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The ICU Manual

Previously Known as ICU Manual

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2nd Edition

JAYPEE

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CHAPTER 15

Sepsis and Septic Shock

Prem Kumar, Jaganathan

INTRODUCTION

Sepsis is a major health problem in the intensive care unit (ICU) and it accounts for a predominant cause of morbidity and mortality in ICU and it varies from 10 to 30% and it increases if the patient ends up with multiple organ dysfunction syndrome (MODS). Although the understanding of the pathophysiology and management of sepsis and septic shock has improved in the past decade, the mortality has still not improved despite antimicrobial therapy and hemodynamic support. This chapter discusses the clinical spectrum of sepsis and septic shock from the latest update of surviving sepsis campaign guidelines 2021. The guidelines emphasize that a systematic screening process is vital to the early identification and management of patients with sepsis.

DEFINITIONS AND DIAGNOSIS

- *Sepsis:* Sepsis is life-threatening organ dysfunction caused by a dysregulated host response to infection
- *Septic shock:* It is manifested by circulatory, cellular, and metabolic dysfunction and defined by hypotension requiring vasopressor therapy to maintain mean arterial pressure >65 mm Hg and serum lactate levels >2 mmol/L after appropriate hypovolemia management.
- *Organ failure:* It is now defined if there is a change in where ≥2 sequential, sepsis-related organ failure assessment (SOFA).

Sepsis can present initially with tachycardia, hypotension, tachypnea, and fever. Altered mental status, hypothermia, organ dysfunction, and leukopenia indicate poor prognosis. Hypoxemia, thrombocytopenia, consumptive coagulopathy, acute tubular necrosis, elevated transaminases, hyperglycemia, pneumonia, and intra-abdominal infection are the other manifestations. Previously gram-negative infections were common in patients with sepsis but the trend of infection is hanging toward gram-positive and fungal infections (gram-negative in India). Abdomen and lung are major sources of infection.

Diagnostic Criteria

Systemic inflammatory response syndrome (SIRS): SIRS is the result of infection or noninfection. The current sepsis guidelines have removed this terminology but still the term is being used by many physicians and intensivists.

It is characterized by two or more of the following:

1. Temperature <36°C or >38°C
2. Heart rate >90 beats/min
3. Respiratory rate >20 breaths/minute or partial pressure of carbon dioxide ($PaCO_2$) <32 mm Hg.
4. White blood cell count <4,000/μL or >12,000/μL or 10% immature band forms.

Modified early warning score (MEWS), SIRS, and National Early Warning Score (NEWS) as a single screening tool for sepsis or septic shock is better than Quick Sequential Organ Failure Score (qSOFA). qSOFA or SOFA criteria, NEWS, and MEWS.

Modified early warning score is a simple scoring that contains five physiological parameters namely respiratory rate, systolic blood pressure, heart rate, level of consciousness, and body temperature which is given in **Table 15.1**. It helps in improving the safety and quality of patient care.

National Early Warning Score is a scoring system done at the bedside to identify acutely ill patients, including those with sepsis. It measures six physiological parameters: Respiratory frequency, oxygen saturation, systolic blood pressure, pulse rate, neurological level of consciousness and body temperature which is given in **Table 15.2**.

PATHOGENESIS

Pathogenesis of septic shock is shown in **Flowchart 15.1**. A Venn diagram depicting the spectrum of sepsis is shown in **Figure 15.1**.

Clinical syndromes associated with sepsis:

- Cardiovascular system—congestive cardiac failure, shock due to reduced intravascular volume. Peripheral vasodilatation results in reduced systemic vascular resistance and vasopressor resistance due to the release of nitric oxide (NO). Myocardial depression with systolic and diastolic dysfunction.
- Respiratory system—acute respiratory distress syndrome (ARDS)
- Central nervous system—septic encephalopathy
- Kidney—acute tubular necrosis
- Gastrointestinal tract (GIT)—paralytic ileus, intestinal ischemia, upper GI bleeding, gastric stress ulcers, and intestinal mucosal injury
- Hematology—disseminated intravascular coagulation (DIC), thrombocytopenia, and leukopenia.

TABLE 15.1: Modified Early Warning Score (MEWS).

