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Application of systemic inflammation score for the assessment of contrast-induced acute kidney injury; a review

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ABSTRACT

Contrast-related acute renal failure is a multifactorial condition that involves oxidative stress, inflammation, and direct tubular toxicity. Risk factors for contrast-induced nephropathy comprise pre-existing kidney dysfunction, diabetes mellitus, advanced age, congestive heart failure, hypotension, anemia, and volume depletion. Preventive measures include identifying high-risk patients and implementing preventive measures such as adequate hydration, minimizing contrast use, and avoiding using contrast media in patients with pre-existing renal dysfunction. The systemic inflammation score is a promising tool for predicting contrast-associated acute kidney injury (CA-AKI) in patients undergoing contrast-enhanced imaging procedures. Further studies are needed to validate the use of SIS in clinical practice and to better understand the underlying mechanisms of inflammation in (CA-AKI).

Keywords: Contrast-associated acute kidney injury, Acute kidney injury, Contrast media, Systemic inflammation score, Contrast nephropathy, Tubular cytotoxicity, Renal vasoconstriction, Oxidative stress, Renal dysfunction

Implication for health policy/practice/research/medical education:
Contrast-associated acute kidney injury (CA-AKI) is a specific form of acute kidney injury caused by the administration of contrast agents during medical imaging procedures. The morphologic lesions associated with CA-AKI are characterized by tubular injury, interstitial inflammation, and vascular damage. The severity of these lesions is correlated with the degree of renal dysfunction. While CA-AKI shares similarities with other types of acute kidney injury regarding symptoms and treatments, its unique association with contrast agents sets it apart regarding cause, risk factors, and prevention strategies.

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Review

Introduction

Contrast-associated acute kidney injury (CA-AKI) is a common iatrogenic complication encountered in patients undergoing contrast-enhanced imaging procedures. The condition is considered by a sudden decline in kidney function following the administration of contrast agents (1). Although the exact pathophysiological mechanisms of CA-AKI remain incompletely understood, it is thought to involve a combination of direct renal tubular cytotoxicity, renal vasoconstriction, and oxidative stress (2). In this review paper, we discuss the use of systemic inflammation score (SIS) as a predictor of CA-AKI. We also sought to summarize the current state of knowledge regarding novel treatment modalities for CA-AKI.

Search strategy

For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ), and Embase using different keywords including contrast-associated acute kidney injury, acute kidney injury, contrast media, systemic inflammation score, contrast nephropathy, tubular cytotoxicity, renal vasoconstriction, oxidative stress, and renal dysfunction.

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A short look at systemic inflammation score

The SIS is a measure used to assess the level of inflammation in the body. It is calculated by measuring the levels of various inflammatory markers, such as C-reactive protein (CRP), interleukin-6 (IL-6), white blood cell (WBC) count, and erythrocyte sedimentation rate (ESR), and tumor necrosis factor-alpha (TNF-alpha), in the blood (3). The score is calculated by considering various markers of inflammation, as mentioned. Each marker is assigned a specific value based on its level, and these values are then summed up to obtain the overall score. This score is conducted in clinical settings to help diagnose and monitor inflammatory diseases, such as rheumatoid arthritis, inflammatory bowel disease, and sepsis (4,5). The SIS has also been studied in COVID-19 patients, where it was used to assess the inflammatory response and monitor changes in inflammatory indices over time. A higher SIS score indicates a higher level of inflammation in the body, which can indicate disease severity and a predictor of poor outcomes. Hence, this score helps healthcare professionals in diagnosing and monitoring inflammatory conditions. It can also be used to assess the effectiveness of treatments aimed at reducing inflammation (6,7).

Systemic inflammation score for CA-AKI

The pathophysiology of CA-AKI is complex and involves multiple factors, including oxidative stress, inflammation, and vasoconstriction. Systemic inflammation is an important contributor to the development of CA-AKI (6,7). The SIS is a simple and objective tool that measures the degree of systemic inflammation based on the levels of CRP and WBC count. The SIS has been detected to be a reliable predictor of adverse outcomes in various clinical settings, including sepsis, acute pancreatitis, and cardiovascular disease (8).

The mechanisms by which systemic inflammation contributes to CA-AKI are not fully understood. However, it is thought that inflammation leads to endothelial dysfunction and impaired renal blood flow, which can result in renal tubular injury and impaired renal function (9).

The use of SIS as a predictor of CA-AKI has several potential benefits. First, SIS is a simple and inexpensive tool that can be easily incorporated into routine clinical practice. Second, SIS can help identify patients at high risk for CA-AKI, allowing for early intervention and preventing this complication. Third, SIS can be used to monitor the response to treatment and guide further management (9,10).

Recently, Zeng et al studied 5726 patients who underwent elective percutaneous coronary intervention to verify whether SIS can assess contrast nephropathy in a group of cases undergoing percutaneous coronary intervention. This study showed SIS was tightly correlated with contrast nephropathy and long-term mortality in individuals following percutaneous coronary intervention. They concluded on the potential benefits of anti-inflammatory treatments in preventing contrast nephropathy and ameliorating the prognosis of cases undergoing percutaneous coronary intervention (11).

Renal pathology of CA-AKI

The pathophysiology of contrast nephropathy is not fully understood, but it is thought to be a sequence of direct toxic effects, vasoconstriction, hypoxia, and ischemia on renal tubular cells (12,13). Therefore, contrast nephropathy is a complex disorder that is characterized by morphologic lesions and systemic inflammation. Contrast media can cause a straightforward cytotoxic consequence on the kidney's proximal tubular cells. This condition will proceed along with enhancing cellular damage by reactive oxygen species, along with increasing resistance to renal blood flow. Contrast media also exacerbates kidney vasoconstriction, principally in the deeper segments of the outer medulla, which can contribute to deteriorating medullary hypoxemia and kidney vasoconstriction in cases that are previously volume-depleted (14,15).

