Systemic inflammatory response after cardiac surgery: Is extracorporeal circulation the main culprit?

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Introduction

Cardiac surgery with extracorporeal circulation (ECC) is associated with a systemic inflammatory response [28, 53]. Kirklin and coworkers described this phenomenon as early as 1981 and coined the term postperfusion syndrome [13]. In the majority of cases, the organism can fully compensate for this systemic inflammatory response syndrome (SIRS). But any postoperative exacerbation will increase morbidity and mortality. Surgical trauma, ischemia/reperfusion, endotoxinemia, and cellular activation induced by shear-stress and by the foreign-surfaces of the heart-lung machine are pathophysiological mechanisms that activate the systemic inflammatory response (Fig. 1).

One of the proposed advantages of off-pump coronary bypass surgery (OPCAB) is that some of these triggers for inflammation are avoided, especially global ischemia/reperfusion and extracorporeal circulation. But clinical signs of a systemic inflammatory response can also be observed after off-pump operations, i.e., fever, decreased vascular resistance, and hypotension. In parallel, inflammatory markers increase postoperatively, e.g., C-reactive protein, lipopolysaccharide binding protein (LBP), and procalcitonin [4, 21]. The following literature review compares the SIRS-related mediator systems in on-pump versus off-pump cardiac surgery.
The complement system is a cascade system, and the cascade can be activated via the classical or the alternate pathway. The anaphylatoxins C3a and C5a are split products of the complement cascade. They are potent leukocyte and platelet activators, raise vascular permeability, cause vasodilatation, and the release of histamine, oxygen-free radicals, and lysosomal enzymes. The terminal complement complex C5b-9 is also called the membrane attack complex. It activates leucocytes and endothelial cells just like the anaphylatoxins C3a and C5a do. Stud-