	3	*2*	*1*	*0*	*1*	*2*	*3*
Systolic blood pressure (mm Hg)	<70	70–80	81–100	101–199		>200	
Heart rate (BPM)		<40	40–50	51–100	101–110	111-129	>130
Respiratory rate (BPM)		<9		9–14	15–20	21–29	>30
Temperature (°C)		<35		35–38.4		>38.5	
AVPU score				Alert	Reacting to voice	Reacting to pain	Unresponsive

TABLE 15.2: National Early Warning Score (NEWS).

Physiological parameters	*3*	*2*	*1*	*0*	*1*	*2*	*3*
Respiration rate	<8		9–11	12–20		21–24	25
Oxygen saturations	<91	92–93	94–95	>96			
Any supplemental		Yes					
Temperature	<35.0		35.1–36.0	36.1–38.0	38.1–39.0	>39.1	
Systolic BP	<90	91–100	101–110	111–219			>220
Heart Rate	<40		41–50	51–90	91–110	111–130	>131
Level of Consciousness				A			V, P, or U

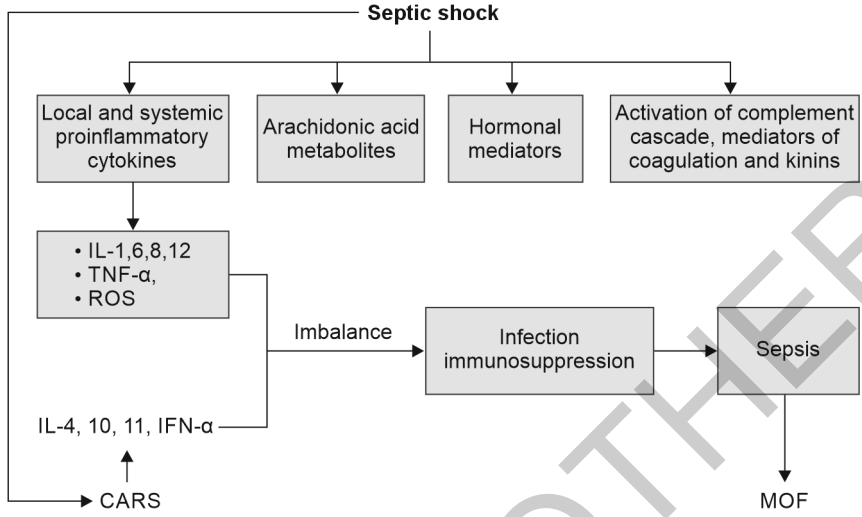
- Neuromuscular—critical illness polyneuropathy and myopathy.
- Hepatic—intrahepatic cholestasis, hepatic failure.
- Endocrine—hyperglycemia from insulin resistance, hypertriglyceridemia, hypoalbuminemia, weight loss, and hypercatabolism.
- Immune—pyrexia, nosocomial pneumonia, and neutrophil dysfunction.

Management of Septic Shock

The three areas to be optimized in sepsis and septic shock are:

1. Trigger
2. Amplification cascade
3. Organ dysfunction

Flowchart 15.1: Pathogenesis of septic shock.



(CARS: compensatory anti-inflammatory response syndrome; IFN: interferon; IL: interleukin; MOF: multiple organ failure; ROS: reactive oxygen species; TNF: tumor necrosis factor)

