Contrast-Induced Acute Kidney Injury
Pathophysiology, Manifestations, Prevention, and Management

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KEYWORDS

• Contrast nephropathy • Contrast-induced acute kidney injury • CI-AKI

KEY POINTS

- There is a large volume of literature confirming the renal effects of iodinated intravenous contrast, but studies with control populations suggest that the risks have been exaggerated.
- A complex interplay of factors is responsible for the development of contrast-induced acute kidney injury and the pathogenesis is incompletely understood.
- Clinical manifestations are typically mild, but there may be serious long-term effects.
- Avoiding contrast is the primary prevention technique, but there are other techniques to decrease risks in patients requiring a contrast-enhanced computed tomography scan.

DISCUSSION OF PROBLEM AND CLINICAL PRESENTATION

Introduction

As medical imaging has advanced, there has been an increased dependence on the information provided by radiologic studies, especially cross-sectional imaging. This has led to dramatic increases in imaging utilization. In fact, more than 85 million computed tomography (CT) scans are performed on an annual basis in the United States alone,[1] with approximately 40 million doses of intravenous (IV) contrast.

Most radiologists in practice today have a basic understanding that these contrast agents have the potential to cause short-term or even permanent renal injury, a finding termed contrast-induced (CI) acute kidney injury (AKI). CI-AKI is defined as an acute decline in renal function after the administration of vascular contrast without other causes.[2] In most patients, it is a nonoliguric, asymptomatic, transient decline in renal function that may go undetected. However, in some cases, CI-AKI may result in more severe renal impairment, resulting in oliguria or requiring dialysis. In these cases, mortality is high.[3] CI-AKI is faced by practitioners in nephrology, cardiology, and radiology. However, as the physicians usually ultimately responsible for the administration of the contrast agent, it is important that radiologists have a more comprehensive understanding of the pathophysiology, clinical presentation, and management of CI-AKI to more fully weigh the risk to a patient. This article provides a practical review of the pathogenesis, clinical manifestations, preventative measures, and, ultimately, treatment of CI-AKI.

Background

Historical perspective

Iodinated contrast was used for years in uroradiology without knowledge of risk to the patient. In the

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1950s, the first cases of nephrotoxicity were reported in patients with preexisting renal impairment.[4] Subsequently, both experimental and clinical studies pointed to an association between use of iodinated contrast material and acute kidney injury.[5,6] Initially, it was thought that appropriate hydration could sufficiently counteract the nephrotoxic effects of contrast material.[7] Since that time, the risks of contrast have been confirmed numerous times.[8] Much of this research was performed in the context of angiocardiography with intra-arterial administration or using high-osmolar contrast before the advent of low-osmolality agents.[9] In the 1980s, the primary contrast media transitioned from high-osmolarity to low-osmolarity contrast media. Studies suggest that these low-osmolar contrast agents are less toxic to the kidneys compared with high-osmolar contrast, as is IV contrast compared with intra-arterial contrast.[10] Thus, much of the data on CI-AKI does not reflect the current clinical scenario in which the method is overwhelmingly IV and the agent is regularly a low-osmolar compound.[11]

Literature overview

Most of the large volume of research has lacked the crucial component of a control population. Additionally, any increase in serum creatinine (SCr) after contrast administration in many of these studies has been attributed to contrast without accounting for the myriad of additional factors that can contribute to increased SCr.[9,10]

Studies with a control population found that renal injury was as frequent in those not exposed to contrast as in those receiving contrast.[12] The initial studies with control populations were small and did not randomly assign patients to receive contrast, leaving open the possibility of a selection bias. Additional studies with larger subject populations and a control cohort have demonstrated that the incidence of CI-AKI was not significantly different from contrast material–independent acute renal failure.[13]

Due to these conflicting data, the risks of IV administration of iodinated contrast must be reevaluated so that radiologists can make informed decisions. A full understanding of the risks associated with intravascular contrast begins with the underlying pathophysiology.

Pathogenesis

The pathophysiology contributing to kidney injury after administration of IV contrast is not completely understood. The conception of the in vivo changes is based mainly on extrapolation from animal and laboratory studies.[14] The effects of IV contrast result from a complicated interplay between hemodynamic changes, oxidative stress, and direct injury to the renal tubular cells (**Fig. 1**). The

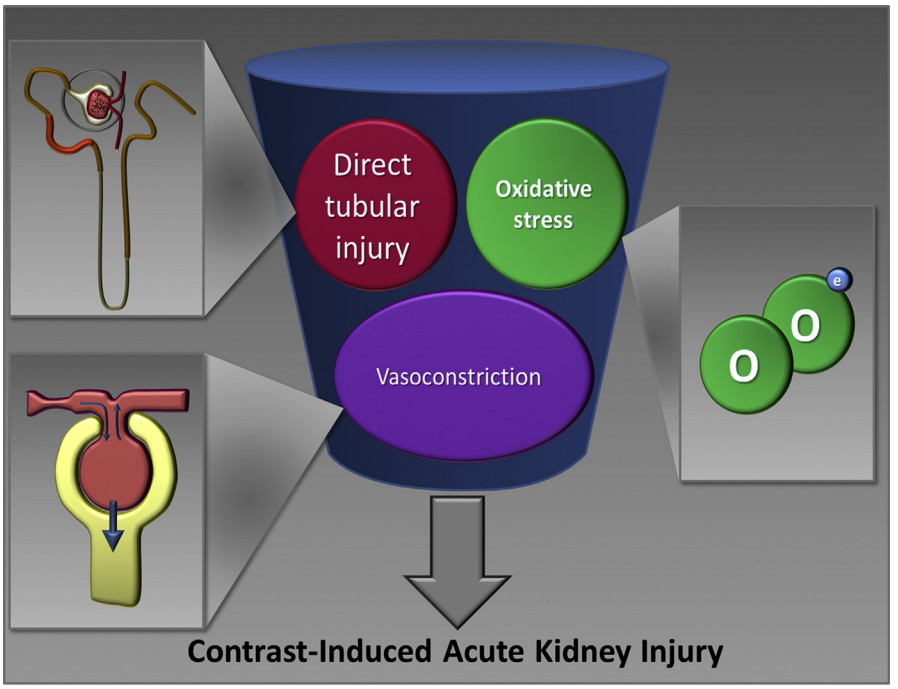

Fig. 1. Three major factors contribute to the development of CI-AKI: hemodynamic changes, oxidative stress, and direct tubular injury. There is overlap between these factors but all contribute to acute tubular necrosis and the resulting kidney injury.