Section V	
Respiratory Emergencies	71
Scope	72
Acute Severe Asthma	72
Acute Exacerbation of COPD (AECOPD)	75
Pneumonia	80
Acute Respiratory Distress Syndrome (ARDS)	85
Acute Respiratory Failure	92
Non-Invasive Ventilation (NIV) & Intubation Criteria	99
Pneumothorax	105
Acute Pulmonary Edema (APE)	109
Algorithms	114
Section VI	
Endocrine & Metabolic Emergencies	116
Scope	117
Introduction	118
Definitions	118
Diabetic Ketoacidosis (DKA)	119
Hyperosmolar Hyperglycemic State (HHS)	135

Hypoglycemia	148
Hyponatremia	151
Hypernatremia	158
Hyperkalemia	160
Thyroid Storm	163
Myxedema Coma	168
Adrenal Crisis	171
Algorithms	174
References	176
Section VII	
Infectious Disease & SEPSIS	177
Sepsis & Septic Shock	178
Dengue Fever	184
Leptospirosis	190
Malaria	195
Scrub Typhus	201
Algorithms	206
References	209
Section VIII	
Trauma Emergencies	210
ATLS Approach	211
Polytrauma	217
Head Injury	218
Chest Trauma	219
Abdominal Trauma	220
Spine Trauma	221
Orthopedic Emergencies	222
Burns	223
Trauma Imaging	224
Massive Transfusion Protocol (MTP)	224

Algorithms	226
Section IX	
Paediatric Emergencies	229
Scope	230
Introduction	231
Definitions	231
Paediatric Triage	232
Paediatric Respiratory Distress	233
Paediatric Shock	240
Febrile Seizure	247
Paediatric Trauma	253
Paediatric Dehydration	261
Paediatric Sepsis	268
Paediatric Poisoning	274

SECTION I
Triage, Initial Assessment and Emergency Department Organization

Scope

This protocol outlines the standard approach for triage, initial assessment, stabilization, prioritization, and disposition of patients presenting to Emergency Departments in Government Medical Institutions across Kerala. The guideline applies to Primary Health Centres, Taluk Hospitals, District Hospitals, Government Medical Colleges, and other tertiary care institutions.

This section establishes a uniform Emergency Medicine framework for:

- Rapid identification of critically ill patients
- Prioritization based on severity
- Standardized ABCDE assessment
- Early resuscitation
- Appropriate referral and transfer
- Timely specialist consultation
- Structured patient flow in Emergency Departments

The protocol is intended for:

- Emergency Physicians
- Casualty Medical Officers
- Junior Residents
- Nurses
- Paramedics
- Emergency Care Assistants
- Trauma Teams
- Critical Care Teams

Introduction

Emergency Departments are high-risk, high-volume clinical environments where timely recognition and intervention significantly influence morbidity and mortality. Delayed identification of critically ill patients may lead to preventable deterioration and death.

Triage is the process of sorting and prioritizing patients according to the urgency of their clinical condition rather than the order of arrival. A structured triage system improves patient safety, optimizes utilization of resources, and ensures early intervention for time-sensitive emergencies including:

- Acute Coronary Syndrome
- Stroke
- Trauma
- Sepsis

- Respiratory Failure
- Poisoning
- Shock
- Obstetric Emergencies
- Paediatric Emergencies

All Government Emergency Departments in Kerala shall adopt a standardized triage and emergency assessment system based on:

- ABCDE approach
- Red–Yellow–Green categorization
- Early identification of critical illness
- Dynamic reassessment

The triage process should be completed within 2–3 minutes of patient arrival.

Objectives

The objectives of this protocol are:

1. To standardize emergency patient assessment across Kerala Government institutions.
2. To ensure timely identification of critically ill patients.
3. To reduce mortality and morbidity due to delayed intervention.
4. To establish a uniform triage policy.
5. To prioritize time-sensitive emergencies.
6. To facilitate appropriate referral and transfer.
7. To improve communication among emergency care teams.
8. To optimize Emergency Department workflow and patient safety.

Principles of Emergency Triage

The following principles shall be followed in all Emergency Departments:

- Triage is based on severity of illness and not on “first come, first served” basis.
- Clinical judgement and common sense are essential.
- Triage is a dynamic process and patients may be upgraded or downgraded after reassessment.
- Critically ill patients should receive immediate attention.
- Children and pregnant women should receive priority assessment.
- Every unconscious patient shall be treated as critically ill until proven otherwise.
- Time-sensitive conditions should be identified immediately.
- Stabilization should begin simultaneously with evaluation.

Emergency Department Zones

A. Red Zone (Priority 1)

Patients with actual or impending ABCDE compromise requiring immediate resuscitation and continuous monitoring.

Conditions requiring Red Zone allocation

Airway

- Stridor
- Obstructed airway
- No spontaneous breathing
- Severe maxillofacial injury
- Blood/vomitus compromising airway

Breathing

- Respiratory rate >30/min or <12/min
- Severe respiratory distress
- SpO_2 <90% (except COPD/ILD)
- Open chest wounds
- Tension pneumothorax
- Significant blunt chest trauma

Circulation

- Absent central pulse
- Pulse >140/min or <50/min
- SBP <90 mmHg
- Shock
- Active severe bleeding
- Massive hematemesis
- Massive hemoptysis
- STEMI with instability

Disability

- GCS <9
- Active seizures
- Stroke within thrombolysis window
- Poisoning with altered sensorium
- Coma
- Hanging
- Drowning

Exposure

- Temperature >103°F with altered sensorium

- Hypoglycemia with altered mental status
- Major trauma

B. Yellow Zone (Priority 2)

Patients who are clinically stable without immediate ABCDE compromise but requiring urgent assessment and observation.

Examples

- Moderate respiratory distress
- Acute abdominal pain
- Hematemesis without shock
- Severe headache
- Fever with bleeding manifestations
- Acute urinary retention
- Stable poisoning
- Stable trauma
- Fractures without vascular compromise
- Hyperglycemia
- Pregnant patients with pain/bleeding

C. Green Zone (Priority 3)

Stable patients with minor illnesses/injuries suitable for ambulatory management.

Examples

- Minor wounds
- Mild fever
- Minor musculoskeletal pain
- Stable chronic complaints
- Prescription refills
- Mild upper respiratory infections

Time Targets for Assessment

Priority	Target Time to Physician
Red	Immediate (within 5 min)
Yellow	Within 15 min
Green	Within 30 min

Initial Assessment – ABCDE Approach

All critically ill patients shall undergo structured ABCDE assessment.

A. Airway (A)

Objectives

- Assess airway patency
- Identify obstruction
- Protect cervical spine when trauma suspected

Assessment

- Ability to speak
- Presence of stridor
- Gurgling/snoring
- Facial trauma
- Vomitus/blood
- Foreign body

Immediate Management

- Positioning
- Head tilt–chin lift
- Jaw thrust if cervical injury suspected
- Suction secretions
- Oropharyngeal/nasopharyngeal airway
- Endotracheal intubation if required

B. Breathing (B)

Assessment

- Respiratory rate
- Work of breathing
- Chest expansion
- Air entry
- Oxygen saturation
- Breath sounds

Management

- Oxygen supplementation
- Bag-mask ventilation if needed
- Nebulization if indicated
- Non-invasive ventilation
- Mechanical ventilation if indicated

Target Oxygen Saturation

- General patients: >94%
- COPD patients: 90–92%

C. Circulation (C)

Assessment

- Heart rate
- Blood pressure
- Peripheral perfusion
- Capillary refill
- Skin temperature
- Urine output

Management

- IV access
- Fluid resuscitation
- Hemorrhage control
- Vasopressors if indicated
- Blood transfusion if needed

Fluid Resuscitation

Adults:

- 500 mL crystalloid bolus initially
- Up to 30 mL/kg in shock

Children:

- 20 mL/kg bolus

D. Disability (D)

Assessment

- AVPU
- Glasgow Coma Scale
- Pupillary examination
- Blood glucose

Management

- Correct hypoglycemia
- Seizure management
- Airway protection if GCS <9
- Neuroimaging when indicated

E. Exposure (E)

Assessment

- Full body examination
- Trauma survey
- Skin lesions/rashes

- Bleeding
- Burns
- Temperature

Management

- Prevent hypothermia
- Cover patient after examination

Time-Sensitive Emergencies

The following conditions require immediate identification and activation of disease-specific protocols:

Condition	Protocol
STEMI	ACS Protocol
Acute Stroke	Stroke Protocol
Sepsis	Sepsis Bundle
Trauma	ATLS Protocol
Snakebite	Snakebite Protocol
Status Epilepticus	Seizure Protocol
DKA/HHS	Endocrine Emergency Protocol

Initial Investigations in Critically Ill Patients

Mandatory

- Random Blood Sugar
- ECG
- CBC
- Renal Function Test
- Electrolytes
- ABG/VBG

As Indicated

- Troponin
- Chest X-ray
- NCCT Brain
- Ultrasound/POCUS
- Lactate
- Blood cultures

- Coagulation profile

Documentation

The following should be documented:

- Triage category
- Vital signs
- Time of arrival
- Initial assessment findings
- Interventions performed
- Time-sensitive decisions
- Referral details
- Consent

Referral and Transfer Policy

Patients requiring higher-level care should be stabilized prior to transfer.

Mandatory before transfer

- Airway secured if indicated
- Hemodynamic stabilization
- IV access
- Oxygen support
- Relevant investigations
- Communication with receiving centre
- Referral documentation

Infection Prevention and Safety

- Hand hygiene mandatory
- PPE use according to risk
- Isolation for infectious diseases
- Biomedical waste segregation
- Needle-stick injury reporting

Staffing Recommendations

Primary Care Hospitals

- Emergency Medical Officer
- Nursing staff trained in triage

District Hospitals

- Emergency physician preferred
- Dedicated triage nurse
- Critical care trained nurses

Tertiary Care Centres

- Dedicated Emergency Medicine Department
- Emergency Physicians
- Trauma teams
- Critical care backup
- Stroke/STEMI pathways

Algorithm

Emergency Department Triage Pathway

Initial Assessment and Management of patient in emergency care unit

ABCDE table

Criteria	Assess	Act / Intervene / Treat
Airway	Protection of patency	Assess & maintain patency by simple measures- Position of comfort, head tilt-chin lift, jaw thrust (if cervical injury), inspect mouth & remove any secretions/ blood/foreign body if visible, apply suction, airway adjuncts (oropharyngeal/ nasopharyngeal airway)
Breathing	Oxygenation - SpO2 Ventilation	Assess Respiratory rate, effort, chest expansion & air movement, lung & airway sounds & SpO2 Assist ventilation if required Commence CPR if patient is unconscious and absence of normal breathing Apply 02 to maintain SpO2 > 94%

Criteria	Assess	Act / Intervene / Treat
Circulation	Perfusion to tissues CRT, Pulse, BP	Assess Skin temperature, colour Heart Rate, rhythm Peripheral & central pulses Capillary refill BP Urine Output Monitor vital signs frequently by multi para monitor Action IV cannulation If SBP <95th percentile for age in children or < 90 mm of Hg in adults- IV/Intra Osseous RL or NS 20 ml/kg bolus in children or 500 ml or 30 ml/kg bolus in adults.
Disability	AVPU/GCS, Pupils, Blood glucose (BGL)	Assess & monitor AVPU/GCS + pupils If GCS < 9 &/or rapidly deteriorating- ET intubation by emergency physician for airway protection Assess & monitor Blood Glucose level by finger prick test If BGL < 40 mg/di or unconscious/confused- Administer IV 50% Glucose 50 ml (if NA- 25% D 100 ml IV) in adults and IV/ Intra Osseous 0.5-1 g/kg (2-4 ml/kg of 25% D or 5-10 ml/kg of 10% D) in children
Exposure	Expose patient for examination	After examination and initial treatment, cover the patient to prevent hypothermia

Key Performance Indicators

- Door-to-triage time
- Door-to-physician time
- Door-to-ECG time
- Door-to-needle time in STEMI
- Door-to-CT time in Stroke
- Sepsis antibiotic time
- Mortality in Red Zone patients

SECTION II
INITIAL RESUSCITATION & CRITICAL CARE PATHWAYS

Scope

This protocol provides a standardized approach for the recognition, assessment, stabilization, and early critical care management of acutely ill patients presenting to Emergency Departments in Government Medical Institutions across Kerala.

The guideline applies to:

- Primary Health Centres
- Taluk Hospitals
- District Hospitals
- Government Medical Colleges

This section focuses on:

- Recognition of critical illness
- Airway management
- Oxygen therapy
- Shock recognition
- Fluid resuscitation
- Vasopressor initiation
- Cardiac arrest recognition
- Mechanical ventilation
- Transfer of critically ill patients

The protocol is intended for:

- Emergency Physicians
- Casualty Medical Officers
- Critical Care Teams
- Nurses
- Paramedics
- Trauma Teams

Introduction

Critically ill patients require rapid identification and early resuscitation to prevent organ dysfunction, cardiac arrest, and mortality. Delayed recognition of airway compromise, respiratory failure, circulatory shock, or altered mental status may lead to irreversible deterioration.

A structured systematic approach using the ABCDE method ensures:

- Immediate recognition of life-threatening conditions
- Prioritization of interventions
- Simultaneous assessment and treatment

- Reduction in preventable mortality

All Emergency Departments shall adopt:

- ABCDE assessment
- Early warning signs identification
- Protocolized resuscitation
- Standard monitoring
- Early escalation of care

Initial stabilization should begin immediately upon identification of critical illness and should not be delayed for investigations.

Recognition of Critical Illness

Clinical Features Suggesting Critical Illness

A patient should be considered critically ill if any of the following are present:

Airway

- Stridor
- Gurgling
- Inability to maintain airway
- Facial trauma
- Blood/vomitus obstructing airway

Breathing

- Respiratory distress
- RR >30/min or <8/min
- Cyanosis
- Use of accessory muscles
- Silent chest
- SpO_2 <90%
- Apnea

Circulation

- SBP <90 mmHg
- MAP <65 mmHg
- Tachycardia >140/min
- Bradycardia <40/min
- Cold clammy extremities
- Delayed capillary refill
- Active bleeding

Disability

- GCS <9

- Altered sensorium
- Seizures
- Unresponsiveness
- Agitation/confusion

Exposure

- Severe trauma
- Burns
- Hypothermia
- Hyperthermia
- Rash suggestive of sepsis/toxidrome

Initial Resuscitation Principles

The following principles shall guide all resuscitation:

1. Simultaneous assessment and treatment
2. Airway takes priority
3. Oxygenation and ventilation should be ensured early
4. Control life-threatening hemorrhage immediately
5. Frequent reassessment is mandatory
6. Early specialist involvement
7. Documentation of interventions and timings
8. Use point-of-care ultrasound where available
9. Early escalation to ICU if needed

Airway Management

A. Objectives

- Maintain airway patency
- Prevent aspiration
- Ensure adequate oxygenation and ventilation

B. Airway Assessment

Signs of Airway Compromise

- Stridor
- Gurgling
- Snoring
- Hoarseness
- Silent airway
- Facial trauma
- Burns to airway
- Altered sensorium

C. Initial Airway Maneuvers

Non-invasive Maneuvers

- Position of comfort
- Head tilt–chin lift
- Jaw thrust (if cervical injury suspected)
- Suction of secretions
- Removal of visible foreign body

Airway Adjuncts

- Oropharyngeal airway
- Nasopharyngeal airway

D. Indications for Endotracheal Intubation

Absolute Indications

- GCS <8
- Cardiac arrest
- Respiratory arrest
- Severe hypoxemia
- Severe hypercapnia
- Inability to protect airway
- Severe shock
- Status epilepticus
- Airway burns

Relative Indications

- Exhaustion
- Severe trauma
- Progressive deterioration

E. Rapid Sequence Intubation (RSI)

Preparation

- Airway assessment
- Monitoring
- Suction ready
- Oxygen source
- Backup airway devices
- Difficult airway cart

Preoxygenation

- 100% oxygen for 3–5 minutes

Drugs (Examples)

Induction Agents

- Etomidate

- Ketamine
- Propofol

Paralytics

- Succinylcholine
- Rocuronium

F. Post-intubation Care

- Confirm tube position clinically and by $ETCO_2$ if available
- Secure tube
- Chest X-ray
- Sedation and analgesia
- Ventilator initiation

Oxygen Therapy

A. Indications

- Hypoxemia
- Respiratory distress
- Shock
- Trauma
- ACS
- Stroke
- Cardiac arrest

B. Target Oxygen Saturation

Patient Group	**Target SpO_2**
General patients	94–98%
COPD/Hypercapnic risk	88–92%
Post cardiac arrest	90–98%

C. Oxygen Delivery Devices

Device	**Flow**
Nasal cannula	1–6 L/min
Face mask	5–10 L/min
Non-rebreathing mask	10–15 L/min
High-flow nasal oxygen	As per device
Bag-mask ventilation	Emergency use

D. Non-Invasive Ventilation (NIV)

Indications

- COPD exacerbation

- Acute pulmonary edema
- Type II respiratory failure

Contraindications

- Cardiac arrest
- Vomiting
- Inability to protect airway
- Severe agitation
- GCS less than 8

Shock Recognition

Shock is a state of circulatory failure resulting in inadequate tissue perfusion and oxygen delivery.

Clinical Features of Shock

A. Hemodynamic Features
- SBP <90 mmHg
- MAP <65 mmHg
- Tachycardia
- Narrow pulse pressure

B. Clinical Features
- Altered sensorium
- Cold extremities
- Delayed CRT
- Oliguria
- Tachypnea

C. Laboratory Features
- Metabolic acidosis
- Elevated lactate
- Base deficit

Types of Shock

Type	Examples
Hypovolemic	Haemorrhage, dehydration
Cardiogenic	ACS, myocarditis
Distributive	Sepsis, anaphylaxis, Neurogenic
Obstructive	Tension pneumothorax, tamponade

Fluid Resuscitation

1. Crystalloids

Preferred fluids:

- Normal saline
- Ringer Lactate

2. Initial Fluid Therapy

Adults

- 500 mL bolus initially
- Up to 30 mL/kg in septic shock

Children

- 20 mL/kg bolus

3. Endpoints of Fluid Resuscitation
- Improved BP
- Improved urine output
- Reduced lactate
- Improved mentation
- Warm extremities

4. Precautions
- Avoid fluid overload
- Use caution in heart failure
- Reassess frequently

Vasopressor Use

1. Indications
- Persistent hypotension despite fluids
- Septic shock
- Cardiogenic shock

2. First-line Vasopressor

Noradrenaline

Preferred vasopressor in:

- Septic shock
- Vasodilatory shock

Can be initiated through large peripheral vein temporarily with close monitoring if central access unavailable.

3. Monitoring During Vasopressor Use
- Continuous BP monitoring
- Heart rate

- Urine output
- Peripheral perfusion
- Extravasation monitoring

Cardiac Arrest Recognition

Features

- Unresponsiveness
- Absence of normal breathing
- Absent carotid pulse

Immediate CPR should be initiated.

1. Initial Cardiac Arrest Management

Immediate Actions

- Activate the team
- Start high-quality CPR
- Attach monitor/defibrillator
- Assess rhythm
- Defibrillate shockable rhythm

2. Reversible Causes (Hs & Ts)

Hs

- Hypoxia
- Hypovolemia
- Hypothermia
- Hydrogen ion (acidosis)
- Hyper/Hypokalemia

Ts

- Tension pneumothorax
- Tamponade
- Toxins
- Thrombosis pulmonary
- Thrombosis coronary

Mechanical Ventilation

1. Indications

- Respiratory arrest
- Severe hypoxemia
- Hypercapnic respiratory failure
- Shock

- Severe head injury
- Severe metabolic acidosis
- Airway protection

2. Initial Ventilator Strategy

General Principles

- Lung protective ventilation
- Low tidal volume
- Adequate PEEP
- Avoid hyperoxia

3. Monitoring

- SpO_2
- $ETCO_2$
- ABG
- Ventilator synchrony
- Peak airway pressure

Altered Mental Status

Patients with altered mental status require immediate assessment for:

- Hypoglycemia
- Stroke
- Sepsis
- Poisoning
- Seizures
- Trauma

Initial Evaluation

- ABCDE assessment
- Blood glucose
- Neurological examination
- CT brain if indicated
- Electrolytes
- ABG/VBG

Interfacility Transfer of Critically Ill Patients

1. Principles

- Stabilize before transfer

- Ensure airway and circulation
- Appropriate escort team
- Pre-transfer communication mandatory

2. Before Transfer

Airway

- Intubate if required

Breathing

- Oxygen/ventilator support

Circulation

- IV access
- Fluids/vasopressors

Documentation

- Referral note
- Investigation reports
- Consent

3. During Transfer

- Monitoring
- Oxygen supply
- Resuscitation drugs
- Defibrillator availability if indicated

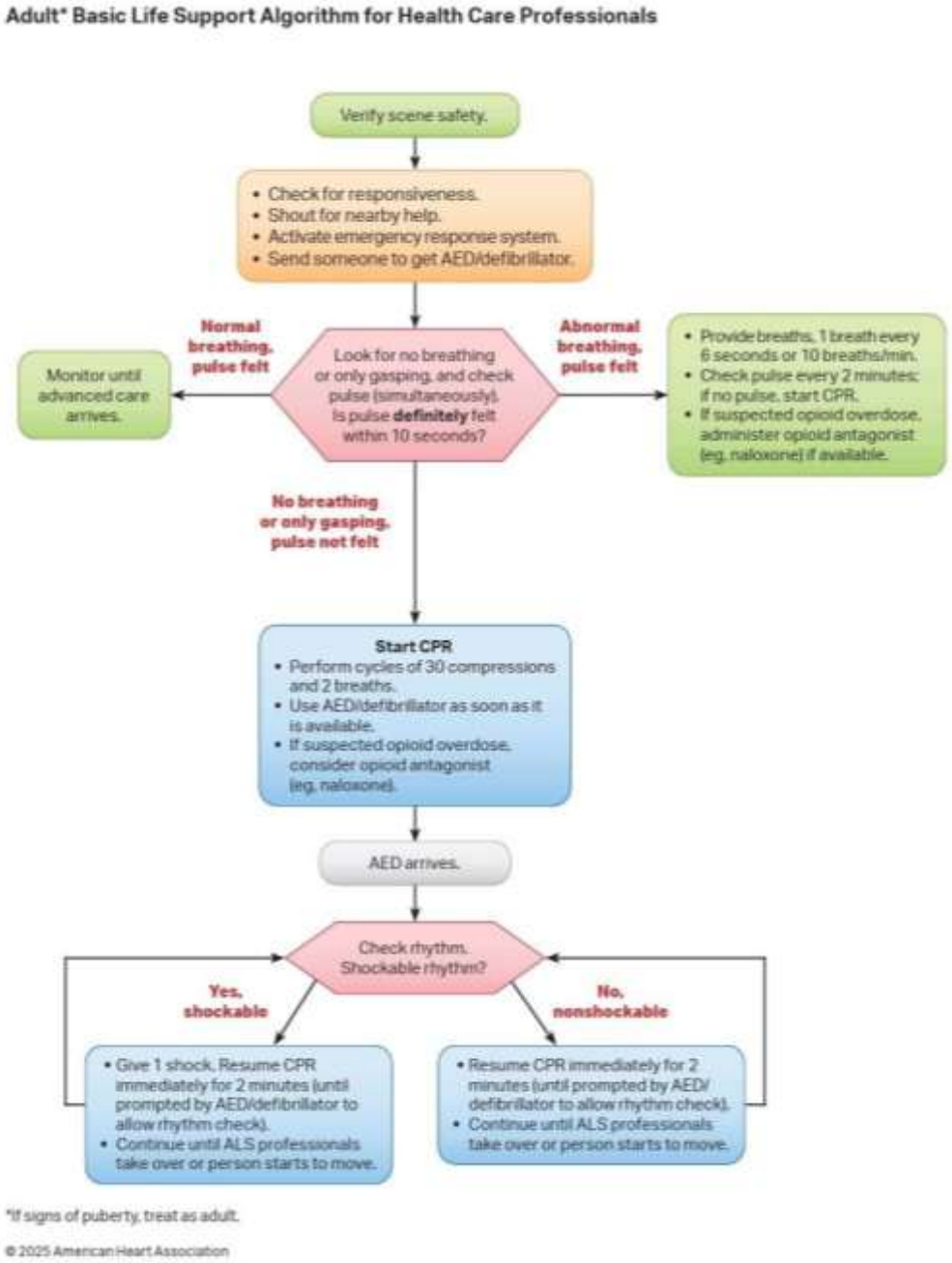
Documentation

The following shall be documented:

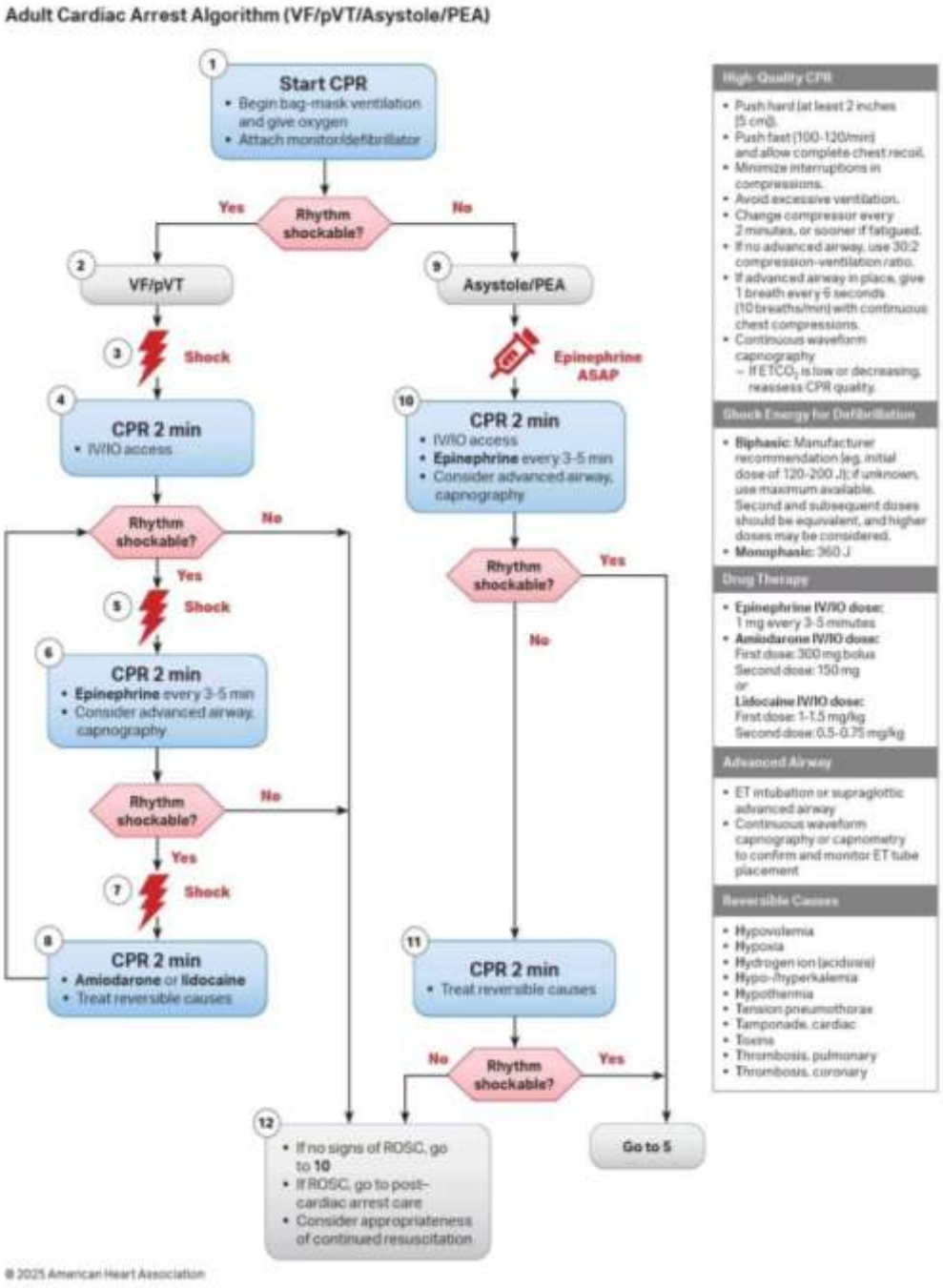
- Time of arrival
- Initial vitals
- ABCDE findings
- Interventions
- Drugs administered
- Response to treatment
- Referral details

Algorithms

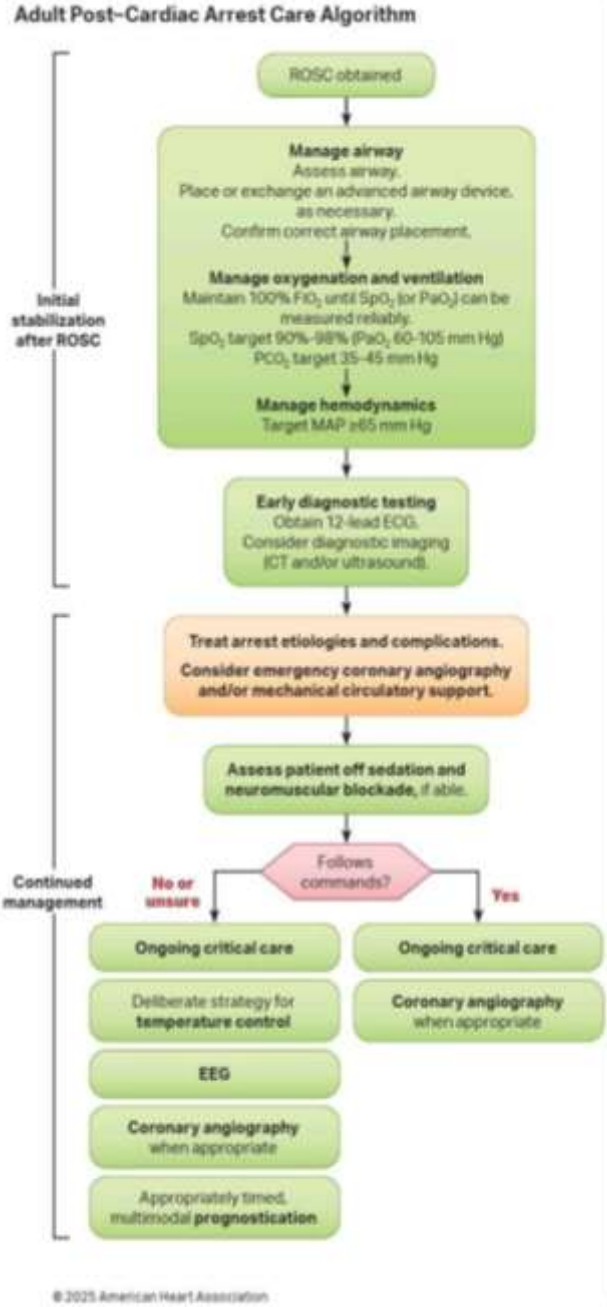
Algorithm 1 - BLS Algorithm



Algorithm 2 - Cardiac Arrest Algorithm



Algorithm 3 – Post Cardiac Arrest Algorithm



Initial Stabilization After ROSC

Resuscitation is ongoing during the post-ROSC phase, and many of these activities can occur concurrently.

Manage airway: Assess and consider placement or exchange of an advanced airway device (usually endotracheal tube or supraglottic device). Confirm correct placement of an advanced airway. This generally includes the use of waveform capnography or capnometry.

Manage oxygenation and ventilation: Titrate FiO_2 for SpO_2 90%-98% (or PaO_2 60-105 mm Hg). Adjust minute ventilation to target PCO_2 35–45 mm Hg in the absence of severe acidemia.

Manage hemodynamics: Initiate or adjust vasopressors and/or fluid resuscitation as necessary for goal MAP ≥65 mm Hg.

Early diagnostic testing: Obtain 12-lead ECG to assess for ischemia or arrhythmia. Consider CT head, chest, abdomen, and/or pelvis to determine cause of arrest or assess for injuries sustained during resuscitation. Point-of-care ultrasound or echocardiography may be reasonable to identify clinically significant diagnoses requiring intervention.

Continued Management

Treat arrest etiologies and complications.

Consider emergency cardiac intervention:

- Persistent ST-segment elevation present
- Cardiogenic shock
- Recurrent or refractory ventricular arrhythmias
- Severe myocardial ischemia

Temperature control: If patient is not following commands off sedation and neuromuscular blockade or is unable to assess, initiate a deliberate strategy of temperature control with goal 32 °C–37.5 °C as soon as possible.

Evaluate for seizure: Evaluate for clinical seizure and obtain EEG to evaluate for seizure in patients not following commands.

Prognostication: Multimodal approach with delayed impressions (≥72 hours from ROSC or achieving normothermia).

Ongoing critical care includes the following:

- Target PaO_2 60-105 mm Hg, PCO_2 35-45 mm Hg (unless severe acidemia); avoid hypoglycemia (glucose <70 mg/dL) and hyperglycemia (glucose >180 mg/dL); target MAP ≥65 mm Hg.
- Consider antibiotics.

Key Performance Indicators

- Time to airway intervention
- Time to oxygen therapy
- Time to fluid resuscitation
- Vasopressor initiation time
- CPR initiation time
- ICU transfer time
- Mortality among critically ill patients

References

1. Initial Assessment and Management of Emergency Patients Protocol Draft.
2. Triage Policy and Procedure Draft.
3. ACS Protocol Draft.
4. COPD Exacerbation Protocol Draft.
5. WHO Emergency Care Systems Framework.
6. Surviving Sepsis Campaign Guidelines.
7. ACLS Guidelines.
8. ATLS Guidelines.

SECTION III
CARDIOVASCULAR EMERGENCIES

Scope

This protocol provides a standardized evidence-based approach for evaluation, resuscitation, stabilization, and definitive management of cardiovascular emergencies presenting to Emergency Departments across Government Medical Institutions in Kerala.

This guideline applies to:

- Primary Health Centres
- Taluk Hospitals
- District Hospitals
- Government Medical Colleges
- Tertiary Cardiac Care Centres

The protocol includes:

- Acute Coronary Syndrome
- STEMI
- NSTEMI/Unstable Angina
- Hypertensive Emergencies
- Arrhythmias
- Acute Heart Failure
- Cardiogenic Shock
- Pulmonary Embolism
- Aortic Dissection

Introduction

Cardiovascular emergencies are major contributors to mortality and morbidity in emergency care settings. Early recognition, rapid diagnosis, timely reperfusion, and structured critical care management significantly improve outcomes. All patients presenting with:

- Chest pain
- Chest pain equivalents
- Dyspnea
- Syncope
- Palpitations
- Shock
- Acute pulmonary edema

should undergo rapid cardiovascular assessment and ECG within 10 minutes of arrival.

Time-sensitive cardiac emergencies requiring immediate intervention include:

- STEMI
- Cardiogenic shock
- Malignant arrhythmias
- Acute pulmonary edema
- Massive pulmonary embolism
- Aortic dissection

Acute Coronary Syndrome (ACS)

1. Definition

Acute Coronary Syndrome refers to acute myocardial ischemia caused by sudden reduction in coronary blood flow.

Includes:

- STEMI
- NSTEMI
- Unstable Angina

2. Chest Pain Equivalent Symptoms

Patients may present with:

- Dyspnea
- Epigastric pain
- Jaw pain
- Neck pain
- Shoulder pain
- Arm pain
- Syncope

3. Initial Assessment of Chest Pain

Immediate Actions

- Triage to Red Zone
- ABCDE assessment
- Continuous cardiac monitoring
- IV access
- 12-lead ECG within 10 minutes
- Record symptom onset time

4. Differential Diagnosis of Chest Pain

Life-Threatening Causes

- ACS
- Aortic dissection

- Pulmonary embolism
- Pneumothorax
- Esophageal rupture
- Pancreatitis

STEMI Pathway

1. Definition of STEMI

ST elevation myocardial infarction is diagnosed by:

- Ischemic symptoms PLUS
- ST elevation in ≥2 contiguous leads

2. 4.2 STEMI Criteria

Limb Leads
ST elevation ≥1 mm
Chest Leads
Men <40 years
≥2.5 mm in V2–V3
Men >40 years
≥2 mm in V2–V3
Women
≥1.5 mm in V2–V3

3. Initial Emergency Management

Oxygen
Only if:

- SpO_2 <90%
- Respiratory distress

Target SpO_2:
Above 90%
Antiplatelet Therapy
Aspirin
300–325 mg chewable stat
P2Y12 Inhibitor

- Clopidogrel 300–600 m

OR

- Ticagrelor 180 mg

Statin
Atorvastatin 80 mg stat

Nitroglycerin
Sublingual if:
- SBP >100 mmHg
- No RV infarction

Analgesia
Morphine 2–4 mg IV OR fentanyl

4. 4.4 Reperfusion Strategy

Primary PCI – Preferred
Indications
- STEMI within 12 hours
- Cardiogenic shock
- Acute pulmonary edema

Target
Door-to-balloon time ≤90 minutes

5. Thrombolysis

Indications
- STEMI within 12 hours
- PCI unavailable within recommended time

Agents
Streptokinase
1.5 mu over 1 hour
OR
Tenecteplase (Weight-based)

Weight	**Dose**
<60 kg	30 mg
60–69 kg	35 mg
70–79 kg	40 mg
80–89 kg	45 mg
≥90 kg	50 mg

Target:
Door-to-needle ≤30 minutes

6. Failed Thrombolysis

Suspect if:
- Persistent chest pain
- <50% ST resolution at 90 min
- Hemodynamic instability

Management
Urgent rescue PCI

NSTEACS

1. Diagnostic Criteria

ECGgs

- ST depression ≥0.5 mm
- T-wave inversion

Biomarkers

- Elevated troponin → NSTEMI
- Normal troponin → UA

2. HEART Score

Component	Score
History	0–2
ECG	0–2
Age	0–2
Risk factors	0–2
Troponin	0–2

Interpretation

Score	Risk
0–3	Low
4–6	Moderate
7–10	High

3. Initial Treatment

- Aspirin 325 mg
- Clopidogrel 300 mg
- Atorvastatin 80 mg
- Anticoagulation
- Cardiology consultation
- ICU admission

4. High-Risk Features Requiring Early PCI

- Cardiogenic shock
- Refractory chest pain
- Dynamic ST changes
- Heart failure
- Life-threatening arrhythmias

Hypertensive Emergency

Definition

Severe hypertension with acute target organ damage.