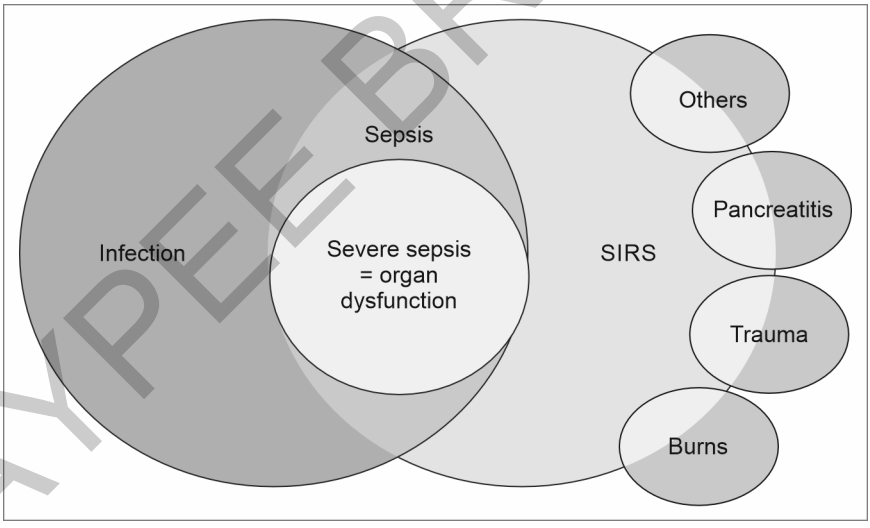


Fig. 15.1: Venn diagram depicting the spectrum of sepsis. (SIRS: systemic inflammatory response syndrome)

Trigger is usually infection, and it is treated by antimicrobial therapy. Amplification is managed by administering activated protein C which exerts anti-inflammatory and antithrombotic effects and reduces the progression of sepsis. Organ dysfunction is treated by supportive measures, such as fluid resuscitation and vasopressors/inotropes.

The management of sepsis and septic shock is done by the following protocol:

- *Measuring blood lactate:*
  - Initial resuscitation
- Mean arterial pressure target
  - ICU admission
  - Screening of sepsis
  - Antimicrobial therapy
  - Source control
  - *Hemodynamic support:*
    - Fluid resuscitation
    - Vasopressors/inotropes
  - Adjunctive therapy
  - Corticosteroids
  - *Supportive therapy:*
    - Blood component administration
    - Immunoglobulins
    - Selenium
    - Recombinant activated protein C (APC)
    - Ventilatory management of ARDS
    - Sedation, analgesia, and neuromuscular blockade
    - Glycemic control
    - Renal replacement therapy
    - Bicarbonate therapy
    - Deep venous thrombosis (DVT) prophylaxis
    - Stress ulcer prophylaxis
    - Nutrition

Initial Resuscitation

- Sepsis and septic shock are medical emergencies, and treatment and resuscitation should begin immediately.
- For patients with sepsis-induced hypoperfusion or septic shock, at least 30 mL/kg of intravenous (IV) crystalloid fluid should be given within the first 3 hours of resuscitation.
- Dynamic hemodynamic measures such as pulse pressure variation (PPV), stroke volume variation (SVV), inferior vena cava (IVC) collapsibility, echocardiography, etc., should be used to guide fluid resuscitation over physical examination or static parameters alone.
- Patients with sepsis or septic shock, guiding resuscitation to reduce serum lactate in patients with elevated lactate levels over not using serum lactate.
- Capillary refill time is used as an adjunct to guide resuscitation compared to other measures of perfusion.

Mean Arterial Pressure Target

For adults with septic shock on vasopressors, an initial target mean arterial pressure (MAP) of 65 mm Hg over higher MAP targets is recommended.

Recommendation for Admission to Intensive Care

Patients with sepsis or septic shock who require ICU admission are admitted to the ICU within 6 hours since this has been shown to reduce hospital stay, mortality, and incidence of mechanical ventilation.

Screening and Diagnosis of Sepsis

The critical component in reducing mortality from sepsis-related multiple organ dysfunction is the time taken to diagnose severe sepsis. Hence, early diagnosis of sepsis and early intervention is recommended. A sample of blood should be sent for blood culture early before administering empirical antibiotics. Cultures of other sites such as urine, cerebrospinal fluid (CSF), wounds, respiratory secretions, or other body fluids that may be the source of infection, should also be obtained before antimicrobial therapy. Early laboratory studies and imaging studies should be done to support or confirm the diagnosis. For adults with suspected sepsis or septic shock but unconfirmed infection, continuously reevaluation and search for alternative diagnoses and discontinue empiric antibiotics if an alternative cause of illness is confirmed or strongly suspected.

