

CONTRAST ASSOCIATED ACUTE KIDNEY INJURY

The American College of Radiology and the National Kidney Foundation reviewed the available evidence and issued updated guidance on the use of intravenous iodinated contrast media in patients with preexisting kidney disease (1).

The risk of contrast-induced acute kidney injury:

- It is near zero in patients with an eGFR of 45 ml/min per 1.73 m² or higher
- It is ≤2% when the eGFR is 30 to 44 ml/min per 1.73 m²
- The evidence is less clear for patients with severe kidney disease (eGFR <30 ml per minute per 1.73 m²) since no single study has been adequately powered to reliably assess the risk of contrast administration in this population.
 - In patients with stage 4 or 5 chronic kidney disease, there is potential for increased risk of acute kidney injury after administration of contrast material but recommended against withholding contrast required for the evaluation or treatment of a potentially life-threatening diagnosis on the basis of kidney function alone.

Contrast associated acute kidney injury instead of contrast induced nephropathy

- Since other factors (e.g., medications, hypotension, or atheroemboli) can precipitate acute kidney injury after exposure to contrast medium, [the term “contrast-associated acute kidney injury” has been suggested by KDIGO to replace contrast induced nephropathy.](#)

The risk of acute kidney injury due to contrast material is overestimated

- Compared with the early, high-osmolality contrast agents, low-osmolality and iso-osmolality agents are associated with a lower risk of kidney injury.

The reports linking contrast-associated acute kidney injury and worse outcome are associational not necessarily cause-effect related. It is plausible that contrast-associated acute kidney injury is a marker of an increased risk of serious adverse outcomes rather than a mediator of such outcomes.

Although currently available data are insufficient to declare that contrast agents are not nephrotoxic, [severe acute kidney injury characterized by substantial decrements in kidney function, the need for renal replacement therapy, or both appears to be very infrequent after intravascular contrast administration.](#)

Definition of contrast-associated acute kidney

- sCr increase by a factor of 1.5 times or more over the baseline value within 7 days after exposure to contrast medium, or
- sCr increase by at least 0.3 mg/dl over the baseline value within 48 hours after exposure to contrast medium, or
- A urinary volume of less than 0.5 ml/Kg/h that persists for at least 6 hours after exposure to contrast medium.

PREVENTIVE STRATEGIES

Intravascular volume expansion with balanced crystalloids or normal saline

Although a recent trial (AMACING) which randomly assigned 660 patients undergoing contrast-enhanced procedures to receive either periprocedural intravenous isotonic saline or no intravenous fluids, showed no significant difference in the incidence of AKI between the groups, it is premature to conclude that intravenous fluids are ineffective or unnecessary.

- Current American College of Radiology guidelines on the administration of contrast material recommend the use of intravenous isotonic saline at an infusion rate of 100 ml per hour for 6 to 12 hours before and 4 to 12 hours after angiography.
- A shorter protocol that is more practical for outpatients and those undergoing urgent procedures comprises an intravenous infusion of isotonic saline at an infusion rate of 100 ml per hour for 1 to 3 hours before and 6 hours after the procedure.

NaHCO₃ and Acetylcysteine

- Numerous clinical trials have investigated the role of intravenous sodium bicarbonate as well as acetylcysteine for the prevention of contrast-associated acute kidney injury. The results of these trials and meta-analyses are highly divergent and inconclusive.
- A more recent double-blind trial (PRESERVE) randomly assigned 5177 high-risk patients undergoing nonemergency angiography to receive intravenous isotonic sodium bicarbonate or intravenous isotonic saline, as well as oral acetylcysteine or oral placebo, for the prevention of AKI was stopped early because of futility. It showed no significant difference in the outcome with the use of NaHCO₃ or acetylcysteine when compared to saline or placebo respectively.

So, it is the conclusion that sodium bicarbonate or acetylcysteine provides no benefit in preventing contrast-associated acute kidney injury.

Statins

- Further studies are needed to definitively clarify the role of prophylactic administration of high dose statins. Nonetheless, because high-intensity statins are commonly indicated for atherosclerotic disease according to clinical practice guidelines, many patients undergoing procedures with contrast administration will have an indication for maintenance therapy with these agents.

References

1. Davenport MS, Perazella MA, Yee J, et al. Use of intravenous iodinated contrast media in patients with kidney disease: consensus statements from the American College of Radiology and the National Kidney Foundation. *Radiology* 2020;294:660-668.
2. Mehran R, George D, Dangas GD, and Weisbord SD. Contrast-Associated Acute Kidney Injury. *N Engl J Med* 2019;380:2146-55.
3. Robert Goulden R et al. Association of Intravenous Radiocontrast With Kidney Function A Regression Discontinuity Analysis. *JAMA Intern Med*. doi:10.1001/jamainternmed.2021.0916 Published online April 5, 2021.