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Multiple Organ Dysfunction Syndrome: A Review

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Abstract: ***Multiple Organ Dysfunction Syndrome (MODS) is a fatal condition characterised by the progressive failure of two or more organs. MODS is a process rather than an event. This article elaborates on overview on the pathophysiology, diagnosis and management of patients with MODS***

Keywords: Multiple Organ failure, Multiple System Organ Failure, Multiple Organ System Failure, Multiple Organ Dysfunction Syndrome (MODS), Septic Shock, SIRS, Sepsis

1. Introduction

Multiple organ dysfunction syndrome (MODS) is a process rather than one event and it can start from Systemic Inflammatory Response Syndrome, Sepsis, Septic Shock leading to MODS[1]. It is defined as a "clinical syndrome characterized by the development of progressive and potentially reversible physiologic dysfunction in 2 or more organs or organ systems that is induced by a variety of acute insults, including sepsis"[2]. It results from progressive physiologic failure of two or more separate organ systems in critically ill patient that require organ support to maintain balance with in the body[3].

Terminology

The following are some of the terminology explained related to MODS. The American College of Chest Physicians/Society of Critical Care Medicine Consensus Panel Guidelines formulated the following criteria[2, 4]. The criteria for SIRS if the patient has two or more of the following such as Temperature > 38 C or < 36 C, Heart rate > 90/min, RR > 20/min, WBC Count > 12000/µL or < 4000/µL or 10% immature forms (bands) [2, 4]. The sepsis is termed as SIRS along with a culture documented infection[2, 4]. The septic shock is defined as a Hypotension despite fluid resuscitation and hypoperfusion[2, 4]. Patient require vasopressor to maintain MAP of 65 mm of Hg or greater and serum lactate level greater than 2 mmol/L in the absence of hypovolemia[2, 4].

Epidemiology

Every 1000 hospitalized patient, an estimated 15 patients will develop sepsis acquired from the hospital[5]. According to CDC report, 1.7 million adults in USA develop sepsis of which atleast 3, 50, 000 die[6].

Risk factors

The following are the risk factors for the patient to develop MODS. They are age more than 65 years old, trauma, Hemorrhage, Sepsis, Shock, Acute pancreatitis, Burns, Aspiration Multiple blood transfusions, Surgical complications[2, 3].

