

SIRS (Systemic inflammatory response syndrome)

Definitions

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

- Hyperthermia $>38^{\circ}\text{C}$ or hypothermia $<36^{\circ}\text{C}$
- Tachycardia >90 bpm
- Tachypnoea >20 r.p.m. or $\text{PaCO}_2 <4.3$ kPa
- Neutrophilia $>12 \times 10^9 \text{ l}^{-1}$ or neutropenia $<4 \times 10^9 \text{ l}^{-1}$.

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-1 α 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 inotrope.

Treatment

Early goal-directed therapy consists of aggressive hemodynamic support in the resuscitation of septic patients that is aimed at achieving specific physiologic targets. : central venous pressure, 8 to 12 mm Hg; mean arterial pressure, greater than 65 mm Hg; urine output, greater than 0.5 mL/kg/hr; central venous oxygen saturation (Scvo₂; superior vena cava), greater than 70%. 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.

High-dose intravenous steroids have little role in established SIRS (probably because of multiple pathways of activation). Steroids for early SIRS are unproven.

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