Examples:

- Hypertensive encephalopathy
- Pulmonary edema
- ACS
- Aortic dissection
- AKI

1. Clinical Features

- Severe headache
- Visual symptoms
- Chest pain
- Dyspnea
- Neurological deficits

2. Initial Management

Goals

Gradual BP reduction.

Avoid rapid overcorrection.

IV Antihypertensives

- Labetalol
- Nitroglycerin
- Nicardipine
- Nitroprusside (ICU setting)

Arrhythmias

1. Tachyarrhythmias

Types

- SVT
- Atrial fibrillation
- VT
- VF

2. Unstable Tachyarrhythmia

Features of Instability

- Hypotension
- Shock
- Altered sensorium
- Pulmonary edema
- Ischemic chest pain

Management

Immediate synchronized cardioversion

3. Stable SVT

Initial Management

- Vagal maneuvers
- Adenosine

4. Ventricular Tachycardia

Stable VT

- Amiodarone infusion
- Pulseless VT/VF
- Defibrillation
- CPR
- ACLS protocol

5. Bradyarrhythmias

Symptomatic Bradycardia

Features:

- Hypotension
- Shock
- Syncope

Management

- Atropine
- Transcutaneous pacing
- Dopamine infusion
- Adrenaline infusion

Acute Heart Failure

Includes:

- Acute pulmonary edema
- Cardiogenic pulmonary edema
- Acute decompensated heart failure

1. Clinical Features

- Severe dyspnea
- Orthopnea
- Pink frothy sputum
- Crepitations
- Hypoxemia

2. Initial Management

Oxygen Therapy

Target SpO_2:

94–98%

COPD:

88–92%

Non-Invasive Ventilation

Early CPAP/BiPAP preferred.

Indications

- Persistent hypoxia
- RR >25/min
- Severe respiratory distress

3. Pharmacological Therapy

Diuretics

Furosemide 20–40 mg IV

Vasodilators

Nitroglycerin infusion if SBP ≥110 mmHg

Inotropes

If shock present:

- Dobutamine
- Noradrenaline

Cardiogenic Shock

1. Definition

Persistent hypotension due to cardiac pump failure causing end-organ hypoperfusion.

2. Clinical Features

- SBP <90 mmHg
- Cold extremities
- Pulmonary edema
- Oliguria
- Altered sensorium

3. Causes

- STEMI
- Myocarditis
- Severe arrhythmias
- Mechanical complications

4. Management

Immediate Actions

- ICU admission
- Oxygen/NIV/intubation
- Vasopressors
- Inotropes
- Urgent PCI if ACS

Pulmonary Embolism

1. Clinical Features
- Sudden dyspnea
- Tachycardia
- Hypoxia
- Pleuritic chest pain
- Syncope

2. Initial Evaluation
- ECG
- ABG
- Troponin
- D-dimer
- CT Pulmonary Angiography

3. Management

Stable PE

- Anticoagulation

Massive PE

- Thrombolysis
- ICU care

Aortic Dissection

1. Clinical Features
- Sudden tearing chest pain
- Pulse deficit
- BP difference between limbs
- Shock

2. Initial Management
- IV access
- Pain control
- BP control

- CT Aortogram
- Cardiothoracic surgery consultation

Monitoring

All critically ill cardiac patients require:

- Continuous ECG
- Pulse oximetry
- BP monitoring
- Urine output monitoring

Disposition

ICU/CCU Admission

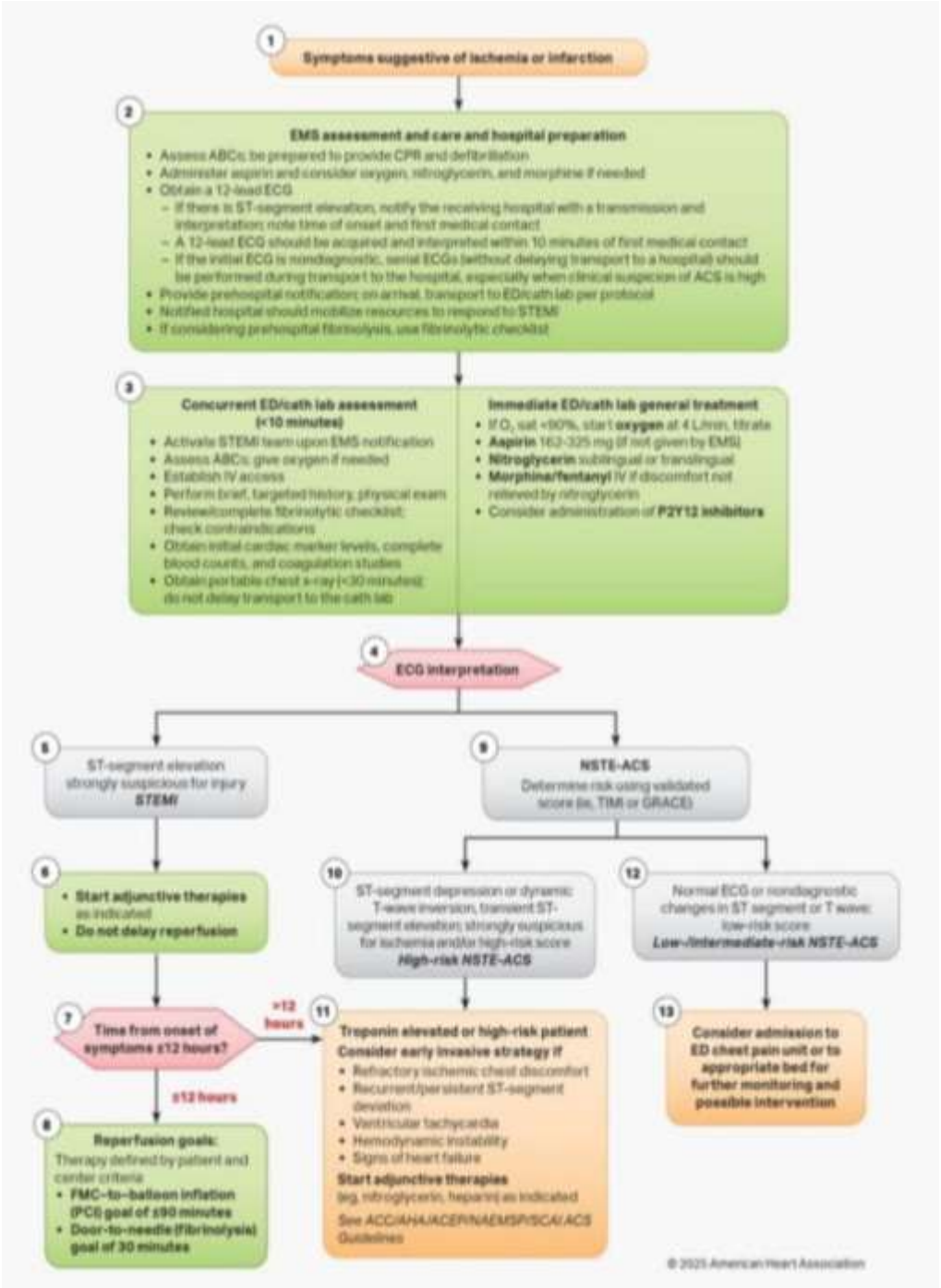
- STEMI
- NSTEMI high risk
- Cardiogenic shock
- Acute pulmonary edema
- Malignant arrhythmias

Ward Admission

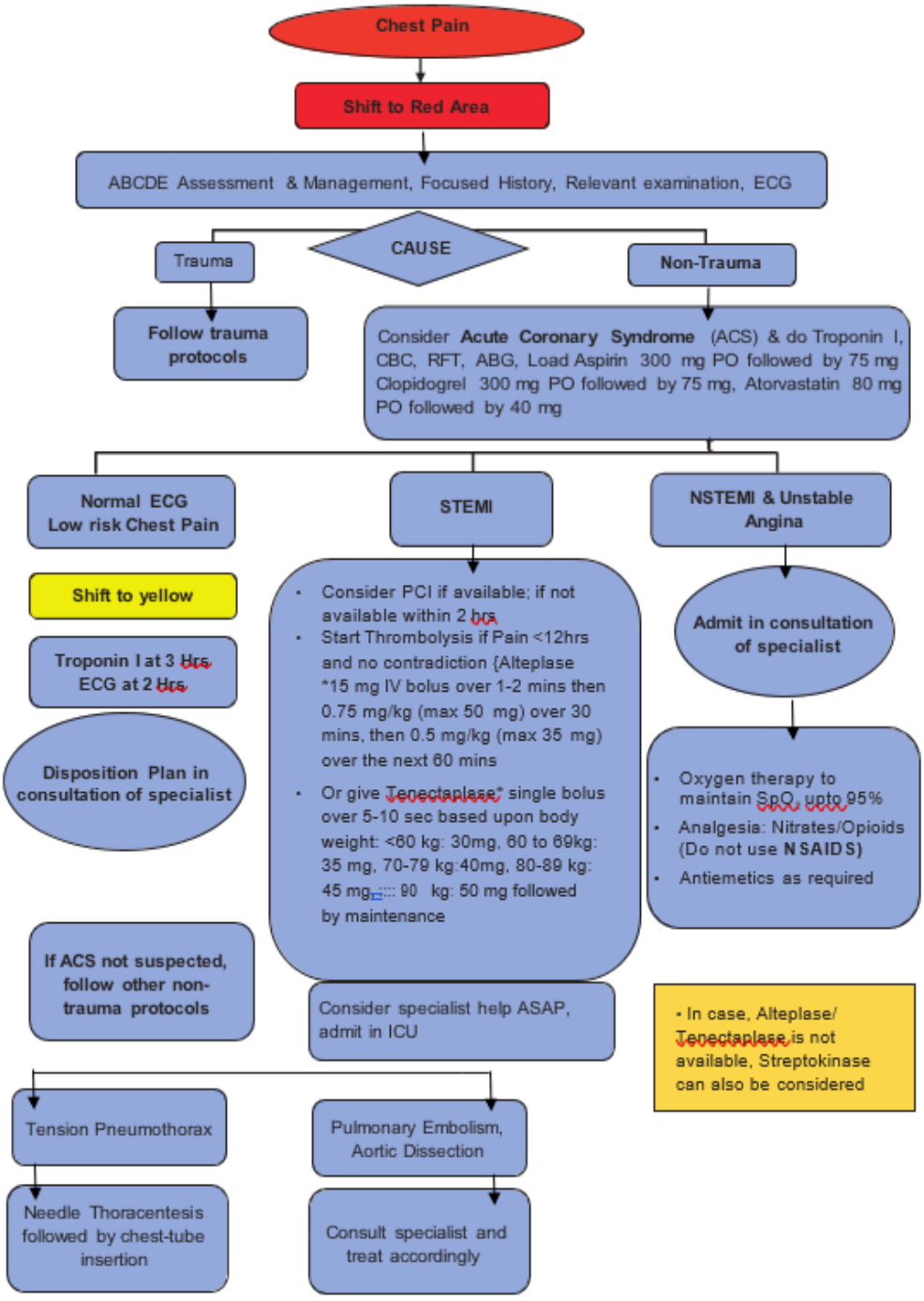
- Stable NSTEMI
- Controlled heart failure

Algorithms

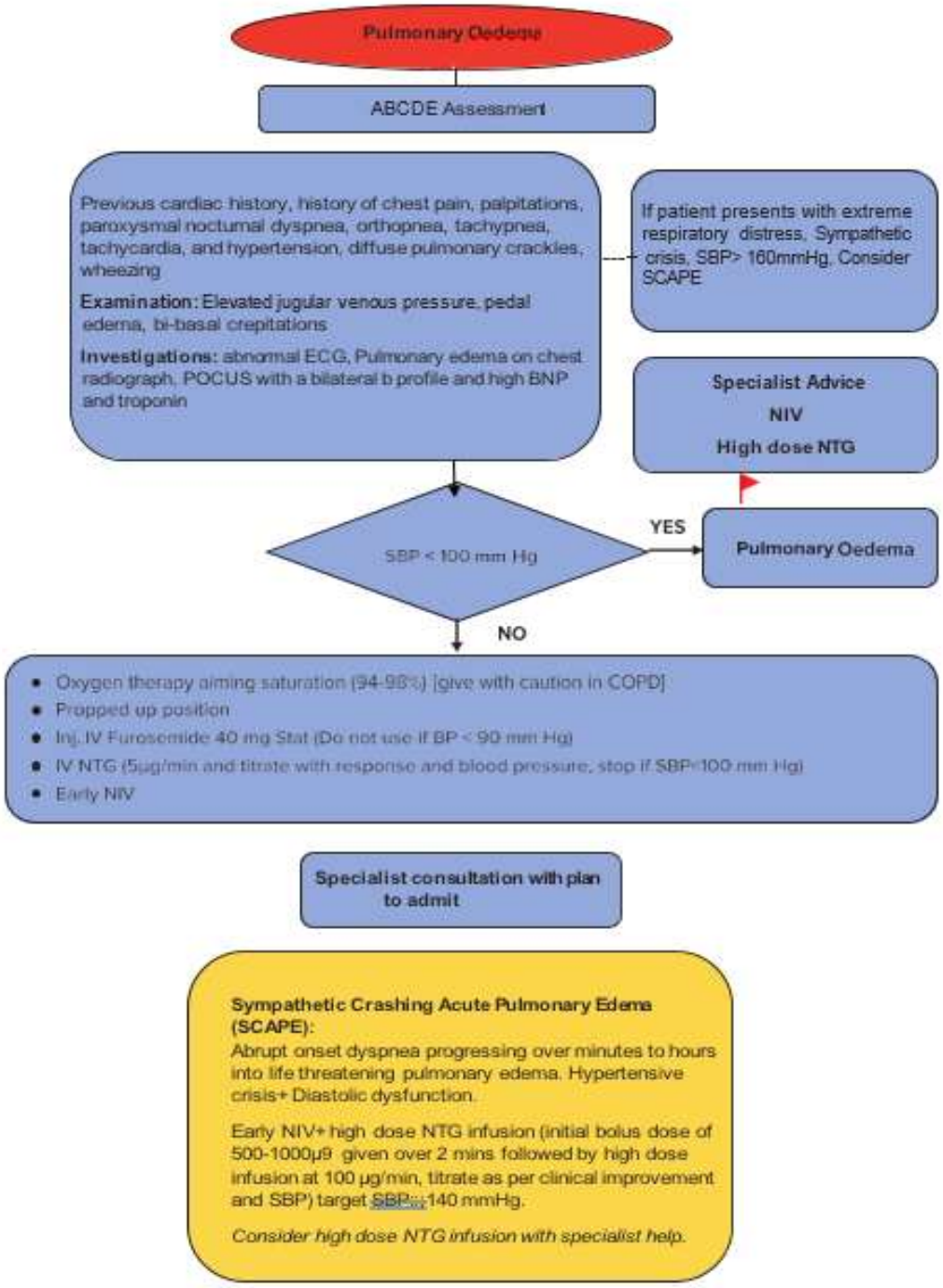
Algorithm 1 - ACS Flowchart



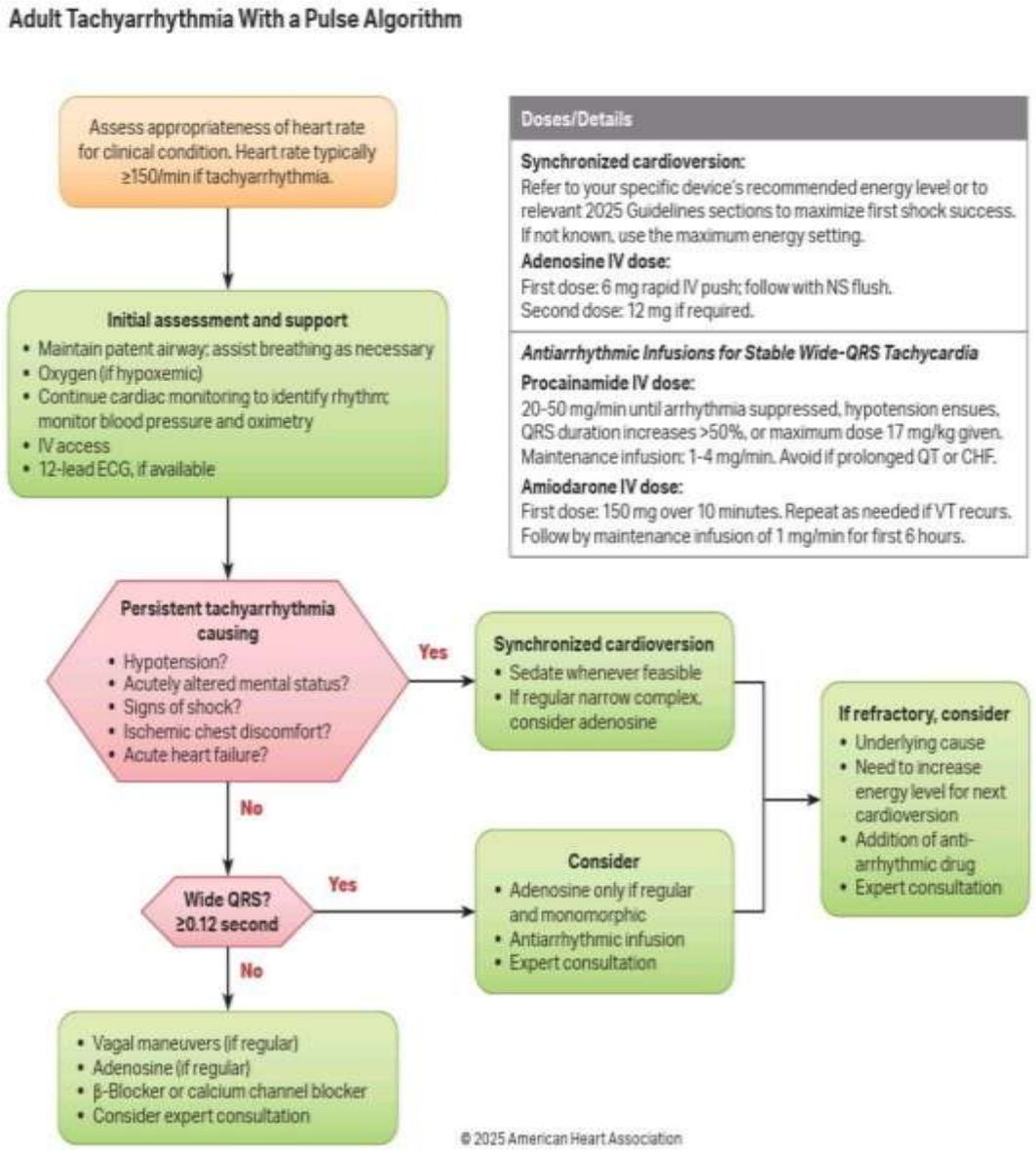
Algorithm 2 - Chest pain approach



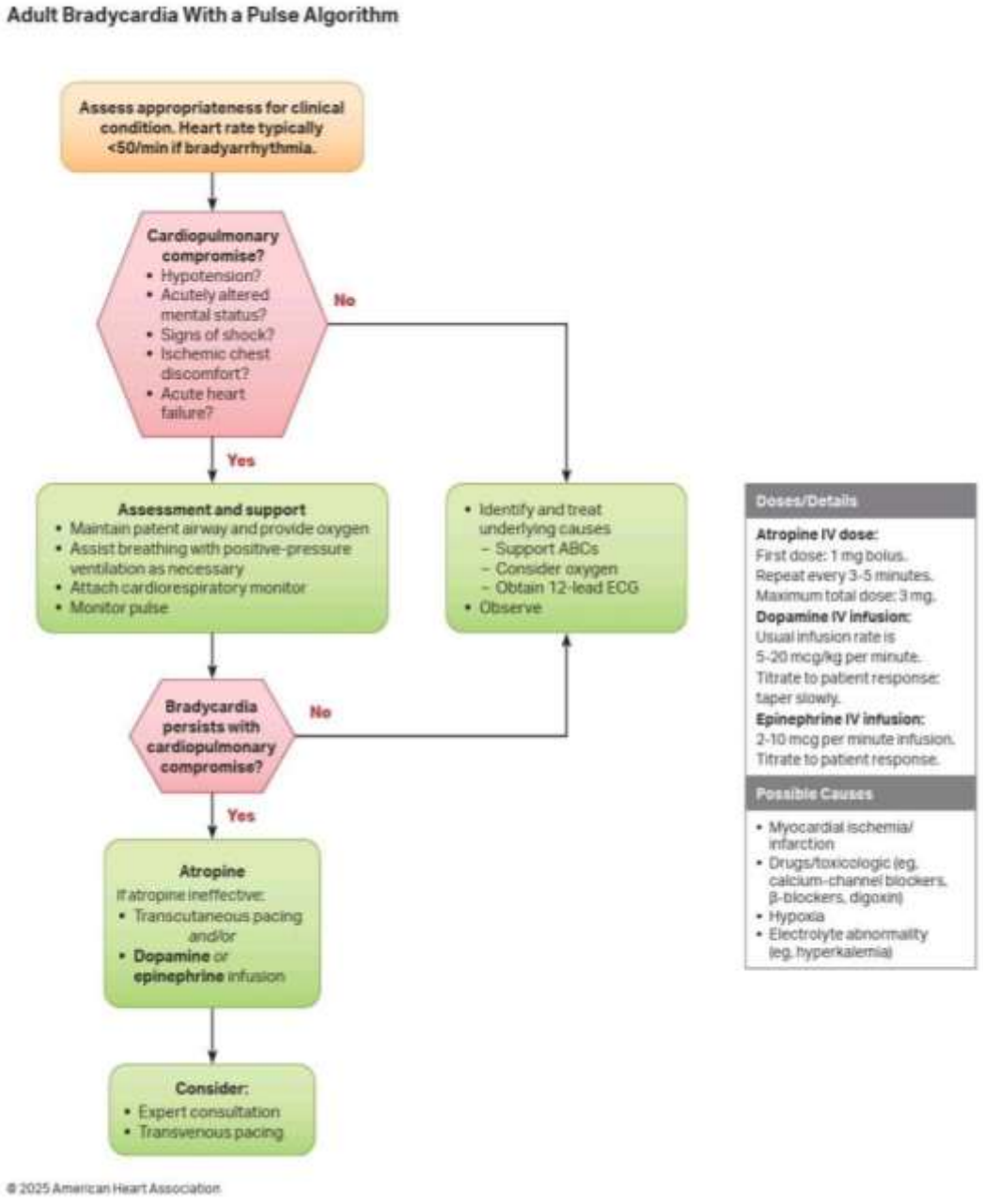
Algorithm 3 - Pulmonary edema



Algorithm 4 - Tachycardia algorithm



Algorithm 5 -Bradycardia algorithm



Quality Indicators

- Door-to-ECG ≤10 min
- Door-to-needle ≤30 min
- Door-to-balloon ≤90 min
- STEMI mortality
- Time to cardiology consultation
- Time to thrombolysis

References

1. ACS Protocol AHA Guidelines
2. STEMI Management ESC guidelines 2023
3. ESC 2023 ACS Guidelines.
4. Fourth Universal Definition of Myocardial Infarction.
5. AHA ACLS Guidelines.

SECTION IV
NEUROLOGICAL EMERGENCIES

Scope

This protocol provides a standardized evidence-based framework for recognition, stabilization, investigation, treatment, referral, and disposition of neurological emergencies presenting to Emergency Departments across Government Medical Institutions in Kerala.

This guideline applies to:

- Primary Health Centres
- Family Health Centres
- Taluk Hospitals
- District Hospitals
- Government Medical Colleges
- Tertiary Neurology and Neurosurgical Centres

This section includes:

1. Acute Ischemic Stroke
2. Intracranial Hemorrhage
3. Seizure & Status Epilepticus
4. Altered Sensorium
5. Meningitis/Encephalitis
6. Head Injury
7. Raised Intracranial Pressure

The protocol is intended for:

- Emergency Physicians
- Casualty Medical Officers
- Neurologists
- Neurosurgeons
- Intensivists
- Nurses
- Paramedics

Introduction

Neurological emergencies are among the leading causes of mortality and long-term disability. Early recognition and timely intervention significantly improve neurological outcomes. Time-sensitive neurological emergencies include:

- Acute ischemic stroke
- Intracranial hemorrhage
- Status epilepticus
- CNS infections
- Raised intracranial pressure
- Traumatic brain injury

All patients presenting with:

- Sudden focal neurological deficits
- Altered mental status
- Seizures
- Severe headache
- Head trauma
- Fever with altered sensorium

must undergo rapid stabilization using the ABCDE approach with simultaneous neurological assessment.

Acute Ischemic Stroke

1. Definition

Acute ischemic stroke is a sudden neurological deficit caused by interruption of cerebral blood flow resulting in cerebral ischemia and infarction.

2. Recognition of Stroke

Common Symptoms

- Sudden limb weakness
- Facial asymmetry
- Slurred speech
- Aphasia
- Sudden visual loss
- Gait imbalance
- Altered sensorium

3. Initial Emergency Department Assessment

Immediate Actions

- Triage to RED zone
- Activate Stroke Code
- ABCDE assessment
- Record exact time of symptom onset or last known well time
- Establish IV access
- Check GRBS immediately
- Obtain ECG
- Perform focused neurological examination

4. Stroke Workflow in Kerala Government Hospitals

Primary/FHC/Taluk Hospitals

- Early stroke recognition
- ABC stabilization
- Immediate GRBS correction

- NCCT Brain if available
- Tele-neurology consultation where possible
- Immediate referral to stroke-ready centre if thrombolysis unavailable

District Hospitals

- 24×7 CT capability preferred
- Emergency physician-led thrombolysis pathway
- ICU/HDU monitoring
- Early thrombectomy referral pathway

Medical Colleges/Tertiary Stroke Centres

- Comprehensive stroke unit
- Mechanical thrombectomy capability
- Neurocritical care
- CTA/MRA/perfusion imaging

5. Emergency Imaging

NCCT Brain

Should be performed urgently in all suspected stroke patients to:

- Exclude hemorrhage
- Identify ischemic changes
- Decide eligibility for thrombolysis
- CT imaging should not be delayed in window-period stroke patients.

6. Stroke Time Targets

Parameter	Target
Door-to-Triage	≤5 min
Door-to-CT	≤20 min
CT Interpretation	≤45 min
Door-to-Needle	≤60 min
Door-to-Groin Puncture	≤90 min

7. Intravenous Thrombolysis

General Principles

- Time is brain.
- Treatment should not be delayed awaiting unnecessary investigations.
- Neurology consultation should occur simultaneously with imaging.
- IV thrombolysis and thrombectomy evaluation should occur in parallel.

8. Alteplase (rtPA) Protocol

Drug of Choice

Alteplase (rtPA)

Dose

- 0.9 mg/kg IV
- Maximum dose: 90 mg

Administration

- 10% of total dose as IV bolus over 1 minute
- Remaining 90% infused over 60 minutes

Example Dosing Table

Weight	Total Dose	Bolus Dose	Infusion Dose
50 kg	45 mg	4.5 mg	40.5 mg
60 kg	54 mg	5.4 mg	48.6 mg
70 kg	63 mg	6.3 mg	56.7 mg
80 kg	72 mg	7.2 mg	64.8 mg
90 kg	81 mg	8.1 mg	72.9 mg
≥100 kg	90 mg	9 mg	81 mg

9. Tenecteplase

Consider In:

- Large vessel occlusion
- Mechanical thrombectomy candidates
- Logistically difficult transfer situations

Dose

- 0.25 mg/kg IV bolus
- Maximum dose: 25 mg

10. Indications for Thrombolysis

Within 4.5 Hours

- Age >18 years
- Acute ischemic stroke causing measurable neurological deficit
- CT excluding hemorrhage

Special Situations

May still be considered after Neurology consultation:

- Wake-up stroke
- Mild but disabling deficit
- Seizure at onset with persistent stroke deficits
- Prior antiplatelet use
- CKD with normal coagulation

11. Contraindications to Thrombolysis

Absolute Contraindications

- Intracranial hemorrhage
- Recent severe head injury
- Ischemic stroke within 3 months
- Platelets <100,000
- INR >1.7

- Active GI bleed
- Suspected aortic dissection
- Uncontrolled BP >185/110 mmHg
- Extensive infarction on CT

12. Blood Pressure Management in Stroke

Before Thrombolysis	After Thrombolysis
Target:	Target:
<185/110 mmHg	<180/105 mmHg

Preferred Agent

IV Labetalol

- 10–20 mg IV over 1–2 minutes
- Repeat if necessary

Continuous infusion:

- 2–8 mg/min if persistent hypertension

13. Mechanical Thrombectomy

Consider if:

- Large vessel occlusion
- ICA/M1 occlusion
- NIHSS ≥6
- Within 24 hours in selected patients

14. Stroke Monitoring

During Thrombolysis

Duration	**Frequency**
First 2 hrs	Every 15 min
Next 6 hrs	Every 30 min
Next 16 hrs	Hourly

Monitor:

- BP
- Neurological status
- Bleeding
- Oxygen saturation

15. Antiplatelet and Statin Therapy

If Thrombolysis NOT Planned

- Aspirin 300 mg loading
- Clopidogrel 225–300 mg loading
- Atorvastatin 80 mg loading

If Thrombolysis Given

- Avoid antiplatelets for 24 hours
- Repeat imaging before starting antiplatelets

Intracranial Hemorrhage

1. Clinical Features

- Sudden severe headache
- Vomiting
- Seizures
- Altered sensorium
- Focal deficits
- Severe hypertension

2. Initial Management

- ABCDE stabilization
- Airway protection
- BP management
- NCCT Brain
- Neurosurgical consultation
- ICU/HDU admission

3. Blood Pressure Management

Avoid excessive BP reduction.

Target individualized based on:

- Hemorrhage type
- ICP
- Clinical condition

4. Reversal of Anticoagulation

If applicable:

- Vitamin K
- PCC
- FFP

Seizure & Status Epilepticus

1. Definitions

Status Epilepticus

- Seizure >5 minutes

  OR

- Recurrent seizures without recovery

2. Initial Assessment

- ABC assessment
- Recovery position
- Oxygen
- Check blood glucose
- IV access

3. First-Line Therapy

Lorazepam

4 mg IV over 2 minutes

OR

Midazolam

IM/IV if IV access unavailable

4. Second-Line Therapy

Options

Phenytoin

15–20 mg/kg IV

OR

Fosphenytoin

15–20 mg PE/kg

OR

Levetiracetam

40–60 mg/kg IV

OR

Sodium Valproate

20–40 mg/kg IV

5. Refractory Status Epilepticus

- ICU admission
- Intubation
- Mechanical ventilation
- Continuous infusion sedation

6. Investigations

- CBC
- Electrolytes
- Calcium
- ABG/VBG
- CT Brain
- Toxicology screen if indicated

Altered Sensorium

1. Causes

Common causes:

- Stroke
- Sepsis
- Hypoglycemia
- Poisoning

- Head injury
- Metabolic encephalopathy
- Postictal state

2. Initial Management
- ABCDE assessment
- Check carotid pulse
- GRBS
- Oxygen
- IV access

3. Airway Protection

GCS <8
- Endotracheal intubation
- ICU admission

4. Evaluation
- CBC
- Electrolytes
- ABG/VBG
- RFT/LFT
- CT Brain
- Toxicology evaluation

Meningitis / Encephalitis

1. Clinical Features
- Fever
- Neck stiffness
- Headache
- Seizures
- Altered mental status

2. Initial Management
- ABC stabilization
- Blood cultures
- Early antibiotics
- Dexamethasone if indicated

3. Empirical Therapy

Adults
- Ceftriaxone + Vancomycin

Add:
- Acyclovir if encephalitis suspected

4. Lumbar Puncture

Perform after CT if:

- Focal deficits
- Papilledema
- GCS deterioration
- Raised ICP suspected

Head Injury

1. Initial Assessment

Follow ATLS principles:

- Airway with C-spine protection
- Breathing
- Circulation
- Disability
- Exposure

2. Red Flag Features

- GCS <13
- Repeated vomiting
- Seizures
- Skull fracture signs
- Focal deficits
- ENT bleed

3. Imaging

NCCT Brain Indications

- GCS <15
- Persistent symptoms
- Focal neurological deficit
- Anticoagulant use

4. Management

- Airway protection
- ICP control
- Neurosurgical referral
- Seizure prophylaxis if indicated

Raised Intracranial Pressure

1. Clinical Features

- Headache
- Vomiting
- Bradycardia

- Hypertension
- Unequal pupils
- Reduced GCS

2. Management

Immediate Measures

- Head elevation 30°
- Oxygenation
- Avoid hypotension
- Sedation

Osmotherapy

Mannitol

0.25–1 g/kg IV

OR

Hypertonic Saline

Indications for Intubation

- GCS <8
- Respiratory failure
- Airway compromise

Monitoring

All neurological emergency patients require:

- GCS monitoring
- Pupillary assessment
- BP monitoring
- SpO_2 monitoring
- Neuro-observation charting

Disposition

ICU/HDU Admission

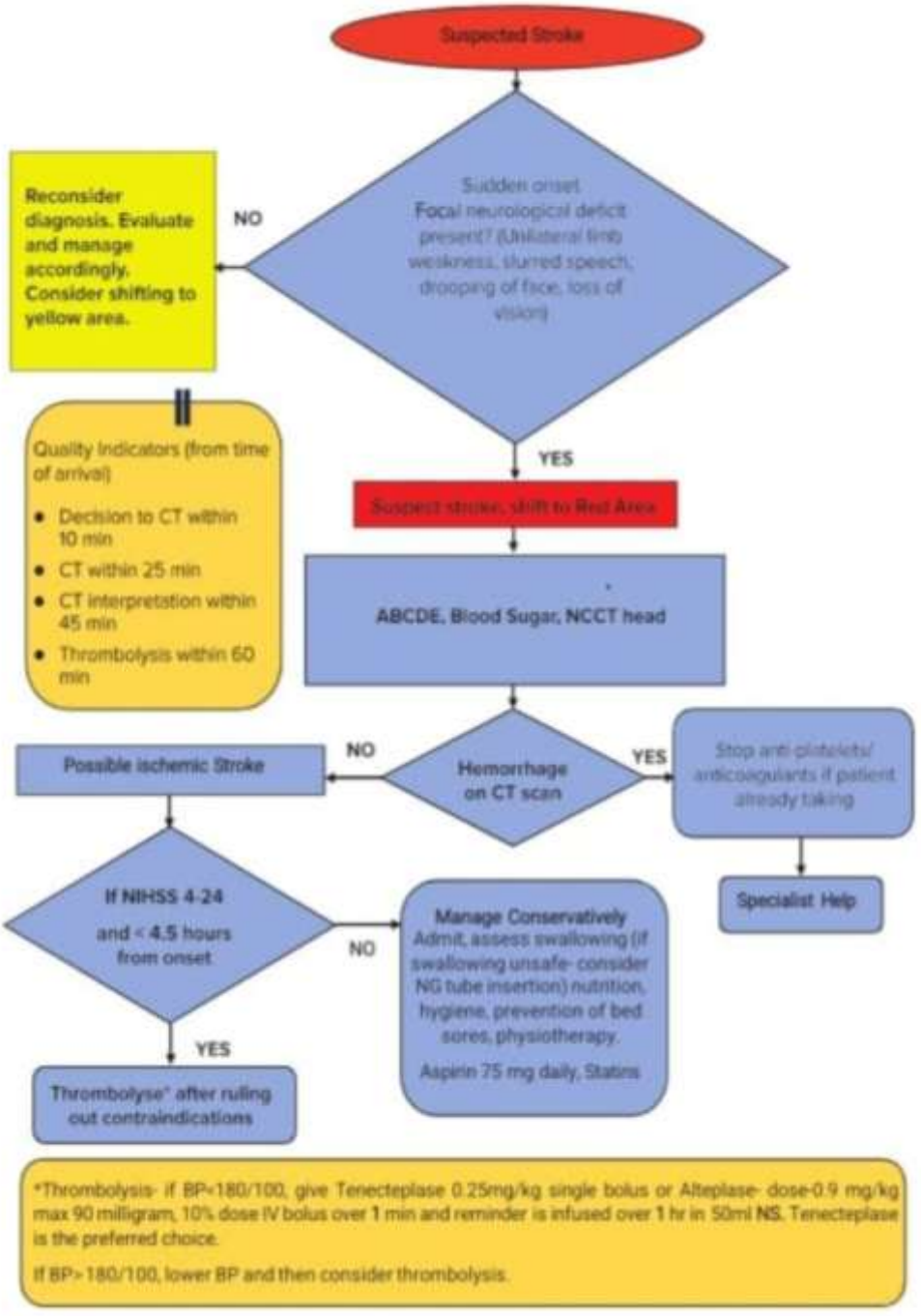
- Thrombolysis
- Intracranial hemorrhage
- Status epilepticus
- Raised ICP
- Ventilator requirement

Ward Admission

- Stable ischemic stroke
- Controlled seizures

Algorithms

Algorithm 1 - Stroke Thrombolysis Pathway



Algorithm 2 - Stroke HIHSS Score and BP management

NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS)*

Item	Title	Responses and Scores
1a	Level of consciousness	0- alert
		1- drowsy
		2-obtunded
		3- coma/unresponsive
1b	Orientation questions (2)	0- answer both correctly
		1- answer one correctly
		2- answer neither correctly
1c	Response to commands (2)	0- performs both tasks correctly
		1- performs one task correctly
		0- performs neither
2	Gaze	0- normal horizontal movements
		1- partial gaze palsy
		2- complete gaze palsy
3	Visual fields	0- no visual field detect
		1-partial hemianopia
		2- complete hemianopia
		3- bilateral hemianopia
4	Facial movement	0- normal
		1- minor facial weakness
		2- partial facial weakness
		3- complete unilateral palsy
5	Major function (arm) a.Left b. Right	0- no drift
		1- drift before 10 seconds
		2- falls before 10 seconds
		3- no effort against gravity
		4- no movements
6	Major function (leg a.Left b. Right	0- no drift
		1- drift before 5 seconds
		2- falls before 5 seconds
		3- no effort against gravity
		4- no movements
7	Limb ataxia	0- no ataxia
		1- ataxia in 1 limb
		2- ataxia in 2 limbs
8	Sensory	0- no sensory loss
		1- mild sensory loss
		2- severe sensory loss
9	Language	0- normal
		1- mild aphasia
		2- severe aphasia
		3- mute or global aphasia

tem	Title	Responses and Scores
10	Articulation	0- normal
		1- mild dysarthria
		2- severe dysarthria
11	Extinction or inattention	0- absent
		1- mild loss (1 sensory modality lost)
		2- severe loss (2 sensory modalities lost)

Score	Stroke Severity
0	No stroke symptoms
1-4	Minor stroke
5-15	Moderate stroke
16-20	Moderate to severe stroke
21-42	Severe stroke

Scoring range is 0-42 points.

