SIRS (Systemic inflammatory response syndrome)

الدكتور مقداد فؤاد
استاذ مساعد

Definitions

SIRS (systemic inflammatory response syndrome) is a systemic inflammatory response characterized by the presence of two or more of the following:

- *Hyperthermia >38°C or hypothermia <36°C*
- *Tachycardia >90 bpm*
- *Tachypnoea >20 b.p.m. or PaCO2 <4.3 kPa*
- *Neutrophilia >12 × 10-9 /L or neutropenia <4 × 10–9 l–1.*

Sepsis syndrome *is a state of SIRS with proven infection.*

Septic shock *is sepsis with systemic shock.*

MODS (*multiple organ dysfunction syndrome*) *is a state of derangement of physiology such that organ function cannot maintain homeostasis.*

The common terminal pathways for organ damage and dysfunction are **vasodilatation**, **capillary leak**, **intravascular coagulation** *and* **endothelial cell activation**.

KEY POINTS

- *SIRS is more common in surgical patients than is diagnosed.*
- *Early treatment of SIRS may reduce the risk of MODS developing.*
- *The role of treatment is to* **eliminate any causative factor** *and* **support the cardiovascular** *and* **respiratory physiology** *until the patient can recover.*
- *Overall mortality is 7% for a diagnosis of SIRS, 14% for sepsis syndrome and 40% for established septic shock.*

Common surgical causes

- *Acute pancreatitis.*
- *Perforated viscus with peritonitis.*
- *Fulminant colitis.*
- *Multiple trauma.*
- *Massive blood transfusion.*
- *Aspiration pneumonia.*
- *Ischaemia reperfusion injury.*

Causation

TNF alpha

• *TNF alpha is both* **released by and activates** *macrophages and neutrophils.*

• *It is* **cytotoxic to endothelial cells and parenchymal cells of end organs**. *There is no clear evidence that anti-TNF alpha therapy is effective in SIRS.*

Lipopolysaccharide (LPS)

• *Released from* **Gram −ve bacterial cell walls**, *activates macrophages via attachment of LPS binding protein and activation of CD14 molecules on the cell surface. There is no proven value for anti-LPS antibody treatment.*

Interleukines

• **IL-6 and IL-1α cause endothelial cell activation and damage**. *They promote complement and chemokines release.*

Platelet activating factor (PAF)

• *This is implicated particularly in acute pancreatitis, no proven role for anti-PAF antibody treatment.*

Inducible nitric oxide synthetase (INOS)

• *This is synthesized by activated endothelial cells, activates endothelial cells and leucocytes, potent negative ionotrope.*

Treatment

- *Aggressive hemodynamic resuscitation of septic patients that is aimed at achieving specific physiologic targets. :*

1. *Central venous pressure, 8 to 12 mm Hg; The goal for central venous pressure in patients who are mechanically ventilated or who have increased abdominal pressure is between 12 and 15 mm Hg.*
2. *Mean arterial pressure, greater than 65 mm Hg;*
3. *Urine output, greater than 0.5 mL/kg/hr;*
4. *Central venous oxygen saturation (Scvo2; superior vena cava), greater than 70%.*
5. *High-dose intravenous steroids have little role in established SIRS (probably because of multiple pathways of activation). Steroids for early SIRS are unproven.*

Mean arterial pressure **MAP**

MAP *= [(2 x (diastolic) + systolic] divided by 4*

- *is considered to be the perfusion* **pressure** *seen by organs in the body.*
- *It is believed that* **MAP** *which is greater than 70 mmHg is enough to sustain the organs of the* **average** *person.*
- *Is normally between 65 and 110 mmHg.*
- **MAP** *may be used similarly to Systolic* **blood pressure** *in monitoring and treating target* **blood pressure***.*

The end