

CONTRAST ASSOCIATED ACUTE KIDNEY INJURY

Supporting information

This guideline has been prepared with reference to the following:

NICE. Point-of-care creatinine devices to assess kidney function before CT imaging with intravenous contrast. 2019. NICE. London

<https://www.nice.org.uk/guidance/htq530>

Stacul F, van der Molen AJ, Reimer P, et al. Contrast induced nephropathy: updated ESUR Contrast Media Safety Committee guidelines. *Europ Radiol* 2011;21:2527-41

<https://esur-cm.org/>

What are the respective merits of sodium chloride 0.9%, sodium bicarbonate 1.26%, and acetylcysteine (NAC)?

A systematic review of RCTs found that low-dose N-acetylcysteine plus IV saline was more beneficial than IV saline in reducing contrast induced nephropathy risk when low-osmolar contrast media (LOCM) were used (risk ratio [RR], 0.75 [95% CI, 0.63 to 0.89]) [Subramaniam, 2016]. It was also found that N-acetylcysteine plus IV saline was more beneficial than IV saline (RR, 0.69 [95% CI, 0.58 to 0.84]). A clinically important but statistically insignificant benefit was also seen in sodium bicarbonate versus IV saline in patients receiving LOCM (RR, 0.65 [95% CI, 0.33 to 1.25]).

A prospective study in 156 patients (Castini, 2010) compared saline infusion vs saline plus NAC vs sodium bicarbonate. Contrast-induced nephropathy developed in 23 patients (14.7%). Incidence of the primary endpoint was similar in the 3 groups of treatment, occurring in 7 patients (14%) in the saline infusion group, in 9 (17%) in the saline infusion plus NAC group, and in 7 (14%) in the SB infusion group. The authors concluded that “neither the addition of NAC nor the administration of SB add further benefit in CIN prevention, compared to standard hydration with isotonic saline infusion.”

A meta-analysis of 7 trials in a total of 1734 patients (Kunadian, 2011) found the odds ratio (OR) for the development of contrast nephropathy for NaHCO(3) versus NaCl was 0.33 (95% confidence interval [CI] 0.16-0.69; P=0.003).

A prospective, randomized trial between March 2005 and December 2009, including 258 consecutive patients with renal insufficiency undergoing intravascular contrast procedures compared Sodium Chloride with Sodium Bicarbonate for prevention of CIN. (Klima et al. 2012) Patients were randomized to receive intravenous volume supplementation with either (A) sodium chloride 0.9% 1 mL/kg/h for at least 12h prior and after the procedure or (B) sodium bicarbonate (166 mEq/L) 3 mL/kg for 1 h before and 1 mL/kg/h for 6 h after the procedure or (C) sodium bicarbonate (166 mEq/L) 3 mL/kg over 20 min before the procedure plus sodium bicarbonate orally (500 mg per 10 kg). The primary endpoint was the change in estimated glomerular filtration rate (eGFR) within 48 h after contrast. Secondary endpoints included the development of CIN. The maximum change in eGFR was significantly greater in Group B compared with Group A {mean difference -3.9 [95% confidence interval (CI), -6.8 to -1] mL/min/1.73 m², P = 0.009} and similar between groups C and B [mean difference 1.3 (95% CI, -1.7-4.3) mL/min/1.73 m², P = 0.39]. The incidence of CIN was significantly lower in Group A (1%) vs. Group B (9%, P = 0.02) and similar between Groups B and C (10%, P = 0.9). The authors concluded that “volume supplementation with 24 h sodium chloride 0.9% is superior to sodium bicarbonate for the prevention of CIN. A short-term regimen with sodium bicarbonate is non-inferior to a 7 h regimen”

Castini D, Lucreziotti S, Bosotti L, et al. Prevention of contrast-induced nephropathy: a single center randomized study. *Clin Cardiol* 2010;33:E63-8

<http://onlinelibrary.wiley.com/doi/10.1002/clc.20576/epdf>

Kunadian V, Zaman A, Spyridopoulos I, et al. Sodium bicarbonate for the prevention of contrast induced nephropathy: a meta-analysis of published clinical trials. *Eur J Radiol* 2011;79:48-55

Klima T, Christ A, Marana I, Kalbermatter, S, Utoff, H, Burri, E, Harwiger, S, Schindler, C, Breidhardt, T, Marenzi, G, and Mueller, C. Sodium chloride vs. sodium bicarbonate for the prevention of contrast medium-induced nephropathy: a randomized controlled trial. *Eur Heart Journal*, 2012;33/16; 2071-9

<http://eurheartj.oxfordjournals.org/content/33/16/2071.long>

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Subramaniam RM, Suarez-Cuervo C, Wilson RF et al. Effectiveness of Prevention Strategies for Contrast-Induced Nephropathy: A Systematic Review and Meta-analysis. Ann Intern Med. 2016;164:406-16

Evidence Level: I

eGFR <60 mL/min increases risk significantly?

A questionnaire survey emailed to 5000 randomly chosen cardiologists in the US (Alhosaini, 2010) received 291 responses. Among responding cardiologists, 70% considered eGFR level less than 60 mL/min a high risk for CIN.

Alhosaini MN, Latta S, Riaz K, et al. Contrast-induced nephropathy: current practices among cardiologists. Renal Failure 2010;32:928-34

Evidence Level: V

What is the evidence that oral hydration is adequate in eGFR >30 to prevent CA-AKI?

A 2017 RCT of patients at risk of contrast-induced nephropathy found no prophylaxis to be non-inferior and cost-saving in preventing contrast-induced nephropathy compared with intravenous hydration. High-risk patients (with an estimated glomerular filtration rate [eGFR] of 30–59 mL per min/1.73 m²) aged 18 years and older, undergoing an elective procedure requiring iodinated contrast material administration were randomly assigned (1:1) to receive intravenous 0.9% NaCl or no prophylaxis. Contrast-induced nephropathy was recorded in eight (2.6%) of 307 non-hydrated patients and in eight (2.7%) of 296 hydrated patients. The absolute difference (no hydration vs hydration) was -0.10% (one-sided 95% CI -2.25 to 2.06). No hydration was cost-saving relative to hydration. No haemodialysis or related deaths occurred within 35 days. 18 (5.5%) of 328 patients had complications associated with intravenous hydration.

Nijssen EC, Rennenberg RJ, Nelemans PJ et al. Prophylactic hydration to protect renal function from intravascular iodinated contrast material in patients at high risk of contrast-induced nephropathy (AMACING): a prospective, randomised, phase 3, controlled, open-label, non-inferiority trial. Lancet. 2017;389:1312-22

Evidence Level: II

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