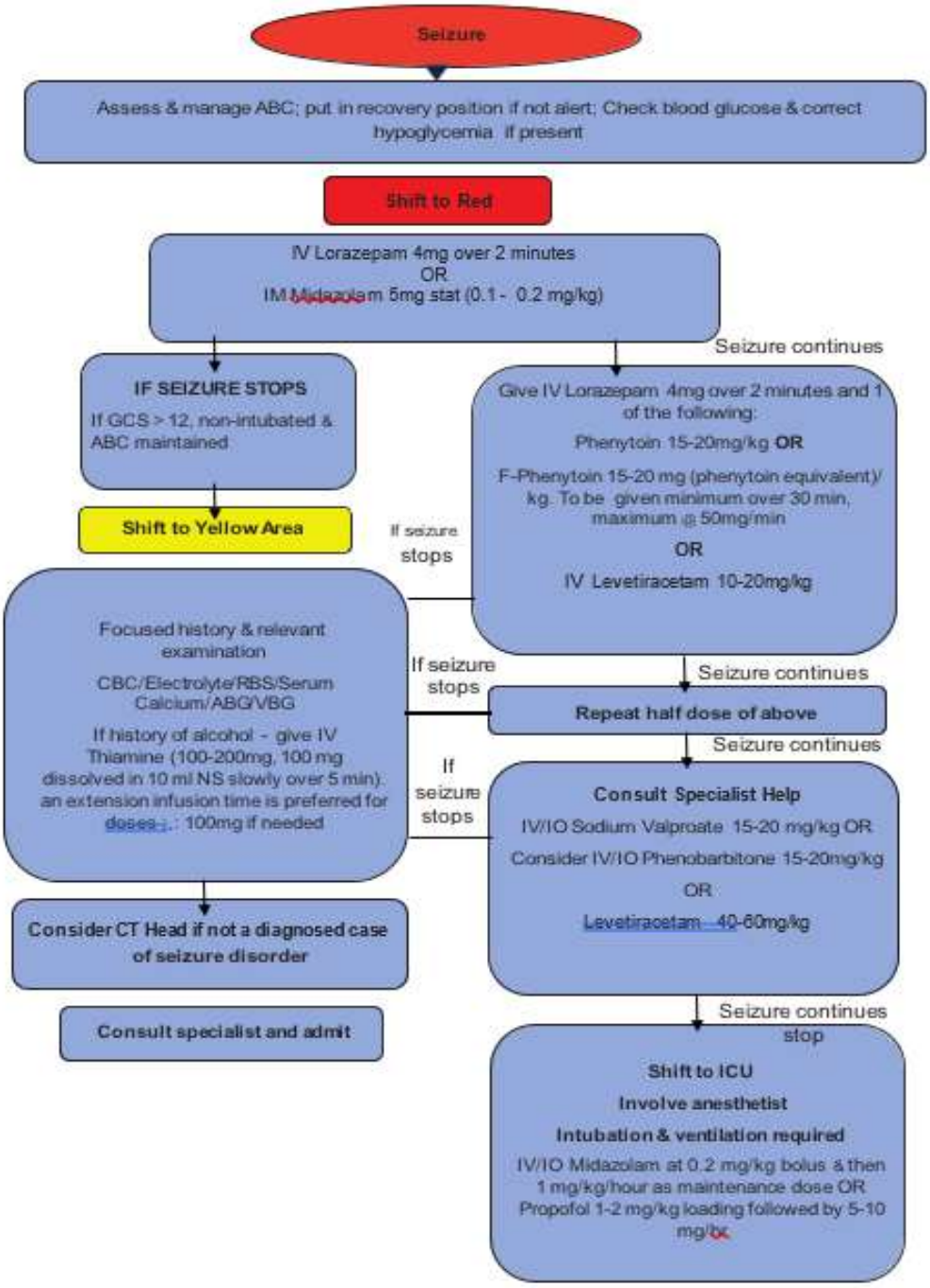
The higher the number, the greater the severity.

Blood Pressure management in Acute Stroke

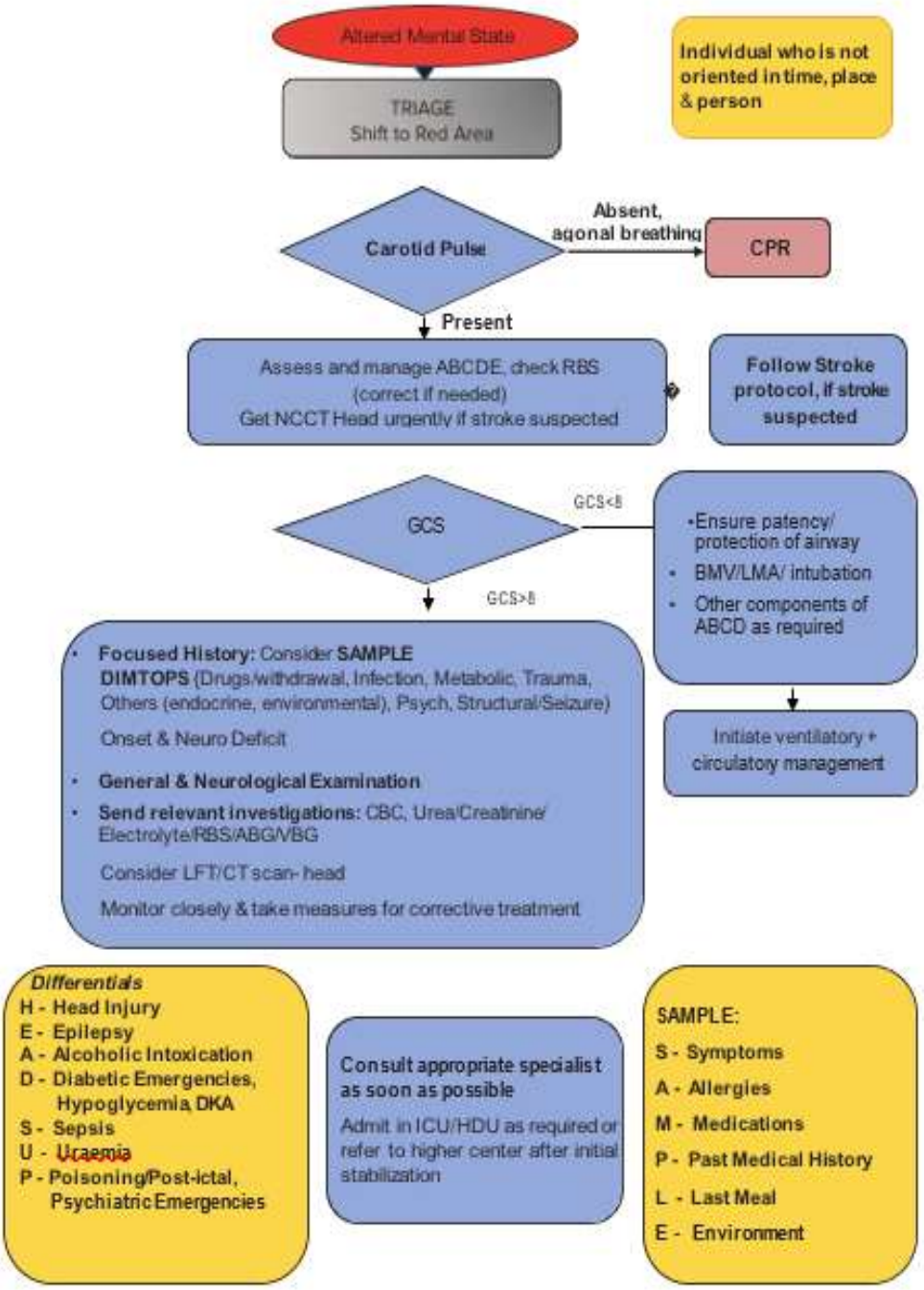
Before starting thrombolysis if, SBP>185 or diastolic > 110 mmHg	Labetalol 10-20 mg IV over 1-2 mins, Nicardipine 5mg/hr, titrate up by 2.5 mg/hr at 5-15 minute intervals, max dose 15mg/hr. If BP does not decline and remains >185/110 do not give thrombolytic agent
Monitor BP evert 15 minutes during thrombolysis treatment and then for another 2 hours, then every 30 mins for 6 hrs, and every hour for 16 hours	
Systolic 180-230 mmHg or diastolic 105-120 mmHg	Labetalol 10 mg IV over 1-2 mins, may repeat every 10-20 mins, max dose 300 mg or Labetalol 10 mg IV followed by an infusion at 2-8 mg/min
Systolic >230 mmHg or diastolic 121-140 mmHg	Labetalol 10 mg IV over 1-2 mins, may repeat every 10-20 mins, max dose 300 mg or Labetalol 10 mg IV followed by an infusion at 2-8 mg/min

Algorithm 3

Seizure & Status Epilepticus Management



Algorithm 4
Altered mental status



Quality Indicators

- Door-to-CT time
- Door-to-needle time
- Stroke code activation time
- NIHSS documentation rate
- Seizure treatment initiation time
- ICU transfer time
- Stroke mortality rate

References

1. AHA/ASA Acute Ischemic Stroke Guidelines 2024.
2. Neurocritical Care Society Guidelines.
3. Status Epilepticus Treatment Guidelines.

SECTION V
RESPIRATORY EMERGENCIES

Scope

This protocol provides a standardized approach for evaluation and management of respiratory emergencies presenting to Emergency Departments in Government Medical Institutions across Kerala.

Conditions covered:

1. Acute Severe Asthma
2. COPD Exacerbation
3. Pneumonia
4. ARDS
5. Acute Respiratory Failure
6. NIV & Intubation Criteria
7. Pneumothorax
8. Acute Pulmonary Edema

1. ACUTE SEVERE ASTHMA

Recognition

Features suggesting severe asthma exacerbation:

- Respiratory rate >30/min
- Unable to complete sentences
- SpO_2 <92%
- Silent chest
- Peak Expiratory Flow (PEF/PEFR) <50% predicted
- Tachycardia >120/min
- Use of accessory muscles
- Agitation, exhaustion, altered sensorium
- Previous ICU/intubation history
- Frequent SABA use or poor ICS adherence

Life-threatening asthma features

- Silent chest
- Cyanosis
- Bradycardia
- Hypotension
- Altered mental status
- Rising $PaCO_2$
- Respiratory exhaustion

Immediate Assessment

ABCDE Approach

Initial Evaluation

- Pulse oximetry

- PEF/PEFR if feasible
- ABG/VBG in severe cases
- ECG if severe tachycardia/chest pain
- Chest X-ray only if:
  - Pneumothorax suspected
  - Pneumonia suspected
  - Poor response to treatment
  - Intubated patient

Oxygen Therapy

Target SpO_2:

- Adults: 94–98%
- Titrate oxygen; avoid unnecessary hyperoxia

Recommended:

- Nasal prongs
- Face mask
- Non-rebreather mask in critical hypoxia

Bronchodilator Therapy

Short-Acting Beta Agonist (SABA)

Nebulized Salbutamol

- 5 mg nebulization every 20 minutes for first hour

OR

- Continuous nebulization in severe cases

MDI + Spacer (preferred where feasible)

- 4–10 puffs every 20 minutes for first hour

Anticholinergic Therapy

Ipratropium Bromide

- 500 mcg nebulization every 20 minutes × 3 doses

Recommended in:

- Severe asthma
- Life-threatening asthma

Corticosteroids

Administer within first hour.

Preferred Options

- Hydrocortisone 100 mg IV every 6 hours

OR

- Methylprednisolone 60–80 mg IV

OR

- Prednisolone 40–50 mg oral (if able to tolerate orally)

Early corticosteroid administration reduces admission and relapse.

Magnesium Sulfate

Intravenous Magnesium Sulfate

- 2 g IV over 20 minutes

Indications:

- Severe asthma
- Poor response after initial bronchodilator therapy
- PEF <25–30%
- Life-threatening asthma

Nebulized magnesium sulfate is NO LONGER recommended.

Non-Invasive Ventilation (NIV)

May be considered selectively in:

- Severe work of breathing
- Hypercapnia without immediate need for intubation

Avoid delaying intubation if deterioration occurs.

Indications for Intubation

- Exhaustion
- Silent chest
- Rising $PaCO_2$
- Altered sensorium
- Persistent hypoxia
- Respiratory arrest
- Hemodynamic instability

Intubation Precautions

- High risk procedure
- Dynamic hyperinflation risk
- Use lung-protective ventilation
- Allow prolonged expiratory time

Monitoring

Continuous monitoring:

- SpO_2
- Respiratory rate
- Heart rate
- Mental status
- PEFR response

Repeat ABG if:

- Deterioration
- Persistent hypoxia
- Hypercapnia suspected

Disposition

Discharge Criteria

- Symptoms improved
- SpO_2 stable
- PEF >60–80%
- Minimal bronchodilator requirement
- Able to ambulate/feed
- Good inhaler technique

Mandatory Discharge Planning

Before discharge:

- Start or optimize ICS-containing therapy
- Avoid SABA-only discharge treatment
- Check inhaler technique
- Provide written asthma action plan
- Educate on trigger avoidance
- Arrange follow-up within 1 week

GINA 2025 strongly discourages SABA-only management because of increased risk of severe exacerbations and asthma-related mortality.

Admission Criteria

Admit if:

- Persistent hypoxia
- PEFR <60%
- Recurrent nebulization requirement
- Previous ICU/intubation
- Poor social support
- Severe/life-threatening attack
- Hypercapnia
- Pregnancy/comorbid illness

2. ACUTE EXACERBATION OF COPD (AECOPD)

Definition

An exacerbation of COPD is an acute worsening of respiratory symptoms occurring over ≤14 days characterized by:

- Increased dyspnea
- Increased cough
- Increased sputum production and/or purulence
- Tachypnea and tachycardia

Usually associated with:

- Respiratory infections
- Environmental pollution
- Other inflammatory triggers

GOLD 2026 emphasizes that alternative diagnoses must always be considered.

Differential Diagnoses / Mimics

Always rule out:

- Pneumonia
- Acute heart failure
- Pulmonary embolism
- Pneumothorax
- Acute coronary syndrome
- Pleural effusion
- Arrhythmias

Initial Assessment

ABCDE Assessment

Clinical Evaluation

Assess:

- Respiratory rate
- Accessory muscle use
- Mental status
- Ability to speak
- Cyanosis
- Hemodynamic stability

Investigations

- Pulse oximetry
- ABG/VBG in moderate–severe exacerbation
- Chest X-ray
- ECG
- CBC
- Renal function/electrolytes
- CRP if available
- Viral testing if indicated
- Sputum culture in severe/recurrent infections

Severity Classification (Modified GOLD/Rome Proposal)

Mild

- Increased symptoms without respiratory failure
- Managed with bronchodilators only

Moderate

Requires:

- Bronchodilators PLUS
- Steroids and/or antibiotics

Severe

Features:

- Acute respiratory failure
- Severe hypoxemia
- Hypercapnia/acidosis
- Altered sensorium
- Need for NIV/intubation

Oxygen Therapy

Controlled Oxygen Therapy

Target SpO_2:

- 88–92%

Preferred methods:

- Venturi mask
- Nasal prongs with titration

Avoid uncontrolled high-flow oxygen due to risk of worsening hypercapnia.

Bronchodilator Therapy

Initial Bronchodilator Treatment

Salbutamol

- Nebulized salbutamol 2.5–5 mg every 20 minutes initially

  OR
- MDI + spacer where feasible

Add Ipratropium

- 500 mcg nebulized every 4–6 hours

GOLD 2026 continues recommending SABA ± short-acting anticholinergics as first-line acute therapy.

Systemic Corticosteroids

Recommended duration:

- Up to 5 days

Options

- Prednisolone 40 mg oral daily × 5 days

  OR
- Hydrocortisone 100 mg IV q6h

  OR
- Methylprednisolone 40–60 mg IV

Benefits:

- Faster recovery
- Reduced treatment failure
- Reduced hospitalization duration

Prolonged steroid courses are generally not recommended.

Antibiotics

Indications

Give antibiotics if:

- Purulent sputum
- Increased sputum volume
- Increased dyspnea
- Need for ventilation
- Pneumonia suspected

Recommended Duration

- Usually 5 days

Suggested Antibiotics

Mild–Moderate

- Amoxicillin-clavulanate
- Azithromycin
- Doxycycline

Severe / ICU

- Piperacillin-tazobactam
- Cefoperazone-sulbactam
- According to local antibiogram

Non-Invasive Ventilation (NIV)

Strongly Recommended in:

- Respiratory acidosis (pH <7.35)
- Hypercapnia
- Severe dyspnea with increased work of breathing
- Persistent hypoxemia despite oxygen

Benefits:

- Reduces intubation
- Reduces mortality
- Shortens hospital stay

Indications for Intubation

- Failure of NIV
- Respiratory arrest
- Severe hypoxemia
- Hemodynamic instability

- Inability to protect airway
- Reduced consciousness
- Severe acidosis worsening

High Flow Nasal Oxygen (HFNO)

Can be considered in:

- Hypoxemic respiratory failure
- NIV intolerance
- Post-extubation support

But NIV remains preferred in hypercapnic respiratory failure.

Monitoring

Continuous monitoring:

- SpO_2
- Respiratory rate
- Mental status
- Heart rate
- Blood pressure

Repeat ABG if:

- Clinical deterioration
- Hypercapnia suspected
- NIV initiated

Maintenance Therapy Before Discharge

Before discharge:

- Restart/optimize long-acting bronchodilator therapy
- Ensure inhaler technique
- Smoking cessation counselling
- Vaccination advice
- Pulmonary rehabilitation advice
- Assess eosinophil count if recurrent exacerbations

Consider ICS Addition If:

- Frequent exacerbations
- Blood eosinophils ≥300 cells/µL
- Coexisting asthma

Discharge Criteria

- Stable SpO_2 on room air or baseline oxygen
- Reduced bronchodilator requirement
- Hemodynamic stability
- Ambulating and eating
- No worsening acidosis

- Adequate home support

Follow-Up Recommendations

Review within:

- 2–4 weeks

Assess:

- Symptoms
- Exacerbation prevention
- Inhaler technique
- Smoking cessation
- Need for pulmonary rehab
- Vaccination status

3. PNEUMONIA

COMMUNITY ACQUIRED PNEUMONIA (CAP)

Definition

Community-acquired pneumonia (CAP) is an acute infection of lung parenchyma acquired outside hospital settings causing:

- Fever
- Cough
- Dyspnea
- Hypoxia
- New infiltrates on chest imaging

Recognition

Clinical Features

- Fever/chills
- Productive or dry cough
- Dyspnea
- Tachypnea
- Hypoxia
- Pleuritic chest pain
- Altered sensorium (elderly/sepsis)
- Crepitations/bronchial breath sounds

Red Flag Signs

- SpO_2 <90%
- RR >30/min
- Hypotension
- Confusion

- Cyanosis
- Septic shock

Initial Assessment

ABCDE Approach

Investigations

Essential

- Pulse oximetry
- Chest X-ray
- CBC
- Renal function/electrolytes
- Blood glucose

If Severe

- ABG/VBG
- Lactate
- Blood cultures BEFORE antibiotics
- ECG
- CRP/Procalcitonin if available

Consider

- Viral panel (Influenza/COVID)
- Sputum Gram stain/culture
- Ultrasound chest if pleural effusion suspected

Severity Assessment

CURB-65 Score

Parameter	Score
Confusion	1
Urea >7 mmol/L	1
Respiratory rate ≥30	1
BP <90 systolic or ≤60 diastolic	1
Age ≥65	1

Interpretation

- 0–1 → Outpatient/ward
- 2 → Admit
- ≥3 → Severe CAP/ICU consideration

Sepsis Screening

Assess for:

- Hypotension
- Lactate elevation

- Organ dysfunction
- Altered mental status

Septic Shock

Requires:

- Vasopressors
- Persistent hypotension despite fluids

Oxygen Therapy

Target Oxygen Saturation

- General patients: >94%
- COPD patients: 88–92%

Methods:

- Nasal prongs
- Face mask
- Non-rebreather mask
- HFNO if severe hypoxemia

Antibiotic Therapy

IMPORTANT PRINCIPLES

1. Start antibiotics EARLY in severe CAP
2. Use local antibiogram
3. Avoid unnecessary broad-spectrum escalation
4. De-escalate based on cultures
5. Shorter antibiotic courses preferred when clinically improving

Empirical Antibiotic Recommendations

Mild CAP (Ward)

Preferred

- Ceftriaxone + Azithromycin

OR

- Amoxicillin-clavulanate + Azithromycin

Moderate–Severe CAP

Preferred

- Piperacillin-tazobactam + Azithromycin

OR

- Cefoperazone-sulbactam + Azithromycin

ICU / Severe CAP

Consider

- Piperacillin-tazobactam

OR

- Meropenem (selected resistant cases)PLUS
- Azithromycin OR Levofloxacin

Add MRSA Coverage If:

- Post-influenza pneumonia
- Necrotizing pneumonia
- Previous MRSA colonization

Options:

- Vancomycin
- Linezolid

Aspiration Pneumonia

Preferred:

- Piperacillin-tazobactam

OR

- Amoxicillin-clavulanate

Routine anaerobic coverage not needed unless:

- Lung abscess
- Empyema
- Severe aspiration

Viral Pneumonia

Influenza

- Oseltamivir if suspected/high-risk

Corticosteroids

NOT Routine in CAP

Consider steroids only in:

- Septic shock
- COPD/asthma overlap
- Refractory severe inflammation

Routine corticosteroids are not universally recommended for uncomplicated CAP.

Fluid Management

- Avoid fluid overload
- Use balanced crystalloids
- Reassess frequently

Respiratory Support

HFNO

Preferred in:

- Moderate hypoxemic respiratory failure

NIV

May be considered in:

- COPD overlap

- Cardiogenic pulmonary edema overlap

Avoid delaying intubation.

Indications for Intubation

- Refractory hypoxemia
- Exhaustion
- Altered sensorium
- Shock
- Severe ARDS
- Respiratory arrest

ICU Referral Criteria

- Need for vasopressors
- Mechanical ventilation
- Severe hypoxemia
- Septic shock
- Multiorgan dysfunction
- CURB-65 ≥3

Antibiotic Duration

Recommended Duration

Usually:

- 5–7 days if clinically improving

Longer duration only if:

- MRSA
- Pseudomonas
- Lung abscess
- Empyema
- Immunocompromised patient

ERS guidelines support shorter antibiotic courses where clinically appropriate.

Monitoring

Monitor:

- Respiratory rate
- SpO_2
- Hemodynamics
- Urine output
- Mental status

Repeat imaging only if:

- Clinical deterioration
- Persistent fever
- Suspected complications

Discharge Criteria

- Hemodynamically stable
- Oral intake adequate
- Improving oxygenation
- Afebrile/improving fever
- Ambulating
- Stable comorbidities

Discharge Planning

Before discharge:

- Smoking cessation advice
- Vaccination advice
  - Influenza
  - Pneumococcal vaccine
- Review antibiotic completion
- Follow-up chest X-ray in selected cases
- Pulmonary rehab if indicated

Suggested Indian ED Antibiotic Protocol

Severity	Suggested Regimen
Mild CAP	Ceftriaxone + Azithromycin
Moderate CAP	Piperacillin-tazobactam + Azithromycin
Severe/ICU	Antipseudomonal beta-lactam + Macrolide
Aspiration	Piperacillin-tazobactam
Suspected MRSA	Add Linezolid/Vancomycin

4. ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

Definition

Acute Respiratory Distress Syndrome (ARDS) is an acute inflammatory lung injury causing:

- Severe hypoxemia
- Diffuse alveolar damage
- Increased pulmonary vascular permeability
- Non-cardiogenic pulmonary edema
- Reduced lung compliance

ARDS commonly occurs secondary to:

- Sepsis
- Severe pneumonia

- Aspiration
- Trauma
- Pancreatitis
- Massive transfusion
- Toxic inhalation
- Near drowning

The Berlin Definition classifies ARDS severity based on PaO_2/FiO_2 ratio with PEEP ≥5 cmH_2O.

Recognition

Clinical Features

- Severe dyspnea
- Tachypnea
- Hypoxemia refractory to oxygen therapy
- Increased work of breathing
- Bilateral crepitations
- Cyanosis
- Altered sensorium
- Respiratory fatigue

Common Risk Factors

- Sepsis
- Severe CAP
- Aspiration
- Polytrauma
- Pancreatitis
- Burns
- Massive transfusion
- Viral pneumonia

Severe Features

- Refractory hypoxemia
- Hemodynamic instability
- Respiratory exhaustion
- Hypercapnia
- Multiorgan dysfunction
- Shock

Diagnostic Criteria (Berlin Definition)

Timing

- Within 1 week of known clinical insult or worsening respiratory symptoms

Imaging

- Bilateral opacities on:
  - Chest X-ray
  - CT chest
  - Lung ultrasound

Origin of Edema

- Respiratory failure not fully explained by:
  - Cardiac failure
  - Fluid overload

Oxygenation Severity

Severity	PaO_2/FiO_2
Mild	201–300
Moderate	101–200
Severe	≤100

PEEP/CPAP ≥5 cmH_2O required.

Initial Assessment

ABCDE Assessment

Immediate Monitoring

- Pulse oximetry
- ECG
- Blood pressure
- Respiratory rate
- Urine output

Investigations

Essential

- ABG/VBG
- Chest X-ray
- CBC
- Renal function/electrolytes
- Lactate
- Blood cultures if sepsis suspected

Additional Investigations

- Procalcitonin/CRP if available
- Echocardiography
- Ultrasound chest
- CT chest in selected cases

POCUS Findings

- Bilateral B-lines

- Consolidation
- Pleural abnormalities

Oxygen Therapy

Oxygen Targets

- Maintain SpO_2 88–95%
- Avoid prolonged severe hyperoxia

Initial Oxygen Devices

- Non-rebreather mask
- High-flow nasal oxygen (HFNO)
- NIV in selected patients

HFNC ≥30 L/min is now recognized within modern ARDS definitions for non-intubated ARDS.

High Flow Nasal Oxygen (HFNO)

Consider In

- Mild ARDS
- Moderate hypoxemic respiratory failure
- Post-extubation support
- NIV intolerance

Benefits

- Improved oxygenation
- Reduced work of breathing
- Better comfort

Important Points

- Closely monitor for failure
- Avoid delaying intubation

HFNO is increasingly accepted as an important noninvasive support strategy in ARDS.

Non-Invasive Ventilation (NIV)

May Be Considered In

- Mild–moderate ARDS
- Cooperative patient
- No shock
- No severe acidosis
- No altered sensorium

Avoid NIV If

- Severe ARDS
- Hemodynamic instability
- Multiorgan failure

- Inability to protect airway

Helmet NIV may improve comfort and reduce ventilator-associated complications in selected patients.

Mechanical Ventilation

Lung Protective Ventilation

Core Strategy

- Low tidal volume ventilation
- Avoid volutrauma/barotrauma
- Prevent ventilator-induced lung injury

Recommended Settings

- Tidal volume: 6 mL/kg predicted body weight
- Plateau pressure <30 cmH_2O
- Driving pressure minimized
- Appropriate PEEP strategy

Low tidal volume ventilation remains the cornerstone of ARDS management.

PEEP Strategy

Goals

- Prevent alveolar collapse
- Improve oxygenation
- Reduce atelectrauma

Approach

- Moderate to high PEEP in moderate–severe ARDS
- Individualize according to:
  - Compliance
  - Oxygenation
  - Hemodynamics

Prone Positioning

Strongly Recommended In

- Moderate–severe ARDS
- PaO_2/FiO_2 <150

Suggested Duration

- ≥16 hours/day

Benefits

- Improved oxygenation
- Reduced mortality
- Better V/Q matching

Early prolonged prone positioning significantly improves outcomes in severe ARDS.

Fluid Management

Conservative Fluid Strategy Preferred

Goals:

- Avoid pulmonary edema worsening
- Maintain organ perfusion

Use

- Balanced crystalloids
- Frequent reassessment

Avoid:

- Excessive fluid administration

Sedation & Analgesia

Principles

- Adequate ventilator synchrony
- Avoid excessive deep sedation when possible

Preferred Agents

- Propofol
- Fentanyl
- Dexmedetomidine (selected patients)

Neuromuscular Blocking Agents (NMBA)

May be considered in:

- Severe ARDS
- Ventilator dyssynchrony
- Refractory hypoxemia
- Early severe ARDS (PaO_2/FiO_2 <100)

Routine prolonged paralysis is not universally recommended.

Recruitment Maneuvers

Important Update

Routine prolonged recruitment maneuvers are NOT recommended.

Avoid:

- Sustained airway pressures >35 cmH_2O for prolonged periods

Because of:

- Barotrauma risk
- Hemodynamic compromise

Corticosteroids

Routine Steroids

NOT universally recommended.

Consider In

- Selected moderate–severe ARDS

- COVID-related ARDS
- Refractory inflammatory state

Common Regimens

- Dexamethasone
  OR
- Methylprednisolone

Decision should be individualized.

ECMO (VV-ECMO)

Consider In Severe Refractory ARDS

Despite:

- Optimal ventilation
- Paralysis
- Proning
- Rescue therapies

Typical Indications

- Severe refractory hypoxemia
- Severe hypercapnia
- Unsafe ventilator pressures required

Requires:

- Experienced center
- Trained ECMO team

Indications for Intubation

- Refractory hypoxemia
- Respiratory exhaustion
- Altered sensorium
- Hemodynamic instability
- Severe acidosis
- Failed HFNO/NIV
- Respiratory arrest

Monitoring

Continuous Monitoring

- SpO_2
- Respiratory rate
- Hemodynamics
- Urine output
- Ventilator parameters

Repeat ABG

- After ventilator changes

- Clinical deterioration
- Persistent hypoxemia

Watch For Complications

- Pneumothorax
- Ventilator-associated pneumonia
- Barotrauma
- Shock
- AKI

ICU Admission Criteria

- Moderate–severe ARDS
- Mechanical ventilation
- NIV/HFNO failure
- Shock
- Multiorgan dysfunction
- Need for proning/ECMO

Disposition

Recovery Criteria

- Improving oxygenation
- Reduced ventilatory support
- Hemodynamic stability
- Improving underlying cause

Weaning Readiness

- Adequate mentation
- Hemodynamic stability
- Improved lung mechanics
- Acceptable oxygenation

5. ACUTE RESPIRATORY FAILURE

Definition

Acute Respiratory Failure (ARF) occurs when the respiratory system fails to maintain adequate:

- Oxygenation

OR

- Ventilation

Leading to:

- Hypoxemia
- Hypercapnia

- Respiratory acidosis
- Tissue hypoxia

Acute respiratory failure is a life-threatening emergency requiring rapid recognition and escalation of respiratory support.

Classification

Type 1 Respiratory Failure (Hypoxemic)

PaO_2 < 60\ mmHg

Features:

- Low PaO_2
- Normal or low $PaCO_2$

Common Causes:

- Pneumonia
- ARDS
- Pulmonary edema
- Pulmonary embolism
- Pneumothorax

Type 2 Respiratory Failure (Hypercapnic)

PaCO_2 > 45\ mmHg

Features:

- Elevated $PaCO_2$
- Respiratory acidosis may occur

Common Causes:

- COPD exacerbation
- Neuromuscular disease
- Obesity hypoventilation
- CNS depression
- Severe asthma
- Chest wall disorders

Recognition

Clinical Features

- Dyspnea
- Tachypnea
- Hypoxia
- Cyanosis
- Accessory muscle use
- Inability to speak full sentences
- Sweating/diaphoresis
- Altered mental status

- Exhaustion
- Reduced air entry

Red Flag Features

- Silent chest
- Bradypnea/apnea
- Agitation or confusion
- Hemodynamic instability
- Severe hypoxemia
- Hypercapnic encephalopathy
- Arrhythmias
- Cardiac arrest

Initial Assessment

ABCDE Assessment

Immediate Priorities

1. Airway patency
2. Oxygenation
3. Ventilation
4. Circulatory support
5. Identify underlying cause

Investigations

Essential

- Pulse oximetry
- ABG/VBG
- Chest X-ray
- ECG
- CBC
- Renal function/electrolytes
- Blood glucose

Additional Tests Depending on Cause

- Troponin
- BNP
- Lactate
- D-dimer
- Viral testing
- Blood cultures
- Ultrasound chest/POCUS
- Echocardiography

Arterial Blood Gas Interpretation

Evaluate:

- pH
- PaO_2
- $PaCO_2$
- HCO_3^-
- Lactate

Important Patterns

ABG Pattern	Suggests
Hypoxemia + low $PaCO_2$	Type 1 failure
Hypercapnia + acidosis	Type 2 failure
Rising $PaCO_2$	Ventilatory fatigue
Severe lactate elevation	Tissue hypoperfusion

Oxygen Therapy

Oxygen Is a Drug

Administer oxygen carefully and titrate to target saturations.

Strongly recommends target-based oxygen therapy rather than indiscriminate high-flow oxygen.

Target Saturations

Patient Group	Target SpO_2
Most acutely ill patients	94–98%
COPD/risk of hypercapnia	88–92%

Oxygen Delivery Devices

Device	Approx FiO_2
Nasal prongs	24–40%
Simple face mask	35–60%
Non-rebreather mask	Up to 90%
Venturi mask	Controlled oxygen
HFNO	Heated high-flow oxygen

High Flow Nasal Oxygen (HFNO)

Consider In

- Hypoxemic respiratory failure
- Pneumonia
- Early ARDS
- Post-extubation support

Benefits

- Improved oxygenation
- Reduced work of breathing
- Better tolerance than NIV in selected patients

Closely monitor for failure and avoid delayed intubation.

Non-Invasive Ventilation (NIV)

Strong Evidence Indications

- COPD exacerbation with hypercapnia
- Acute cardiogenic pulmonary edema

Selected Uses

- Obesity hypoventilation
- Neuromuscular weakness
- Mild ARDS

NIV Criteria in Hypercapnic Failure

pH < 7.35

AND

PaCO_2 > 45\ mmHg

Despite optimal medical therapy.

Contraindications to NIV

Absolute

- Respiratory arrest
- Inability to protect airway
- Massive aspiration
- Facial trauma
- Severe encephalopathy

Relative

- Agitation
- Copious secretions
- Hemodynamic instability
- Severe hypoxemia

Indications for Intubation

Airway Failure

- Reduced consciousness
- Aspiration risk
- Inability to protect airway

Oxygenation Failure

- Refractory hypoxemia despite maximal support

Ventilatory Failure

- Rising $PaCO_2$

- Worsening acidosis
- Respiratory fatigue

Clinical Deterioration

- Shock
- Exhaustion
- Cardiac arrest
- Failed NIV/HFNO

RCEM emphasizes avoiding delayed escalation to invasive ventilation in deteriorating patients.

Mechanical Ventilation Principles

Lung Protective Ventilation

Recommended Strategy

- Tidal volume 6–8 mL/kg predicted body weight
- Plateau pressure <30 cmH_2O
- Appropriate PEEP

Especially important in:

- ARDS
- Severe pneumonia

Management of Common Causes

COPD Exacerbation

- Controlled oxygen (88–92%)
- Bronchodilators
- Steroids
- NIV early if hypercapnic

Acute Pulmonary Edema

- CPAP/NIV
- Nitrates
- Diuretics
- Treat precipitating cause

Pneumonia

- Oxygen therapy
- Early antibiotics
- HFNO/NIV in selected patients

ARDS

- Lung protective ventilation
- Prone positioning
- Conservative fluids

Severe Asthma

- Bronchodilators
- Steroids
- Magnesium sulfate
- Careful ventilatory strategy if intubated

Monitoring

Continuous Monitoring

- SpO_2
- Respiratory rate
- Mental status
- Hemodynamics

Repeat ABG

- 30–60 minutes after intervention
- Clinical deterioration
- NIV initiation

Complications

Respiratory

- Barotrauma
- Pneumothorax
- Ventilator-associated pneumonia

Cardiovascular

- Arrhythmias
- Shock
- Cardiac arrest

Neurological

- Hypercapnic encephalopathy
- Hypoxic brain injury

ICU/HDU Referral Criteria

- NIV requirement
- Mechanical ventilation
- Persistent hypoxemia
- Severe acidosis
- Shock
- Multiorgan dysfunction

Disposition

Discharge Criteria

- Stable oxygenation
- Improved work of breathing
- Stable ABG

- No escalation requirement
- Treatable underlying cause

6. NON-INVASIVE VENTILATION (NIV) & INTUBATION CRITERIA

Scope

This section outlines:

- Indications for NIV
- Contraindications to NIV
- Initial NIV settings
- Monitoring during NIV
- Criteria for NIV failure
- Indications for intubation and invasive ventilation

NON-INVASIVE VENTILATION (NIV)

Definition

NIV provides ventilatory support without endotracheal intubation using:

- CPAP (Continuous Positive Airway Pressure)
- BiPAP/Bilevel ventilation

Goals:

- Improve oxygenation
- Reduce work of breathing
- Correct hypercapnia
- Avoid intubation complications

Common Emergency Department Indications

Strong Evidence Indications

- Acute exacerbation of COPD with hypercapnic respiratory failure
- Acute cardiogenic pulmonary edema
- Obesity hypoventilation syndrome
- Neuromuscular respiratory weakness (selected cases)

Selected/Conditional Uses

- Mild–moderate ARDS
- Post-extubation respiratory support
- Immunocompromised hypoxemic respiratory failure
- Chest wall deformity disorders

NIV CRITERIA

COPD Exacerbation

Indications

Start NIV if:

- pH <7.35
  AND
- $PaCO_2$ >45 mmHg (6 kPa)
  despite optimal medical therapy

Additional supportive findings:

- RR >24–25/min
- Increased work of breathing
- Accessory muscle use

Recommends NIV initiation within 60 minutes of qualifying ABG results.

