Inflammation and thoracic surgery: a complex interaction


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Letter to the Editor

Inflammation and thoracic surgery: a complex interaction

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We appreciated the reading of the work of Amar et al. [1]. The authors reported data suggesting that markers of inflammation such as C-reactive protein (CRP) and IL-6 can help to classify patients who are at high risk for major postoperative complications (PC) following thoracic surgery. Systemic inflammation is thus considered to be important in the pathogenesis of important PC. Defining the precise inflammatory response represents a sensitive issue, frequently debated within scientific community. Plasma levels of cytokines or CRP are probably not sufficient to determine whether a patient or experimental animal is hyper-inflammatory or hypo-inflammatory. Conversely, if only cytokine antagonists or anti-inflammatory mediators are measured, then the patient will appear hyper-inflammatory. Conversely, if only cytokine antagonists or anti-inflammatory mediators are measured, the subject appears to be hypo-inflammatory. Indeed, both pro- and anti-inflammatory mediators may be circulating at the same time in the plasma [2]. Better methods for determining the precise immunological status may be achieved via either a multiplex format for cytokine measurements or an evaluation of cellular function. Many cellular aspects become dysfunctional in inflammation, for example in chronic obstructive pulmonary disease (COPD), and may be characterized as either excessive activation or depressed function. Excessive activation refers to cells that are primed so that they respond in a very vigorous manner to a second stimulus (for example, neutrophils generating excess toxic products that cause damage to nearby cells [3]). Conversely, an example of depressed function would be neutrophils’ failure to phagocytize any clearly invading pathogens. One of the current areas of active investigation concerning cellular function is the induction of cellular apoptosis or necrosis. The signalling mechanisms and molecules that induce apoptosis are currently being described in great detail by several investigators. The pathobiology of COPD (a disease that can be considered a model of pulmonary inflammation) encompasses multiple injurious processes including inflammation (excessive or inappropriate innate and adaptive immunity), cellular apoptosis, altered cellular and molecular alveolar maintenance program, abnormal cell repair, extracellular matrix destruction (protease and anti-protease imbalance), and oxidative stress (oxidant and antioxidant imbalance) [4]. Apoptosis may contribute to the pathogenesis of sepsis by the delayed removal of those cells that should be removed, i.e., neutrophils, and early removal of those cells that should not be removed, i.e., lymphocytes. Activated protein C has emerged as a novel therapeutic agent for use in selected patients with severe sepsis, even though the mechanism of its benefit is not well established. It has anticoagulant, anti-inflammatory, antiapoptotic, and pro-fibrinolytic properties, but it is not clear through which of these mechanisms APC exerts its benefit in severe sepsis. Focus has recently turned to the role of APC in maintaining endothelial barrier function, and in vitro and in vivo studies have examined this relationship [5].

In conclusion, CRP and IL-6 alterations are extremely non-specific, and an accurate diagnostic test for sepsis would be a welcome addition in the management of specific patients undergoing thoracic surgery, whereas the condition supporting the surgical indication could easily be correlated with a previous pulmonary inflammation, i.e., lung cancer arising in COPD patients.

References


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Reply to the Letter to the Editor

Reply to Paleari et al.

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