ANTIMICROBIAL THERAPY

The recommendation is to administer IV antibiotics within the 1st hour of recognition of sepsis or septic shock. Initial empirical antimicrobial therapy should include one or more drugs that have activity against bacterial, viral, and fungal organisms presumed to be the source of infection. The most common pathogens causing septic shock in hospitalized patients are gram-positive bacteria, followed by gram-negative and mixed-bacterial microorganisms. For patients with sepsis with hemodynamic stability, rapid assessment (history and clinical examination, tests—this should be completed within 3 hours of presentation so that a decision about timely antimicrobial therapy can be made) to be done for detecting infectious versus noninfectious causes of acute illness which is given in **Figure 15.2**. Candidiasis and toxic shock syndromes though uncommon should be suspected in certain patients (immunosuppressed or neutropenic state, prior intense antibiotic therapy, or colonization in multiple sites). Procalcitonin level cannot be used as a guide to discontinue empiric antibiotics because the evidence is very little for its routine use in patients with sepsis with a known source of infection whereas it can be used as a guide when there is an unclear source control or unclear

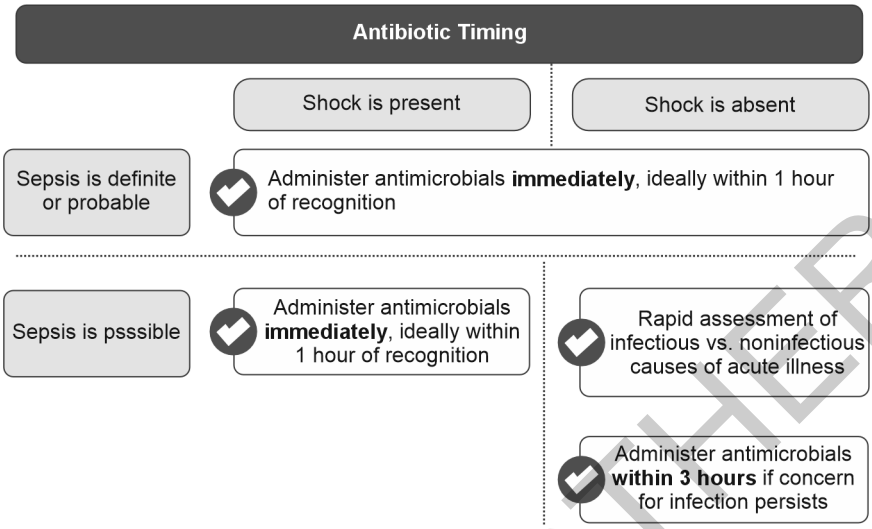


Fig. 15.2: Timing of antibiotics in sepsis.

initial diagnosis. Empiric antibiotic therapy should not be administered for >3–5 days and deescalation should be done as soon as possible. The typical duration of therapy is 7–10 days but may be needed longer in patients with neutropenia, viral, or fungal infections. Antimicrobial therapy should not be used for patients with severe inflammatory states of noninfectious cause. Methicillin-resistant *Staphylococcus aureus* (MRSA) accounts for around 5% of culture-positive infections among critically ill patients. Risk factors for MRSA include recent IV antibiotics, history of recurrent skin infections, history of chronic wounds, previous history of MRSA infection, presence of invasive devices, hemodialysis, recent hospital admissions, and increased disease severity. Risk factors for MDR pathogen include hospital-acquired infection, broad-spectrum antibiotic use within the preceding 90 days, confirmed infection with antibiotic-resistant organisms within the preceding year, local prevalence of antibiotic-resistant organisms, concurrent use selective digestive decontamination (SDD), travel to a highly endemic country within the preceding 90 days. Antibiotics are given as a prolonged extended infusion (antibiotic infused over at least 50% of the dosing interval) of beta-lactams for maintenance (after an initial bolus) over conventional bolus infusion and optimizing dosing strategies of antimicrobials based on accepted pharmacokinetic (PK)/pharmacodynamic (PD) principles and specific properties of the drug. Daily assessment for de-escalation of antimicrobials over using fixed durations of therapy is recommended. The recommendations for antibiotics for specific infections are given in **Table 15.3**.

TABLE 15.3: Recommendation for specific infections.