Acute Pulmonary Edema

Consider CPAP/NIV If

- Persistent hypoxia
- Severe dyspnea
- Increased work of breathing
- Pulmonary edema despite oxygen therapy

Benefits:

- Reduces preload/afterload
- Improves oxygenation
- Reduces intubation rate

ARDS / Hypoxemic Respiratory Failure

NIV May Be Considered Only If

- Mild ARDS
- Hemodynamically stable
- Cooperative patient
- No multiorgan failure

Avoid prolonged NIV trial if worsening hypoxemia.

Contraindications to NIV

Absolute Contraindications

- Respiratory arrest
- Inability to protect airway
- Severe altered sensorium
- Massive aspiration/vomiting
- Facial trauma preventing mask seal
- Upper airway obstruction
- Untreated pneumothorax (without drain)

Relative Contraindications

- Severe agitation
- Copious secretions

- Hemodynamic instability
- Severe hypoxemia
- Recent upper GI surgery

Initial NIV Setup

Patient Preparation

- Explain procedure clearly
- Upright positioning (>30°)
- Choose appropriate mask size
- Minimize leaks

Suggested Initial BiPAP Settings

Parameter	Initial Setting
IPAP	10 cmH_2O
EPAP	4–5 cmH_2O
Backup Rate	12–16/min
FiO_2	Titrate to target saturation

Oxygen Targets

Condition	SpO_2 Target
COPD/Hypercapnic failure	88–92%
Others	92–96%

Settings should be titrated according to:

- Respiratory effort
- ABG response
- Patient comfort

CPAP Settings

Acute Pulmonary Edema

Suggested:

- CPAP 5–10 cmH_2O
- FiO_2 titrated to oxygenation

Monitoring During NIV

Continuous Monitoring

- SpO_2
- Respiratory rate
- Heart rate
- Blood pressure
- Mental status

Repeat ABG

- 1 hour after NIV initiation
- 1 hour after major setting changes

Signs of NIV Success

Clinical Improvement

- Reduced respiratory distress
- Reduced RR
- Improved comfort
- Reduced accessory muscle use

ABG Improvement

- Rising pH
- Falling $PaCO_2$
- Improved oxygenation

NIV Failure Criteria

Immediate Failure

- Respiratory arrest
- Severe agitation
- Inability to tolerate mask
- Hemodynamic collapse

Progressive Failure

- Worsening acidosis
- Persistent/worsening hypoxemia
- Rising $PaCO_2$
- Altered mental status
- Exhaustion
- Inability to clear secretions

Do NOT delay intubation when NIV is failing.

INTUBATION CRITERIA

General Indications for Intubation

Airway Failure

- Inability to protect airway
- Aspiration risk
- GCS deterioration

Oxygenation Failure

- Refractory hypoxemia despite maximal support
- Severe ARDS
- Persistent SpO_2 failure

Ventilatory Failure

- Rising $PaCO_2$ with worsening acidosis

- Respiratory muscle fatigue
- Apnea

Clinical Deterioration

- Exhaustion
- Hemodynamic instability
- Cardiac arrest
- Severe sepsis/shock

Specific Intubation Triggers

Condition	Important Trigger
COPD	Worsening acidosis despite NIV
Asthma	Silent chest/exhaustion
Pulmonary edema	NIV failure
ARDS	Severe refractory hypoxemia
Sepsis	Shock/encephalopathy
Trauma	Airway protection

Emergency Airway Preparation

Pre-Intubation Checklist

- Airway assessment
- Difficult airway equipment ready
- Suction ready
- Backup airway plan
- Vasopressors prepared if unstable
- Continuous monitoring

Preoxygenation

Preferred Methods

- Non-rebreather mask
- HFNO
- NIV/CPAP in severe hypoxemia

Critically ill patients desaturate rapidly during intubation. Oxygen optimization before induction is essential.

Rapid Sequence Intubation (RSI)

Common Induction Agents

Agent	Advantages
Ketamine	Hemodynamic stability
Etomidate	Minimal hypotension
Propofol	Useful if hypertensive

Paralytics

Agent	Notes
Succinylcholine	Rapid onset
Rocuronium	Alternative when succinylcholine is contraindicated

Post-Intubation Priorities

Immediate Steps

- Confirm tube placement with waveform capnography
- Secure ET tube
- Start lung-protective ventilation
- Sedation and analgesia
- Post-intubation chest X-ray

Mechanical Ventilation Principles

Lung Protective Strategy

Recommended:

- Tidal volume 6–8 mL/kg predicted body weight
- Plateau pressure <30 cmH_2O
- Appropriate PEEP

Particularly important in:

- ARDS
- Severe asthma
- Pulmonary edema

Complications of NIV

Common

- Mask discomfort
- Air leaks
- Dryness
- Gastric distension

Serious

- Aspiration
- Pneumothorax
- Delayed intubation
- Hemodynamic compromise

Complications of Intubation

Immediate

- Hypotension
- Hypoxia
- Esophageal intubation

- Aspiration
- Cardiac arrest

Ventilator Related

- Barotrauma
- Ventilator-associated pneumonia
- Delirium

7. PNEUMOTHORAX

Definition

Presence of air in the pleural cavity causing partial or complete lung collapse.

Types:

- Primary spontaneous pneumothorax (PSP)
- Secondary spontaneous pneumothorax (SSP)
- Traumatic pneumothorax
- Tension pneumothorax

Recognition

Clinical Features

- Sudden pleuritic chest pain
- Dyspnea
- Tachypnea
- Reduced air entry
- Hyperresonance
- Tachycardia

Severe Features

- Hypoxia
- Cyanosis
- Hemodynamic instability

Initial Assessment

ABCDE Assessment

Investigations

- Pulse oximetry
- Chest X-ray
- Point-of-care ultrasound (POCUS) if available
- ABG in severe cases

CT Chest

Not routinely required in ED.

Consider if:

- Uncertain diagnosis
- Underlying lung disease
- Persistent air leak
- Recurrent pneumothorax

Strongly supports ultrasound use for rapid bedside diagnosis where expertise exists.

Tension Pneumothorax

Clinical Diagnosis — DO NOT WAIT FOR X-RAY

Features:

- Severe respiratory distress
- Hypotension
- Tachycardia
- Distended neck veins
- Tracheal deviation (late sign)
- Severe hypoxia
- Cardiac arrest (PEA)

Management of Tension Pneumothorax

Immediate Decompression

Preferred Initial Approach

- Finger thoracostomy where trained personnel available

OR

Needle Decompression

- Large bore cannula
- 4th or 5th intercostal space anterior-to-mid axillary line preferred

Lateral decompression rather than traditional 2nd intercostal midclavicular approach is favoured because of lower failure rates.

Definitive Treatment

- Intercostal chest drain insertion

Primary Spontaneous Pneumothorax (PSP)

Small PSP

- Minimal symptoms
- Hemodynamically stable

Management

- Observation
- Ambulation
- Analgesia
- Follow-up imaging

Conservative management is now strongly supported in selected stable patients.

Large or Symptomatic PSP

Preferred Initial Management

- Needle aspiration

  OR
- Small-bore chest drain

Ambulatory Management

Can be considered in selected stable patients using:

- One-way valve systems
- Ambulatory pleural devices

If:

- Stable
- Reliable follow-up available
- No severe hypoxia

Secondary Spontaneous Pneumothorax (SSP)

Usually occurs in:

- COPD
- ILD
- Tuberculosis
- Cystic lung disease

Higher risk than PSP.

Management

Generally admit all SSP patients.

Treatment

- Oxygen
- Chest drain if symptomatic/significant
- Treat underlying disease

Observation alone only in very small stable SSP.

Chest Drain Recommendations

Preferred

- Small-bore chest drains (≤14F)

Avoid routine large-bore drains unless:

- Hemothorax
- Trauma
- Massive air leak

Recommends small-bore drains in most spontaneous pneumothoraces

Oxygen Therapy

Supplemental Oxygen

Can increase pleural air absorption.

Use:

- In hypoxic patients
- Carefully in COPD patients

Avoid unnecessary high-flow oxygen in COPD.

Persistent Air Leak

Consider thoracic surgery referral if:

- Air leak >5–7 days
- Failure of lung re-expansion
- Recurrent pneumothorax

Surgical Referral Indications

- Recurrent ipsilateral pneumothorax
- Bilateral pneumothorax
- Persistent air leak
- High-risk professions
    - Pilot
    - Diver

Surgical Options

- VATS pleurodesis
- Bullectomy

Traumatic Pneumothorax

Consider Associated Injuries

- Rib fractures
- Hemothorax
- Pulmonary contusion

Management

- Oxygen
- Chest drain if significant
- Trauma evaluation

Ultrasound in Pneumothorax

POCUS Findings

- Absent lung sliding
- Barcode sign
- Lung point

Useful in:

- Unstable patients
- Trauma
- ICU
- Difficult X-ray interpretation

Disposition

Discharge Criteria

- Stable vitals
- Minimal symptoms
- No progression
- Reliable follow-up
- Adequate understanding of warning signs

Patient Advice Before Discharge

Avoid:

- Air travel until radiological resolution
- Scuba diving permanently unless definitive surgery performed
- Smoking

Smoking cessation strongly advised.

Follow-Up

- Repeat chest X-ray
- Pulmonary/thoracic surgery review if recurrent
- Evaluate underlying lung disease

8.ACUTE PULMONARY EDEMA (APE)

Definition

Acute pulmonary edema is rapid accumulation of fluid within alveoli and interstitial lung tissue causing:

- Severe dyspnea
- Hypoxemia
- Respiratory distress

Most commonly cardiogenic due to:

- Acute heart failure
- Hypertensive emergency
- Acute coronary syndrome
- Arrhythmias

Recognition

Clinical Features

- Sudden severe dyspnea
- Orthopnea
- Tachypnea
- Diaphoresis
- Pink frothy sputum

- Crepitations
- Hypertension (common in SCAPE)
- Hypoxia
- Anxiety/agitation

Severe Features

- Altered sensorium
- Exhaustion
- Hypotension
- Cardiogenic shock

Initial Assessment

ABCDE Approach

Immediate Monitoring

- Cardiac monitor
- Pulse oximetry
- Blood pressure
- ECG

Investigations

- Chest X-ray
- ABG/VBG
- CBC
- Renal function/electrolytes
- Troponin
- BNP/NT-proBNP if available
- Bedside echocardiography
- POCUS lung (B-lines)

Point-of-care ultrasound is increasingly recommended for rapid diagnosis of pulmonary edema.

Positioning

- Sit patient upright
- Legs dependent if tolerated

This rapidly reduces venous return and improves symptoms.

Oxygen Therapy

Oxygen Targets

- Maintain SpO_2 >94%
- COPD patients: 88–92%

Oxygen Devices

- Nasal prongs
- Face mask

- Non-rebreather mask

Avoid unnecessary oxygen in non-hypoxemic patients.

Non-Invasive Ventilation

NIV/CPAP SHOULD BE CONSIDERED EARLY

Preferred Modalities

- CPAP
- BiPAP

Indications

- Moderate–severe respiratory distress
- Persistent hypoxemia
- Increased work of breathing
- Hypercapnia

Benefits:

- Reduces preload/afterload
- Improves oxygenation
- Reduces intubation
- Improves survival

Current evidence strongly supports early NIV as one of the most effective therapies in acute pulmonary edema.

Suggested Initial Settings

- CPAP: 5–10 cmH_2O
- FiO_2 titrated to oxygenation

Vasodilator Therapy

NITRATES ARE NOW EARLY FIRST-LINE THERAPY

Especially useful in:

- Hypertensive pulmonary edema
- SCAPE (Sympathetic Crashing Acute Pulmonary Edema)

Nitroglycerin

Sublingual GTN

- 0.4–0.6 mg every 5 minutes initially

IV Nitroglycerin

If SBP >100 mmHg:

- Start 10–20 mcg/min
- Rapid titration according to BP

Benefits:

- Reduces preload
- Reduces afterload
- Rapid symptom improvement

Modern management emphasizes aggressive vasodilator therapy rather than relying only on diuretics.

Diuretics

IV Furosemide

Suggested Dose

- 40–80 mg IV

Higher dose if:

- Chronic diuretic use
- CKD

Important Update

Diuretics are most useful when:

- True fluid overload present

Not always primary therapy in:

- Flash pulmonary edema (fluid redistribution states)

Evidence suggests nitrates + NIV are often more important initially than aggressive diuresis alone.

Hypertensive Pulmonary Edema (SCAPE)

Key Features

- Severe hypertension
- Sudden pulmonary edema
- Sympathetic surge

Preferred Treatment

1. NIV/CPAP early
2. High-dose nitrates
3. Avoid delay in vasodilators

SCAPE management now emphasizes rapid afterload reduction. (EMCrit Project)

Hypotensive Pulmonary Edema

If SBP <90 mmHg

Avoid:

- Aggressive nitrates
- Excess diuretics

Consider:

- Dobutamine
- Vasopressors if shock

Search for:

- MI
- Cardiogenic shock
- Arrhythmia

Hypotensive pulmonary edema requires separate management strategy.
(Dr.Oracle)
Morphine (IMPORTANT UPDATE)
Routine Morphine NOT Recommended
Because of:

- Respiratory depression
- Worse outcomes
- Delayed recognition of deterioration

Use only selectively for:

- Severe distress/anxiety

Indications for Intubation

- NIV failure
- Severe hypoxemia
- Altered sensorium
- Respiratory arrest
- Exhaustion
- Cardiogenic shock

Treat Underlying Cause
Always evaluate for:

- Acute coronary syndrome
- Arrhythmia
- Hypertensive emergency
- Valvular disease
- Renal failure
- Noncompliance

Monitoring
Continuous:

- SpO_2
- ECG
- Blood pressure
- Urine output

Repeat:

- ABG if worsening
- Electrolytes/renal function after diuretics

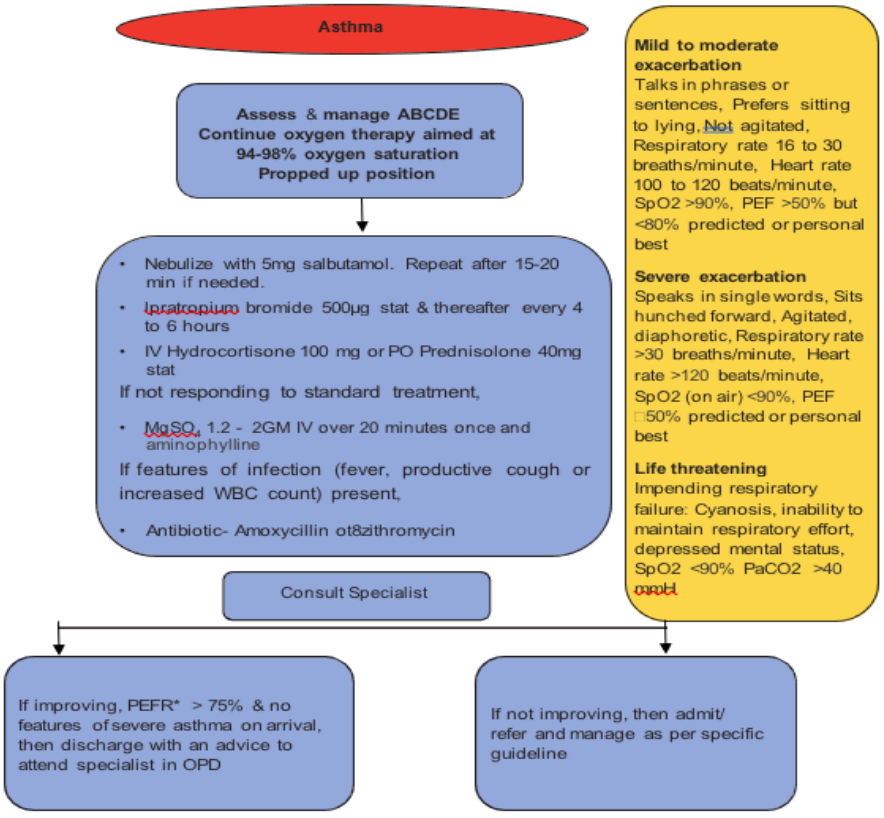
Disposition
ICU/HDU Indications

- NIV requirement
- Persistent hypoxia

- Hemodynamic instability
- ACS
- Arrhythmias
- Intubation

Algorithm

Algorithm for the management of Acute Severe Asthma in Adults



Peak Expiratory Flow Rate (PEFR)

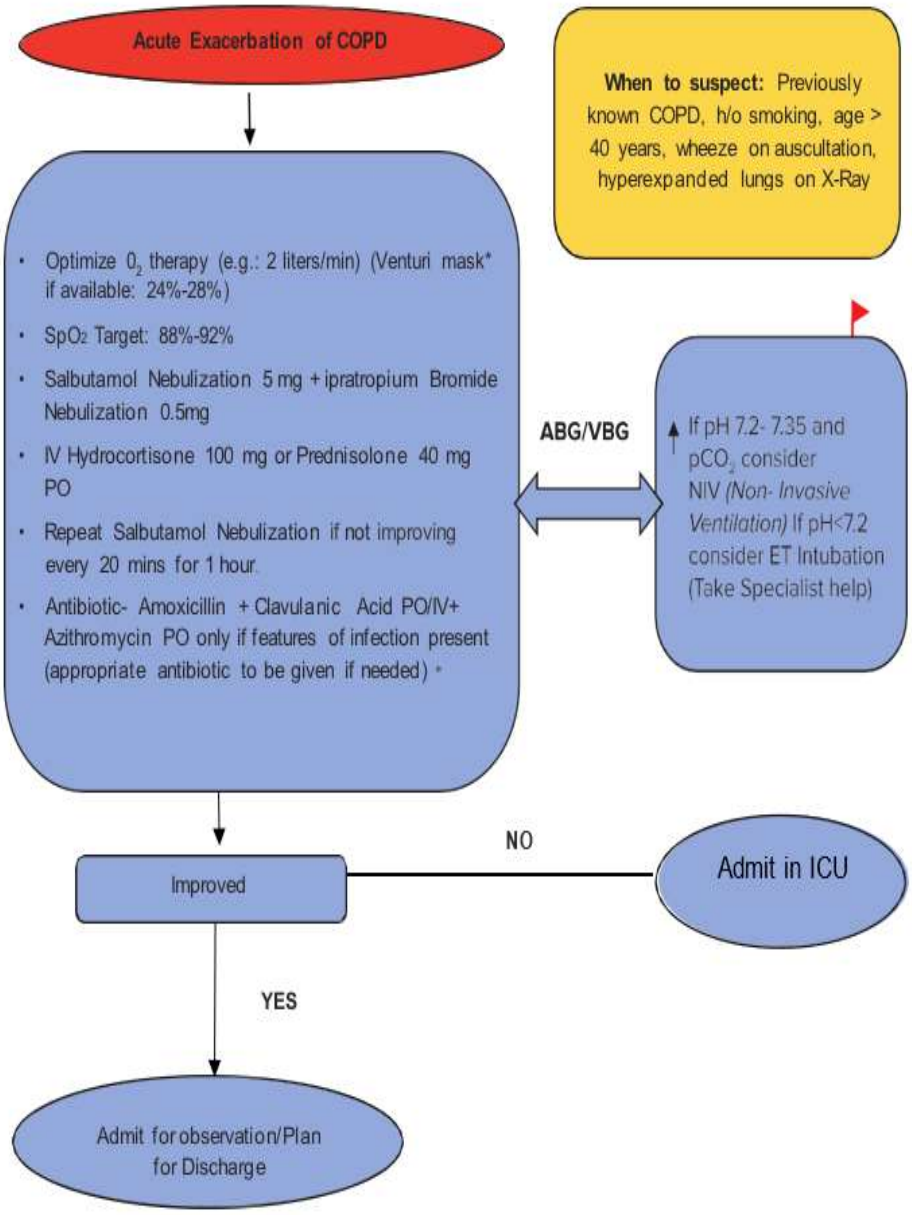
Serial measurements of Peak Expiratory Flow Rate (PEFR) is a key component in asthma disease assessment and response to therapy in emergency department.

Initial Assessment

PEFR>=40% (of percentage predicted) ➔ mild to moderate asthma

PEFR<40% (of percentage predicted) ➔ severe asthma

Algorithm for the management of Acute Exacerbation of Chronic Obstructive Pulmonary Disease



SECTION VI
ENDOCRINE & METABOLIC EMERGENCIES

Scope

This protocol provides a standardized approach for recognition, assessment, resuscitation, investigation, stabilization, and early management of endocrine and metabolic emergencies presenting to Emergency Departments in Government Medical Institutions across Kerala.

The guideline applies to:

1. Primary Health Centres
2. Taluk Hospitals
3. District Hospitals
4. Government Medical Colleges
5. Critical Care Units

This section focuses on:

1. Diabetic Ketoacidosis (DKA)
2. Hyperosmolar Hyperglycemic State (HHS)
3. Hypoglycemia
4. Hyponatremia
5. Hypernatremia
6. Hyperkalemia
7. Thyroid Emergencies
8. Adrenal Crisis

Acute metabolic derangements causing life-threatening physiological instability

The protocol is intended for:

1. Emergency Physicians
2. Casualty Medical Officers
3. Internal Medicine Teams
4. Critical Care Teams
5. Nurses
6. Junior Residents
7. Paramedics

Special emphasis is placed on:

1. Early recognition of metabolic emergencies
2. Protocolized fluid and electrolyte correction
3. Prevention of cardiac and neurological complications
4. Identification of precipitating factors
5. Timely ICU referral where required

Introduction

Endocrine and metabolic emergencies are acute life-threatening disorders caused by severe hormonal dysfunction or metabolic derangement leading to disturbances in:

1. Glucose homeostasis
2. Electrolyte balance
3. Acid-base physiology
4. Fluid balance
5. Cellular metabolism

These conditions may rapidly progress to:

1. Shock
2. Cardiac arrhythmias
3. Respiratory failure
4. Seizures
5. Cerebral edema
6. Coma
7. Multi-organ dysfunction

Management principles include:

1. ABCDE stabilization
2. Rapid correction of life-threatening abnormalities
3. Identification of precipitating causes
4. Frequent monitoring
5. Prevention of overcorrection complications
6. Timely escalation of care

All critically ill endocrine patients should undergo:

1. Continuous monitoring
2. Electrolyte assessment
3. Cardiac monitoring where indicated
4. Frequent reassessment

Definitions

Endocrine Emergency

An acute disorder caused by severe hormonal dysfunction resulting in actual or impending life-threatening physiological instability.

DIABETIC KETOACIDOSIS (DKA)

1. INTRODUCTION

Diabetic Ketoacidosis (DKA) is an acute life-threatening metabolic emergency caused by absolute or relative insulin deficiency associated with increased counter-regulatory hormones including glucagon, catecholamines, cortisol, and growth hormone.

The metabolic consequences include:

1. Hyperglycemia
2. Ketogenesis
3. Metabolic acidosis
4. Osmotic diuresis
5. Severe fluid depletion
6. Electrolyte derangements

DKA most commonly occurs in:

1. Type 1 Diabetes Mellitus
2. Ketosis-prone Type 2 Diabetes Mellitus
3. Newly diagnosed diabetes mellitus
4. Patients with severe physiological stress
5. SGLT2 inhibitor associated euglycemic ketoacidosis

Mortality in DKA is usually related to:

1. Delayed diagnosis
2. Sepsis
3. Shock
4. Severe electrolyte abnormalities
5. Cerebral edema
6. Acute kidney injury
7. Acute respiratory failure
8. Cardiac arrhythmias
9. Underlying precipitating illness

Early protocolized management significantly reduces morbidity, ICU utilization, and mortality.

2. PATHOPHYSIOLOGY

Insulin deficiency combined with excess counter-regulatory hormones results in:

1. Increased hepatic gluconeogenesis and glycogenolysis
2. Reduced peripheral glucose utilization
3. Accelerated lipolysis
4. Increased free fatty acid delivery to the liver
5. Hepatic ketone production

6. Metabolic acidosis due to accumulation of ketoacids

Hyperglycemia produces osmotic diuresis leading to:

1. Severe dehydration
2. Sodium losses
3. Potassium depletion
4. Magnesium depletion
5. Phosphate depletion

Although serum potassium may initially appear normal or elevated, total body potassium stores are markedly depleted.

3. DEFINITIONS

Diabetic Ketoacidosis (DKA)

DKA is defined by the presence of:

1. Hyperglycemia or known diabetes mellitus
2. Ketosis
3. Metabolic acidosis

4. DIAGNOSTIC CRITERIA

Diagnostic Criteria for DKA

Parameter	**Diagnostic Criteria**
Hyperglycemia	Blood glucose ≥200 mg/dL OR known diabetes mellitus
Ketosis	Beta-hydroxybutyrate ≥3 mmol/L OR urine ketones ≥2+
Metabolic acidosis	pH <7.3 OR serum bicarbonate <18 mmol/L

Important Diagnostic Principles

1. Direct serum beta-hydroxybutyrate measurement is preferred over urine ketone testing whenever available.
2. Venous blood gas (VBG) is acceptable in most patients and may replace arterial blood gas unless severe respiratory failure is suspected.
3. Anion gap should not be used as the sole diagnostic criterion.
4. Euglycemic DKA may occur with only mildly elevated glucose values, particularly in patients using SGLT2 inhibitors.

5. SEVERITY CLASSIFICATION

Severity	**Beta-hydroxybutyrate**	**pH**	**Serum Bicarbonate**	**Mental Status**
Mild DKA	≤6 mmol/L	>7.25	≥15 mmol/L	Alert
Moderate DKA	≤6 mmol/L	7.0–7.25	10 to <15 mmol/L	Alert/drowsy
Severe DKA	>6 mmol/L	<7.0	<10 mmol/L	Stupor/coma may occur

6. PRECIPITATING FACTORS

Common Precipitating Causes

Infectious Causes

1. Pneumonia
2. Urinary tract infection
3. Skin and soft tissue infection
4. Sepsis
5. Intra-abdominal infection

Medication Related Causes

1. Insulin omission
2. Inadequate insulin dosing
3. Corticosteroids
4. Sympathomimetics
5. SGLT2 inhibitors
6. Antipsychotics

Acute Medical Conditions

1. Myocardial infarction
2. Stroke
3. Pancreatitis
4. Trauma
5. Surgery
6. Gastrointestinal bleeding
7. Pulmonary embolism

Other Causes

1. Newly diagnosed diabetes mellitus
2. Pregnancy
3. Alcohol use
4. Substance abuse
5. Starvation

7. CLINICAL FEATURES

Symptoms

1. Polyuria
2. Polydipsia
3. Vomiting
4. Abdominal pain
5. Generalized weakness
6. Weight loss
7. Dyspnea

8. Altered sensorium
9. Fever or infective symptoms

Signs

1. Dehydration
2. Tachycardia
3. Hypotension
4. Poor peripheral perfusion
5. Kussmaul respiration
6. Acetone breath odor
7. Altered mental status
8. Reduced urine output
9. Shock in severe cases

8. INITIAL ASSESSMENT AND RESUSCITATION

Immediate Priorities

1. Rapid recognition of DKA
2. Assessment of airway, breathing, and circulation
3. Identification of precipitating factors
4. Early fluid resuscitation
5. Correction of electrolyte abnormalities
6. Timely insulin administration
7. Prevention of complications

9. ABCDE ASSESSMENT

Airway

Assess for:

1. Airway protection
2. Vomiting
3. Aspiration risk
4. Reduced consciousness

Intubation May Be Required In

1. Severe coma
2. Respiratory failure
3. Refractory shock
4. Inability to protect airway
5. Cardiac arrest

Avoid unnecessary intubation whenever possible because abrupt correction of compensatory hyperventilation may worsen acidosis.

Breathing

Assess:

1. Respiratory rate
2. Oxygen saturation
3. Work of breathing
4. Kussmaul respiration
5. Pulmonary edema
6. Respiratory fatigue

Provide supplemental oxygen if hypoxemic.

Circulation

Assess:

1. Pulse rate
2. Blood pressure
3. Capillary refill
4. Peripheral perfusion
5. Urine output
6. Shock state

Immediate Measures

1. Establish two large-bore intravenous lines
2. Initiate cardiac monitoring
3. Begin fluid resuscitation immediately
4. Insert urinary catheter if strict urine monitoring required

Disability

Assess:

1. Glasgow Coma Scale
2. Mental status
3. Capillary blood glucose
4. Seizures
5. Focal neurological deficits

Exposure

Assess for:

1. Infection source
2. Trauma
3. Injection sites
4. Pressure injuries
5. Surgical abdomen
6. Skin infections

10. INVESTIGATIONS

Mandatory Investigations

1. Capillary and laboratory blood glucose

2. Venous blood gas or arterial blood gas
3. Serum electrolytes
4. Renal function tests
5. Serum bicarbonate
6. Serum beta-hydroxybutyrate if available
7. Urine ketones
8. Complete blood count
9. ECG
10. Serum osmolality if indicated

Additional Investigations as Indicated

1. Chest radiograph
2. Blood cultures
3. Urine culture
4. Lactate
5. Troponin
6. Serum lipase/amylase
7. Pregnancy test
8. Toxicology screening
9. CT brain in unexplained altered sensorium

11. FLUID MANAGEMENT

Principles of Fluid Therapy

Patients with DKA typically have a fluid deficit of approximately 6–9 liters.

Fluid replacement aims to:

1. Restore intravascular volume
2. Improve tissue perfusion
3. Reduce counter-regulatory hormones
4. Improve renal perfusion
5. Facilitate glucose clearance
6. Correct dehydration gradually and safely

Initial Fluid Therapy

Preferred Fluids

1. Balanced crystalloids such as:
    - Ringer Lactate
    - Plasma-Lyte
2. Normal saline is acceptable if balanced crystalloids unavailable.

Initial Rate

Administer:

500–1000 mL/hour during the first 2–4 hours depending on:

1. Severity of dehydration
2. Hemodynamic status
3. Cardiac function
4. Renal function
5. Age
6. Comorbid illness

Subsequent Fluid Therapy

After initial resuscitation:

1. Reduce infusion rate to approximately 250 mL/hour
2. Adjust according to:
    - Corrected sodium
    - Urine output
    - Hemodynamics
    - Ongoing losses
    - Cardiac and renal status

Special Situations

Elderly Patients

Use cautious fluid administration due to risk of:

1. Pulmonary edema
2. Heart failure
3. Fluid overload

Chronic Kidney Disease or Dialysis

1. Smaller fluid boluses
2. Frequent reassessment
3. Close monitoring for pulmonary edema

Dextrose Addition

When serum glucose falls below 250 mg/dL:

1. Add 5–10% dextrose to IV fluids
2. Continue insulin infusion
3. Prevent hypoglycemia while ketoacidosis resolves

Insulin must not be discontinued solely because glucose normalizes.

12. POTASSIUM MANAGEMENT

Principles

Despite normal or elevated serum potassium at presentation, total body potassium depletion is substantial in DKA.

Insulin administration drives potassium intracellularly and may precipitate life-threatening hypokalemia.

Potassium Replacement Protocol

Serum Potassium	Management
<3.5 mmol/L	Hold insulin and aggressively replace potassium
3.5–5.0 mmol/L	Add 20–30 mmol potassium per liter of IV fluid
>5.0 mmol/L	No replacement initially; monitor closely

Monitoring

1. Check potassium at baseline
2. Reassess 2 hours after insulin initiation
3. Repeat every 4 hours thereafter
4. Continuous ECG monitoring recommended in significant potassium abnormalities

13. INSULIN THERAPY

Principles

Insulin therapy:

1. Suppresses ketogenesis
2. Reduces hepatic glucose production
3. Facilitates glucose utilization
4. Corrects acidosis

Intravenous Insulin Therapy

Indications

IV insulin infusion is preferred in:

1. Severe DKA
2. Moderate DKA with instability
3. Altered sensorium
4. Shock
5. ICU patients
6. Persistent vomiting
7. Severe acidosis

Standard IV Insulin Regimen

Administer:

Regular insulin infusion at 0.1 units/kg/hour IV.

Routine IV insulin bolus is not recommended.

Therapeutic Goals

Expected glucose reduction:

50–75 mg/dL/hour

If glucose does not fall adequately:

1. Reassess fluid therapy
2. Check IV access

3. Confirm infusion accuracy
4. Evaluate for ongoing sepsis or shock
5. Increase insulin infusion incrementally if required

14. SUBCUTANEOUS INSULIN PATHWAY

Indications

Subcutaneous rapid-acting insulin analog protocols may be considered in:

1. Mild DKA
2. Selected uncomplicated moderate DKA
3. Hemodynamically stable patients
4. Patients without altered mental status
5. Patients without severe vomiting
6. Settings capable of close nursing and biochemical monitoring

This pathway may reduce:

1. ICU utilization
2. Emergency Department boarding time
3. Cost of care
4. Resource burden in high-volume centers

Eligibility Criteria

All of the following should ideally be present:

1. pH >7.0
2. Serum bicarbonate ≥10 mmol/L
3. Alert and cooperative patient
4. Preserved peripheral perfusion
5. No vasopressor requirement
6. No respiratory failure
7. Ability to perform frequent glucose and electrolyte monitoring

Suggested Regimen

Initial Dose

Administer:

1. Insulin lispro or insulin aspart:
    - 0.2–0.3 units/kg subcutaneously initially

Maintenance Dosing

Followed by:

1. 0.1–0.2 units/kg subcutaneously every 1–2 hours

Monitoring Requirements

1. Hourly glucose monitoring
2. Electrolytes every 4 hours
3. Frequent reassessment of:

- Mental status
- Hydration
- Hemodynamics
- Ketosis

Contraindications to SC Insulin Pathway

1. Severe DKA
2. Shock
3. Pregnancy
4. Altered mental status
5. Severe acidosis
6. Persistent vomiting
7. Respiratory failure
8. Poor peripheral circulation
9. Requirement for ICU-level support
10. Inability to monitor closely

15. BICARBONATE THERAPY

Recommendations

Routine bicarbonate therapy is not recommended in DKA.

Studies have not demonstrated clear benefit in most patients and inappropriate use may lead to complications.

Potential Risks of Bicarbonate Therapy

1. Hypokalemia
2. Delayed ketone clearance
3. Hypernatremia
4. Cerebral edema
5. Volume overload
6. Paradoxical central nervous system acidosis

Indications for Bicarbonate Therapy

Consider bicarbonate only if:

1. Arterial or venous pH <6.9
2. Severe life-threatening hyperkalemia
3. Severe cardiovascular compromise attributable to acidosis

Suggested Regimen

Prepare:

1. Sodium bicarbonate 100 mmol
2. In 400 mL sterile water
3. Add 20 mmol potassium chloride

Administer over 2 hours.

Repeat every 2 hours until:

1. pH >7.0
2. Hemodynamic improvement occurs

16. PHOSPHATE MANAGEMENT

General Principles

Total body phosphate depletion is common in DKA due to osmotic diuresis. However, routine phosphate replacement has not shown consistent clinical benefit.

Indications for Replacement

Consider phosphate replacement if:

1. Serum phosphate <1 mg/dL
2. Respiratory muscle weakness
3. Cardiac dysfunction
4. Severe anemia
5. Prolonged critical illness

Monitoring During Replacement

Monitor closely for:

1. Hypocalcemia
2. Arrhythmias
3. Soft tissue calcification
4. Hyperphosphatemia

17. MAGNESIUM MANAGEMENT

Importance

Magnesium depletion frequently accompanies potassium depletion in DKA. Hypomagnesemia may contribute to:

1. Refractory hypokalemia
2. Cardiac arrhythmias
3. Neuromuscular instability

Replacement

Consider magnesium replacement if:

1. Serum magnesium low
2. Arrhythmias present
3. Refractory hypokalemia present

18. MONITORING

Clinical Monitoring

Hourly Monitoring

1. Pulse rate
2. Blood pressure

3. Respiratory rate
4. Oxygen saturation
5. Neurological status
6. Capillary blood glucose
7. Urine output
8. Fluid balance

Biochemical Monitoring

Every 2–4 Hours

1. Serum electrolytes
2. Potassium
3. Serum bicarbonate
4. Venous pH
5. Creatinine
6. Serum ketones or beta-hydroxybutyrate where available

Therapeutic Targets

1. Glucose reduction of 50–75 mg/dL/hour
2. Potassium maintained between 4–5 mmol/L
3. Gradual correction of osmolality
4. Progressive clearance of ketosis

Monitor Closely For

1. Hypoglycemia
2. Hypokalemia
3. Fluid overload
4. Pulmonary edema
5. Hyperchloremic metabolic acidosis
6. Acute respiratory distress syndrome
7. Cerebral edema
8. Arrhythmias

19. CEREBRAL EDEMA

Importance

Cerebral edema is uncommon in adults but potentially fatal.

