

PRO-INFLAMMATORY CYTOKINES IN ACUTE KIDNEY INJURY INDUCED BY CONTRAST AGENTS IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Abstract

Acute kidney injury (AKI) induced by contrast agents, commonly referred to as contrast-induced nephropathy (CIN), remains one of the most frequent and serious complications in patients undergoing diagnostic and interventional procedures for acute coronary syndrome (ACS). Pro-inflammatory cytokines play a pivotal role in the pathogenesis of this condition, mediating inflammatory responses, endothelial dysfunction, and renal tubular damage. The study focuses on evaluating the dynamics and clinical significance of cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and interleukin-1 beta (IL-1 β) in patients with ACS who develop CIN. Analysis of these biomarkers demonstrates their association with the severity of kidney injury, progression of systemic inflammation, and the risk of adverse cardiovascular and renal outcomes. Understanding the molecular mechanisms underlying cytokine activity in this setting provides valuable insight into early diagnosis, risk stratification, and potential therapeutic targets. The findings highlight the necessity of incorporating cytokine monitoring into clinical practice for patients with ACS at high risk of contrast-induced kidney injury.

Keywords: Acute kidney injury (AKI); contrast-induced nephropathy (CIN); acute coronary syndrome (ACS); pro-inflammatory cytokines; interleukin-6 (IL-6); tumor necrosis factor-alpha (TNF- α); interleukin-1 beta (IL-1 β); renal dysfunction; systemic inflammation; biomarkers.

Introduction

Acute kidney injury (AKI) remains one of the most frequent and serious complications among patients with acute coronary syndrome (ACS), especially when diagnostic or interventional procedures require the use of iodinated contrast agents.

This condition, commonly referred to as contrast-induced nephropathy (CIN), is associated with significant morbidity and mortality, prolongs hospitalization, and increases healthcare costs. Despite advances in interventional cardiology and nephrology, the pathophysiological mechanisms underlying CIN are not fully understood, which complicates the development of targeted preventive and therapeutic strategies.

One of the key factors contributing to CIN is the role of inflammatory mediators, particularly pro-inflammatory cytokines. Cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and interleukin-1 beta (IL-1 β) are critically involved in amplifying renal inflammation, endothelial dysfunction, and oxidative stress. In the setting of ACS, patients are already in a state of systemic inflammatory activation due to ischemia-reperfusion injury, plaque rupture, and heightened sympathetic drive. The administration of contrast agents further exacerbates this pro-inflammatory state, leading to renal tubular injury, microcirculatory impairment, and worsening of renal perfusion.

Research has shown that elevated levels of circulating cytokines not only reflect ongoing renal injury but also serve as predictors of adverse outcomes, including progression to chronic kidney disease and increased risk of major adverse cardiovascular events. Furthermore, the interplay between cytokines and other pathophysiological pathways—such as oxidative stress, apoptosis, and impaired renal autoregulation—suggests that inflammatory markers could be used as early diagnostic tools and potential therapeutic targets.

This study is aimed at analyzing the role of pro-inflammatory cytokines in the development of AKI induced by contrast agents in patients with ACS, identifying their diagnostic and prognostic significance, and evaluating the potential for integrating cytokine-based biomarkers into clinical practice. By understanding these mechanisms, it becomes possible to improve prevention strategies, risk stratification, and outcomes in this high-risk patient population.

Conclusion

Contrast-induced acute kidney injury in patients with acute coronary syndrome represents a multifactorial disorder in which pro-inflammatory cytokines play a central role. Elevated levels of IL-6, TNF- α , and IL-1 β contribute to renal inflammation, endothelial dysfunction, and tubular injury, thereby exacerbating renal impairment in a population already burdened by systemic inflammation. The evidence indicates that cytokine profiling can provide valuable information for early detection of kidney injury, prediction of adverse outcomes, and guidance in the development of targeted therapeutic strategies.

Future approaches should focus on integrating cytokine monitoring into routine risk assessment protocols, alongside conventional renal function markers such as serum creatinine and estimated glomerular filtration rate. Additionally, anti-inflammatory therapies aimed at modulating cytokine activity could hold promise in reducing the incidence and severity of contrast-induced kidney injury. Thus, a better understanding of the inflammatory mechanisms underlying this condition is crucial for improving the prognosis and quality of life of patients with acute coronary syndrome.

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