Renal harms from IV contrast: overstated?

Story From The Front Lines

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A 48 year old man with type 2 diabetes mellitus presented with a week of polyuria and polydipsia as well as new onset right foot pain that was associated with redness and swelling for three days. He was found to have diabetic ketoacidosis with an acute kidney injury in setting of Methicillin-sensitive Staphylococcus aureus bacteremia presumed secondary to cellulitis. His foot was notable for diffuse tenderness, erythema and swelling along the dorsum of his foot without purulence. Imaging including X-ray, venous ultrasound, and magnetic resonance imaging were unremarkable for fractures, thrombus, osteomyelitis, or abscess. A transthoracic echocardiograph did not show valve vegetations and subsequent blood cultures cleared on intravenous (IV) cefazolin. Notably, his hospital course was complicated by persistently elevated serum creatinine felt consistent with acute tubular necrosis along with little improvement in tenderness, erythema and swelling of his right extremity two weeks into his hospitalization. There was new fluctuance appreciated thus a soft tissue ultrasound was obtained with was no evidence of abscess. Two days later the patient received approval for a rehabilitation facility but given his persistent infectious findings infectious disease recommended computed tomography (CT) with contrast to rule out abscess. There were concerns for causing contrast nephropathy given his serum creatinine was 1.6 (his baseline 0.7) with estimated glomerular filtration rate (eGFR) 43. A CT without contrast was obtained instead which again did not appreciate an organized fluid collection; however radiology noted that non-contrast studies are insensitive for evaluation of abscess. In this setting not only was the imaging non-conclusive but the patient also received potentially unnecessary radiation.

A Teachable Moment

Contrast-induced nephropathy (CIN) is a term used to described acute kidney injury (AKI) within three days of IV contrast administration when other etiologies have been ruled out. The correlation was first observed in the 1950's when patients developed renal failure after IV contrast dye[1]. Since then, the concept has remained pervasive in how clinicians make medical decisions to investigate acute and chronic illnesses, often times forgoing the appropriate study due to concern patient may develop acute kidney injury, or worsened renal failure requiring dialysis. However, in the past several years this concept has been challenged with many studies indicating no clear causal relationship between contrast administration and worsened renal function.

McDonald et al examined 13 controlled studies (25,950 patients) that evaluated the incidence of AKI, dialysis and death in patients exposed to IV contrast compared to those who were not and found similar incidence between these two groups; propensity scoring was used to matched patients with similar comorbidities and creatinine and the results were sustained[2]. Similarly another study examining 17,934 patients divided among three groups (CT with contrast, CT without contrast and no CT scan at all) showed contrast did not increase the risk of AKI even among patients with chronic kidney disease (CKD) at

baseline. Additionally, at six months those who received contrast did not show increased risk of CKD, need for renal replacement therapy or kidney transplant when compared to those who did not receive contrast[3]. Interestingly, Davenport et al. did show evidence of correlation in patients with eGFR less than 30 mL/min/1.73 m^2 (P= 0.04; odds ratio, 2.96; 95% confidence interval: 1.22, 7.17) with a trend toward significance in patients with stable eGFR between 30-44 mL/min/1.73 m2[4].

In conclusion, the risk of CIN has likely been overestimated due to multiple confounders for AKI especially among low-risk patients. The nonuse of IV contrast material among these patients impairs diagnostic abilities in many instances such as in the case presented when it did not have to be. However, in certain high-risk populations with strongest evidence in patients with eGFR below 30 mL/min/1.73 m2 intravenous contrast media should continue to be considered as nephrotoxic risk factor when used to guide clinical decision-making.

References:

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