It is more frequently observed in:

1. Young adults
2. Severe DKA
3. Marked hyperosmolality
4. Rapid osmotic shifts

Warning Signs

1. Sudden headache

2. Bradycardia
3. Hypertension
4. Reduced consciousness
5. Seizures
6. Cranial nerve abnormalities
7. Behavioral change

Immediate Management

1. Reduce fluid administration rate
2. Urgent ICU transfer
3. Obtain neuroimaging
4. Elevate head end
5. Consider:
    - Mannitol 0.5–1 g/kg IV
    - Hypertonic saline
6. Neurospecialist consultation

20. RESOLUTION OF DKA

Criteria for Resolution

DKA is considered resolved when ALL of the following are achieved:

1. Clinical improvement
2. Hemodynamic stability
3. Ability to tolerate oral intake
4. Serum bicarbonate ≥15 mmol/L
5. pH >7.3
6. Beta-hydroxybutyrate <1 mmol/L where available

If beta-hydroxybutyrate unavailable:

1. Ketones negative or trace
2. Clinical recovery present

Closure of anion gap is not mandatory for defining DKA resolution.

21. TRANSITION TO SUBCUTANEOUS INSULIN

Principles

Transition should occur only after:

1. Resolution of ketoacidosis
2. Stable hemodynamics
3. Ability to eat orally
4. Acceptable electrolyte status

Transition Strategy

1. Administer long-acting basal insulin before discontinuing IV insulin.
2. Continue IV insulin infusion for 1–2 hours after SC insulin administration.

3. Avoid interruption of insulin delivery.
4. Resume appropriate nutritional insulin regimen.

Basal Insulin Considerations

Early administration of basal insulin may reduce:

1. Rebound hyperglycemia
2. Recurrent ketoacidosis
3. Transition failures

22. SPECIAL SITUATIONS

SGLT2 Inhibitor Associated Euglycemic DKA

Clinical Features

Patients may present with:

1. Significant ketosis
2. Metabolic acidosis
3. Mildly elevated or normal glucose levels

Common Triggers

1. Fasting
2. Surgery
3. Infection
4. Reduced carbohydrate intake
5. Reduced insulin administration
6. Pregnancy
7. Excess alcohol intake

Management Principles

1. Early recognition is critical.
2. Treatment principles remain similar to standard DKA.
3. Dextrose-containing fluids may be required earlier.
4. SGLT2 inhibitor therapy should be discontinued.

DKA in Pregnancy

Important Considerations

1. DKA may occur at lower glucose levels.
2. Fetal mortality risk is high.
3. Rapid treatment is essential.

Management Principles

1. Early ICU involvement
2. Obstetric consultation
3. Continuous fetal monitoring in viable pregnancies
4. Aggressive correction of metabolic abnormalities

DKA in Chronic Kidney Disease and Dialysis

Important Considerations

1. Reduced osmotic diuresis may mask dehydration.
2. Fluid overload risk is increased.
3. Potassium abnormalities may be severe.

Management Principles

1. Smaller fluid boluses
2. Frequent reassessment
3. Early nephrology involvement
4. Careful potassium replacement
5. Monitoring for pulmonary edema

23. COMPLICATIONS OF DKA AND ITS TREATMENT

1. Hypoglycemia
2. Hypokalemia
3. Hyperkalemia
4. Cerebral edema
5. Pulmonary edema
6. Acute respiratory distress syndrome
7. Hyperchloremic metabolic acidosis
8. Acute kidney injury
9. Venous thromboembolism
10. Cardiac arrhythmias
11. Aspiration pneumonia

24. ICU ADMISSION CRITERIA

ICU Admission Recommended if Any of the Following Present

1. Severe DKA
2. pH <7.0
3. Beta-hydroxybutyrate >6 mmol/L
4. Shock
5. Vasopressor requirement
6. Respiratory failure
7. Altered mental status
8. Severe electrolyte abnormalities
9. Significant comorbid illness
10. Requirement for invasive monitoring
11. Pregnancy with severe DKA
12. Persistent deterioration despite therapy

25. DISPOSITION

Ward Admission

Appropriate for patients with:

1. Mild or improving moderate DKA
2. Stable hemodynamics
3. No organ failure
4. No ICU indications

ICU Admission

Required for:

1. Severe DKA
2. Hemodynamic instability
3. Respiratory compromise
4. Altered consciousness
5. Severe metabolic derangements
6. Requirement for invasive monitoring

26. PREVENTION AND DISCHARGE COUNSELING

Patient Education

Patients should receive counseling regarding:

1. Insulin adherence
2. Sick-day rules
3. Ketone testing during illness
4. Adequate hydration
5. Recognition of warning symptoms
6. Home glucose monitoring
7. Infection management
8. SGLT2 inhibitor precautions
9. Avoidance of insulin omission

Discharge Planning

Ensure:

1. Appropriate insulin prescription
2. Follow-up appointment arrangement
3. Diabetes educator review
4. Medication reconciliation
5. Nutritional counseling
6. Social support assessment where needed

Indications

Subcutaneous rapid-acting insulin analogs may be considered in:

1. Mild DKA

2. Selected uncomplicated moderate DKA
3. Hemodynamically stable patients
4. Patients without altered mental status
5. Settings with close monitoring capability

Suggested Regimen

1. Initial rapid-acting insulin analog:
    - Lispro or aspart 0.2–0.3 units/kg SC
2. Followed by:
    - 0.1–0.2 units/kg SC every 1–2 hours

Contraindications to SC Insulin Pathway

1. Severe DKA
2. Shock
3. Pregnancy
4. Altered sensorium
5. Persistent vomiting
6. ICU requirement
7. Poor peripheral perfusion

HYPEROSMOLAR HYPERGLYCEMIC STATE (HHS)

1. INTRODUCTION

Hyperosmolar Hyperglycemic State (HHS) is a life-threatening metabolic emergency characterized by:

1. Severe hyperglycemia
2. Hyperosmolarity
3. Profound dehydration
4. Minimal or absent ketosis
5. Altered mental status of variable severity

HHS most commonly occurs in:

1. Elderly patients
2. Patients with Type 2 Diabetes Mellitus
3. Undiagnosed diabetes mellitus
4. Patients with significant medical comorbidities

Compared with Diabetic Ketoacidosis (DKA), HHS is associated with:

1. Greater fluid deficits
2. More severe hyperosmolarity
3. Higher mortality
4. Greater neurological dysfunction

5. Increased thrombotic risk

Mortality is usually related to:

1. Delayed recognition
2. Severe dehydration
3. Sepsis
4. Stroke
5. Myocardial infarction
6. Renal failure
7. Thromboembolic complications
8. Underlying precipitating illness

Early recognition and careful controlled correction of hyperosmolarity are essential to reduce morbidity and mortality.

2. PATHOPHYSIOLOGY

Relative insulin deficiency combined with elevated counter-regulatory hormones results in:

1. Marked hyperglycemia
2. Severe osmotic diuresis
3. Progressive dehydration
4. Electrolyte depletion
5. Hyperosmolarity

Unlike DKA, residual insulin activity is usually sufficient to suppress significant ketogenesis.

Progressive dehydration may lead to:

1. Reduced renal perfusion
2. Worsening hyperglycemia
3. Severe hypernatremia
4. Acute kidney injury
5. Cerebral dysfunction
6. Circulatory collapse

Estimated fluid deficits in HHS are frequently between 8–12 liters.

3. DEFINITION

Hyperosmolar Hyperglycemic State (HHS) is defined as a hyperglycemic emergency characterized by:

1. Severe hyperglycemia
2. Hyperosmolarity
3. Severe dehydration
4. Minimal or absent ketoacidosis

4. DIAGNOSTIC CRITERIA

Diagnostic Criteria for HHS

Parameter	Diagnostic Criteria
Plasma glucose	≥600 mg/dL
Effective serum osmolality	>300 mOsm/kg
Total serum osmolality	>320 mOsm/kg
Beta-hydroxybutyrate	<3 mmol/L or absent ketosis
pH	≥7.3
Serum bicarbonate	≥15 mmol/L
Mental status	Altered sensorium common

Important Diagnostic Principles

- ✓ Mixed DKA-HHS presentations may occur.
- ✓ Significant ketosis suggests overlap with DKA.
- ✓ Serum osmolality should be calculated and monitored serially.
- ✓ Neurological dysfunction correlates more closely with osmolality than glucose level.
- ✓ Venous blood gas is acceptable in most patients.

5. CALCULATION OF EFFECTIVE SERUM OSMOLALITY

Effective serum osmolality may be calculated using:

Effective Serum Osmolality (mOsm/kg) = $2 \times Na^{+} + [Glucose\ (mg/dL) / 18]$

Where:

- Sodium is measured in mEq/L
- Glucose is measured in mg/dL

Rapid decline in osmolality should be avoided because it may precipitate cerebral edema.

6. PRECIPITATING FACTORS

Infectious Causes

1. Pneumonia
2. Urinary tract infection
3. Skin and soft tissue infection
4. Sepsis
5. Intra-abdominal infection

Cardiovascular and Neurological Causes

1. Stroke
2. Myocardial infarction
3. Pulmonary embolism
4. Mesenteric ischemia

Medication Related Causes

1. Poor medication adherence

2. Corticosteroids
3. Thiazide diuretics
4. Antipsychotics
5. Sympathomimetics
6. SGLT2 inhibitors

Other Causes

- ✓ Severe dehydration
- ✓ Renal failure
- ✓ Pancreatitis
- ✓ Trauma
- ✓ Surgery
- ✓ Undiagnosed diabetes mellitus
- ✓ Substance abuse

7. CLINICAL FEATURES

Symptoms

1. Polyuria
2. Polydipsia
3. Progressive weakness
4. Reduced oral intake
5. Weight loss
6. Confusion
7. Lethargy
8. Visual disturbances
9. Seizures in severe cases

Signs

1. Severe dehydration
2. Tachycardia
3. Hypotension
4. Poor peripheral perfusion
5. Dry mucous membranes
6. Altered mental status
7. Focal neurological deficits
8. Seizures
9. Reduced urine output
10. Shock in severe cases

8. INITIAL ASSESSMENT AND RESUSCITATION

Immediate Priorities

1. Rapid recognition of HHS

2. Assessment of airway, breathing, and circulation
3. Restoration of intravascular volume
4. Gradual correction of hyperosmolarity
5. Identification and treatment of precipitating causes
6. Prevention of neurological complications
7. Monitoring for thromboembolic events

9. ABCDE ASSESSMENT

Airway

Assess for:

1. Airway protection
2. Reduced consciousness
3. Aspiration risk
4. Vomiting

Intubation May Be Required In

1. Severe coma
2. Respiratory failure
3. Cardiac arrest
4. Inability to protect airway

Breathing

Assess:

1. Respiratory rate
2. Oxygen saturation
3. Work of breathing
4. Pulmonary edema
5. Respiratory fatigue

Provide supplemental oxygen if hypoxemic.

Circulation

Assess:

1. Pulse rate
2. Blood pressure
3. Capillary refill
4. Peripheral perfusion
5. Urine output
6. Shock state

Immediate Measures

1. Establish intravenous access
2. Initiate cardiac monitoring
3. Begin fluid resuscitation immediately

4. Insert urinary catheter if strict monitoring required

Disability

Assess:

1. Glasgow Coma Scale
2. Neurological deficits
3. Mental status
4. Capillary blood glucose
5. Seizures

Exposure

Assess for:

1. Infection source
2. Trauma
3. Pressure injuries
4. Dehydration
5. Precipitating illness

10. INVESTIGATIONS

Mandatory Investigations

1. Capillary and laboratory blood glucose
2. Serum electrolytes
3. Renal function tests
4. Serum osmolality
5. Venous or arterial blood gas
6. Serum ketones or beta-hydroxybutyrate
7. Complete blood count
8. ECG
9. Urinalysis

Additional Investigations as Indicated

1. Chest radiograph
2. Blood cultures
3. Urine culture
4. Lactate
5. Troponin
6. CT brain if focal neurological deficits present
7. Toxicology screening
8. Serum lipase/amylase
9. Coagulation profile

11. FLUID MANAGEMENT

Principles of Fluid Therapy

Fluid replacement is the cornerstone of HHS management.
Therapeutic goals include:

1. Restoration of circulating volume
2. Gradual reduction of osmolality
3. Improvement in renal perfusion
4. Reduction of glucose concentration
5. Correction of dehydration
6. Prevention of cerebral edema

Patients with HHS often have very large fluid deficits.
Initial Fluid Therapy
Preferred Fluids
1. Balanced crystalloids such as:
- Ringer Lactate
- Plasma-Lyte

2. Normal saline remains acceptable if balanced crystalloids unavailable.

Initial Rate
Administer:
1. 500–1000 mL/hour during the first 2–4 hours depending on:
- Hemodynamic status
- Severity of dehydration
- Cardiac function
- Renal function
- Age
- Comorbid illness

Subsequent Fluid Therapy
After initial stabilization:
Adjust fluids according to:
- Serum sodium
- Osmolality
- Urine output
- Hemodynamics
- Cardiac and renal function

If Corrected Sodium Normal or Elevated
Use:
1. 0.45% saline

If Corrected Sodium Low
Continue:
1. 0.9% saline

Rate of Osmolality Correction

Rapid osmotic shifts should be avoided.

Target decline in serum osmolality:

1. Approximately 3–8 mOsm/kg/hour

Excessively rapid reduction may precipitate:

1. Cerebral edema
2. Neurological deterioration

Special Situations

Elderly Patients

Use cautious fluid administration due to increased risk of:

1. Pulmonary edema
2. Heart failure
3. Renal dysfunction

Chronic Kidney Disease and Dialysis

1. Smaller fluid boluses
2. Frequent reassessment
3. Close monitoring for fluid overload
4. Early nephrology involvement

Addition of Dextrose

When serum glucose falls below 250–300 mg/dL:

1. Add 5–10% dextrose to IV fluids
2. Continue insulin infusion
3. Prevent rapid osmolar shifts and hypoglycemia

12. POTASSIUM MANAGEMENT

Principles

Total body potassium depletion is common despite normal or elevated serum potassium at presentation.

Insulin therapy may rapidly lower serum potassium.

Potassium Replacement Protocol

Serum Potassium	**Management**
<3.5 mmol/L	Hold insulin and replace potassium aggressively
3.5–5.0 mmol/L	Add 20–30 mmol potassium per liter IV fluid
>5.0 mmol/L	No replacement initially; monitor closely

Monitoring

1. Check potassium at baseline
2. Reassess every 2–4 hours
3. Continuous ECG monitoring in significant abnormalities

13. INSULIN THERAPY

Principles

Fluid replacement alone may significantly reduce glucose concentration.

Insulin therapy should generally be initiated only after adequate initial fluid resuscitation.

Rapid glucose lowering should be avoided.

Intravenous Insulin Regimen

Administer:

1. Regular insulin infusion at 0.05 units/kg/hour IV

Routine insulin bolus is not recommended.

Therapeutic Goals

1. Glucose reduction of approximately 50–75 mg/dL/hour
2. Gradual correction of osmolality
3. Avoidance of rapid osmotic shifts

If Glucose Falls Too Rapidly

1. Reduce insulin infusion rate
2. Add dextrose-containing fluids earlier
3. Reassess osmolality trend

14. SODIUM MANAGEMENT

Corrected Sodium

Hyperglycemia lowers measured serum sodium.

Corrected sodium should be calculated to guide fluid therapy.

Corrected Sodium (mEq/L) = Measured Sodium + 1.6 × [(Glucose − 100) / 100]

Clinical Importance

1. Rising sodium during treatment may occur despite improving hyperglycemia.
2. Effective osmolality trend is more clinically important than isolated sodium values.
3. Rapid sodium shifts should be avoided.

15. PHOSPHATE MANAGEMENT

Routine phosphate replacement is not recommended.

Consider replacement if:

1. Serum phosphate <1 mg/dL
2. Respiratory muscle weakness
3. Cardiac dysfunction
4. Severe anemia

Monitor calcium closely during replacement.

16. MAGNESIUM MANAGEMENT

Hypomagnesemia may contribute to:

1. Arrhythmias
2. Neuromuscular instability
3. Refractory hypokalemia

Consider magnesium replacement if:

1. Serum magnesium low
2. Arrhythmias present
3. Refractory hypokalemia present

17. THROMBOEMBOLIC RISK

Importance

HHS is associated with increased risk of:

1. Deep vein thrombosis
2. Pulmonary embolism
3. Stroke
4. Arterial thrombosis

This risk is related to:

1. Severe dehydration
2. Hyperviscosity
3. Immobility
4. Prothrombotic state

Prevention

Consider venous thromboembolism prophylaxis unless contraindicated.

18. MONITORING

Clinical Monitoring

Hourly Monitoring

1. Pulse rate
2. Blood pressure
3. Respiratory rate
4. Oxygen saturation
5. Neurological status
6. Capillary blood glucose
7. Urine output
8. Fluid balance

Biochemical Monitoring

Every 2–4 Hours

1. Electrolytes
2. Serum osmolality
3. Potassium
4. Creatinine

5. Venous pH if indicated
6. Serum glucose

Therapeutic Targets

1. Gradual decline in osmolality
2. Controlled glucose reduction
3. Restoration of renal perfusion
4. Improvement in mental status
5. Correction of dehydration

Monitor Closely For

1. Cerebral edema
2. Hypoglycemia
3. Hypokalemia
4. Fluid overload
5. Pulmonary edema
6. Acute kidney injury
7. Thromboembolic complications

19. CEREBRAL EDEMA

Importance

Rapid reduction in osmolality may precipitate cerebral edema.

Although uncommon in adults, it is potentially fatal.

Warning Signs

1. Sudden headache
2. Reduced consciousness
3. Bradycardia
4. Hypertension
5. Seizures
6. Neurological deterioration

Immediate Management

1. Reduce rate of osmolar correction
2. Reduce fluid administration rate
3. Urgent ICU transfer
4. Obtain neuroimaging
5. Consider:
   - Mannitol
   - Hypertonic saline
6. Neurospecialist consultation

20. RESOLUTION OF HHS

Criteria for Resolution

HHS is considered resolved when ALL of the following are achieved:

1. Serum osmolality normalized or substantially improved
2. Mental status improved
3. Hemodynamic stability achieved
4. Oral intake tolerated
5. Adequate hydration restored
6. Glucose controlled safely

21. TRANSITION TO SUBCUTANEOUS INSULIN

Transition Criteria

1. Clinically stable
2. Eating orally
3. Hyperosmolar state resolved
4. Electrolytes reasonably corrected

Transition Principles

1. Administer basal insulin before stopping IV insulin.
2. Continue IV insulin infusion for 1–2 hours after first SC dose.
3. Avoid interruption in insulin delivery.

22. SPECIAL SITUATIONS

Mixed DKA-HHS Presentation

Some patients present with features of both DKA and HHS.

These patients may have:

1. Severe hyperglycemia
2. Hyperosmolarity
3. Significant ketosis
4. Acidosis

Management should address:

1. Volume depletion
2. Ketosis
3. Electrolyte abnormalities
4. Hyperosmolarity

HHS in Elderly Patients

Elderly patients are at higher risk of:

1. Delirium
2. Falls
3. Renal failure
4. Fluid overload
5. Cardiovascular complications

Careful individualized fluid therapy is essential.

HHS in Renal Failure

1. Smaller fluid boluses
2. Close monitoring for pulmonary edema
3. Frequent electrolyte reassessment
4. Early nephrology involvement

23. COMPLICATIONS OF HHS AND ITS TREATMENT

1. Cerebral edema
2. Hypoglycemia
3. Hypokalemia
4. Fluid overload
5. Pulmonary edema
6. Acute kidney injury
7. Venous thromboembolism
8. Stroke
9. Cardiac arrhythmias
10. Aspiration pneumonia
11. Acute respiratory distress syndrome

24. ICU ADMISSION CRITERIA

ICU Admission Recommended if Any of the Following Present

1. Severe altered sensorium
2. Shock
3. Severe electrolyte abnormalities
4. Respiratory failure
5. Significant comorbid illness
6. Need for invasive monitoring
7. Multi-organ dysfunction
8. Severe hyperosmolarity
9. Persistent deterioration despite therapy
10. Seizures

25. DISPOSITION

Ward Admission

Appropriate for patients with:

1. Improving clinical status
2. Stable hemodynamics
3. No organ failure
4. No ICU indication

ICU Admission

Required for:

1. Hemodynamic instability
2. Severe neurological impairment
3. Respiratory compromise
4. Severe metabolic derangement
5. Multi-organ dysfunction

26. PREVENTION AND DISCHARGE COUNSELING

Patient Education

Patients should receive counseling regarding:

1. Medication adherence
2. Adequate hydration
3. Sick-day management
4. Home glucose monitoring
5. Recognition of warning symptoms
6. Infection management
7. Early medical consultation

Discharge Planning

Ensure:

1. Appropriate diabetes therapy
2. Follow-up appointment arrangement
3. Diabetes educator review
4. Nutritional counseling
5. Medication reconciliation
6. Social support assessment where needed

HYPOGLYCEMIA

Definition

Hypoglycemia is classified according to the American Diabetes Association (ADA) Standards of Care:

Level	Definition
Level 1 Hypoglycemia	Glucose <70 mg/dL and ≥54 mg/dL
Level 2 Hypoglycemia	Glucose <54 mg/dL
Level 3 Hypoglycemia	Severe hypoglycemia associated with altered mental and/or physical status requiring assistance, regardless of glucose value

Clinical Features

Autonomic (Adrenergic) Symptoms

1. Sweating

2. Tremors
3. Palpitations
4. Anxiety
5. Hunger
6. Tachycardia

Neuroglycopenic Symptoms

1. Confusion
2. Behavioral changes
3. Drowsiness
4. Visual disturbance
5. Slurred speech
6. Seizures
7. Focal neurological deficits
8. Coma

Initial Assessment

Assess:

1. Airway protection
2. Mental status
3. Capillary blood glucose
4. Ability to swallow safely
5. Underlying precipitating cause

Look for:

1. Insulin overdose
2. Sulfonylurea use
3. Sepsis
4. Renal failure
5. Liver disease
6. Alcohol use
7. Missed meals or starvation

Management

Conscious Patient Able to Swallow

Preferred Treatment: Oral Rapid-Acting Carbohydrate

Administer:

1. 15–20 g oral glucose or fast-acting carbohydrate such as:
   * Glucose tablets
   * Glucose gel
   * Fruit juice
   * Sugared beverage

Examples:

1. 3–4 glucose tablets
2. 150–200 mL fruit juice
3. 1 tablespoon sugar or honey

Reassessment

Repeat capillary glucose after 15 minutes.

If glucose remains <70 mg/dL:

1. Repeat 15–20 g oral glucose
2. Reassess again after 15 minutes

Once glucose normalizes:

1. Provide longer-acting carbohydrate meal or snack to prevent recurrence.

This is commonly referred to as the "15–15 rule."

Unconscious Patient or Severe Hypoglycemia

Intravenous Therapy

Administer:

1. 25 g IV dextrose:
   * 50 mL of 50% dextrose
   OR
   * 100 mL of 25% dextrose depending on institutional practice

Follow with:

1. Continuous dextrose infusion if recurrent hypoglycemia risk present.

If IV Access Unavailable

Administer glucagon:

1. 1 mg IM or SC glucagon

OR

2. Intranasal glucagon where available.

Special Situations

Sulfonylurea-Induced Hypoglycemia

High risk of recurrent prolonged hypoglycemia.

Management may require:

1. Prolonged observation
2. Repeated glucose monitoring
3. Continuous dextrose infusion
4. Consideration of octreotide in recurrent cases

Alcohol-Related Hypoglycemia

Consider thiamine administration in malnourished or chronic alcohol use patients before glucose where appropriate.

Monitoring

Immediate Monitoring

1. Repeat blood glucose after 15 minutes
2. Reassess neurological status
3. Monitor airway and vital signs

Ongoing Monitoring

1. Repeat glucose monitoring every 15–30 minutes initially
2. Observe for recurrent hypoglycemia
3. Identify and treat underlying cause

Disposition

Discharge Criteria

Patient may be discharged if:

1. Fully conscious and neurologically normal
2. Able to eat safely
3. No recurrent hypoglycemia
4. Underlying cause addressed
5. Reliable follow-up available

Admission Criteria

Consider admission for:

1. Persistent or recurrent hypoglycemia
2. Sulfonylurea-associated hypoglycemia
3. Altered mental status
4. Seizures
5. Lack of social support
6. Significant comorbid illness

Prevention and Counseling

Patients should receive education regarding:

1. Recognition of hypoglycemia symptoms
2. Appropriate glucose correction
3. Medication adherence
4. Meal regularity
5. Sick-day management
6. Carrying glucose sources
7. Glucagon availability for high-risk individuals

HYPONATREMIA

1. INTRODUCTION

Hyponatremia is the most common electrolyte abnormality encountered in hospitalized patients.

It is associated with:

1. Increased mortality
2. Neurological complications
3. Falls and fractures
4. Prolonged hospitalization
5. Increased ICU utilization

Hyponatremia results from excess water relative to sodium and may occur in association with:

1. Hypovolemia
2. Euvolemia
3. Hypervolemia

The severity of symptoms depends on:

1. Absolute sodium level
2. Rapidity of sodium decline
3. Chronicity
4. Cerebral adaptation

Rapid correction of chronic hyponatremia may cause osmotic demyelination syndrome (ODS), a potentially irreversible neurological injury.

2. DEFINITION

Hyponatremia is defined as:

Hyponatremia is defined as serum sodium <135 mEq/L.

3. CLASSIFICATION

By Severity

Severity	Serum Sodium
Mild	130–134 mEq/L
Moderate	125–129 mEq/L
Severe	<125 mEq/L

By Symptom Severity

Mild Symptoms

1. Nausea
2. Headache
3. Mild confusion

Moderate Symptoms

1. Vomiting
2. Marked confusion
3. Agitation

4. Drowsiness

Severe Symptoms

1. Seizures
2. Coma
3. Respiratory arrest
4. Severe neurological dysfunction

By Chronicity

Type	Duration
Acute	<48 hours
Chronic	≥48 hours or unknown duration

4. ETIOLOGY

Hypovolemic Hyponatremia

1. Vomiting
2. Diarrhea
3. Diuretics
4. Burns
5. Adrenal insufficiency

Euvolemic Hyponatremia

1. SIADH
2. Hypothyroidism
3. Glucocorticoid deficiency
4. Psychogenic polydipsia
5. Drug-induced hyponatremia

Hypervolemic Hyponatremia

- Heart failure
- Cirrhosis
- Nephrotic syndrome
- Renal failure

5. CLINICAL FEATURES

Symptoms

1. Nausea
2. Vomiting
3. Headache
4. Weakness
5. Confusion
6. Lethargy
7. Seizures
8. Reduced consciousness

9. Coma

Signs

- Altered mental status
- Neurological deficits
- Signs of dehydration
- Edema
- Hypotension
- Tachycardia

6. INITIAL ASSESSMENT

Immediate Priorities

1. Assess symptom severity
2. Determine chronicity if possible
3. Identify volume status
4. Exclude life-threatening neurological complications
5. Prevent overcorrection

7. ABCDE ASSESSMENT

Airway

Assess airway protection and aspiration risk.

Intubation may be required in:

1. Severe coma
2. Status epilepticus
3. Respiratory failure

Breathing

Assess:

1. Respiratory rate
2. Oxygen saturation
3. Pulmonary edema

Circulation

Assess:

1. Pulse
2. Blood pressure
3. Peripheral perfusion
4. Volume status

Disability

Assess:

1. GCS
2. Seizures
3. Focal neurological deficits

4. Mental status

Exposure

Assess for:

1. Dehydration
2. Edema
3. Infection
4. Drug exposure

8. INVESTIGATIONS

Mandatory Investigations

1. Serum electrolytes
2. Serum osmolality
3. Urine osmolality
4. Urine sodium
5. Renal function tests
6. Blood glucose
7. ECG

Additional Investigations

1. Thyroid function tests
2. Serum cortisol
3. Liver function tests
4. Chest radiograph
5. CT brain if neurological deficit present

9. MANAGEMENT PRINCIPLES

Treatment depends on:

1. Symptom severity
2. Chronicity
3. Volume status
4. Underlying cause

The primary goal in severe symptomatic hyponatremia is controlled partial correction to reduce cerebral edema.

10. SEVERE SYMPTOMATIC HYPONATREMIA

Indications

1. Seizures
2. Coma
3. Severe confusion
4. Respiratory compromise

Hypertonic Saline Therapy

Administer:

1. 3% hypertonic saline
2. 150 mL IV over 20 minutes

Repeat up to 2 additional times if severe symptoms persist.

Therapeutic Target

Initial target:

Initial target: Increase serum sodium by 4–6 mEq/L

This is usually sufficient to reduce cerebral edema and improve symptoms.

11. CORRECTION LIMITS

Rapid correction must be avoided.

Recommended Limits

Standard Risk Patients

Correction ≤10 mEq/L in 24 hours

High Risk for Osmotic Demyelination

Includes:

1. Alcoholism
2. Malnutrition
3. Liver disease
4. Hypokalemia
5. Very low sodium

Correction target:

Correction ≤8 mEq/L in 24 hours

12. HYPOTONIC HYPONATREMIA MANAGEMENT BY VOLUME STATUS

Hypovolemic Hyponatremia

Treatment:

1. 0.9% saline
2. Volume replacement
3. Treat underlying losses
4. Stop offending diuretics

Euvolemic Hyponatremia (SIADH)

Treatment:

1. Fluid restriction
2. Treat underlying cause
3. Increase solute intake if needed
4. Consider loop diuretics in selected cases

Hypervolemic Hyponatremia

Treatment:

1. Fluid restriction
2. Salt restriction

3. Loop diuretics
4. Treat underlying heart failure/cirrhosis/renal disease

13. OVERCORRECTION MANAGEMENT

If sodium rises excessively:

1. Stop ongoing correction
2. Consider:
   - o IV dextrose
   - o Desmopressin
3. Frequent sodium monitoring

14. MONITORING

Severe Hyponatremia

1. Serum sodium every 2–4 hours
2. Neurological assessment
3. Urine output
4. Fluid balance

Monitor Closely For

1. Osmotic demyelination syndrome
2. Cerebral edema
3. Seizures
4. Fluid overload

15. OSMOTIC DEMYELINATION SYNDROME

Clinical Features

1. Dysarthria
2. Dysphagia
3. Quadriparesis
4. Locked-in syndrome
5. Altered consciousness

Prevention

1. Avoid rapid correction
2. Frequent sodium monitoring
3. Lower correction targets in high-risk patients

16. ICU ADMISSION CRITERIA

1. Severe symptomatic hyponatremia
2. Seizures
3. Coma
4. Need for hypertonic saline infusion
5. Severe neurological dysfunction
6. Respiratory failure

17. DISPOSITION

Ward Admission

1. Moderate stable hyponatremia
2. Improving symptoms

ICU Admission

1. Severe neurological symptoms
2. Rapid sodium shifts
3. Hypertonic saline requirement

HYPERNATREMIA

1. DEFINITION

Hypernatremia is defined as: Serum Sodium >145 mEq/L

2. INTRODUCTION

Hypernatremia reflects a deficit of water relative to sodium.

It is associated with:

1. High mortality
2. Neurological dysfunction
3. Dehydration
4. Renal injury

Hypernatremia is particularly common in:

1. Elderly patients
2. Critically ill patients
3. Patients with impaired thirst or access to water

3. CLASSIFICATION

Severity	Serum Sodium
Mild	146–150 mEq/L
Moderate	151–159 mEq/L
Severe	≥160 mEq/L

4. ETIOLOGY

Water Loss

1. Gastrointestinal losses
2. Fever
3. Sweating
4. Osmotic diuresis
5. Diabetes insipidus

Sodium Gain

1. Hypertonic saline

2. Sodium bicarbonate excess
3. Salt poisoning

5. CLINICAL FEATURES
   1. Thirst
   2. Weakness
   3. Irritability
   4. Confusion
   5. Seizures
   6. Coma
   7. Neuromuscular irritability
6. INVESTIGATIONS
   1. Serum electrolytes
   2. Serum osmolality
   3. Urine osmolality
   4. Renal function tests
   5. Urine output monitoring
7. MANAGEMENT PRINCIPLES
   1. Identify acute vs chronic hypernatremia
   2. Correct free water deficit gradually
   3. Avoid rapid osmotic shifts
   4. Treat underlying cause
8. FREE WATER DEFICIT

   Water Deficit = TBW × [(Current Na / 140) − 1]

   Where TBW:
   1. Approximately 0.6 × body weight in men
   2. Approximately 0.5 × body weight in women
9. RATE OF CORRECTION

   Chronic Hypernatremia

   Recommended correction: Correction Rate12 mEq/L/day

   Rapid correction may cause cerebral edema.

   Acute Hypernatremia

   More rapid correction may be tolerated.
10. FLUID THERAPY

Hypovolemic Hypernatremia
   1. Restore intravascular volume first using isotonic crystalloids
   2. Then replace free water deficit

Euvolemic Hypernatremia
   1. Free water replacement

2. Treat diabetes insipidus if present

Hypervolemic Hypernatremia

1. Free water
2. Diuretics
3. Treat sodium overload

11. MONITORING
    1. Sodium every 4–6 hours initially
    2. Neurological assessment
    3. Urine output
    4. Fluid balance
12. COMPLICATIONS
    1. Cerebral edema
    2. Seizures
    3. Fluid overload
    4. Neurological deterioration
13. ICU ADMISSION CRITERIA
    1. Severe hypernatremia
    2. Seizures
    3. Coma
    4. Hemodynamic instability
    5. Severe dehydration

HYPERKALEMIA

1. DEFINITION

Hyperkalemia is defined as: Serum Potassium >5.0 mEq/L

2. CLASSIFICATION

Severity	**Potassium Level**
Mild	5.1–5.5 mEq/L
Moderate	5.6–6.0 mEq/L
Severe	>6.0 mEq/L

3. INTRODUCTION

Hyperkalemia is a potentially life-threatening electrolyte emergency associated with:

1. Cardiac arrhythmias
2. Conduction abnormalities
3. Cardiac arrest
4. Neuromuscular dysfunction

Rapid recognition and treatment are essential.

4. ETIOLOGY

Reduced Renal Excretion

1. Acute kidney injury
2. Chronic kidney disease
3. Hypoaldosteronism
4. Potassium-sparing diuretics

Cellular Shift

1. Metabolic acidosis
2. Rhabdomyolysis
3. Tumor lysis syndrome
4. Insulin deficiency

Excess Intake

1. Potassium supplements
2. Excess IV potassium

5. CLINICAL FEATURES
    1. Weakness
    2. Paresthesia
    3. Paralysis
    4. Palpitations
    5. Syncope
    6. Cardiac arrest
6. ECG CHANGES
    1. Peaked T waves
    2. PR prolongation
    3. QRS widening
    4. Sine wave pattern
    5. Ventricular arrhythmias
7. INVESTIGATIONS
    1. Serum electrolytes
    2. Renal function tests
    3. Blood gas
    4. ECG
    5. Blood glucose
8. MANAGEMENT PRINCIPLES

Treatment priorities:

1. Stabilize myocardium
2. Shift potassium intracellularly

3. Remove potassium from body
4. Treat underlying cause

9. CARDIAC MEMBRANE STABILIZATION

Indications

1. ECG changes
2. Severe hyperkalemia
3. Arrhythmias

Calcium Therapy

Administer:

1. Calcium gluconate 10%
2. 10 mL IV over 2–5 minutes

Repeat if ECG changes persist.

10. INTRACELLULAR SHIFT OF POTASSIUM

Insulin and Glucose

Administer:

1. Regular insulin 10 units IV
2. With 25 g glucose IV

Beta-2 Agonist

1. Nebulized salbutamol

Sodium Bicarbonate

Consider if severe metabolic acidosis present.

11. POTASSIUM REMOVAL

1. Loop diuretics
2. Potassium binders
3. Hemodialysis

Dialysis is definitive therapy in severe or refractory hyperkalemia.

12. MONITORING

1. Continuous cardiac monitoring
2. Repeat potassium levels
3. Repeat ECG
4. Glucose monitoring after insulin therapy

13. ICU ADMISSION CRITERIA

1. Severe hyperkalemia
2. ECG abnormalities
3. Arrhythmias
4. Dialysis requirement
5. Hemodynamic instability

14. KEY PRACTICE POINTS

1. ECG changes may precede cardiac arrest.
2. Calcium stabilizes myocardium but does not lower potassium.
3. Insulin shifts potassium intracellularly rapidly.
4. Dialysis is definitive treatment in refractory cases.
5. Continuous monitoring is essential.

THYROID STORM

1. INTRODUCTION

Thyroid storm is a rare but life-threatening endocrine emergency resulting from severe thyrotoxicosis with systemic decompensation.

It is associated with:

1. High mortality
2. Cardiovascular collapse
3. Arrhythmias
4. Hyperthermia
5. Multi-organ dysfunction
6. Neurological impairment

Thyroid storm most commonly occurs in patients with untreated or poorly controlled hyperthyroidism precipitated by acute physiological stress.

Early recognition and aggressive treatment are essential.

2. DEFINITION

Thyroid storm is a severe decompensated state of thyrotoxicosis characterized by:

1. Hypermetabolism
2. Adrenergic excess
3. Multi-system dysfunction
4. Altered mental status

3. PRECIPITATING FACTORS

1. Infection
2. Surgery
3. Trauma
4. Myocardial infarction
5. Stroke
6. Diabetic ketoacidosis
7. Pulmonary embolism
8. Abrupt discontinuation of antithyroid drugs
9. Iodinated contrast exposure
10. Parturition

4. CLINICAL FEATURES

Symptoms	Signs
1. Fever	Hyperthermia
2. Palpitations	Tachycardia/ Atrial fibrillation
3. Dyspnea	Heart failure
4. Agitation	Delirium
5. Tremors	Psychosis
6. Diarrhea	Seizures
7. Vomiting	Coma
8. Altered sensorium	Goiter, Ophthalmopathy

5. DIAGNOSTIC ASSESSMENT

Diagnosis is primarily clinical.

