Contrast and the Kidney

The risk was overstated for decades · Withholding a needed scan is now the commoner harm

eGFR < 30
the prophylaxis threshold

WHAT CHANGED, AND WHY *the old teaching was built on studies without control groups*

TWO DIFFERENT THINGS

Contrast-associated AKI is injury that happens **after** contrast — a purely temporal association. **Contrast-induced AKI** is injury **caused by** it. **Older studies could not separate the two** because they lacked non-contrast comparison groups, so they attributed to contrast the acute kidney injury of the illness that prompted the scan.

THE CORRECTED PICTURE

Studies using **propensity-matched patients who did not receive contrast** show that **the attributable risk from modern intravenous contrast is far smaller than was taught**, and is very low above an eGFR of 30. The kidney injury largely belonged to the sepsis, hypotension and nephrotoxins, not to the dye.

WHY IT MATTERS CLINICALLY

The real harm today is the scan that does not happen. Withholding contrast means **missed pulmonary embolism, missed dissection, missed abscess and missed cancer** — or a substituted, less accurate test. **Do not decline a genuinely indicated contrast study on renal grounds alone**; weigh the diagnostic cost explicitly.

WHO ACTUALLY NEEDS PROPHYLAXIS *a much smaller group than the old protocols implied*

Situation	Action
eGFR 30 or above	**No prophylaxis required** for intravenous contrast, and **no need to delay the scan** for a creatinine in a well outpatient with no risk factors. Give the study and treat the patient.
eGFR below 30, or established AKI	**Give intravenous isotonic saline** before and after, in those without a contraindication such as decompensated heart failure. This is the group in whom prophylaxis is actually recommended.
eGFR 30–44 with multiple risk factors	**Prophylaxis may be considered** on an individual basis — for example diabetic nephropathy with heart failure, hypotension, or concurrent nephrotoxins. It is a judgement, not a protocol trigger.
On maintenance dialysis	**Anuric dialysis patients need no prophylaxis and no urgent extra dialysis session** after contrast — there is no residual function left to protect. In those with meaningful residual urine output, protect it as you would any low eGFR.
What does not work	**N-acetylcysteine has no role** — large trials showed no benefit, and it also interferes with creatinine assays. **Sodium bicarbonate offers nothing over saline. Volume expansion with isotonic saline is the only intervention with a reasonable basis**, and even that matters mainly at the lowest eGFRs.

THE REST OF THE CHECKLIST *the things that genuinely reduce risk*

Item	Detail
Fix the hemodynamics	**Hypovolemia and hypotension are the dominant modifiable risks**, and far more important than the contrast itself. Resuscitate the patient first; a normotensive, euvolemic patient tolerates contrast well.
Review the nephrotoxins	**Stop NSAIDs and aminoglycosides** where possible around the study. **ACE inhibitors and ARBs do not need routine withholding** for contrast alone, but reconsider them in a hypotensive or volume-depleted patient.
Metformin	**Withhold only if there is AKI or an eGFR below 30**, in line with the general rule for metformin at that level of function. **Routine 48-hour holds in patients with preserved renal function are unnecessary** and cause avoidable hyperglycemia.
Minimize the dose	Use **the lowest volume that answers the question**, use **low- or iso-osmolar agents**, and **avoid repeated studies in quick succession**. Intra-arterial administration with first-pass renal exposure, as in coronary angiography, carries higher risk than intravenous contrast.
Gadolinium	A different question entirely. **Nephrogenic systemic fibrosis is now very rare with modern group II agents**, and these can be given at low eGFR when the scan is needed. **Do not reflexively substitute a contrast MRI for a contrast CT** assuming it is renally safer.
Know who is genuinely higher risk	The risk concentrates in **advanced chronic kidney disease, established or evolving AKI, diabetic nephropathy, heart failure, hypotension or hypovolemia, sepsis, and concurrent nephrotoxins** — and rises further with **intra-arterial administration and large or repeated contrast volumes**. **An otherwise well patient with a stable eGFR above 45 and none of these is at negligible risk**, and treating them as high-risk generates delay without benefit.
Follow up sensibly	**Recheck renal function at 48 to 72 hours** in higher-risk patients. If the creatinine rises, **look for the real cause** — sepsis, hypotension, a nephrotoxin, obstruction — rather than closing the case as contrast nephropathy and missing something treatable.

HOW TO HAVE THE CONVERSATION *with radiology, with the team, and with the patient*

FRAME IT AS A TRADE-OFF

State the question the scan answers and what happens if it is not answered. "This is a possible aortic dissection" carries different weight from a routine surveillance study. **Document the reasoning**, including the eGFR, the indication, and the alternative considered. **A delayed diagnosis is a real harm with a real probability**, and it deserves to be weighed against a risk that has turned out to be small.

WHEN TO PAUSE

Defer or modify when the study is genuinely elective and the patient is unwell — actively hypotensive, in established AKI that is still evolving, or acutely volume-depleted. **Optimize and rescan rather than cancelling outright.** Consider whether an **unenhanced study, ultrasound or MRI** would answer the question adequately, but only if it truly would. **Never let the debate itself delay imaging in a time-critical presentation.**

PEARLS & PITFALLS

- **Metformin is about lactic acidosis, not the kidney.** It does not cause contrast nephropathy. **Continue it if eGFR is 30 or above with no AKI**; hold it when eGFR is below 30, there is AKI, or intra-arterial contrast is going to the renal arteries, and restart after **48 h** once renal function is confirmed stable.
- **If you do give fluid, give enough to matter: isotonic saline 1-1.5 mL/kg/h for 3-12 h before and 6-12 h after** contrast. When time is short, **3 mL/kg/h for 1 h before and 1-1.5 mL/kg/h for 6 h after**. A single token bag achieves nothing.
- **The bigger risk is now the scan you did not do.** Missed dissection or embolism outweighs a small and often theoretical renal risk.
- **Prophylaxis belongs below an eGFR of 30**, or in established AKI. Above that, give the contrast.
- **N-acetylcysteine does not work**, and it also distorts creatinine measurement. Stop prescribing it for this.
- **Saline, not bicarbonate.** Bicarbonate has repeatedly failed to beat plain isotonic saline.
- **Hold metformin only below an eGFR of 30 or in AKI**, not routinely for every contrast study.
- **A creatinine rise after contrast is usually caused by something else.** Look for sepsis, hypotension or a nephrotoxin before blaming the dye.

References: ACR and National Kidney Foundation consensus statements on the use of intravenous iodinated contrast media in patients with kidney disease · ACR Manual on Contrast Media · PRESERVE · AMACING · KDIGO guidance on acute kidney injury.

Educational aid; verify against local radiology and renal protocol and patient factors.