Infection	*Recommendation*
High risk of methicillin-resistant *Staphylococcus aureus* (MRSA)	Empiric antimicrobials with MRSA coverage
High risk for multidrug-resistant (MDR) organisms	Two antimicrobials with gram-negative coverage for empiric treatment over one gram-negative agent
High risk of fungal infection	Empiric antifungal therapy over no antifungal therapy
Antivirals	Not recommended and specific antivirals can be initiated in case of diagnosis
Specific antibiotic recommendations	• Severe sepsis with neutropenia and multidrug-resistant organisms such as *Acinetobacter* and *Pseudomonas*—combination empirical therapy • Severe infection with respiratory failure and septic shock—combination therapy with an extended spectrum beta-lactam and aminoglycoside/fluoroquinolone for *P. aeruginosa* bacteremia • *Streptococcus pneumoniae* with septic shock—combination of beta-lactam and macrolide • IV drug abusers—vancomycin • In the case of neutropenic patients, empirical antifungal agents can be started empirically if neutropenia persists for >5 days • Empirical antifungal drugs—amphotericin B is used for all life-threatening fungal infections. Fluconazole or echinocandins is recommended by recent Infectious Diseases Society of America (IDSA) guidelines for severe candidiasis. Echinocandins are used for severe illness or when the patient is recently treated with antifungal agents • Severe sepsis with neutropenia with suspected *Pseudomonas* infection—ceftazidime or piperacillin–tazobactam or meropenem + aminoglycoside

Source Control

Rapid identification and exclusion of a specific anatomical diagnosis of infection that requires immediate source control and implementation of required source control intervention is done as soon as possible. Prompt removal of intravascular access devices which can be a possible source of sepsis has been established.

HEMODYNAMIC SUPPORT

Fluid Therapy

Crystalloids are the initial fluid of choice to be used in the resuscitation of severe sepsis and septic shock. Colloids are not recommended. Albumin has a role when patients require substantial amounts of crystalloids. An initial fluid challenge of 30 mL/kg of crystalloids (preferably balanced salt solutions rather than normal saline) is administered in patients with septic shock when there is suspicion of hypovolemia and the fluid challenge is continued until the patient has improvement in hemodynamics as indicated by dynamic parameters such as pulse pressure variation (PPV), stroke volume variation (SVV) and static parameters, such as arterial blood pressure, central venous pressure (CVP), and heart rate. The former guidelines which recommended early goal-directed therapy is no longer followed because 3 trials [Protocolized Care for Early Septic Shock (ProCESS) trial], (Australasian Resuscitation in Sepsis Evaluation [ARISE] trial), and [Protocolized Management in Sepsis (ProMISe) trial] has challenged early goal-directed therapy demonstrating that early goal-directed therapy (EGDT) did not lead to an improvement in outcome. These studies demonstrated that there was no significant difference in mortality at 90 days among those receiving 6 hours of EGDT and those receiving usual resuscitation. The ongoing Crystalloid Liberal or Vasopressors Early Resuscitation in Sepsis (CLOVERS) trial and the Conservative versus liberal fluid therapy in septic shock (CLASSIC) trial is not in favor of any fluid strategy. Current evidence suggests no evidence of recommending liberal or restrictive fluid strategy in the first 24 hours of septic shock or sepsis who have signs of hypovolemia or hypoperfusion.

Vasopressors/Inotropes

All patients requiring vasopressors or inotropes should have an invasive arterial blood pressure monitoring. Norepinephrine is the first-choice vasopressor in septic shock and the target mean arterial pressure is 65 mm Hg. Dopamine is an alternative to norepinephrine. Epinephrine can be added if an additional vasopressor is required and low-dose dopamine for renal vasodilatation is not recommended. Vasopressin and phenylephrine are not recommended as the first choice but can be used for refractory cases of septic shock. The vasopressin dose is 0.03 U/min. Phenylephrine is used when norepinephrine is associated with serious arrhythmias, persistently low blood pressure with high cardiac output, or as salvage therapy when combined inotrope/vasopressor drugs and low-dose vasopressin have failed to achieve the target MAP. In the presence of myocardial dysfunction and low cardiac output or ongoing signs of hypoperfusion despite achieving adequate intravascular volume and adequate MAP, dobutamine infusion of 20 μg/kg/min or epinephrine infusion of 0.01–0.1 μg/kg/minute is started.

TABLE 15.4: Recommendation for vasopressors.

Use norepinephrine as a first-line vasopressor	If central access is not available, consider initiating vasopressors peripherally	If cardiac dysfunction with persistent hypoperfusion is present despite adequate volume status and blood pressure, consider adding dobutamine or switching to epinephrine
For patients with septic shock on vasopressor target a MAP of 65 mm Hg	If MAP is inadequate despite a low to moderate dose of norepinephrine, consider adding vasopressin	
Consider invasive monitoring of arterial blood pressure		

For adults with sepsis and septic shock and cardiac dysfunction with persistent hypoperfusion despite adequate volume status and arterial blood pressure, levosimendan is not recommended. Recommendations for vasopressors are given in **Table 15.4**.