Burch-Wartofsky Point Scale (BWPS)

BWPS ≥45 strongly suggests thyroid storm.

Laboratory Findings

1. Suppressed TSH
2. Elevated free T4 and/or T3
3. Hyperglycemia
4. Elevated liver enzymes
5. Leukocytosis
6. Hypercalcemia may occur

6. INITIAL ASSESSMENT

Immediate Priorities

1. ABC stabilization
2. Cardiac monitoring
3. Temperature control
4. Identification of precipitating cause
5. Early ICU involvement

7. ABCDE ASSESSMENT

Airway

Assess airway protection and mental status.

Intubation may be required in:

1. Severe agitation
2. Respiratory failure
3. Coma

Breathing

Assess:

1. Respiratory rate

2. Oxygen saturation
3. Pulmonary edema
4. Respiratory fatigue

Circulation

Assess:

1. Heart rate
2. Blood pressure
3. Arrhythmias
4. Signs of heart failure
5. Shock

Disability

Assess:

1. GCS
2. Agitation
3. Delirium
4. Seizures
5. Coma

Exposure

Assess for:

1. Infection
2. Trauma
3. Surgical wounds
4. Hyperthermia

8. INVESTIGATIONS

Mandatory Investigations

1. Thyroid function tests
2. CBC
3. Renal function tests
4. Liver function tests
5. Electrolytes
6. Blood glucose
7. ECG
8. Chest radiograph

Additional Investigations

1. Troponin
2. Blood cultures
3. Arterial or venous blood gas
4. Echocardiography if heart failure suspected

9. MANAGEMENT PRINCIPLES

Treatment priorities include:

1. Supportive care
2. Beta blockade
3. Inhibition of thyroid hormone synthesis
4. Inhibition of thyroid hormone release
5. Reduction of peripheral T4 to T3 conversion
6. Treatment of precipitating cause

10. SUPPORTIVE MANAGEMENT

Fluid Therapy

1. Correct dehydration cautiously
2. Avoid fluid overload in heart failure

Temperature Control

1. Paracetamol
2. Cooling blankets
3. Ice packs

Avoid aspirin because it may increase free thyroid hormone levels.

Oxygen and Ventilation

1. Supplemental oxygen
2. Mechanical ventilation if respiratory failure develops

11. BETA BLOCKADE

Preferred Therapy

Propranolol

Administer:

1. Propranolol 40–80 mg orally every 4–6 hours

OR

2. Propranolol 0.5–1 mg IV slowly over 10 minutes

May repeat cautiously every 10–15 minutes based on:

1. Heart rate
2. Blood pressure
3. Cardiac function
4. Clinical response

Alternative

Esmolol Infusion

Loading Dose -- Esmolol 250–500 mcg/kg IV over 1 minute

Maintenance Infusion -- Start infusion at 50 mcg/kg/min IV

Titrate gradually every 5–10 minutes according to:

1. Heart rate

2. Blood pressure
3. Perfusion status
4. Cardiac function

Typical infusion range: 50–200 mcg/kg/min IV infusion

Monitoring

Continuous monitoring required:

1. ECG
2. Blood pressure
3. Oxygen saturation
4. Signs of heart failure
5. Bronchospasm

12. ANTITHYROID THERAPY

Preferred Initial Therapy

Propylthiouracil (PTU)

Administer:

1. Loading dose 500–1000 mg orally or via NG tube
2. Followed by 250 mg every 4 hours

PTU additionally inhibits peripheral conversion of T4 to T3.

Alternative

Methimazole

1. Methimazole 20 mg every 4–6 hours

13. IODINE THERAPY

Administer iodine at least 1 hour after antithyroid drug administration.

This prevents iodine from being used for new hormone synthesis.

Options

1. Lugol solution - Lugol's iodine: 8–10 drops orally every 6–8 hours, administered at least 1 hour after antithyroid medication.
2. Potassium iodide- Potassium iodide (SSKI): 5 drops orally every 6 hours, administered at least 1 hour after antithyroid medication.

14. GLUCOCORTICOID THERAPY

Recommended Regimen

Hydrocortisone 100 mg IV every 8 hours

Benefits include:

1. Reduced peripheral T4 to T3 conversion
2. Treatment of relative adrenal insufficiency
3. Hemodynamic support

15. TREATMENT OF PRECIPITATING CAUSES

1. Broad-spectrum antibiotics if infection suspected

2. Management of myocardial infarction
3. DKA treatment if present
4. Pulmonary embolism treatment if present

16. MONITORING
    1. Continuous cardiac monitoring
    2. Temperature monitoring
    3. Neurological assessment
    4. Electrolytes
    5. Thyroid function trends
    6. Fluid balance
17. COMPLICATIONS
    1. Arrhythmias
    2. Heart failure
    3. Cardiogenic shock
    4. Hepatic dysfunction
    5. Respiratory failure
    6. Multi-organ dysfunction
    7. Death
18. ICU ADMISSION CRITERIA
    1. Thyroid storm diagnosis
    2. Arrhythmias
    3. Heart failure
    4. Severe hyperthermia
    5. Respiratory failure
    6. Shock
    7. Altered mental status

MYXEDEMA COMA

1. INTRODUCTION

Myxedema coma is a severe life-threatening manifestation of decompensated hypothyroidism.

It is associated with:

1. Hypothermia
2. Respiratory failure
3. Bradycardia
4. Altered consciousness
5. Multi-organ dysfunction

Mortality remains high despite treatment.

2. PRECIPITATING FACTORS
    1. Infection
    2. Stroke
    3. Myocardial infarction
    4. Cold exposure
    5. Sedative medications
    6. Trauma
    7. Surgery
    8. Failure to take thyroid medication
3. CLINICAL FEATURES
    1. Hypothermia
    2. Bradycardia
    3. Hypotension
    4. Hypoventilation
    5. Altered mental status
    6. Dry skin
    7. Non-pitting edema
    8. Delayed reflex relaxation
    9. Hyponatremia
    10. Hypoglycemia
4. INVESTIGATIONS
    1. Thyroid function tests
    2. CBC
    3. Electrolytes
    4. Blood glucose
    5. ABG
    6. ECG
    7. Chest radiograph
5. MANAGEMENT PRINCIPLES
    1. Early thyroid hormone replacement
    2. Stress-dose steroids
    3. Ventilatory support
    4. Passive rewarming
    5. Treatment of precipitating illness
6. THYROID HORMONE REPLACEMENT

Levothyroxine

Administer:

1. IV levothyroxine loading dose 200–400 mcg
2. Followed by 50–100 mcg IV daily
3. Liothyronine (T3): 5–20 mcg IV loading dose followed by 2.5–10 mcg IV every 8 hours in selected severe myxedema coma patients.

Use lower doses in:

1. Elderly patients
2. Coronary artery disease
3. Arrhythmias

7. GLUCOCORTICOID THERAPY

Administer empiric steroids before thyroid hormone therapy.

Hydrocortisone 100 mg IV every 8 hours

8. SUPPORTIVE MANAGEMENT
    1. Passive rewarming
    2. Oxygen therapy
    3. Mechanical ventilation if required
    4. Correction of hypoglycemia
    5. Correction of hyponatremia
    6. Treatment of infection

Avoid aggressive active warming because it may precipitate cardiovascular collapse.

9. MONITORING
    1. Cardiac monitoring
    2. Temperature monitoring
    3. Electrolytes
    4. Blood glucose
    5. ABG monitoring
    6. Neurological assessment
10. ICU ADMISSION CRITERIA
    1. Myxedema coma diagnosis
    2. Respiratory failure
    3. Shock
    4. Severe hypothermia
    5. Altered consciousness
11. KEY PRACTICE POINTS
    1. Treat empirically if strongly suspected.
    2. Administer steroids before thyroid hormone.
    3. Passive rewarming is preferred.
    4. Respiratory failure is common.

5. ICU care is usually required.

ADRENAL CRISIS

1. INTRODUCTION

Adrenal crisis is a life-threatening endocrine emergency caused by acute glucocorticoid deficiency.

It is associated with:

1. Shock
2. Severe hypotension
3. Hypoglycemia
4. Hyponatremia
5. Hyperkalemia
6. Multi-organ dysfunction

Immediate treatment is essential and should not be delayed while awaiting investigations.

2. PRECIPITATING FACTORS
    1. Infection
    2. Surgery
    3. Trauma
    4. Gastroenteritis
    5. Abrupt steroid withdrawal
    6. Pituitary disease
    7. Adrenal hemorrhage
    8. Missed steroid doses
3. CLINICAL FEATURES
    1. Weakness
    2. Vomiting
    3. Abdominal pain
    4. Hypotension
    5. Shock
    6. Fever
    7. Confusion
    8. Hypoglycemia
    9. Dehydration
4. INVESTIGATIONS
    1. Serum cortisol
    2. Electrolytes

3. Blood glucose
4. CBC
5. Renal function tests
6. Blood cultures if infection suspected

Treatment must not be delayed for cortisol results.

5. MANAGEMENT PRINCIPLES
    1. Immediate steroid replacement
    2. Aggressive fluid resuscitation
    3. Correction of hypoglycemia
    4. Treatment of precipitating illness
    5. Electrolyte correction
6. GLUCOCORTICOID THERAPY

Initial Therapy --- Hydrocortisone 100 mg IV immediately
Maintenance Therapy-- Hydrocortisone 200 mg/24 hours
This may be administered as:

1. Continuous infusion OR
2. 50 mg IV every 6 hours

7. FLUID RESUSCITATION
    1. 0.9% saline
    2. Rapid IV crystalloid resuscitation
    3. Add dextrose if hypoglycemic
8. HYPOGLYCEMIA MANAGEMENT
    1. IV dextrose if required
    2. Frequent glucose monitoring
9. ELECTROLYTE MANAGEMENT

Correct:

1. Hyponatremia
2. Hyperkalemia
3. Dehydration

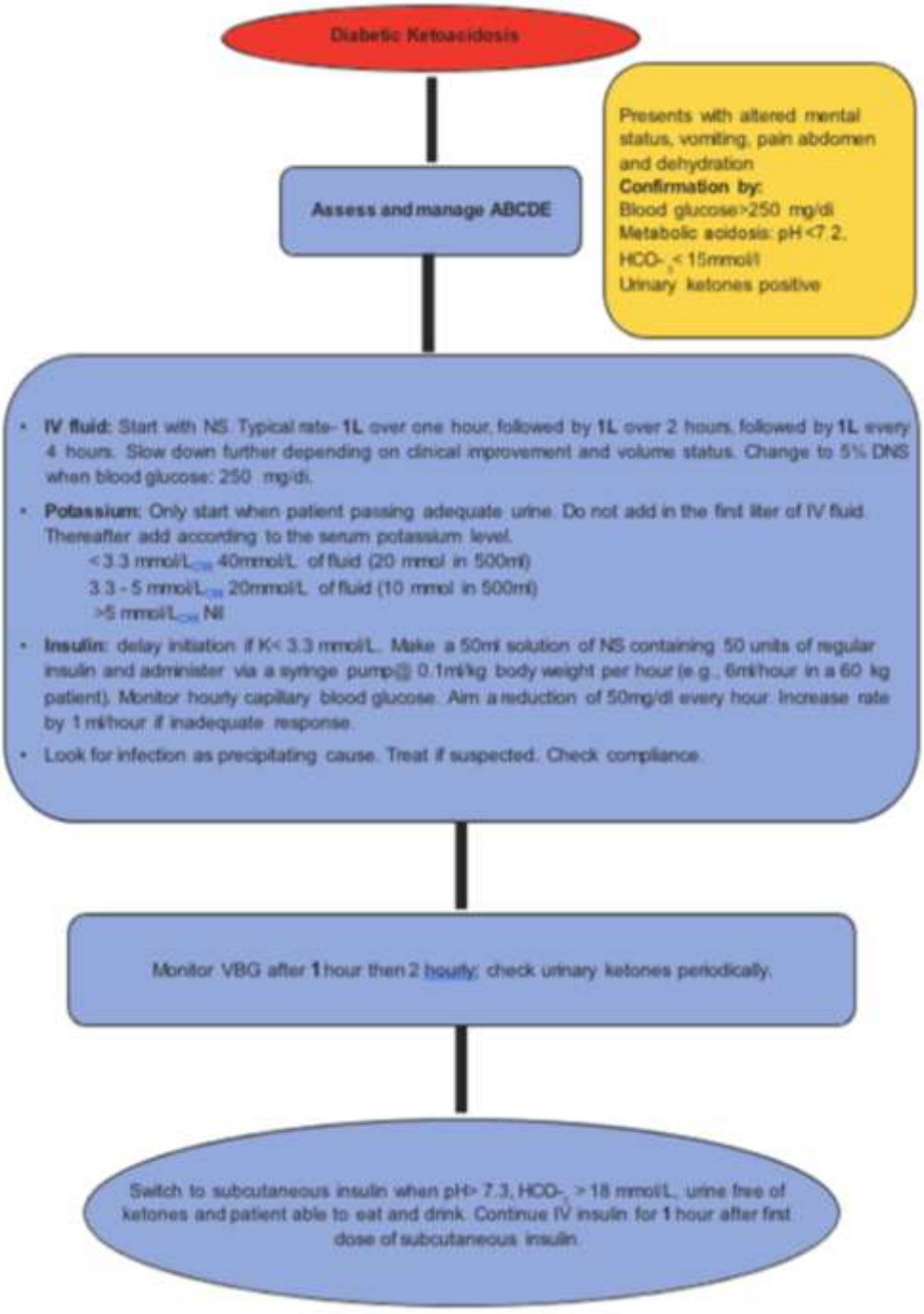
10. TREATMENT OF PRECIPITATING CAUSES
    1. Antibiotics if infection suspected
    2. Management of trauma
    3. Treatment of GI losses
    4. Management of adrenal hemorrhage if present
11. MONITORING
    1. Blood pressure
    2. Urine output
    3. Electrolytes

4. Blood glucose
5. Cardiac monitoring
6. Mental status

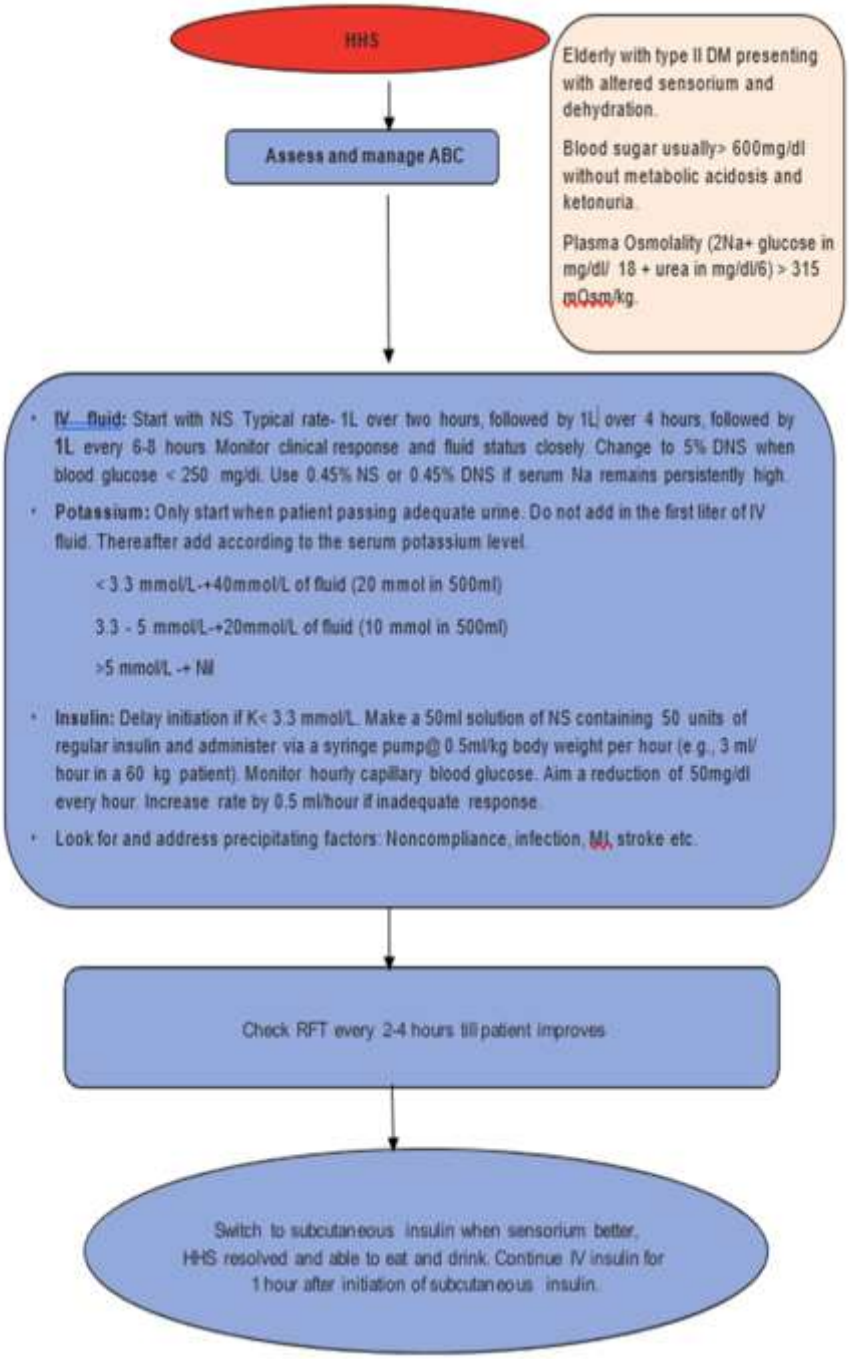
12. COMPLICATIONS
    1. Refractory shock
    2. Arrhythmias
    3. Hypoglycemia
    4. Acute kidney injury
    5. Multi-organ failure
13. ICU ADMISSION CRITERIA
    1. Shock
    2. Persistent hypotension
    3. Severe electrolyte abnormalities
    4. Altered mental status
    5. Multi-organ dysfunction

Algorithm

Algorithm for management of DKA



Algorithm for management of HHS



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SECTION VII
INFECTIOUS DISEASE & SEPSIS

1. Sepsis & Septic Shock

Definition

Sepsis

Sepsis is a life-threatening acute organ dysfunction due to infection.

Septic Shock

Septic shock is a subset of sepsis characterized by:

- Persistent hypotension requiring vasopressors to maintain MAP ≥65 mmHg
  AND
- Serum lactate >2 mmol/L despite adequate fluid resuscitation

Clinical Features

General Features

1. Fever or hypothermia
2. Tachycardia
3. Tachypnea
4. Hypotension
5. Altered sensorium
6. Reduced urine output

Features Suggesting Organ Dysfunction

1. Hypoxia
2. Oliguria
3. Elevated lactate
4. Coagulopathy
5. Confusion/agitation
6. Metabolic acidosis

Initial Assessment

ABCDE Assessment

Airway

Assess airway patency and mental status.

Breathing

Assess:

1. Respiratory rate
2. SpO_2
3. Work of breathing

Provide oxygen therapy as required.

Circulation

Assess:

1. Pulse

2. Blood pressure
3. Peripheral perfusion
4. Urine output

Establish:

1. Two large-bore IV lines
2. Cardiac monitoring

Disability

Assess:

1. GCS
2. Blood glucose
3. Mental status

Exposure

Look for:

1. Rash
2. Cellulitis
3. Wounds
4. Surgical sites
5. Indwelling catheters

Screening and Recognition of Sepsis

Sepsis should be suspected in patients with:

1. Suspected or confirmed infection AND
2. Evidence of organ dysfunction or physiological deterioration

Early Warning Signs

1. Hypotension
2. Tachypnea
3. Hypoxia
4. Altered mentation
5. Reduced urine output
6. Elevated lactate
7. Mottled or poorly perfused skin
8. Metabolic acidosis

Recommended Screening Tools

Preferred:

- NEWS
- NEWS2
- MEWS
- SIRS

qSOFA should not be used as the sole screening tool for identification of sepsis.

Investigations

Mandatory Investigations

1. CBC
2. Renal function test
3. Electrolytes
4. ABG/VBG
5. Serum lactate
6. Blood glucose
7. ECG

Microbiological Investigations

Obtain before antibiotics where feasible:

1. Blood cultures
2. Urine culture
3. Sputum culture
4. Wound culture

Blood cultures should ideally be obtained before antimicrobial therapy but should not delay treatment.

Imaging

As indicated:

1. Chest X-ray
2. Ultrasound
3. CT imaging
4. Echocardiography where indicated

Management of Sepsis

Initial Fluid Resuscitation

Preferred Fluids

Balanced crystalloids preferred:

- Ringer Lactate
- Plasma-Lyte

Initial Fluid Bolus

30 mL/kg IV crystalloid within first 3 hours

Recommended for:

- Sepsis-induced hypoperfusion
- Septic shock

Fluid resuscitation should be individualized with frequent reassessment to avoid under- or over-resuscitation.

Fluid Responsiveness Assessment

Fluid therapy should be guided by:

1. Blood pressure
2. Urine output
3. Peripheral perfusion
4. Lactate trend
5. Capillary refill time
6. Dynamic fluid responsiveness

Dynamic Markers

1. Passive leg raise
2. Stroke volume variation
3. Pulse pressure variation
4. IVC assessment
5. Bedside echocardiography

Antimicrobial Therapy

Timing

Septic Shock

Administer broad-spectrum antibiotics ideally within 1 hour of recognition.

Sepsis Without Shock

Administer antibiotics as early as possible after rapid evaluation.

Principles of Antibiotic Selection

Consider:

1. Suspected source of infection
2. Local antibiogram
3. Healthcare-associated infection risk
4. Immunocompromised status
5. Prior antibiotic exposure

Empirical antibiotics should later be de-escalated according to:

- Culture results
- Clinical response
- Biomarker trends

Management of Septic Shock

Vasopressor Therapy

Indications

Persistent hypotension despite adequate fluid resuscitation.

First-Line Vasopressor

Noradrenaline (Norepinephrine)

Initial MAP Target

MAP≥65 mmHg

For adults ≥65 years, a lower permissive target may be reasonable:

MAP=60–65 mmHg may be reasonable if adequately perfused.

Timing of Vasopressors

Initial IV crystalloid bolus should generally precede vasopressor initiation. However, in unstable septic shock with:

- Profound hypotension
- Mottling
- Cyanosis
- Altered mentation
- Severe hypoperfusion

concurrent early vasopressor initiation may be warranted.

Peripheral Vasopressor Administration

Peripheral vasopressor initiation may be considered temporarily through a large proximal vein with close monitoring if central access unavailable.

Additional Vasopressors

Vasopressin

Dose

0.03 units/min

May be added in refractory shock to reduce norepinephrine requirements.

Adrenaline (Epinephrine)

May be considered if hypotension persists despite norepinephrine.

Inotropic Support

Dobutamine

May be considered in:

1. Myocardial dysfunction
2. Persistent hypoperfusion despite fluids and vasopressors

Corticosteroids

Hydrocortisone

May be considered in refractory septic shock requiring ongoing vasopressor support.

Suggested Regimen

Hydrocortisone 200 mg/day IV

Source Control

Examples include:

1. Drainage of abscess
2. Removal of infected catheter
3. Debridement of infected tissue
4. Surgical intervention where indicated

Source control should be achieved as early as feasible.

Oxygen and Respiratory Support

Provide:

1. Supplemental oxygen
2. NIV where appropriate
3. Mechanical ventilation if respiratory failure develops

Use lung-protective ventilation strategies in ventilated patients.

Monitoring

Hourly Monitoring

1. Vital signs
2. Urine output
3. Mental status
4. Vasopressor requirement

Repeat Monitoring

1. Serum lactate
2. Electrolytes
3. Renal function
4. ABG/VBG

Lactate Monitoring

Serial lactate measurements may help assess response to resuscitation.

Lactate Clearance Target

Lactate clearance > 10%

Lactate should not be the sole endpoint of resuscitation.

Complications

1. ARDS
2. Acute kidney injury
3. DIC
4. Multi-organ dysfunction
5. Myocardial dysfunction

ICU Admission Criteria

1. Septic shock
2. Vasopressor requirement
3. Respiratory failure
4. Elevated lactate with instability
5. Altered sensorium
6. Multi-organ dysfunction

Disposition

Ward Admission

- Stable sepsis without major organ dysfunction

ICU Admission

1. Septic shock
2. Respiratory support requirement
3. Hemodynamic instability
4. Severe organ dysfunction

2. Dengue Fever

Definition

Dengue Fever

An acute mosquito-borne viral illness caused by dengue virus (DENV-1 to DENV-4) transmitted by *Aedes* mosquitoes.

Severe Dengue

Severe dengue is characterized by:

1. Severe plasma leakage leading to shock or respiratory distress
2. Severe bleeding
3. Severe organ dysfunction

Clinical Phases of Dengue

Febrile Phase

Usually lasts 2 to 7 days.

Features:

1. High-grade fever
2. Headache
3. Retro-orbital pain
4. Myalgia
5. Arthralgia
6. Rash
7. Vomiting
8. Facial flushing

Critical Phase (Leakage Phase)

Usually occurs around day 3 to 7 of illness and lasts 24 to 48 hours.

Characterized by:

1. Plasma leakage
2. Hemoconcentration
3. Shock
4. Bleeding manifestations
5. Organ dysfunction

Recovery Phase

Features:

1. Hemodynamic stabilization
2. Reabsorption of extravasated fluid
3. Improving appetite
4. Diuresis
5. Recovery of platelet count

Risk:

- Pulmonary edema from excessive IV fluids

Warning Signs

Warning Symptoms and Signs

1. Persistent vomiting
2. Abdominal pain/tenderness
3. Lethargy or restlessness
4. Mucosal bleeding
5. Syncope/giddiness
6. Clinical fluid accumulation
7. Hepatomegaly >2 cm
8. Rising hematocrit with falling platelet count

Initial Assessment

ABCDE Assessment

Airway

Assess airway patency and sensorium.

Breathing

Assess:

1. Respiratory rate
2. SpO_2
3. Pleural effusion
4. Respiratory distress

Circulation

Assess:

1. Pulse pressure
2. Capillary refill
3. Peripheral perfusion
4. Urine output
5. Postural hypotension

Disability

Assess:

1. GCS

2. Encephalopathy
3. Seizures

Exposure

Look for:

1. Petechiae
2. Rash
3. Bleeding manifestations
4. Tourniquet test positivity

Classification

Group A – Dengue Without Warning Signs

1. Stable hemodynamics
2. Adequate oral intake
3. No warning signs
4. No severe bleeding

Group B – Dengue With Warning Signs / High-Risk Factors

1. Warning signs present
2. Comorbid illness
3. Elderly
4. Pregnancy
5. Extremes of age

Group C – Severe Dengue

1. Shock
2. Severe bleeding
3. Respiratory distress
4. Severe metabolic acidosis
5. Organ dysfunction

Investigations

Mandatory Investigations

1. CBC with hematocrit
2. Platelet count
3. LFT
4. RFT
5. Electrolytes
6. Blood glucose
7. ABG/VBG if severe disease

Specific Tests

1. NS1 antigen
2. Dengue IgM ELISA

3. RT-PCR where available

Imaging

As indicated:

1. Chest X-ray
2. Ultrasound for ascites/pleural effusion
3. Echocardiography if myocarditis suspected

Management

Mild Dengue (Group A)

Symptomatic Management

Antipyretic

Paracetamol 10-15mg/kg/dose

Avoid:

1. NSAIDs
2. Aspirin
3. Ibuprofen

Encourage:

1. Oral fluids
2. ORS
3. Adequate rest

Moderate Dengue (Group B)

IV Fluid Therapy

Preferred crystalloids:

1. Ringer Lactate
2. Normal Saline

Initial Fluid Rate

5–7 mL/kg/hr

Reassess frequently using:

1. Pulse pressure
2. Hematocrit
3. Urine output
4. Peripheral perfusion
5. Capillary refill time

Severe Dengue / Dengue Shock Syndrome (Group C)

Initial Fluid Bolus

10–20 mL/kg crystalloid 15–30 min

Repeat according to:

1. Hemodynamic response
2. Pulse pressure

3. Urine output
4. Hematocrit

Features of Compensated Shock

1. Cold extremities
2. Delayed capillary refill
3. Tachycardia
4. Narrow pulse pressure

Narrow Pulse Pressure

Pulse Pressure < 20 mmHg

Features of Decompensated Shock

1. Hypotension
2. Feeble pulses
3. Altered sensorium
4. Mottled skin
5. Oliguria/anuria
6. Severe metabolic acidosis

Blood Products

Platelet Transfusion

Not indicated solely for thrombocytopenia.

Indications:

1. Major bleeding
2. Intracranial hemorrhage
3. Severe GI bleed
4. Invasive procedures with severe thrombocytopenia

Packed RBC

Indicated in:

1. Massive bleed
2. Hemodynamic instability
3. Falling hematocrit with shock

Organ Dysfunction in Severe Dengue

Neurological

1. Encephalopathy
2. Encephalitis
3. Intracranial hemorrhage
4. GBS

Hepatic

1. Hepatitis
2. Fulminant liver failure

Cardiac

1. Myocarditis
2. Arrhythmias
3. Pericarditis

Respiratory

1. ARDS
2. Pulmonary hemorrhage

Renal

1. Acute kidney injury

Monitoring

Hourly Monitoring

1. Pulse pressure
2. Blood pressure
3. Urine output
4. Capillary refill time
5. Mental status

Serial Monitoring

1. Hematocrit
2. Platelet count
3. Lactate if shock
4. ABG/VBG if severe disease

ICU Admission Criteria

1. Dengue shock syndrome
2. Severe bleeding
3. Respiratory distress
4. Encephalopathy
5. Severe hepatitis
6. Myocarditis
7. Multi-organ dysfunction

Disposition

Home Management

Suitable if:

1. No warning signs
2. Hemodynamically stable
3. Adequate oral intake
4. Reliable follow-up available

Ward Admission

1. Warning signs

2. Rising hematocrit
3. Falling platelet count
4. Dehydration
5. High-risk comorbidities

ICU Admission

1. Shock
2. Organ dysfunction
3. Severe bleeding
4. Respiratory failure

3. Leptospirosis

Definition

Leptospirosis

A zoonotic spirochetal infection caused by *Leptospira* species, transmitted through exposure to water or soil contaminated with urine of infected animals.

Severe Leptospirosis (Weil's Disease)

Characterized by:

1. Jaundice
2. Acute kidney injury
3. Hemorrhage
4. Shock
5. Myocarditis
6. Pulmonary hemorrhage/ARDS
7. Multi-organ dysfunction

Risk Factors

Common exposures:

1. Flood water exposure
2. Paddy field work
3. Sewage exposure
4. Rodent exposure
5. Animal handling
6. Walking barefoot in contaminated water

Clinical Features

Septicemic Phase

Common Features

1. Acute febrile illness
2. Severe myalgia (especially calf/back)

3. Headache
4. Conjunctival suffusion
5. Vomiting
6. Rash
7. Photophobia

Immune Phase

Features

1. Jaundice
2. AKI
3. Meningitis
4. Pulmonary hemorrhage
5. Myocarditis
6. Shock
7. Thrombocytopenia

Red Flag Signs

1. Hypotension
2. Oliguria
3. Hemoptysis
4. Dyspnea
5. Altered sensorium
6. Severe jaundice
7. Metabolic acidosis
8. Arrhythmias

Initial Assessment

ABCDE Assessment

Airway

Assess airway patency and mental status.

Breathing

Assess:

1. Respiratory distress
2. SpO_2
3. Pulmonary hemorrhage
4. ARDS

Circulation

Assess:

1. Blood pressure
2. Peripheral perfusion
3. Shock

4. Urine output

Disability

Assess:

1. GCS
2. Meningeal signs
3. Encephalopathy

Exposure

Look for:

1. Conjunctival suffusion
2. Icterus
3. Rash
4. Muscle tenderness

Investigations

Mandatory Investigations

1. CBC
2. Platelet count
3. RFT
4. Electrolytes
5. LFT
6. ABG/VBG
7. Urinalysis
8. ECG

Specific Investigations

1. Leptospira IgM ELISA
2. MAT where available
3. PCR where available

Imaging

As indicated:

1. Chest X-ray
2. Ultrasound abdomen
3. Echocardiography if myocarditis suspected

Severity Assessment

Mild Disease

1. Stable vitals
2. No organ dysfunction
3. Tolerating oral intake

Severe Disease

1. AKI

2. Pulmonary hemorrhage
3. ARDS
4. Shock
5. Severe jaundice
6. Myocarditis
7. CNS involvement
8. Severe thrombocytopenia

Antibiotic Therapy

Mild Leptospirosis

Doxycycline

Doxycycline100 mg PO BD for 7 days

Alternative:

- Amoxicillin if doxycycline contraindicated

Severe Leptospirosis

Preferred Therapy

Ceftriaxone

Ceftriaxone 1–2 g IV OD

OR

Penicillin G

Penicillin G 1.5 million units IV 6 hourly

Duration usually:

- 7 days

Fluid Management

Principles

1. Careful fluid resuscitation
2. Avoid overload in pulmonary involvement
3. Monitor urine output closely

Use balanced crystalloids unless contraindicated.

Pulmonary Hemorrhage Syndrome

Features

1. Hemoptysis
2. Rapid desaturation
3. Bilateral infiltrates
4. Severe hypoxemia

Management

1. ICU care
2. Oxygen therapy
3. Mechanical ventilation if required

4. Lung-protective ventilation
5. Blood products where indicated

Acute Kidney Injury

Management

1. Fluid optimization
2. Avoid nephrotoxins
3. Electrolyte correction
4. Dialysis if indicated

Indications for Dialysis

1. Refractory hyperkalemia
2. Severe metabolic acidosis
3. Pulmonary edema
4. Uremic complications
5. Severe azotemia with oliguria

Monitoring

Hourly Monitoring

1. Vital signs
2. Urine output
3. SpO_2
4. Mental status

Repeat Monitoring

1. Creatinine
2. Electrolytes
3. Platelets
4. Bilirubin
5. ABG/VBG

Complications

1. AKI
2. Pulmonary hemorrhage
3. ARDS
4. Myocarditis
5. Arrhythmias
6. DIC
7. Hepatic dysfunction
8. Meningitis

ICU Admission Criteria

1. Shock
2. Pulmonary hemorrhage

3. Respiratory failure
4. Severe AKI
5. Dialysis requirement
6. Altered sensorium
7. Myocarditis
8. Multi-organ dysfunction

Disposition

Home Management

Suitable if:

1. Mild disease
2. Stable vitals
3. No organ dysfunction
4. Reliable follow-up available

Ward Admission

1. Jaundice
2. AKI
3. Dehydration
4. Thrombocytopenia
5. Moderate disease

ICU Admission

1. Pulmonary hemorrhage
2. Shock
3. Respiratory failure
4. Severe AKI
5. Multi-organ dysfunction

4. Malaria

Definition

Malaria

A protozoal infection caused by *Plasmodium* species transmitted by female *Anopheles* mosquitoes.

Common species:

1. Plasmodium falciparum
2. Plasmodium vivax
3. Plasmodium malariae
4. Plasmodium ovale
5. Plasmodium knowlesi

Severe Malaria

Usually associated with *Plasmodium falciparum* infection.

Characterized by:

1. Cerebral malaria
2. Severe anemia
3. Shock
4. Metabolic acidosis
5. Hypoglycemia
6. Acute kidney injury
7. Pulmonary edema/ARDS
8. Hemoglobinuria
9. Bleeding/DIC
10. Multi-organ dysfunction

Clinical Features

Uncomplicated Malaria

1. Fever with chills and rigors
2. Sweating
3. Headache
4. Myalgia
5. Vomiting
6. Malaise
7. Splenomegaly

Severe Malaria

1. Altered sensorium
2. Repeated seizures
3. Respiratory distress
4. Jaundice
5. Oliguria
6. Severe anemia
7. Shock
8. Hypoglycemia
9. Metabolic acidosis

Initial Assessment

ABCDE Assessment

Airway

Assess airway patency and mental status.

Breathing

Assess:

1. Respiratory distress
2. ARDS
3. SpO_2

Circulation

Assess:

1. Blood pressure
2. Shock
3. Peripheral perfusion
4. Urine output

Disability

Assess:

1. GCS
2. Seizures
3. Cerebral malaria

Exposure

Look for:

1. Pallor
2. Icterus
3. Rash
4. Hepatosplenomegaly

Investigations

Mandatory Investigations

1. CBC
2. Peripheral smear for MP
3. Rapid diagnostic test (RDT)
4. Blood glucose
5. RFT
6. LFT
7. Electrolytes
8. ABG/VBG if severe disease

Additional Investigations

As indicated:

1. Chest X-ray
2. ECG
3. Coagulation profile
4. Ultrasound abdomen

Diagnosis

All fever cases should preferably be investigated for malaria by:

1. Microscopy
   OR
2. Rapid Diagnostic Kit (RDK/RDT)

Classification

Uncomplicated Malaria

1. Stable vitals
2. No organ dysfunction
3. Able to tolerate oral therapy

Severe/Complicated Malaria

1. Cerebral malaria
2. Severe anemia
3. Renal failure
4. Shock
5. ARDS
6. Acidosis
7. Multi-organ dysfunction

Treatment

Plasmodium vivax Malaria

Chloroquine Regimen

Chloroquine 25 mg/kg over 3 days

Primaquine Regimen

Primaquine 0.25 mg/kg/day for 14 days

Primaquine should be given under medical supervision only.

Plasmodium falciparum Malaria

ACT Therapy

Artesunate + Sulphadoxine-Pyrimethamine (AS+SP)

Artesunate 4 mg/kg/day for 3 days

PLUS

Sulphadoxine-Pyrimethamine 25/1.25 mg/kg single dose

Primaquine

Single-dose primaquine may reduce gametocyte carriage:

Primaquine 0.75 mg/kg single dose

Contraindicated in:

1. Pregnancy
2. Infants

Severe and Complicated Malaria

First-Line Therapy

IV Artesunate

Artesunate 2.4 mg/kg IV at 0,12,24 hours then daily

Preferred treatment for severe malaria.

