

Strategies for prevention and management

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Contrast induced nephropathy (CIN) is the third leading cause of acute renal failure in hospitalized patients, and it is responsible for 11% of cases of hospital-acquired acute renal failure. The single most important risk factor for CIN is preexisting renal failure; diabetes mellitus is the second most important risk factor. The incidence of CIN in the general population is estimated at 1% to 2%. However, the risk for developing CIN may be as high as 50% in patients with concomitant diabetes mellitus and preexisting renal failure. There is no specific treatment once contrast-induced acute kidney injury (AKI) develops, and management must be as for any cause of acute tubular necrosis, with the focus on maintaining fluid and electrolyte balance. The best treatment of contrast-induced kidney injury is prevention. Optimal therapy to prevent contrast-induced acute kidney injury (AKI) remains uncertain. Patients with near-normal renal function are at little risk, and few precautions are necessary, only to avoid volume depletion. In patients with increased risk, some preventive strategies had been proposed. Among them, volume expansion might be the least controversial. But it was usually intolerable by aging patients, especially if they have renal or heart diseases. The optimal intravenous solution and its volume and duration of administration pre- and post-contrast for prevention of contrast nephropathy are unclear. In general, intravenous fluids should be administered at a rate of 1 mL/kg/h for 6 to 12 hours before contrast-media and continued for at least 6 to 12 hours after the procedure in the absence of fluid overload. Contrast media-related risk factors for CIN include osmolality, viscosity, and administration volume. According to the current evidences, for patients with increased risk, it is reasonable to use low-osmolal contrast media (LOCM) to minimize the risk for CIN. The superiority of iso-osmolal contrast media (IOCM) in preventing CIN in patients with renal insufficiency remains controversial. Although the administration of sodium bicarbonate may be potentially superior to the administration of isotonic saline for the prevention of CIN, the 2012 KDIGO guideline Work Group did not make a specific recommendation for the use of bicarbonate preferentially to saline due to concern for potential harm from errors in compounding of the bicarbonate solutions at point of care or in the hospital pharmacy. Although several pharmacologic agents have been evaluated for CIN prevention, none consistently has been shown to reduce the risk for this condition. Among these agents, NAC has recently drawn much attention. But data regarding the efficacy of NAC are still conflicting. Importantly, only the trials in which NAC reduced serum creatinine below baseline values demonstrated a beneficial effect on reducing rates of renal injury. Thus, NAC appears to falsely lower serum creatinine, which was proven in healthy volunteers, but it does not fundamentally protect against CIN. However, because of NAC's low cost, limited side effects, general availability, and ease of administration, It might be suggested that NAC be administered the day before and the day of the procedure. If NAC is administered, the recommended dose of 1200 mg orally twice daily rather than 600 mg twice daily.

Contrast media are excreted mainly by glomerular filtration. The elimination is slow in patients with preexisting renal failure, so these vulnerable patients are exposed to contrast media longer. Although contrast media can be removed effectively and rapidly from the blood of patients with chronic renal failure by hemodialysis, even faster than by the kidneys in normal subjects, the effect of prophylactic hemodialysis in preventing CIN addressed in previous studies is controversial. It may be due to the heterogeneity of the previous studies. However, based on our study before, we strongly suggest that patients with a creatinine clearance less than $25 \text{ mL/min/1.73 m}^2$ may benefit from prophylactic hemodialysis after coronary angiography. Like hemodialysis, continuous venovenous hemofiltration (CVVH) before and after coronary angiography has been shown to be effective in preventing CIN. However, because of the relatively high cost, length of treatment, limited availability of beds in intensive care units, lengthy immobilization of patients, and lower efficiency of contrast media removal inherent in CVVH, hemodialysis might be more practical for preventing CIN after coronary angiography.