Role of Steroids

Intravenous hydrocortisone is not routinely used to treat patients with septic shock if hemodynamic stability is achieved with fluids and vasopressors. If this is not possible, IV hydrocortisone is used as a continuous infusion of 200 mg/day given as 50 mg intravenously every 6 hours or as a continuous infusion. It is started at least 4 hours after initiation when the dose of norepinephrine or epinephrine exceeds >0.25 μg/kg/min. Corticosteroids should not be administered for the treatment of sepsis in the absence of shock and an adrenocorticotropic hormone (ACTH) stimulation test is done to identify patients who require steroids. Hydrocortisone infusion is tapered when vasopressors are not required.

Ventilation in Patients with Sepsis-induced Respiratory Failure

A tidal volume of 6 mL/kg predicted body weight is initiated in sepsis-induced respiratory failure with or without acute respiratory distress syndrome (ARDS) and the plateau pressure is limited to ≤30 cmH_2O. Positive end-expiratory pressure (PEEP) is applied to prevent alveolar collapse at end-expiration and higher PEEP is required in case of hypoxemia not responding to low PEEP in case of moderate-to-severe sepsis-induced ARDS. Conservative oxygen targets (defined as PaO_2 55–70 mm Hg;

SpO_2 88–92%) have insufficient evidence, hence not recommended in sepsis-induced type 1 respiratory failure, and high flow nasal oxygen is preferred over noninvasive ventilation and there is no preference for noninvasive ventilation over invasive ventilation in type 1 respiratory failure. Head end elevation of the head of 30–45° is given to prevent aspiration and the development of ventilator-associated pneumonia (VAP). Recruitment maneuver (traditional recruitment maneuvers are preferred overincremental PEEP titration/strategy) and prone positioning (>12 hours daily) is used in moderate-to-severe refractory hypoxemia and when PaO_2/FiO_2 ratio is ≤100 mm Hg. A weaning protocol is recommended with frequent spontaneous breath trials in patients who meet weaning criteria and are extubated if fit for extubation. Noninvasive ventilation can be considered in specific groups of ARDS patients. SSC guidelines do not recommend the routine use of pulmonary artery catheter (PAC) and beta-2 agonists in ARDS patients unless there are specific indications.

EXTRACORPOREAL MEMBRANE OXYGENATION

If there is adequate infrastructure in the hospital with sufficient experience, venovenous extracorporeal membrane oxygenation (ECMO) is used when conventional mechanical ventilation fails for adults with sepsis-induced severe ARDS.

Supportive Therapy

Blood Component Administration

After resolution of tissue hypoperfusion packed RBC transfusion is recommended when Hb is <7 g/dL in the absence of cardiac disease, acute hemorrhage, or severe hypoxemia, and the target Hb concentration is 7–9 g/dL. The use of erythropoietin and antithrombin is not recommended in severe sepsis and septic shock. Fresh frozen plasma is not used in clotting disorders in the absence of bleeding. In patients with severe sepsis, prophylactic platelets are administered when counts are <10,000/mm^3 (10×10^9/L) in the absence of apparent bleeding. Prophylactic platelet transfusion when counts are <20,000/mm^3 (20×10^9/L) if the patient has a significant risk of bleeding. Higher platelet counts [≥50,000/mm^3 (50×10^9/L)] are taken as cut-off for active bleeding, surgery, or invasive procedures.

Immunoglobulins

They are not recommended for severe sepsis and septic shock.

Recombinant Activated Protein C

The first ever trial showing the role of recombinant human activated protein C (rhAPC) in severe sepsis was the PROWESS (Recombinant Human-activated

Protein C Worldwide Evaluation in Severe Sepsis) trial in 2001 which demonstrated reduction in mortality but the PROWESS SHOCK trial done in 2011, showed no benefit of rhAPC in patients with septic shock and hence not recommended in severe sepsis and septic shock.