Alternative Therapy

IV Quinine

Quinine 10 mg/kg IV every 8 hours

Maximum duration:

- 7 days total therapy including oral + IV therapy

Supportive Management

Fluid Therapy

1. Careful fluid balance
2. Avoid fluid overload
3. Monitor urine output closely

Hypoglycemia

Management

25–50 mL of 50% dextrose IV

Repeat glucose monitoring frequently.

Seizure Management

Lorazepam

Lorazepam 0.1 mg/kg IV

Severe Anemia

Blood Transfusion

Indicated if:

1. Severe anemia
2. Hemodynamic instability
3. Symptomatic hypoxia

Acute Kidney Injury

Management

1. Fluid optimization
2. Electrolyte correction
3. Avoid nephrotoxins
4. Dialysis if indicated

Monitoring

Hourly Monitoring

1. Vital signs
2. GCS
3. Urine output
4. Blood glucose
5. SpO_2

Repeat Monitoring

1. Peripheral smear
2. CBC
3. Creatinine
4. Electrolytes
5. ABG/VBG if severe disease

Complications

1. Cerebral malaria
2. ARDS
3. Acute kidney injury
4. Severe anemia
5. Hypoglycemia
6. DIC
7. Shock
8. Multi-organ dysfunction

ICU Admission Criteria

1. Cerebral malaria
2. Shock
3. Respiratory failure
4. Severe metabolic acidosis
5. Acute kidney injury
6. Recurrent seizures
7. Severe anemia
8. Multi-organ dysfunction

Disposition

Home Management

Suitable if:

1. Uncomplicated malaria
2. Oral intake adequate
3. Stable vitals
4. Reliable follow-up available

Ward Admission

1. Vomiting
2. Dehydration
3. Moderate anemia
4. Inability to tolerate oral drugs

ICU Admission

1. Cerebral malaria

2. Shock
3. ARDS
4. Severe acute kidney injury
5. Severe metabolic acidosis
6. Multi-organ dysfunction

5. Scrub Typhus

Definition

Scrub Typhus

An acute febrile zoonotic illness caused by *Orientia tsutsugamushi*, transmitted through the bite of larval trombiculid mites (chiggers).

Risk Factors

1. Farming
2. Forest exposure
3. Grassland exposure
4. Monsoon/post-monsoon season
5. Outdoor occupational exposure
6. Rural residence

Clinical Features

Common Features

1. Acute febrile illness
2. Severe headache
3. Myalgia
4. Dry cough
5. Nausea/vomiting
6. Rash
7. Lymphadenopathy

Characteristic Feature

Eschar

A painless necrotic black crust at the site of mite bite.

Common sites:

1. Axilla
2. Groin
3. Inframammary region
4. Genital region
5. Neck folds

Severe Scrub Typhus

1. ARDS
2. Shock
3. Meningoencephalitis
4. Acute kidney injury
5. Hepatitis
6. Myocarditis
7. Multi-organ dysfunction

Initial Assessment

ABCDE Assessment

Airway

Assess airway patency and sensorium.

Breathing

Assess:

1. Respiratory distress
2. ARDS
3. SpO_2

Circulation

Assess:

1. Blood pressure
2. Peripheral perfusion
3. Shock
4. Urine output

Disability

Assess:

1. GCS
2. Encephalopathy
3. Seizures

Exposure

Look carefully for:

1. Eschar
2. Rash
3. Lymphadenopathy
4. Hepatomegaly/splenomegaly

Investigations

Mandatory Investigations

1. CBC
2. Platelet count
3. RFT

4. LFT
5. Electrolytes
6. Blood glucose
7. ABG/VBG if severe disease

Specific Investigations

1. Scrub typhus IgM ELISA
2. Rapid antibody tests
3. PCR where available

Imaging

As indicated:

1. Chest X-ray
2. Ultrasound abdomen
3. CT brain if neurological involvement
4. Echocardiography if myocarditis suspected

Common Laboratory Findings

1. Thrombocytopenia
2. Elevated transaminases
3. Hyponatremia
4. Leukocytosis or leukopenia
5. Elevated creatinine
6. Elevated CRP

Treatment

First-Line Therapy

Doxycycline

Doxycycline 100 mg PO IV BD

Usual duration:

- 5 to 7 days
- Continue at least 48 hours after defervescence

Alternative Therapy

Azithromycin

Azithromycin 500 mg OD

Preferred in:

1. Pregnancy
2. Doxycycline intolerance
3. Children where indicated

Severe Disease

Combination Therapy

May use:

1. IV doxycycline
2. IV azithromycin

in critically ill patients.

Shock Management

Initial Fluid Therapy

Use isotonic crystalloids with careful reassessment.

Initial Bolus

10–20 mL/kg crystalloid

Vasopressors

Indicated if hypotension persists despite fluids.

First-Line

- Noradrenaline (Norepinephrine)

Respiratory Failure

Management

1. Oxygen therapy
2. NIV where appropriate
3. Mechanical ventilation if required
4. Lung-protective ventilation strategy

Acute Kidney Injury

Management

1. Fluid optimization
2. Electrolyte correction
3. Avoid nephrotoxins
4. Dialysis if indicated

CNS Involvement

Features

1. Altered sensorium
2. Seizures
3. Meningoencephalitis

Management

1. ICU monitoring
2. Antiedema measures if indicated
3. Seizure control
4. Supportive care

Monitoring

Hourly Monitoring

1. Vital signs
2. Urine output

3. SpO_2
4. Mental status

Repeat Monitoring

1. CBC
2. Platelet count
3. Creatinine
4. Electrolytes
5. LFT
6. ABG/VBG if severe disease

Complications

1. ARDS
2. Shock
3. Acute kidney injury
4. Hepatitis
5. Meningoencephalitis
6. Myocarditis
7. DIC
8. Multi-organ dysfunction

ICU Admission Criteria

1. Shock
2. Respiratory failure
3. ARDS
4. CNS involvement
5. Severe acute kidney injury
6. Multi-organ dysfunction
7. Severe metabolic acidosis

Disposition

Home Management

Suitable if:

1. Mild disease
2. Stable vitals
3. No organ dysfunction
4. Tolerating oral medications

Ward Admission

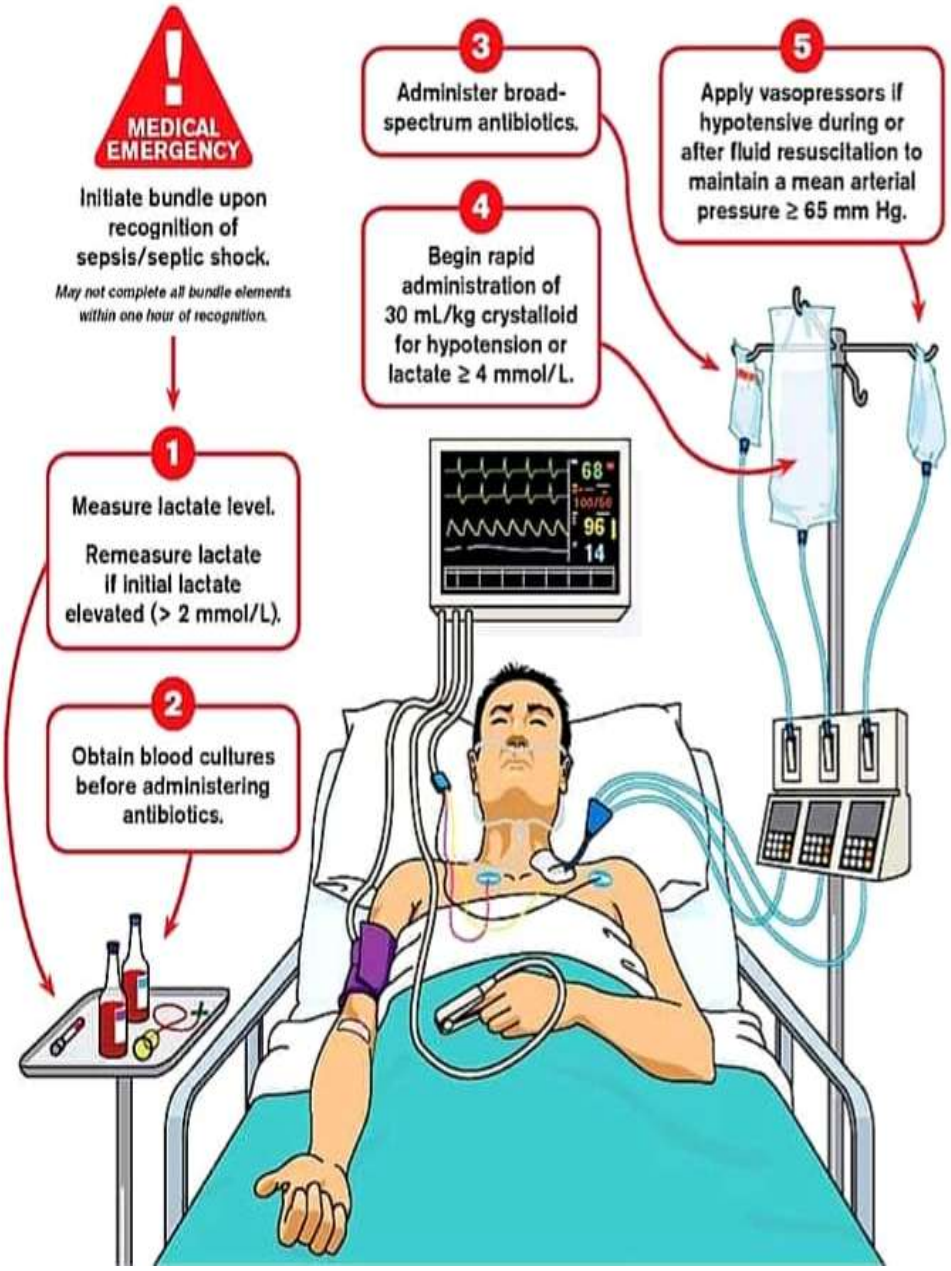
1. Persistent vomiting
2. Dehydration
3. Thrombocytopenia
4. Moderate organ dysfunction

ICU Admission

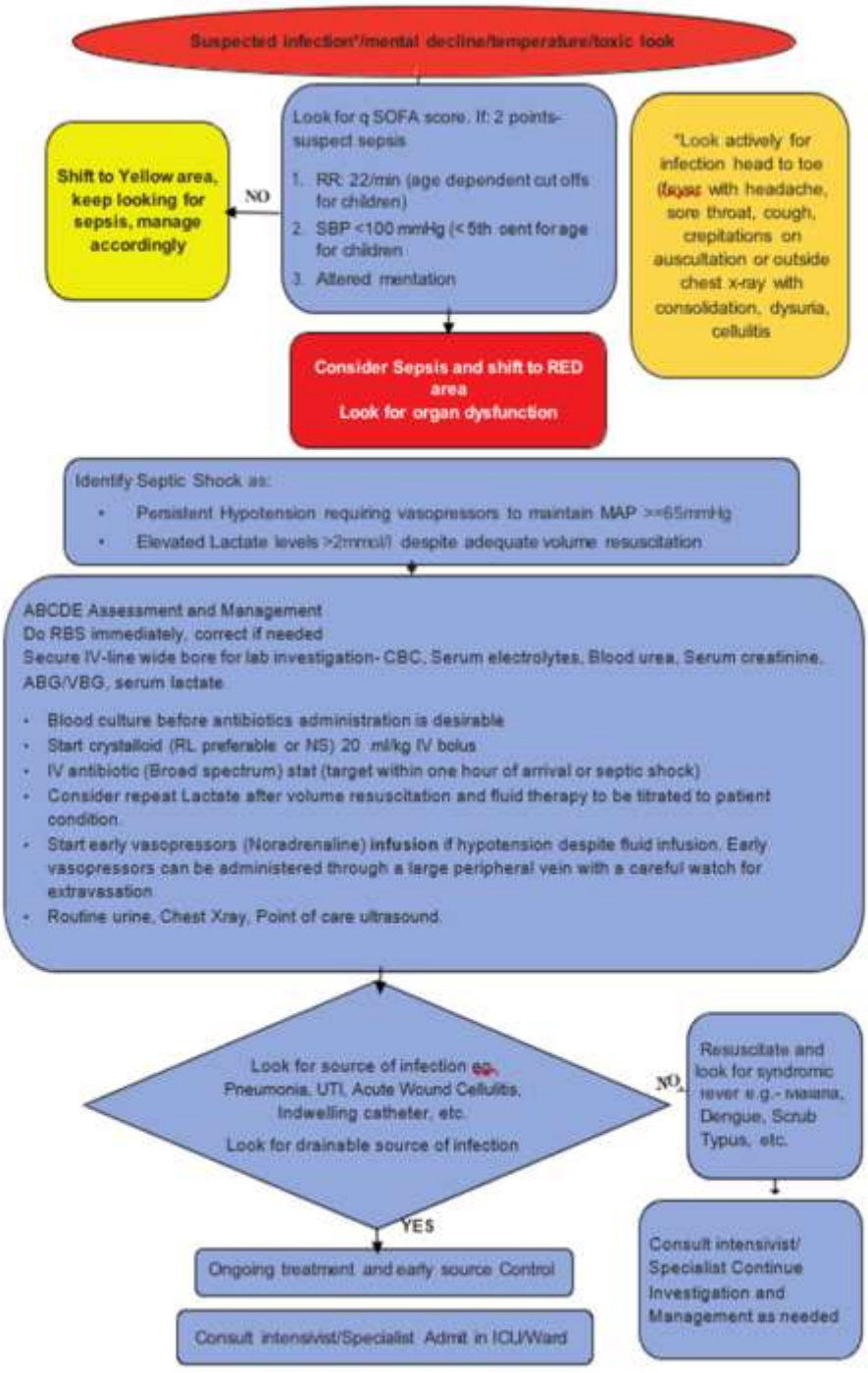
1. Shock
2. ARDS
3. CNS involvement
4. Severe acute kidney injury
5. Multi-organ dysfunction

Algorithm

Sepsis one hour bundle



Algorithm for the Management of Sepsis



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8. Directorate of Health Services Kerala Leptospirosis Protocols
9. National Centre for Disease Control (NCDC) Guidelines
10. WHO Human Leptospirosis Guidelines
11. National Vector Borne Disease Control Programme Standard Treatment Guidelines for Malaria.

SECTION VIII
TRAUMA EMERGENCIES

CONTENTS

1. ATLS Approach
2. Polytrauma
3. Head Injury
4. Chest Trauma
5. Abdominal Trauma
6. Spine Trauma
7. Orthopedic Emergencies
8. Burns
9. Trauma Imaging
10. Massive Transfusion Protocol

Algorithms

- Primary Survey
- Trauma Team Activation
- Massive Hemorrhage
- Burn Resuscitation

1. ATLS APPROACH

Scope

This protocol establishes a standardized ATLS-based approach for initial evaluation, resuscitation, stabilization, and transfer of trauma patients presenting to Emergency Departments across Kerala Government institutions.

Principles of Trauma Care

The ATLS 11th Edition emphasizes:

- Treat greatest threat to life first
- Simultaneous assessment and resuscitation
- Prioritized management
- Prevention of secondary injury
- Team-based trauma care
- Standardized communication

ATLS 11th edition introduces the concept of: xABCDE approach where:

- x = Exsanguinating hemorrhage control
- A = Airway with cervical spine protection
- B = Breathing and ventilation
- C = Circulation and hemorrhage control
- D = Disability (neurological assessment)

- E = Exposure and environmental control

Trauma Team Activation

Activation Criteria

Physiological

- SBP <90 mmHg
- RR <10 or >29/min
- GCS ≤13
- Need for airway intervention

Anatomical

- Penetrating torso injury
- Flail chest
- Pelvic fracture
- Amputation proximal to wrist/ankle
- Paralysis
- Multiple long bone fractures

Mechanism

- High-speed RTC
- Fall >20 feet
- Ejection from vehicle
- Pedestrian struck
- Blast injury

PRIMARY SURVEY – xABCDE

x – Exsanguinating Hemorrhage

Assess For

- Massive external bleeding
- Junctional hemorrhage
- Amputation
- Pelvic hemorrhage

Management

- Direct pressure
- Tourniquet application
- Hemostatic dressing
- Pelvic binder
- Activate MTP

Hemorrhage control takes priority over airway in selected exsanguinating trauma.

A – Airway with Cervical Spine Protection

Assessment

Assess:

- Ability to speak
- Gurgling
- Stridor
- Facial trauma
- Blood/vomitus
- Airway burns

Assume cervical spine injury in all major trauma.

Airway Adjuncts

- Jaw thrust
- Oropharyngeal airway
- Nasopharyngeal airway
- Suction

Indications for Intubation

- GCS ≤8
- Airway obstruction
- Hypoxia
- Severe facial trauma
- Respiratory fatigue
- Anticipated deterioration

RSI Considerations

Avoid hypotension during induction.

Preferred agents may include:

- Ketamine
- Etomidate

Surgical Airway

Indications:

- Failed airway
- Severe facial trauma
- Cannot intubate/cannot oxygenate

B – Breathing and Ventilation

Assessment

- RR
- SpO_2
- Chest rise
- Tracheal deviation
- Percussion
- Air entry

Immediate Life-Threatening Chest Injuries

Identify and treat:

1. Tension pneumothorax
2. Open pneumothorax
3. Massive hemothorax
4. Flail chest
5. Cardiac tamponade

Management

Tension Pneumothorax

Immediate:

- Finger thoracostomy or needle decompression

Followed by:

- Chest tube insertion

Open Pneumothorax

- Three-sided dressing
- Chest tube away from wound

Massive Hemothorax

- Large chest tube
- Blood transfusion
- Thoracic surgery consultation

C – Circulation and Hemorrhage Control

Assessment

- Pulse
- BP
- Skin perfusion
- External bleeding
- Pelvic stability

Initial Management

- Two large bore IV cannulas
- Blood sampling
- Warm fluids
- Early blood products
- TXA within 3 hours

Shock in Trauma

Assume hemorrhage until proven otherwise.

Common sites:

- Chest
- Abdomen

- Pelvis
- Long bones
- External bleeding

Damage Control Resuscitation

Principles:

- Permissive hypotension (except TBI)
- Hemostatic resuscitation
- Minimize crystalloids
- Correct coagulopathy
- Prevent hypothermia

Tranexamic Acid (TXA)

Indications

Major trauma with suspected bleeding.

Dose

- 1 g IV over 10 min
  THEN
- 1 g IV over 8 hours

Best benefit within 3 hours of injury.

D – Disability

Assessment

- GCS
- Pupils
- Blood glucose
- Lateralizing signs

GCS Classification

- Mild: 13–15
- Moderate: 9–12
- Severe: ≤8

Prevent Secondary Brain Injury

Avoid:

- Hypoxia
- Hypotension
- Hyperthermia

Target:

- SBP >100–110 mmHg

E – Exposure and Environmental Control

Exposure

Completely expose patient.

Look for:

- Hidden injuries
- Penetrating wounds
- Deformities
- Burns

Prevent Hypothermia

- Warm blankets
- Fluid warmers
- Warm environment
- Remove wet clothes

Hypothermia contributes to:

- Coagulopathy
- Acidosis
- Mortality

Adjuncts to Primary Survey

- FAST/eFAST
- Portable chest X-ray
- Pelvic X-ray
- ABG/VBG
- Lactate
- ECG

Secondary Survey

Performed after stabilization.

Includes:

- Full head-to-toe examination
- AMPLE history

AMPLE History

- Allergies
- Medications
- Past illness/pregnancy
- Last meal
- Events/environment

Reassessment

Continuous reassessment is mandatory.

Any deterioration requires repeat primary survey.

2. POLYTRAUMA

Definition

Injuries involving multiple body systems associated with physiological instability.

High-Risk Features

- Hypotension
- Altered sensorium
- Pelvic instability
- Penetrating torso injury
- Multiple fractures
- Severe chest trauma

Management Principles

- xABCDE approach
- Simultaneous multidisciplinary care
- Early hemorrhage control
- Damage control surgery
- ICU planning

Investigations

- CBC
- Lactate
- ABG
- Coagulation profile
- FAST/eFAST
- CT trauma protocol if stable

Pelvic Trauma

Suspect major hemorrhage.

Management:

- Pelvic binder
- MTP activation
- IR/surgical consultation

ICU Admission

- Persistent shock
- Severe TBI
- Ventilation
- Massive transfusion
- Multi-organ injury

3. HEAD INJURY

Classification

Severity	GCS
Mild	13–15
Moderate	9–12
Severe	≤8

Initial Priorities

- Prevent hypoxia
- Prevent hypotension
- Early airway control
- Cervical spine protection

Indications for CT Brain

- GCS <15 after resuscitation
- Repeated vomiting
- Focal deficit
- Skull fracture signs
- Anticoagulant use
- Seizure
- Dangerous mechanism
- Age >65 years

Raised ICP Management

Measures

- Head elevation 30°
- Analgesia/sedation
- Normoxia
- Normocapnia
- Avoid fever

Hyperosmolar Therapy

Hypertonic Saline

Preferred in many settings.

OR

Mannitol

Avoid in hypotension.

Signs of Herniation

- Unilateral dilated pupil
- Bradycardia
- Hypertension

- Irregular respiration

Requires immediate neurosurgical consultation.

Seizure Prophylaxis

Consider in severe TBI.

Neurosurgical Referral

- Intracranial bleed
- Midline shift
- Deteriorating GCS
- Penetrating injury

4. CHEST TRAUMA

Life-Threatening Chest Injuries

1. Tension pneumothorax
2. Open pneumothorax
3. Massive hemothorax
4. Cardiac tamponade
5. Flail chest

Investigations

- Chest X-ray
- eFAST
- ABG
- CT chest if stable

Rib Fractures

High risk in elderly patients.

Management:

- Aggressive analgesia
- Pulmonary hygiene
- Incentive spirometry

Flail Chest

- Adequate analgesia
- Oxygen
- NIV/mechanical ventilation if required

Cardiac Tamponade

Features:

- Hypotension
- Raised JVP
- Muffled heart sounds

Management:

- Emergency thoracotomy/pericardiocentesis in selected settings

Chest Tube Indications

- Pneumothorax
- Hemothorax
- Hemopneumothorax

5. ABDOMINAL TRAUMA

Clinical Features

- Pain
- Distension
- Tenderness
- Shock
- Seatbelt marks

Assessment

- FAST/eFAST
- CT abdomen if stable

FAST Examination

Assesses:

- Pericardial fluid
- RUQ fluid
- LUQ fluid
- Pelvic free fluid

Hemodynamically Unstable Patient

Positive FAST + shock:

→ Immediate surgical consultation

Penetrating Trauma

Requires:

- Surgical evaluation
- CT if stable
- Serial examination

Red Flags

- Peritonitis
- Evisceration
- Persistent hypotension
- Free fluid

Damage Control Surgery

Indications:

- Severe hemorrhage
- Coagulopathy
- Hypothermia
- Acidosis

6. SPINE TRAUMA

Clinical Features

- Neck pain
- Weakness
- Sensory loss
- Paralysis
- Respiratory weakness

Principles

- Assume unstable injury until ruled out
- Minimize spinal movement
- Early imaging
- Frequent neurological reassessment

Imaging

Preferred:

- CT spine

MRI:

- Cord injury
- Ligamentous injury
- Neurological deficits

Cervical Spine Clearance

Use:

- Clinical assessment
- CT imaging

High Cervical Injury

May cause:

- Respiratory failure
- Neurogenic shock

Neurogenic Shock

Features:

- Hypotension
- Bradycardia

- Warm extremities

Management:

- Vasopressors
- Fluids cautiously

7. ORTHOPEDIC EMERGENCIES

Common Emergencies

- Open fractures
- Pelvic fractures
- Compartment syndrome
- Dislocations
- Traumatic amputations

Open Fractures

Management:

- Sterile dressing
- IV antibiotics early
- Tetanus prophylaxis
- Orthopedic consultation

Compartment Syndrome

Features

- Pain out of proportion
- Pain on passive stretch
- Tense compartment

Late signs:

- Pulselessness
- Paralysis

Requires emergency fasciotomy.

Pelvic Fracture

Major source of hemorrhage.

Management:

- Pelvic binder
- MTP
- IR/surgery

Traumatic Amputation

- Control bleeding
- Wrap amputated part in moist gauze
- Place in sealed bag on ice

8. BURNS

Burn Assessment

Assess:

- TBSA
- Burn depth
- Airway involvement
- Circumferential burns

Burn Depth

- Superficial
- Partial thickness
- Full thickness

Inhalational Injury

Suspect if:

- Facial burns
- Singed nasal hair
- Hoarseness
- Carbonaceous sputum

Consider early intubation.

Burn Fluid Resuscitation

Modified Parkland Formula

Fluid\ Requirement = 2 ml LR x kg x % TBSA/16 hrs

Preferred fluid:

- Ringer Lactate

Administration:

- Total in 16 hours

Urine Output Targets

Adults:

- ≥0.5 mL/kg/hr

Children:

- ≥1 mL/kg/hr

Burn Referral Criteria

- 10% TBSA
- Facial burns
- Electrical burns
- Pediatric burns
- Inhalational injury
- Perineal burns

9. TRAUMA IMAGING

Imaging Principles

- Imaging should not delay lifesaving interventions
- Hemodynamic stability determines imaging strategy

Initial Trauma Imaging

- Portable chest X-ray
- Pelvic X-ray
- FAST/eFAST

CT Trauma Protocol

Indications:

- Stable major trauma
- Polytrauma
- Unexplained shock

eFAST

Detects:

- Hemoperitoneum
- Pneumothorax
- Hemothorax
- Pericardial effusion

Imaging in Unstable Trauma

Avoid unnecessary CT.

Proceed to:

- OR
- Damage control intervention

10. MASSIVE TRANSFUSION PROTOCOL (MTP)

Definition

Protocolized blood product administration for severe hemorrhage.

Activation Criteria

- Hemorrhagic shock
- Massive bleeding
- Pelvic trauma
- Multiple penetrating injuries
- Anticipated massive transfusion

Components

Balanced resuscitation:

- PRBC

- FFP
- Platelets

Target ratio:

1:1:1 where feasible

Adjuncts

- TXA
- Calcium replacement
- Warming measures

Monitoring

- Hemoglobin
- INR
- Fibrinogen
- Lactate
- Calcium
- Temperature

Lethal Diamond in Trauma

1. Hypothermia
2. Acidosis
3. Coagulopathy
4. Hypocalcemia

Goals

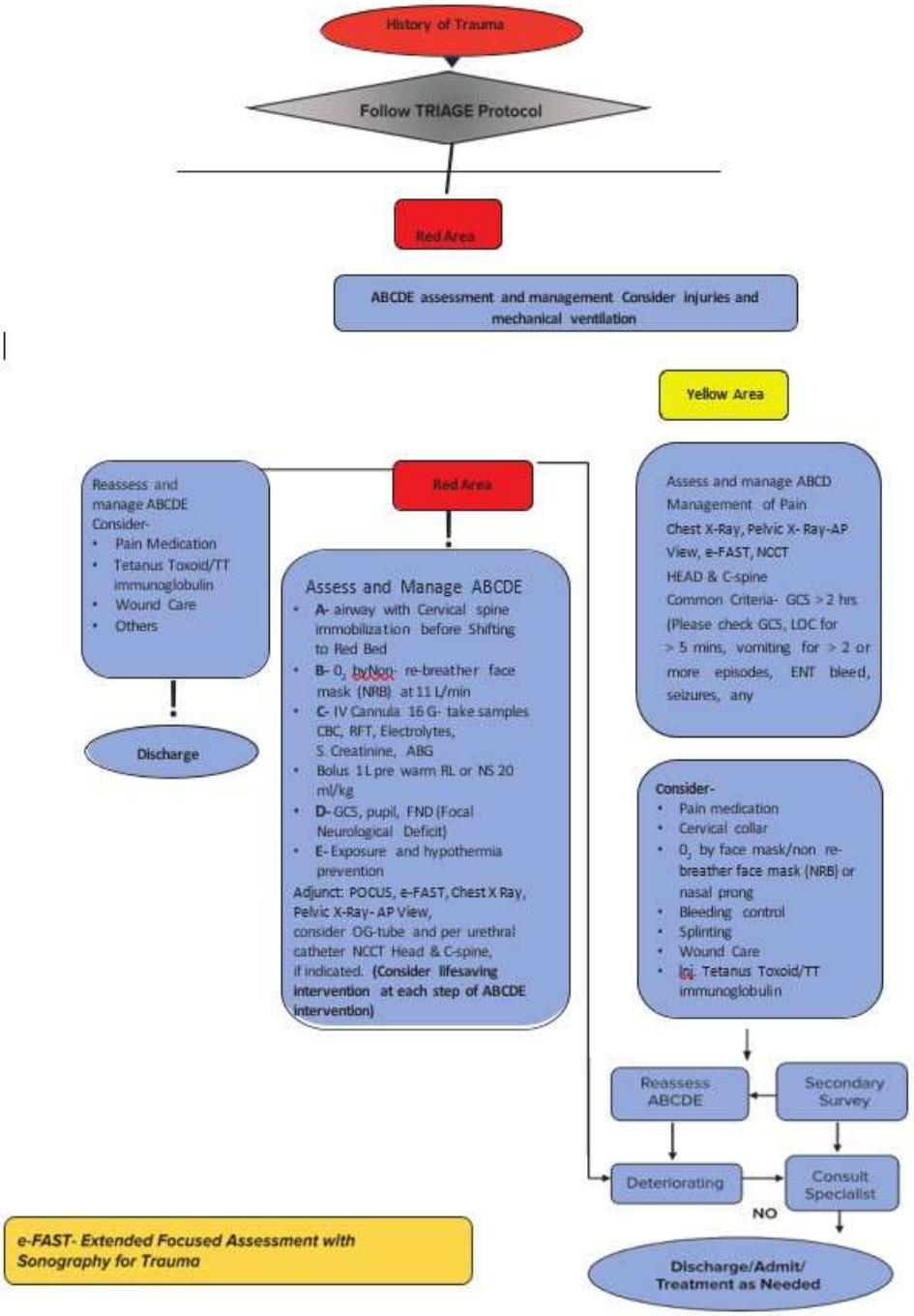
- Control bleeding
- Restore perfusion
- Prevent coagulopathy
- Avoid dilutional shock

References:

- ATLS 11th Edition Manual

Algorithm

1. Algorithm for the management of Trauma



1. Blood loss sites

SITE	DIAGNOSTIC	TIMING	FINDINGS	Comments/Caveats
Abdomen	FAST	Can be performed as a part of the primary survey (xABCDE — FAST)	Positive in any window	A negative FAST is nondiagnostic and should be repeated at intervals in the persistently unstable patient
	Diagnostic peritoneal aspiration (aspiration or lavage)	Usually after excluding hemorrhage in other sites	Grossly positive	Used when other sources of bleeding have not been clearly identified Should only be performed by an available surgeon
Chest	Chest radiograph	As soon as possible after arrival, either during or immediately after primary survey	'White-out' of hemithorax	A wide mediastinum is a danger sign, but not likely active bleeding Pneumonectomy may mimic hemothorax
Pelvis	Pelvis radiograph	As soon as possible after arrival, either during or immediately after primary survey	Pelvis fracture patterns associated with bleeding: open book, vertical shear, lateral compression	
Retroperitoneum	By elimination	Moving to the CT scanner should only occur when there is a modicum of stability and reserved for responders and transient responders	There are no current bedside diagnostics to evaluate this area	This is a closed space and often these patients will be transient responders, allowing time for CT scanning
Floor	Observation/History from field	At time of arrival and during primary survey	Gross blood on the gurney at patient arrival; report from transporting providers	If patient is a transient or non-responder and no gross bleeding is observed, look for other sources
Muscular and subcutaneous tissues; Fracture sites	Rapid TEG, INR, low hematocrit, skeletal x-rays		Discuss TEG INR may not change early Low hematocrit is a late sign, but any low hematocrit detected at first lab draw should raise suspicion for bleeding	This can stem from multiple injuries that are nonsurgical in nature (e.g., scalp laceration, multiple bony and soft tissue injuries), resulting in trauma-induced coagulopathy Low hematocrit may be chronic but could be determined by looking at RBC indices

2. Hemorrhage Severity

	HEMORRHAGE SEVERITY →			
PARAMETER	**MINOR**		**MODERATE**	**MAJOR**
Heart Rate (beats/min)	Unchanged	Unchanged to Increased	Increased (>100)	Markedly Increased (>120)
Systolic Pressure (mm Hg)	Unchanged	Unchanged	Unchanged to Decreased	Markedly Decreased (<90)
Pulse Pressure	Unchanged	Decreased	Decreased	Decreased
Respiratory Rate	Unchanged	Unchanged to Increased	Increased	Increased
Level of Consciousness	Unchanged	Unchanged	Decreased	Markedly Decreased
Base Deficit (meq/L)	0 to -2	-2 to -6	-6 to -10	-10 and less
Blood Products	Unlikely	Possibly	Likely	Massive Transfusion Protocol
Response to Resuscitation	Rapid and Sustained	Fairly Rapid and Sustained	Transient	Minimal to None
Operative Hemorrhage Control	Unlikely	Possibly	Likely	Extremely Likely

3. Burns management

Category of Burn	Age and Weight	Adjusted Hourly Fluid Rate (ml/hr)	Urine Output Goal
Thermal or Chemical Burn	Adults and Older Children ≥13 years old	$\frac{2 \text{ ml LR} \times \text{kg} \times \% \text{ TBSA}}{16}$	0.5 ml/kg/hr (30–50 ml/hr)
	Children <13 years old	$\frac{3 \text{ ml LR} \times \text{kg} \times \% \text{ TBSA}}{16}$	1 ml/kg/hr
	Infants and young children ≤ 30 kg	$\frac{3 \text{ ml LR} \times \text{kg} \times \% \text{ TBSA}}{16}$ Plus D5LR at maintenance rate	1 ml/kg/hr
Electrical Injury	All ages	$\frac{4 \text{ ml LR} \times \text{kg} \times \% \text{ TBSA}}{16}$ Children <13 years old require D5LR at maintenance rate	1–1.5 ml/kg/hr until urine is clear of pigment

SECTION IX
PAEDIATRIC EMERGENCIES

Scope

This protocol provides a standardized approach for recognition, assessment, stabilization, resuscitation, investigation, and early management of Paediatric emergencies presenting to Emergency Departments in Government Medical Institutions across Kerala.

The guideline applies to:

1. Primary Health Centres
2. Taluk Hospitals
3. District Hospitals
4. Government Medical Colleges
5. Paediatric Emergency and Critical Care Units

This section focuses on:

1. Paediatric triage
2. Respiratory distress
3. Paediatric shock
4. Febrile seizures
5. Paediatric trauma
6. Dehydration
7. Paediatric sepsis
8. Poisoning

The protocol is intended for:

1. Emergency Physicians
2. Paediatricians
3. Casualty Medical Officers
4. Critical Care Teams
5. Nurses
6. Junior Residents
7. Paramedics

Special emphasis is placed on:

1. Early recognition of deterioration
2. Weight-based emergency management
3. Prevention of respiratory failure and shock
4. Timely referral and escalation of care
5. Early identification of sepsis and trauma

Introduction

Paediatric emergencies differ significantly from adult emergencies due to anatomical, physiological, and developmental differences. Children may initially compensate well during critical illness and subsequently deteriorate rapidly.

Early recognition of respiratory distress, shock, sepsis, neurological compromise, and trauma is essential to reduce morbidity and mortality.

Children commonly present with:

1. Fever
2. Respiratory distress
3. Seizures
4. Dehydration
5. Trauma
6. Poisoning
7. Altered sensorium

All critically ill children should undergo:

1. Structured Paediatric Assessment Triangle (PAT)
2. ABCDE assessment
3. Weight-based drug dosing
4. Continuous reassessment

The major priorities in Paediatric emergency care include:

1. Airway stabilization
2. Adequate oxygenation and ventilation
3. Recognition and treatment of shock
4. Prevention of hypoglycemia
5. Early treatment of sepsis
6. Timely referral to higher centres where required

Definitions

1. Paediatric Emergency

An acute illness or injury in a child requiring urgent evaluation and intervention to prevent morbidity or mortality.

2. Paediatric Age Group

Children from birth up to 12 years of age.

3. Paediatric Shock

A state of inadequate tissue perfusion resulting in impaired oxygen delivery to organs.

4. Sepsis

Life-threatening organ dysfunction caused by dysregulated host response to infection.

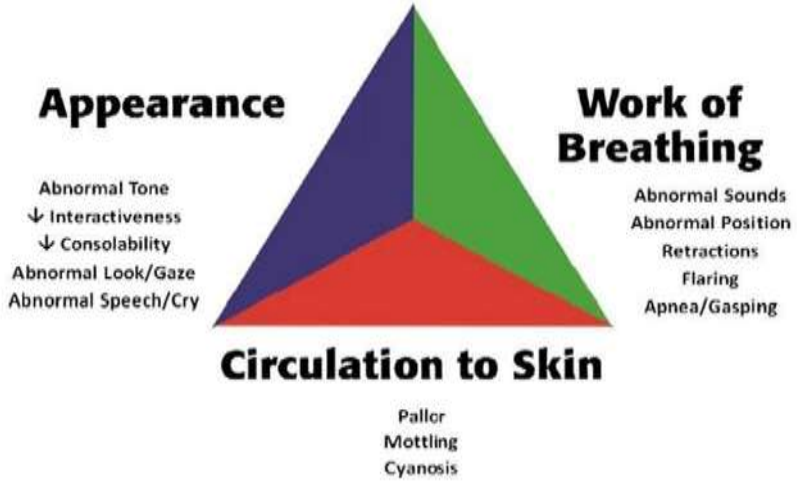
1. PAEDIATRIC TRIAGE

Principles

Children should be prioritized according to:

1. Severity of illness
2. Airway compromise
3. Respiratory distress
4. Shock
5. Neurological status
6. Red Flag Signs
7. Cyanosis
8. Severe respiratory distress
9. Altered sensorium
10. Shock
11. Active seizures
12. Severe dehydration

Paediatric Assessment Triangle (PAT)



Appearance

Assess:

1. Tone
2. Interactiveness
3. Consolability
4. Cry/speech

5. Gaze

Work of Breathing

Assess:

1. Respiratory effort
2. Retractions
3. Nasal flaring
4. Grunting

Circulation to Skin

1. Assess:
2. Pallor
3. Mottling
4. Cyanosis

2. PAEDIATRIC RESPIRATORY DISTRESS

Definition

Paediatric respiratory distress is increased work of breathing caused by respiratory compromise while adequate gas exchange is still maintained.

