COMMENTARIES ON MEDICAL INNOVATIONS, NEW TECHNOLOGIES, AND CLINICAL TRIALS

Evidence-based medicine and the misconception of contrast-induced kidney disease

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In clinical practice we are frequently confronted with the difficulties in performing contrast enhanced imaging in patients with impaired kidney function (glomerular filtration rate, GFR <45 ml/min) which is particularly observed when the imaging studies are performed in outpatient radiology centres.

Contrast-induced acute kidney injury (CI-AKI), formerly termed ‚contrast-induced nephropathy', implies a causal relationship between intravenous contrast media and the development of AKI. CI-AKI is usually defined as a frequently clinically insignificant, small increase in creatinine within the days following contrast administration (e.g. 0.3 mg/dL) [1].

The 'historical' concept of CI-AKI dates back from the 1950's, where in a small non-randomised (case report) study, the authors observed that some patients developed renal failure following injection of intravenous (IV) contrast media for intravenous pyelography [2]. This phenomenon, according to the current status of knowledge, might have led to a true nephrotoxic reaction, i.e. older contrast media probably were nephrotoxic. However, modern contrast media (with lower osmolarity) do not seem to cause renal failure.

Recently, numerous studies have been performed to investigate whether intravenous contrast administration for computed tomography (CT) is independently associated with increased risk for AKI and adverse clinical outcomes. In one of the largest well-controlled studies, Hinson and co-authors revealed that intravenous contrast was not associated with an increased frequency of AKI. Similarly, McDonald et al., examined creatinine changes in patients who had underwent both a contrast CT scan and also a non-contrast CT scan at different points in time [3]. The analysis revealed no significant difference in AKI incidence between contrast enhanced and unenhanced CT scans in the same patient i.e. changes in creatinine following both scans were the same, regardless of whether or not the patient was administered contrast. Furthermore, Kooiman et al., in a randomised controlled trial, observed no increase in kidney injury molecule-1 and neutrophil gelatinase-associated lipocalin excretion following intravenous contrast enhanced-CT meaning that that CI-AKI was not accompanied by any kidney damage i.e. renal tubular injury. There is also no obvious evidence that contrast increases directly the risk of death or renal failure requiring dialysis [4].

Considering the above, the best available evidence indicates that contrast dye is safe but can we then

forget about kidney function prior to contrast imaging? The answer is probably not that straightforward. Based on the consensus statements from the American College of Radiology and the National Kidney Foundation „Although the true risk of CI-AKI remains unknown, prophylaxis with intravenous normal saline is indicated for patients without contraindication (e.g. heart failure) who have AKI or an estimated GFR (eGFR) less than 30 mL/min/1.73 m^2 who are not undergoing maintenance dialysis. In individual high-risk circumstances, prophylaxis may be considered in patients with an eGFR of 30–44 mL/min/1.73 m^2 at the discretion of the ordering clinician. The presence of a solitary kidney should not independently influence decision making regarding the risk of CI-AKI [5]".

To sum up, to the best of our knowledge, there is no point in withholding contrast media for fear of AKI irrespective of GFR/kidney status in any case that seems beneficial for patient (i.e. when the use of contrast is clinically indicated) in patients requiring urgent assessment in emergency departments and/or those with life threatening conditions such as sepsis.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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