Sedation, Analgesia, and Neuromuscular Blockade

Sedation is minimized in patients put on mechanical ventilation and neuromuscular blocking agents are avoided in patients without ARDS and if used, it should be used with train-of-four monitoring. In patients with ARDS, neuromuscular blocking agents are not used for >48 hours and. intermittent boluses are preferred over continuous infusion.

Glycemic Control

Blood glucose should be managed by a protocol in patients with severe sepsis and insulin is started when two consecutive blood glucose levels are >180 mg/dL with a target of 144–180 mg/dL and blood glucose should be monitored every 1–2 hours until glucose values and insulin infusion rates are stable and then every 4 hours thereafter. Point-of-care monitors should be interpreted with caution because of their unreliability. Treatment should avoid hyperglycemia (>180 mg/dL), hypoglycemia, and wide swings in glucose levels. Many studies have shown that the variability in glucose levels over time is an important determinant of mortality.

Renal Replacement Therapy

There is no mortality benefit between continuous renal replacement therapy (CRRT) and intermittent hemodialysis in patients with severe sepsis and acute renal failure. CRRT is used in hemodynamically unstable sepsis patients for fluid balance.

Bicarbonate Therapy

Sodium bicarbonate therapy is not recommended for improving hemodynamics or reducing vasopressor requirements in patients with tissue hypoperfusion-induced lactic acidosis with pH ≥7.2. It is indicated in severe metabolic acidosis (pH ≤7.2) and AKI (AKIN score 2 or 3).

Goals of Care

The goals of care and long-term outcomes are outlined in **Table 15.5**.

Deep Vein Thrombosis Prophylaxis

Intensive care unit patients are at risk for deep vein thrombosis and hence patients with severe sepsis are recommended daily pharmacoprophylaxis

TABLE 15.5: Goals of care and long-term outcomes.

Transition of care	Handoff process of critically important information
Economic or social support screening	Screening for economic and social support is done, and referral is applied, wherever it is appropriate
Sepsis education for patients and families	• Written document • Verbal sepsis education (diagnosis, treatment, and postintensive care unit (ICU)/postsepsis syndrome) prior to hospital discharge
Decision making	Clinical team should create opportunities in the following areas: • Participation in shared decision making • Hospital discharge planning
Discharge planning	• Critical care transition program • Reconciling medications • Information about ICU stay, sepsis and related diagnosis, treatments, and common impairments after sepsis • Follow-up with clinicians for patients with new impairment
Postdischarge follow-up	• Assessment and follow-up for physical, cognitive, and emotional problems after discharge • For survivors of septic shock. Postcritical illness follow-up clinic referral • For survivors, mechanical ventilation for >2 days or ICU stay of >3 days, referral to a posthospital rehabilitation clinic

against venous thromboembolism (VTE). Daily subcutaneous low-molecular-weight heparin (LMWH) which is preferred or twice daily unfractionated heparin (UFH) or thrice daily UFH is given. If creatinine clearance is <30 mL/min, dalteparin or UFH is used. A combination of heparin and intermittent pneumatic compression device is better than heparin alone. In patients where heparin is contraindicated, graduated compression stockings or intermittent compression devices is used.

Stress Ulcer Prophylaxis

H_2 blocker or proton pump inhibitor is given to patients with severe sepsis/septic shock who have risk factors for bleeding. Patients without risk factors for bleeding are not given prophylaxis routinely.

Nutrition

Once the patient is diagnosed with severe sepsis or septic shock, oral or enteral feeding are started within 72 hours in low-dose feeding rather than full caloric feeding in the first week and the patient should not be left with fasting or only given IV glucose. Underfeeding (60–70% of target) or trophic feeding (upper limit of 500 kcal) is probably a better nutritional strategy

in the first week of severe sepsis/septic shock. The recommendation is to use IV glucose and enteral nutrition rather than total parenteral nutrition (TPN) alone or parenteral nutrition in conjunction with enteral feeding in the first week. The use of immunomodulators such as arginine, glutamine, and omega-3 fatty acids supplementation with enteral nutrition is not advised by the Surviving Sepsis Campaign (SSC) 2021 guidelines.

CONCLUSION

Sepsis is a life-threatening condition and very much dependent on time for many interventions. Although there are many advances in the management, this condition still has poor prognosis. Early diagnosis and multidisciplinary management with different therapeutic aspects remains the mainstay in the management of sepsis.

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