Without timely intervention, respiratory distress may progress to:

- Respiratory failure
- Bradycardia
- Cardiac arrest

Recognition

Paediatric Assessment Triangle (PAT)

Appearance

Assess:

- Tone
- Interactiveness
- Consolability
- Cry/speech
- Gaze

Work of Breathing

Assess:

- Tachypnea
- Nasal flaring
- Retractions
- Grunting
- Head bobbing

- Accessory muscle use

Circulation to Skin

Assess:

- Pallor
- Cyanosis
- Mottling

Rapid PAT assessment helps identify critically ill children within seconds.

Severity Assessment

Mild Respiratory Distress

- Mild tachypnea
- Minimal retractions
- Normal mental status
- Feeding/speaking preserved

Moderate Respiratory Distress

- Marked tachypnea
- Intercostal/subcostal retractions
- Nasal flaring
- Reduced feeding/activity
- Tachycardia

Severe Respiratory Distress

- Severe retractions
- Grunting
- Head bobbing
- Cyanosis
- Inability to feed/speak
- Altered sensorium
- Exhaustion

Respiratory Failure Warning Signs

Impending Respiratory Failure

- Poor air entry
- Bradypnea
- Cyanosis
- Altered mental status
- Weak respiratory effort
- Apnea
- Hypoxia despite oxygen
- Bradycardia

Bradycardia in a hypoxic child is a pre-arrest sign requiring immediate intervention.

Classification of Respiratory Distress

Upper Airway Obstruction

Clinical Features

- Stridor
- Barking cough
- Hoarse voice
- Drooling

Common Causes

- Croup
- Epiglottitis
- Foreign body aspiration
- Anaphylaxis

Lower Airway Obstruction

Clinical Features

- Wheeze
- Prolonged expiration
- Accessory muscle use

Common Causes

- Asthma
- Bronchiolitis

Lung Tissue Disease

Clinical Features

- Crackles
- Hypoxia
- Grunting

Common Causes

- Pneumonia
- ARDS
- Pulmonary edema

Disordered Control of Breathing

Clinical Features

- Irregular breathing
- Reduced respiratory effort

Causes

- CNS infection
- Poisoning

- Seizures
- Sedative overdose

Initial Assessment

ABCDE Approach

Airway

Assess:

- Patency
- Secretions
- Obstruction
- Stridor

Position airway appropriately.

Breathing

Assess:

- Respiratory rate
- Work of breathing
- Air entry
- Oxygen saturation

Provide oxygen immediately if hypoxic.

Circulation

Assess:

- Heart rate
- Perfusion
- Blood pressure
- Pulses

Disability

Assess:

- AVPU/GCS
- Blood glucose

Exposure

Look for:

- Rash
- Trauma
- Fever
- Cyanosis

Oxygen Therapy

Recommended

Provide supplemental oxygen for:

- Hypoxia

- Moderate/severe respiratory distress

Oxygen Target

- SpO_2 ≥94%

Oxygen Delivery Devices

Device	Indication
Nasal prongs	Mild distress
Face mask	Moderate distress
Non-rebreather mask	Severe hypoxia
HFNC	Moderate-severe distress
NIV/CPAP	Selected cases

High-Flow Nasal Cannula (HFNC)

Consider In

- Bronchiolitis
- Pneumonia
- Moderate hypoxemia
- Increased work of breathing

Benefits

- Improved oxygenation
- Reduced work of breathing
- Better secretion clearance

Monitor closely for deterioration.

Nebulized Therapy

Bronchodilator Therapy

Salbutamol

2.5-5 mg

Nebulized with oxygen.

Indications

- Asthma
- Wheezing disorders

Nebulized Adrenaline

Consider In

- Moderate-severe croup

Dose

5 mL of 1:1000 adrenaline

Nebulized.

Corticosteroids

Dexamethasone

0.15-0.6 mg/kg

Maximum:

- 10 mg

Indications

- Croup
- Asthma exacerbation

Fluid Management

Principles

- Avoid fluid overload
- Maintain hydration

Children with severe respiratory distress fatigue rapidly during feeding.

Consider

- IV fluids
- NG hydration if unable to tolerate orally

Monitoring

Continuous Monitoring

- Respiratory rate
- SpO_2
- Heart rate
- Mental status
- Work of breathing

Blood Gas Assessment

Consider ABG/VBG If

- Severe distress
- Persistent hypoxia
- Suspected hypercapnia
- Deterioration
- Fatigue

Indications for Escalation

Urgent Senior/PICU Review If

- Persistent hypoxia
- Increasing work of breathing
- Exhaustion
- Altered sensorium
- Bradycardia
- Apnea
- Hemodynamic instability

Non-Invasive Ventilation (NIV)

Consider In Selected Patients

- Severe asthma
- Pulmonary edema
- Neuromuscular weakness

Requires:

- Experienced staff
- Close monitoring

Indications for Intubation

Consider Early Intubation If

- Respiratory failure
- Exhaustion
- Persistent hypoxia
- Reduced consciousness
- Apnea
- Poor respiratory effort
- Severe shock

Bag-Mask Ventilation

Important PALS Principle

Effective bag-mask ventilation is a core Paediatric resuscitation skill and may be preferable to immediate advanced airway placement in many Paediatric emergencies.

Common Etiology-Based Management

Condition	Important Management
Asthma	Bronchodilators + steroids
Bronchiolitis	Oxygen + suction + HFNC
Croup	Neb adrenaline + dexamethasone
Pneumonia	Oxygen + antibiotics
Anaphylaxis	IM adrenaline
Foreign body aspiration	Airway management

Disposition

Discharge Criteria

- Minimal/no respiratory distress
- Stable SpO_2 on room air
- Adequate feeding
- Caregiver comfortable
- No escalation requirement

Admission Criteria

- Oxygen requirement
- Moderate/severe distress
- Feeding difficulty
- Recurrent apnea
- HFNC/NIV requirement
- Significant comorbidity

ICU Referral Criteria

- Respiratory failure
- Need for NIV/intubation
- Exhaustion
- Persistent hypoxia
- Altered sensorium
- Shock

3. PAEDIATRIC SHOCK

Definition

Paediatric shock is a state of inadequate tissue perfusion resulting in impaired oxygen delivery, cellular dysfunction, and organ failure.

Children may maintain blood pressure until late stages of shock because of strong compensatory vasoconstriction and tachycardia. Hypotension is therefore a late and pre-terminal sign.

Current Paediatric Advanced Life Support (PALS) guidelines emphasize:

- Early recognition
- Rapid vascular access
- Frequent reassessment
- Timely vasoactive support
- Prevention of progression to cardiac arrest

Types of Paediatric Shock

1. Hypovolemic Shock

Most common type in children.

Causes:

- Gastroenteritis
- Vomiting
- Hemorrhage
- Burns
- Dehydration

2. Septic Shock

Distributive shock caused by severe infection leading to:

- Vasodilation
- Capillary leak
- Myocardial dysfunction

3. Cardiogenic Shock

Failure of myocardial pump function.

Causes:

- Myocarditis
- Cardiomyopathy
- Arrhythmias
- Congenital heart disease

4. Obstructive Shock

Mechanical obstruction to circulation.

Causes:

- Tension pneumothorax
- Cardiac tamponade
- Massive pulmonary embolism

5. Distributive Shock

Abnormal vasodilation causing maldistribution of blood flow.

Causes:

- Anaphylaxis
- Neurogenic shock
- Sepsis

Recognition

Early Compensated Shock

Clinical Features

- Tachycardia
- Delayed capillary refill time (CRT)
- Cool extremities
- Weak peripheral pulses
- Tachypnea
- Reduced urine output
- Irritability/anxiety
- Poor feeding in infants

Children compensate initially with vasoconstriction and tachycardia before hypotension develops.

Decompensated Shock

Features

- Hypotension
- Altered sensorium
- Mottled/cyanotic skin
- Weak or absent pulses
- Severe oliguria/anuria
- Bradycardia (pre-terminal sign)

Hypotension in children indicates severe circulatory failure requiring immediate aggressive management.

Paediatric Assessment Triangle (PAT)

Appearance

Assess:

- Tone
- Interactiveness
- Consolability
- Cry/speech
- Gaze

Work of Breathing

Assess:

- Tachypnea
- Retractions
- Nasal flaring
- Grunting

Circulation to Skin

Assess:

- Pallor
- Mottling
- Cyanosis

Initial Assessment

ABCDE Assessment

Airway

- Ensure airway patency
- Position appropriately
- Suction secretions if needed

Breathing

- Oxygen therapy
- Assess respiratory effort
- Pulse oximetry

Circulation

Assess:

- Heart rate
- Pulses
- CRT
- Blood pressure
- Perfusion

Obtain:

- IV/IO access rapidly

Disability

- AVPU/GCS
- Blood glucose

Exposure

Look for:

- Rash
- Trauma
- Bleeding
- Infection source

Initial Stabilization

Oxygen Therapy

Recommended

High-flow oxygen via:

- Non-rebreather mask
- HFNC if required

Oxygen Target

- SpO_2 ≥94%

Vascular Access

Rapid Access Is Essential

Preferred

- Peripheral IV access

If IV Delayed (>90 seconds or 2 failed attempts)

- Establish IO access immediately

PALS strongly supports early IO access in critically ill children when IV access is difficult.

Fluid Therapy

Initial Fluid Resuscitation

Standard Initial Bolus

10-20\ mL/kg

Use:

- Isotonic crystalloids
  - Normal saline
  - Ringer lactate

Administration

- Give over 5–10 minutes
- Reassess after EACH bolus

Frequent reassessment is emphasized in current PALS recommendations.

Reassessment After Every Bolus

Assess:

- Heart rate
- CRT
- Pulse quality
- Blood pressure
- Mental status
- Urine output
- Hepatomegaly
- Respiratory distress/rales

Caution With Fluids

Use Smaller Boluses (5–10 mL/kg) and Frequent Reassessment In:

- Severe malnutrition
- Suspected cardiogenic shock
- Renal failure
- Dengue shock syndrome

Watch for:

- Pulmonary edema
- Hepatomegaly
- Worsening respiratory distress

Septic Shock

Recognition

Cold Shock Features

- Cold extremities
- Delayed CRT
- Weak pulses
- Narrow pulse pressure

Warm Shock Features

- Bounding pulses
- Flash CRT
- Wide pulse pressure

Children commonly present with cold shock unlike adults.

Septic Shock Management

First Hour Priorities

1. Oxygen therapy
2. IV/IO access
3. Early fluid resuscitation
4. Broad-spectrum antibiotics
5. Frequent reassessment
6. Early vasoactive support if fluid refractory

Antibiotics

Administer Early

Broad-spectrum antibiotics should be administered as early as possible after cultures if feasible.

Delayed antibiotics increase mortality in Paediatric septic shock.

Vasoactive Support

Early Vasoactive Therapy Recommended in Fluid-Refractory Shock

Epinephrine

Preferred in:

- Cold shock
- Low cardiac output states

Norepinephrine

Preferred in:

- Warm vasodilatory shock

Peripheral or IO vasoactive infusion may be initiated while central access is being secured in critically ill children.

Cardiogenic Shock

Suspect If

- Hepatomegaly
- Gallop rhythm
- Pulmonary edema
- Murmur
- Cardiomegaly
- Poor response to fluids

Management

- Smaller fluid boluses (5–10 mL/kg)
- Early inotropes
- Avoid fluid overload
- Early ICU/cardiology involvement

Anaphylactic Shock

Intramuscular Adrenaline

0.01\ mg/kg

Maximum:

- 0.5 mg IM

Repeat every 5–15 minutes if needed.

Additional Measures:

- Oxygen
- IV fluids
- Nebulized bronchodilators
- Antihistamines
- Steroids

Monitoring

Continuous Monitoring

- Heart rate
- Respiratory rate
- SpO_2
- Blood pressure
- Mental status
- Urine output

Urine Output Goal

Investigations

Recommended

- CBC
- Blood glucose
- ABG/VBG
- Lactate
- Renal function
- Blood cultures
- Chest X-ray if indicated
- ECG/Echocardiography in suspected cardiogenic shock

Indications for Intubation

Consider Early Intubation If

- Respiratory failure
- Persistent shock
- Severe encephalopathy
- Refractory hypoxemia
- Exhaustion

- Recurrent apnea

Paediatric cardiac arrest is commonly preceded by respiratory failure or shock. Early stabilization may prevent arrest.

CPR Considerations in Shock Progression

If Cardiac Arrest Occurs

High-quality CPR remains the cornerstone of Paediatric resuscitation.

- Early epinephrine in nonshockable arrest
- Rapid vascular access (IV/IO)
- Avoid hyperventilation
- Treat reversible causes aggressively

ICU Referral Criteria

- Fluid-refractory shock
- Need for vasoactive agents
- Respiratory failure
- Altered sensorium
- Multiorgan dysfunction
- Persistent hypotension

4. FEBRILE SEIZURE

Definition

A febrile seizure is a seizure occurring in children aged:

- 6 months to 5 years

Associated with:

- Fever ≥38°C

Without:

- CNS infection
- Acute metabolic abnormality
- Previous afebrile seizures

Classification

Simple Febrile Seizure

Features

- Generalized tonic-clonic seizure
- Duration <15 minutes
- Single episode within 24 hours
- Complete recovery
- No focal neurological deficits

Most febrile seizures are simple febrile seizures.

Complex Febrile Seizure

Features

Any ONE of:

- Duration >15 minutes
- Focal seizure features
- Recurrent seizure within 24 hours

Higher risk of recurrence and future epilepsy compared to simple febrile seizures.

Febrile Status Epilepticus

Definition

Seizure duration >30 minutes

OR recurrent seizures without regaining consciousness.

Requires urgent aggressive management.

Recognition

Clinical Features

- Fever
- Generalized tonic-clonic movements
- Eye deviation
- Limb stiffening/jerking
- Postictal drowsiness
- Transient altered sensorium

Red Flag Features

Consider Alternative Diagnoses If

- Age <6 months
- Persistent altered sensorium
- Neck stiffness
- Bulging fontanelle
- Focal neurological deficits
- Purpuric rash
- Persistent seizures
- Signs of raised ICP
- Developmental delay/regression
- Trauma history

Always evaluate for:

- Meningitis
- Encephalitis
- Metabolic disturbances

Initial Assessment

ABCDE Assessment

Airway

- Position airway
- Suction secretions if needed

Breathing

- Oxygen therapy
- Monitor SpO_2

Circulation

- Heart rate
- Blood pressure
- IV/IO access if required

Disability

- AVPU/GCS
- Blood glucose
- Seizure duration

Exposure

- Identify source of fever
- Rash
- Trauma
- Signs of infection

Immediate Stabilization

During Active Seizure

Safety Measures

- Place child in lateral position
- Prevent injury
- Do NOT restrain forcibly
- Do NOT place objects in mouth

Oxygen Therapy

Recommended

- High-flow oxygen if hypoxic

Target SpO_2

- ≥94%

Blood Glucose

Mandatory Early Check

Treat hypoglycemia immediately.

Active Seizure Management

First-Line Benzodiazepines

IV Lorazepam

0.1\ mg/kg

Maximum:

- 4 mg

OR

IV/IN/BUCCAL Midazolam

0.2 mg/kg

OR

Rectal Diazepam

0.5\ mg/kg

If IV access unavailable.

Repeat ONE dose after 5 minutes if seizure persists.

Refractory Seizure Management

If Seizure Persists After Benzodiazepines

Consider

- Levetiracetam

  OR
- Fosphenytoin/phenytoin

  OR
- Valproate (selected cases)

Escalate Early

- PICU/critical care involvement
- Prepare for airway management

Fever Management

Antipyretics

Paracetamol

15 mg/kg

OR

Ibuprofen

10 mg/kg

If age appropriate.

Antipyretics improve comfort but do NOT reliably prevent recurrence of febrile seizures.

Investigations

Simple Febrile Seizure

Routine investigations usually NOT required in well-appearing children.

Avoid routine:

- CT brain
- MRI

- EEG
- Extensive laboratory testing

Investigations If Indicated

Consider:

- CBC
- Blood glucose
- Electrolytes
- Lumbar puncture
- Neuroimaging

If:

- CNS infection suspected
- Persistent altered sensorium
- Complex febrile seizure
- Signs of raised ICP
- Toxic appearance

Lumbar Puncture

Consider If

- Meningitis suspected
- Incompletely immunized child
- Persistent altered mental status
- Infants <12 months with unclear presentation

EEG

NOT Routinely Recommended

In:

- Simple febrile seizures
- Neurologically normal child

Neuroimaging

NOT Routine

CT/MRI only if:

- Focal deficits
- Trauma
- Raised ICP
- Persistent altered consciousness

Admission Criteria

Admit If

- Complex febrile seizure
- Febrile status epilepticus
- Persistent altered sensorium

- CNS infection suspected
- Respiratory compromise
- Recurrent seizures
- Poor oral intake/dehydration
- Unreliable home observation

Discharge Criteria

Safe Discharge If

- Child returned to baseline
- Simple febrile seizure
- Source of fever identified/acceptable
- Tolerating oral intake
- Parents reassured and educated
- No red flag signs

Parent Counselling

Important Counselling Points

Explain:

- Febrile seizures are usually benign
- Most children recover completely
- Recurrence may occur
- Risk of epilepsy is low after simple febrile seizure

Teach parents:

- Recovery position
- Seizure first aid
- When to return urgently

Return Immediately If

- Seizure >5 minutes
- Breathing difficulty
- Persistent drowsiness
- Recurrent seizures
- Neck stiffness
- Poor feeding
- Rash
- Child appears very ill

Follow-Up

Paediatric Follow-Up Recommended If

- Complex febrile seizure
- Developmental concerns
- Recurrent febrile seizures

- Neurological abnormalities

5. PAEDIATRIC TRAUMA

Definition

Paediatric trauma is physical injury occurring in infants and children requiring rapid identification and treatment of life-threatening conditions.

Trauma is one of the leading causes of death in children worldwide.

"Treat the greatest threat to life first."

Important Paediatric Trauma Principles

Unique Paediatric Considerations

Children have:

- Larger head-to-body ratio
- Smaller airway diameter
- More compliant chest wall
- Higher oxygen consumption
- Less blood volume
- Greater heat loss risk

Children may maintain blood pressure until late shock.

Hypotension is a late and ominous sign.

xABCDE Approach

xABCDE

Where:

- x = control of exsanguinating hemorrhage
- A = airway
- B = breathing
- C = circulation
- D = disability
- E = exposure/environment

x – Exsanguinating Hemorrhage

Immediate Priorities

Control life-threatening bleeding FIRST.

Methods

- Direct pressure
- Tourniquet
- Hemostatic dressing
- Pelvic binder if indicated

Uncontrolled hemorrhage rapidly leads to shock and cardiac arrest.

A – Airway With Cervical Spine Protection

Assess For

- Airway obstruction
- Stridor
- Gurgling
- Facial trauma
- Altered consciousness

Maintain:

- Inline cervical stabilization

Airway Adjuncts

Consider

- Oropharyngeal airway
- Nasopharyngeal airway
- Suction

Indications for Definitive Airway

Consider Early Intubation If

- GCS ≤8
- Airway compromise
- Severe facial trauma
- Hypoventilation
- Refractory hypoxemia
- Severe shock

B – Breathing

Assess

- Respiratory rate
- Chest expansion
- Air entry
- Oxygen saturation
- Work of breathing

Provide:

- High-flow oxygen

Life-Threatening Thoracic Injuries

Tension Pneumothorax

Features

- Severe respiratory distress
- Reduced unilateral breath sounds
- Shock
- Tracheal deviation (late)

Management

Immediate decompression.

Open Pneumothorax

- Three-sided occlusive dressing
- Chest tube

Massive Hemothorax

Suspect if:

- Shock
- Reduced breath sounds
- Dull percussion

Requires:

- Chest drainage
- Blood products

Pulmonary Contusion

More common in children due to compliant chest wall.

C – Circulation

Assess

- Heart rate
- Pulses
- Blood pressure
- Capillary refill
- Skin perfusion

Hemorrhagic Shock

Early Signs

- Tachycardia
- Delayed CRT
- Weak pulses
- Cool extremities

Late Signs

- Hypotension
- Altered sensorium
- Bradycardia

Vascular Access

Rapid Access Essential

Preferred

- Peripheral IV access

If Delayed

- IO access

ATLS supports rapid vascular access during trauma resuscitation.

Fluid Resuscitation

Initial Bolus

20 mL/kg

Use:

- Isotonic crystalloids

Reassess after each bolus.

Avoid excessive crystalloid administration.

Blood Product Resuscitation

Consider Early If

- Major hemorrhage
- Persistent shock
- Poor response to fluids

Pelvic Trauma

Suspect If

- High-energy trauma
- Pelvic instability
- Shock without obvious source

Management

- Pelvic binder
- Massive transfusion consideration

D – Disability (Neurological Assessment)

Assess

- AVPU/GCS
- Pupils
- Seizures
- Limb movement
- Blood glucose

Paediatric Glasgow Coma Scale

Severe Traumatic Brain Injury

GCS <8

Requires:

- Definitive airway consideration
- Neurosurgical consultation

Signs of Raised ICP

Features

- Altered sensorium
- Bradycardia

- Hypertension
- Unequal pupils
- Posturing

E – Exposure & Environment

Fully Expose Child

Look for:

- Bleeding
- Deformities
- Burns
- Penetrating injuries

Prevent:

- Hypothermia aggressively

Children lose heat rapidly during trauma resuscitation.

Secondary Survey

Perform After Stabilization

Includes

- Full head-to-toe examination
- AMPLE history
- Detailed neurological assessment
- Reassessment of vitals

ATLS emphasizes continuous reassessment throughout resuscitation.

Paediatric Head Trauma

Red Flag Features

- Altered mental status
- Persistent vomiting
- Severe headache
- Seizure
- Loss of consciousness
- Signs of skull fracture
- Severe mechanism

PECARN Head Injury Rules

Purpose

PECARN helps identify children at very low risk of clinically important traumatic brain injury (ciTBI).

Reduces unnecessary CT scans.

PECARN Criteria (<2 Years)

High Risk

- Altered mental status

- Palpable skull fracture

Intermediate Risk

- Occipital/parietal hematoma
- Severe mechanism
- LOC >5 seconds
- Abnormal behavior per parent

PECARN Criteria (≥2 Years)

High Risk

- Altered mental status
- Signs of basilar skull fracture

Intermediate Risk

- Vomiting
- Severe headache
- LOC
- Severe mechanism

Observation vs CT based on clinical judgment.

Signs of Basilar Skull Fracture

Features

- Battle sign
- Raccoon eyes
- Hemotympanum
- CSF leak

Cervical Spine Trauma

Suspect If

- High-risk mechanism
- Neck pain
- Neurological deficits
- Altered consciousness

Children commonly sustain upper cervical injuries.

Thoracoabdominal Trauma

Assess For

- Abdominal tenderness
- Distension
- Seatbelt sign
- Guarding
- Shock

FAST Ultrasound

Useful For

- Hemoperitoneum
- Pericardial effusion
- Pneumothorax

Negative FAST does not completely exclude injury in children.

Splenic & Liver Injury

Most Paediatric solid organ injuries are managed non-operatively if:

- Hemodynamically stable

Musculoskeletal Trauma

Assess

- Deformity
- Distal pulses
- Sensation
- Compartment syndrome

Compartment Syndrome Signs

Features

- Severe pain
- Pain on passive stretch
- Pallor
- Weak pulses (late)

Pain Management

Important Principle

Children require adequate analgesia early.

Common Agents

- Paracetamol
- Ibuprofen
- Fentanyl
- Morphine

Avoid undertreating pain.

Procedural Sedation

Consider For

- Fracture reduction
- Painful procedures

Requires:

- Monitoring
- Airway preparedness
- Resuscitation equipment

Non-Accidental Trauma (NAT)

Suspect If

- Injury inconsistent with history
- Delayed presentation
- Multiple injuries
- Different healing stages
- Retinal hemorrhage

Safeguarding evaluation is mandatory.

Monitoring

Continuous Monitoring

- Heart rate
- Respiratory rate
- BP
- SpO_2
- GCS
- Urine output

ICU Referral Criteria

Refer Early If

- Severe TBI
- Respiratory failure
- Persistent shock
- Massive transfusion requirement
- Polytrauma
- Need for invasive monitoring

Admission Criteria

Admit If

- Moderate/severe injury
- Persistent symptoms
- Abnormal imaging
- Significant mechanism
- Need for observation
- Safeguarding concerns

Discharge Criteria

Safe Discharge If

- Normal neurological status
- Stable vitals
- Ambulating appropriately
- Tolerating oral intake
- Reliable caregivers
- No concerning findings

Return Immediately If

- Repeated vomiting
- Worsening headache
- Seizure
- Drowsiness
- Breathing difficulty
- Behavioral changes
- Weakness

6. PAEDIATRIC DEHYDRATION

Definition

Paediatric dehydration is a deficit of body water resulting from excessive fluid loss, inadequate intake, or both.

Common causes include:

- Acute gastroenteritis
- Vomiting
- Fever
- Poor oral intake
- Excessive sweating

Severe dehydration may progress to:

- Hypovolemic shock
- Electrolyte imbalance
- Acute kidney injury
- Cardiac arrest

Classification of Dehydration

No Dehydration

Clinical Features

- Alert and active
- Normal thirst
- Moist mucosa
- Normal skin pinch
- Normal urine output

Some Dehydration

Clinical Features

Presence of TWO or more:

- Restlessness/irritability
- Sunken eyes

- Thirsty/drinks eagerly
- Dry mucosa
- Reduced tears
- Slow skin pinch

Equivalent to moderate dehydration.

Severe Dehydration

Clinical Features

Presence of TWO or more:

- Lethargy/unconsciousness
- Unable to drink
- Very sunken eyes
- Very slow skin pinch
- Weak pulses
- Tachycardia
- Hypotension
- Poor perfusion

Severe dehydration is a medical emergency requiring rapid fluid resuscitation.

Recognition

Symptoms

- Vomiting
- Diarrhea
- Reduced urine output
- Poor feeding
- Fever
- Lethargy
- Excessive thirst

Signs of Severe Illness

Red Flag Features

- Altered sensorium
- Persistent vomiting
- Shock
- Severe malnutrition
- Seizures
- Bloody diarrhea
- Severe abdominal distension
- Cyanosis
- Respiratory distress

Initial Assessment

ABCDE Assessment

Airway

- Ensure patency
- Protect airway if altered sensorium

Breathing

Assess:

- Respiratory distress
- Deep acidotic breathing
- Oxygen saturation

Provide oxygen if hypoxic.

Circulation

Assess:

- Heart rate
- Blood pressure
- CRT
- Pulses
- Urine output

Obtain:

- IV/IO access if severe dehydration

Disability

Assess:

- AVPU/GCS
- Blood glucose

Exposure

Assess:

- Weight
- Nutritional status
- Rash
- Fever
- Signs of infection

Management Principles

Goals

1. Restore circulation
2. Replace fluid deficit
3. Correct electrolyte abnormalities
4. Continue feeding
5. Prevent complications

ORS Therapy

Oral Rehydration Solution (ORS)

ORS remains the cornerstone of Paediatric dehydration management.

Continue:

- Breastfeeding
- Feeding as tolerated

Plan A: No Dehydration

Home Therapy

Give Extra Fluids After Each Loose Stool

Age	ORS After Stool
<2 years	50–100 mL
2–10 years	100–200 mL
Older children	As desired

Zinc Supplementation

Age <6 Months

10 mg/day for 14 days

Age ≥6 Months

20 mg/day for 14 days

Zinc reduces:

- Severity
- Duration
- Recurrence of diarrhea

Plan B: Some Dehydration

ORS Rehydration Therapy

75 mL/kg over 4 hours

Important Points

- Reassess frequently
- Continue breastfeeding
- Replace ongoing losses

Reassessment After ORS

Assess:

- Mental status
- Thirst
- CRT
- Urine output
- Skin pinch
- Heart rate

If worsening:

- Escalate to IV therapy

Plan C: Severe Dehydration

Immediate IV Fluid Therapy

Preferred Fluid

- Ringer lactate

  OR
- Normal saline if RL unavailable

Initial Fluid Resuscitation

If Shock Present

20 mL/kg

Rapid bolus over:

- 5–10 minutes

Repeat as required with reassessment.

Severe Dehydration Without Shock

WHO/MOHFW Plan C

Infants (<12 months)

100 mL/kg

- First 30 mL/kg over 1 hour
- Remaining 70 mL/kg over 5 hours

Children (>12 months)

100 mL/kg

- First 30 mL/kg over 30 minutes
- Remaining 70 mL/kg over 2.5 hours

If IV Access Difficult

Establish IO Access

PALS strongly supports early IO access in critically ill children if IV access fails.

Ongoing Loss Replacement

Continue ORS

Replace:

- Ongoing diarrhea
- Vomiting losses

Feeding Recommendations

Continue Feeding Early

- Continue breastfeeding
- Resume age-appropriate diet early
- Avoid prolonged fasting

Investigations

Usually Not Required in Mild Cases

Consider If Severe Illness

- Blood glucose
- Electrolytes
- Renal function
- ABG/VBG
- CBC
- Stool studies if indicated

Electrolyte Abnormalities

Watch For

- Hyponatremia
- Hypernatremia
- Hypokalemia
- Metabolic acidosis

Correct gradually and cautiously.

Hypernatremic Dehydration

Suspect If

- Doughy skin
- Irritability
- Neurological symptoms
- Seizures

Requires:

- Slow correction
- Careful sodium monitoring

Rapid correction may cause cerebral edema.

Severe Acute Malnutrition (SAM)

Important Modification

Children with SAM require:

- Smaller slower rehydration
- Careful monitoring
- Specialized WHO protocols

Avoid aggressive fluid boluses unless shock is present.

Monitoring

Continuous Monitoring in Severe Cases

- Heart rate
- Respiratory rate
- Blood pressure
- CRT
- Urine output

- Mental status

Urine Output Goal

Indications for Admission

Admit If

- Severe dehydration
- Persistent vomiting
- Shock
- Electrolyte abnormality
- Failed oral therapy
- Severe malnutrition
- Altered sensorium
- Significant comorbidity

ICU Referral Criteria

- Hypovolemic shock
- Persistent hypotension
- Severe electrolyte abnormalities
- Refractory acidosis
- Altered mental status
- Multiorgan dysfunction

Discharge Criteria

Safe Discharge If

- Hydrated clinically
- Tolerating oral intake
- Good urine output
- Caregivers educated
- No ongoing severe losses

Parent Counselling

Important Advice

Teach caregivers:

- ORS preparation
- Zinc completion
- Continued feeding
- Danger signs requiring return

Return Immediately If

- Unable to drink
- Persistent vomiting
- Blood in stool
- Lethargy

- Reduced urine output
- Fast breathing
- Seizures
- Worsening diarrhea

7. PAEDIATRIC SEPSIS

Definition

Paediatric sepsis is a life-threatening condition caused by a dysregulated response to infection resulting in organ dysfunction.

Children may deteriorate rapidly from compensated sepsis to:

- Septic shock
- Respiratory failure
- Multiorgan dysfunction
- Cardiac arrest

Recognition

Common Sources of Infection

- Pneumonia
- Gastroenteritis
- Meningitis
- Urinary tract infection
- Skin/soft tissue infection
- Tropical infections

Early Recognition

Compensated Sepsis

Clinical Features

- Fever or hypothermia
- Tachycardia
- Tachypnea
- Delayed capillary refill time (CRT)
- Reduced urine output
- Irritability
- Poor feeding
- Weak peripheral pulses

Children often maintain blood pressure until late stages.

Decompensated Septic Shock

Severe Features

- Hypotension

- Altered sensorium
- Mottled/cyanotic skin
- Weak or absent pulses
- Severe oliguria
- Bradycardia (pre-terminal sign)
- Respiratory failure

Hypotension in Paediatric septic shock is a late and ominous sign.

Types of Septic Shock

Cold Shock

Most common in children.

Features

- Cold extremities
- Delayed CRT
- Weak pulses
- Narrow pulse pressure

Suggests:

- Low cardiac output
- Increased SVR

Warm Shock

Features

- Bounding pulses
- Flash CRT
- Wide pulse pressure
- Warm extremities

Suggests:

- Vasodilatory shock

Paediatric Assessment Triangle (PAT)

Appearance

Assess:

- Tone
- Interactiveness
- Consolability
- Cry/speech
- Mental status

Work of Breathing

Assess:

- Tachypnea
- Retractions

- Grunting
- Respiratory distress

Circulation to Skin

Assess:

- Pallor
- Cyanosis
- Mottling

Rapid PAT assessment helps identify critically ill children early.

Initial Assessment

ABCDE Approach

Airway

- Ensure airway patency
- Position airway
- Suction secretions if needed

Breathing

Assess:

- Respiratory rate
- Work of breathing
- SpO_2
- Air entry

Provide oxygen immediately if hypoxic.

Circulation

Assess:

- Heart rate
- Blood pressure
- CRT
- Peripheral pulses
- Perfusion
- Urine output

Obtain:

- IV/IO access rapidly

Disability

Assess:

- AVPU/GCS
- Blood glucose
- Seizures

Exposure

Look for:

- Rash
- Trauma
- Infection focus
- Petechiae/purpura

Initial Stabilization

Oxygen Therapy

Recommended

- High-flow oxygen

Oxygen Target

- SpO_2 ≥94%

Vascular Access

Rapid Access Essential

Preferred

- Peripheral IV access

If Delayed

- IO access immediately

Do not delay resuscitation waiting for central access.

Fluid Resuscitation

Initial Fluid Bolus

10-20 mL/kg

Use:

- Isotonic crystalloids
  - Normal saline
  - Ringer lactate

Administration

- Over 5–10 minutes
- Reassess after EACH bolus

Reassessment After Every Bolus

Assess:

- Heart rate
- CRT
- Blood pressure
- Pulse quality
- Urine output
- Mental status
- Hepatomegaly
- Respiratory distress/rales

Fluid-Refractory Shock

Suspect If

Persistent poor perfusion despite fluids.

Avoid excessive fluid overload.

Vasoactive Support

Early Vasoactive Therapy Recommended

Epinephrine

Preferred in:

- Cold shock
- Low cardiac output states

Norepinephrine

Preferred in:

- Warm vasodilatory shock

Peripheral or IO vasoactive infusions may be started while securing central access in critically ill children.

Antibiotics

Early Broad-Spectrum Antibiotics

Important Principle

Administer as early as possible after cultures if feasible.

Delay in antibiotics increases mortality.

Blood Cultures

Obtain If Possible

BUT:

- Do not delay antibiotics in unstable children.

Glucose Management

Monitor Carefully

Children are prone to:

- Hypoglycemia
- Hyperglycemia

Correct abnormalities promptly.

Electrolyte & Metabolic Abnormalities

Monitor For

- Metabolic acidosis
- Lactate elevation
- Electrolyte disturbances
- AKI

Respiratory Support

Consider HFNC/NIV If

- Moderate respiratory distress

- Hypoxemia

Requires:

- Close monitoring
- Early escalation if deteriorating

Indications for Intubation

Consider Early Intubation If

- Respiratory failure
- Persistent shock
- Altered mental status
- Refractory hypoxemia
- Exhaustion
- Recurrent apnea

Mechanical Ventilation Principles

Goals

- Reduce oxygen consumption
- Improve oxygenation
- Support ventilation
- Optimize hemodynamics

Avoid:

- Hypoxia
- Hyperventilation
- Hypotension

Investigations

Recommended

- CBC
- Blood glucose
- Electrolytes
- Renal function
- Lactate
- Blood cultures
- ABG/VBG
- Chest X-ray
- Urine studies

Monitoring

Continuous Monitoring

- Heart rate
- Respiratory rate
- Blood pressure

- SpO_2
- Mental status
- Urine output

Urine Output Goal

Complications

Major Complications

- ARDS
- AKI
- DIC
- Myocardial dysfunction
- Multiorgan failure
- Cardiac arrest

ICU Referral Criteria

Refer Early If

- Fluid-refractory shock
- Need for vasoactive support
- Respiratory failure
- Altered sensorium
- Persistent hypotension
- Multiorgan dysfunction

Disposition

Admission Required For

- Septic shock
- Persistent tachycardia
- Respiratory distress
- Abnormal perfusion
- Altered sensorium
- Significant comorbidity

8. PAEDIATRIC POISONING

Definition

Exposure to toxic substances resulting in harmful physiological effects in children.

Common Causes

1. Household chemicals
2. Medications
3. Kerosene/hydrocarbon ingestion
4. Pesticides

Clinical Features

1. Vomiting
2. Altered sensorium
3. Respiratory distress
4. Seizures
5. Abnormal pupils

Initial Assessment

1. ABCDE assessment
2. Blood glucose
3. ECG
4. Toxicology history

Management

1. Initial Stabilization
2. Airway protection
3. Oxygen therapy
4. IV access
5. Decontamination

Activated charcoal where indicated.

Not recommended in:

1. Hydrocarbon ingestion
2. Corrosive poisoning

Admission Criteria

1. Symptomatic poisoning
2. Altered sensorium
3. Respiratory distress
4. Intentional poisoning
5. Unknown substance ingestion