Direct effects

Contrast media can directly damage the renal tubular cells. This is thought to occur through oxidative stress and direct cytotoxic effects. The damage can lead to tubular cell death, detachment, and dysfunction (16,17). Moreover, contrast media can cause vasoconstriction of the renal blood vessels, including the afferent arterioles. This reduces renal blood flow and impairs oxygen delivery to the renal tubules, leading to ischemic injury (18,19).

Indirect effects

Contrast media can induce the production of reactive oxygen species, including free radicals and reactive oxygen/nitrogen species. These reactive oxygen species can cause oxidative stress, leading to cellular damage and dysfunction (20,21). Furthermore, contrast media can stimulate an inflammatory response within the kidney. This involves the activation of immune cells, release of cytokines and chemokines, and recruitment of inflammatory cells to the site of injury. The inflammation can further contribute to renal injury and dysfunction (22,23).

The renal pathology associated with CA-AKI can manifest in various ways:

Acute tubular necrosis

Contrast-associated acute kidney injury is often characterized by ATN, which is the death of renal tubular cells. This can lead to a reduction in urine production,

electrolyte imbalances, and accumulation of waste products (24,25).

Interstitial edema and inflammatory infiltrates

Inflammation and tubular injury can lead to interstitial edema, which is the accumulation of fluid in the tissue spaces between the tubules. Inflammatory infiltrates consisting of immune cells may also be present (10,26).

Acute glomerular injury

In some cases, CA-AKI can result in acute glomerular injury, such as glomerular endothelial cell damage or disruption of the glomerular basement membrane. This can contribute to proteinuria and impaired filtration function (10,27).

In addition to morphologic lesions, systemic inflammation has also been implicated in the pathogenesis of contrast nephropathy. Inflammatory markers such as CRP, IL-6, and TNF-alpha are elevated in patients with contrast nephropathy. These markers are thought to contribute to the development of renal injury by promoting oxidative stress, endothelial dysfunction, and tubular apoptosis (28,29).

Diagnostic criteria for CA-AKI

The diagnostic criteria for CA-AKI typically involve increased serum creatinine levels after contrast exposure. Here are the commonly used diagnostic criteria for CA-AKI (30,31).

Kidney Disease Improving Global Outcomes (KDIGO) criteria: KDIGO defines CA-AKI as a rise in serum creatinine to more than 1.5 times the baseline serum creatinine concentration along seven days following contrast media administration or a rise of ≥0.3 mg/dL (26.5 µmol/L) (32,33).

Follow-up creatinine levels: CA-AKI can be diagnosed by monitoring creatinine levels two to three days after contrast exposure (12).

It is important to note that the diagnosis of CA-AKI is clinical and should also involve ruling out other potential causes of AKI.

These diagnostic criteria help identify patients who have experienced kidney injury following contrast administration. Early detection and appropriate management are crucial in preventing further kidney damage and improving patient outcomes (34,35).

Risk factors for CA-AKI

Previous studies show the Risk factors for developing contrast-related acute renal failure are older age, Pre-existing renal failure, presence of hypertension, diabetes mellitus, and metabolic syndrome. Additionally, the presence of anemia, multiple myeloma, hypoalbuminemia, and kidney transplantation was detected as the risk of developing CA-AKI (36,37).

Clinical presentation

Patients suffering from contrast-related acute renal failure frequently have a previous history of contrast administration 24-48 hours before the presentation while undergoing a diagnostic or therapeutic procedure, like percutaneous coronary intervention. In this condition, acute renal failure is mainly non-oliguric. Following contrast exposure, serum creatinine levels peak between 2 and 5 days and usually return to normal in 14 days. The primary goal of treatment is to prevent contrast-induced nephropathy by identifying high-risk patients and implementing preventive measures (12,38).

General treatment strategies for CA-AKI

Discontinuation of nephrotoxic medications: If you take any medications that could harm your kidneys, your healthcare provider may temporarily stop them until your kidney function improves (39,40).

Fluid management: Adequate hydration is essential for maintaining kidney function and preventing further damage. Your healthcare provider may recommend increasing your fluid intake, especially if you have no contraindications to fluids (41,42).

Monitoring kidney function: Regular monitoring of serum creatinine and urine output is necessary to assess kidney function and guide treatment decisions (43).

Medications: Several strategies have been proposed to prevent or mitigate the effects of contrast nephropathy. These include the use of low-osmolar contrast media, hydration before and after the procedure, and the administration of N-acetylcysteine and other antioxidants. However, the efficacy of these interventions remains controversial (44,45).

Avoidance of nephrotoxic agents: It is crucial to stay away from substances that can further damage your kidneys. These may include certain medications, illicit drugs, excessive alcohol, and certain herbal supplements. Your healthcare provider can give you a comprehensive list of such substances (46,47).

Conclusion

Contrast-associated acute kidney injury, also known as contrast-induced nephropathy, is a condition that refers to damage to the kidneys due to the administration of contrast agents used during medical imaging procedures, such as computed tomography (CT) scans or angiograms. It is considered a common cause of hospital-acquired AKI. Overall, the SIS is a valuable tool for assessing systemic inflammation and has shown potential in predicting and monitoring various medical conditions. Further research is needed to fully understand its clinical utility and establish

standardized guidelines for its application.

Authors' contribution

Conceptualization: Sam Mirfendereski, Hadi Taghavinejad.
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Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

Ethical issues (including plagiarism, data fabrication, and double publication) have been completely observed by the authors.

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