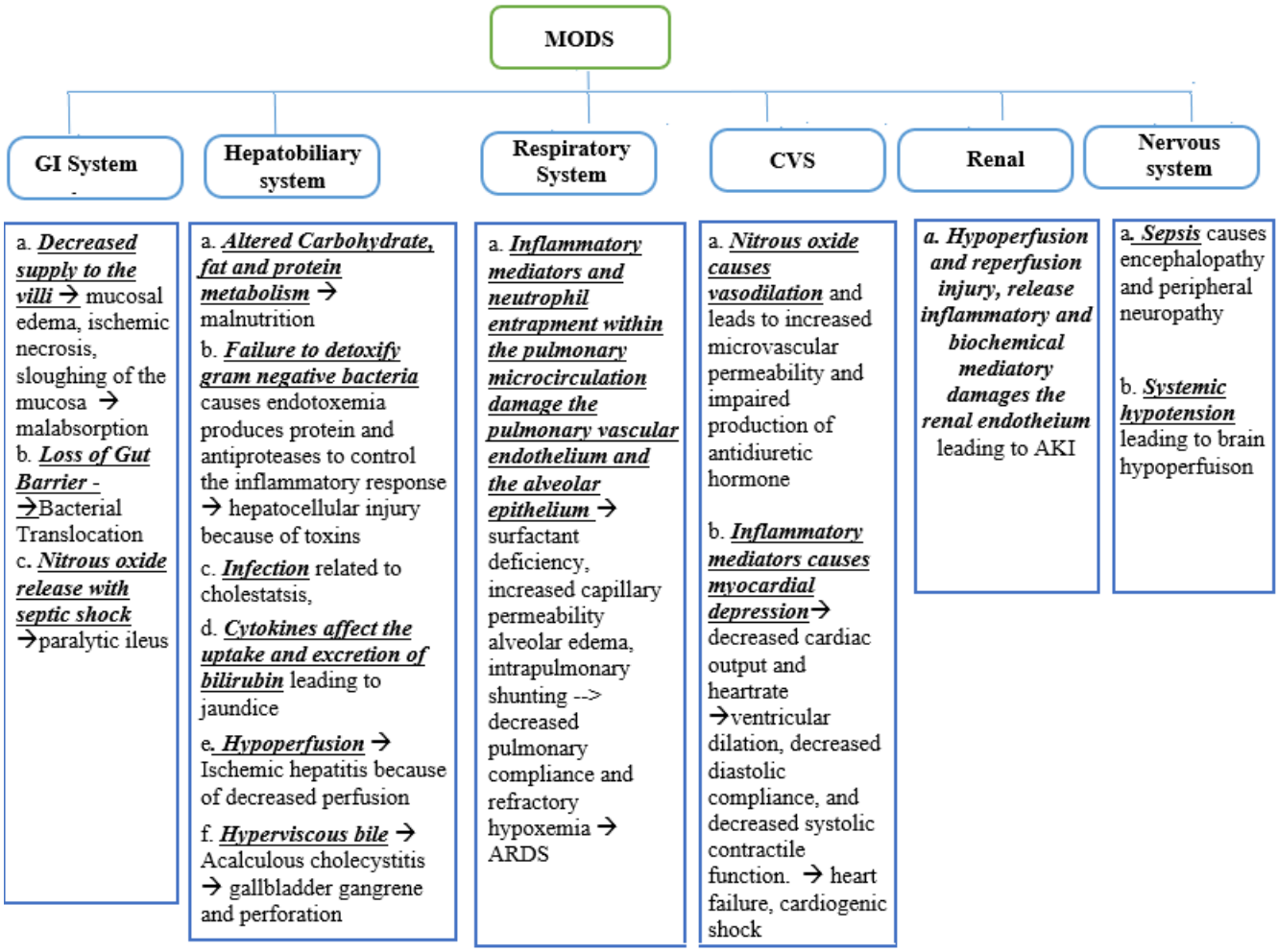
Types of MODS

MODS has two types. They are Primary and Secondary. Primary MODS is well defined insult to the organ in which organ dysfunction occurs. The patient diagnosed with AKI or pulmonary failure are the examples of primary MODS. Secondary MODS is a complication of extensive sustained systemic inflammation and sepsis is a common condition leading to it[2, 3].

Pathophysiology[2, 3]

the various inflammatory cells and biochemical mediators or plasma protein systems are produced by the body in response to SIRS and Sepsis. These inflammatory cells and biochemical mediators damages the tissues, epithelial cells, blood vessels leading to increased capillary wall permeability, formation of thrombus, bleeding in various organs, ischemia and hypoxia.

Inflammatory cells	Biochemical mediators
Neutrophils	Tumor Necrosis Factor - Alpha
Monocytes	Arachidonic Acid Metabolites
Macrophages	Platelet Activating Factor
Mast cells	Reactive Oxygen Species
Lymphocytes	Interleukin 1, 2
	Proteases



Clinical Manifestations[2, 3]

The clinical manifestations may differ depend on type and site of organ injury and source of infection. The patient will present with a signs of inflammation or infection or shock. They are fever, chills, perspiration, tachycardia, tachypnea/hyperventilation, altered mental status may vary from mild disorientation or confusion, apprehension, anxiety and agitation, warm extremities, and normal Capillary Refill Time in an initial phase of shock and in later phase when compensatory mechanisms fail, the patient will manifest with the following signs cool skin and clammy extremities, delayed CRT.

GI- Stress ulcers, malnutrition
HPB- vague right upper quadrant pain and tenderness, abdominal distention, unexplained fever, loss of bowel sounds
RS- low grade fever, tachycardia, dyspnea, and mental confusion, hypoxemia
CVS & Hematologic- Bleeding, thrombus/emboli
Renal- oliguria, anuria, increased creatinine level, prerenal azotemia.
Nervous system- Confusion, agitation, coma

Investigations[2]

1) CBC will reveal elevated or decreased WBC count
2) ABG will reveal metabolic and lactic acidosis
3) Serum lactate will be elevated because of tissue hypoperfusion
4) Hematologic: Elevated PT, APTT and decreased platelet count.
5) Dyanamic parameters for fluid management – Passive Leg Raising Test, Pulse Pressure Variation or Stroke Volume Variation will be assessed (Medscape)
6) Blood culture should be taken preferably 2 sets prior administration of antibiotic therapy
7) Chest X - ray is performed to identify ARDS
8) Depends on the source of an infection: culture, LP, imaging such as CT abdomen, chest, can be done

Severity of MODS[2]

The following table will give the details of the severity of MODS.

Organ system	Mild Criteria	Severe criteria
Pulmonary	Hypoxia or hypercapnia requiring assisted ventilation for 3 - 5 days	ARDS requiring PEEP >10 cm H2 O and FiO2< 0.5
Hepatic	Bilirubin 2 - 3 mg/dL or other liver function tests >2 × normal, PT elevated to 2 × normal	Jaundice with bilirubin 8 - 10 mg/dL
Renal	Oliguria (< 500 mL/day) or increasing creatinine (2 - 3 mg/dL)	Dialysis
GI	Intolerance of gastric feeding for more than 5 days	Stress ulceration with need for transfusion, acalculous cholecystitis
Hematologic	aPTT >125% of normal, platelets < 50 - 80, 000	DIC

Cardiovascular	Decreased ejection fraction with persistent capillary leak	Coma
CNS	Confusion	Hyperdynamic state not responsive to vasopressors
Peripheral Nervous system	Mild sensory neuropathy	Combined motor and sensory deficit

Medical management

The management includes early recognition, Fluid resuscitation, hemodynamic support, prevention and treatment of infection, maintenance of tissue oxygenation, nutritional and metabolic support, provide comfort and emotional support.

1) Early recognition

- Recognition of SIRS, Septic shock and perform resuscitation accordingly[2].

2) Fluid resuscitation

- The goal is to maintain MAP > > 65 mm Hg, urine output > 0.5 ml/kg/hr, CV or mixed venous oxygen saturation 70% or 65 %[7].
- Administer atleast 30 ml/Kg or IV isotonic crystalloids within the first 3 hours (Medscape - SCCM). Normal saline causes hyperchloremic metabolic acidosis and hence RL or plasmolyte can be recommended[2, 7].
- Albumin can be administered for impending shock[2, 7].

3) Hemodynamic support

- If MAP < 65 mm of Hg and then start on Dopamine/Noradrenaline. Start Noradrenaline at the rate of 0.2 – 1.35 mcg/kg/min to 3.3 mcg/kg/min. Dopamine is also first line of drug if the patient did not have any history of arrhythmias[8, 9].
- Administer vasopressin if the noradrenaline dosage between 0.25 to 0.5 mcg/kg/min. Administer/Start epinephrine if MAP is not maintained with norepinephrine and vasopressin[7]
- Administer IV hydrocort 200mg/day - 50 mg Q6H or as a continuous infusion for 7 days[10] if the patient requires noradrenaline support ≥ 0.25 mcg/kg/min atleast 4 hours after initiation[7] or until no longer requirement of vasopressor support[2]
- Administer soda bicarbonate when pH ≤7.2 and AKI[7]
- Blood transfusion if Hb < 7 g/dl[2]

4) Maintenance of tissue oxygenation

- The main goal is to increase oxygen delivery and decrease oxygen demand. It can be achieved by sedation, neuromuscular blocking agent, mechanical ventilation with PEEP, rest, temperature and pain control, prone position > 12 hours. Set tidal volume as 6 ml/Kg to avoid Ventilator induced lung injury and maintain plateau pressure to 30 cm H2O[2].
- If the patient is conscious and start on High Frequency Nasal Cannula Over Non - invasive Ventilation for Type I Respiratory failure[2].
- Venovenous Extracorporeal membrane oxygenation is indicated if regular mechanical ventilation is failed[2].

5) Nutrition and metabolic support

- The goal is to preserve the organ structure and function.
- Start on enteral nutrition within 72 hours that limits bacterial translocation[2, 3].
- Start on stress ulcer prophylaxis[2].
- Maintain normoglycemia and if hyperglycmeia (RBS > 180 mg/dl) persists start on inslin infusion and the target is to maintain ≤ 110 mg/dl[2, 3].
- Correct any electrolyte deficiency
- Start on dialysis if there is no urine output or if the patient is developing Acute Kidney Injury[2].

6) Prevention and treatment of infection

- The measures need to be taken to prevent further infection such as removing invasive catheters and avoid invasive catheter[7], administering Prophylactic dose of antibiotics during perioperative phase and start on adequate nutrition with enteral nutrition
- Identify infection and administer appropriate antibiotics to be prescribed - Broad spectrum antibiotics[7]. Preferably carbapenem start within 1 hour of suspicion of sepsis[2, 7]
- Surgical procedures such as debridement or incision and drainage can be done to control the source of infection[2, 7]

7) Comfort and emotional support

- Provide comfort and emotional support for the patients and relatives[2].

2. Prognosis

The research highlights that that severity of 28 - day mortality rate ranges from SIRS to sepsis, severe sepsis, and septic shock as approximately 10%, 20%, 20 - 40%, and 40 - 60%, respectively[11].

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