

Post-angiography Acute Kidney Injury

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L'insufficienza renale acuta da mezzo di contrasto
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INTRODUCTION

The association of a decrease in glomerular filtration rate following the administration of iodinated contrast was first described over 60 years ago [1]. The name has changed from contrast-induced nephropathy (CIN) to contrast-associated acute kidney injury (CA-AKI) to post-angiography acute kidney injury (PA-AKI), reflecting an ongoing controversy regarding the association. Since the original publication, over 1700 publications have documented this association, attempted to unravel the pathophysiologic mechanism, described the short and long-term consequences, and advised practitioners on how to prevent this association. The most common definition is an absolute increase in serum creatinine of 0.5 mg/dl or a relative increase of 25% compared to baseline that occurs over the 48-72 hours following exposure to contrast. However, many controversies remain. Guidelines from the major groups using iodinated contrast have been published and revised over the years [2,3]. In this brief editorial, we will review the three major tenets of these guidelines.

1. IDENTIFY PATIENTS MOST AT RISK FOR DEVELOPING PA-AKI

It is generally agreed that not all patients are at equal risk for PA-AKI. Figure 1 describes the pathogenesis of this form of AKI and divides it between patient-related factors and procedure-related factors.

Central to the heightened risk of this form of AKI is a vulnerable kidney. The kidney may be vulnerable because of hemodynamic alterations that either decrease blood flow (for example congestive heart failure) or perfusion pressure (hypotension), and/or impair the renal vasculature's ability to respond to contrast-induced decreases in blood flow (for example, renal insufficiency, diabetes) or drugs such as nonsteroidal anti-inflammatory drugs (NSAIDs) and renin-angiotensin-aldosterone system (RAAS) inhibitors. Chronic kidney disease is associated with both vascular changes and decreases in renal reserve that also make the kidney more vulnerable to subsequent injury.

A number of risk models have been presented to enable anticipation of kidney injury and to focus prophylactic efforts on those more in danger of developing PA-AKI. In cardiology, the most widely used

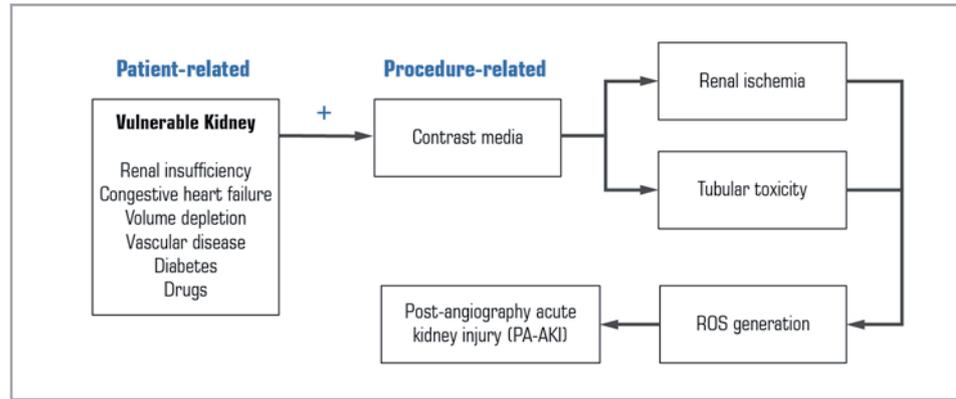
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Figure 1. Model of post-angiography acute kidney injury (PA-AKI) pathogenesis that emphasizes the factors that make the kidney vulnerable to contrast. How contrast gets into body (IV or IA) doesn't alter how it gets to the kidney.

ROS = reacting oxygen species



risk model uses a small number of patient- and procedure-related factors to divide patients into 4 categories of risk [4]. The categories are not only predictive of AKI but also the need for dialysis and in-hospital mortality. Patients in the 2 highest risk categories are targeted for specific prophylactic interventions (see below).

2. REDUCE THE AMOUNT OF CONTRAST ADMINISTERED

The evidence from animal and *in vitro* studies suggests that iodinated contrast is directly nephrotoxic (see Figure 1) [5]. Review of large patient databases indicates that patients who receive more contrast have a higher incidence of AKI [6]. Therefore, another recommendation in the guidelines points to using as little contrast as necessary. This includes consideration of other imaging techniques that don't require use of contrast. There have also been a number of attempts to diminish the volume of contrast administered using pressure sensitive manifolds [7], automatic injectors [8], or coronary si-

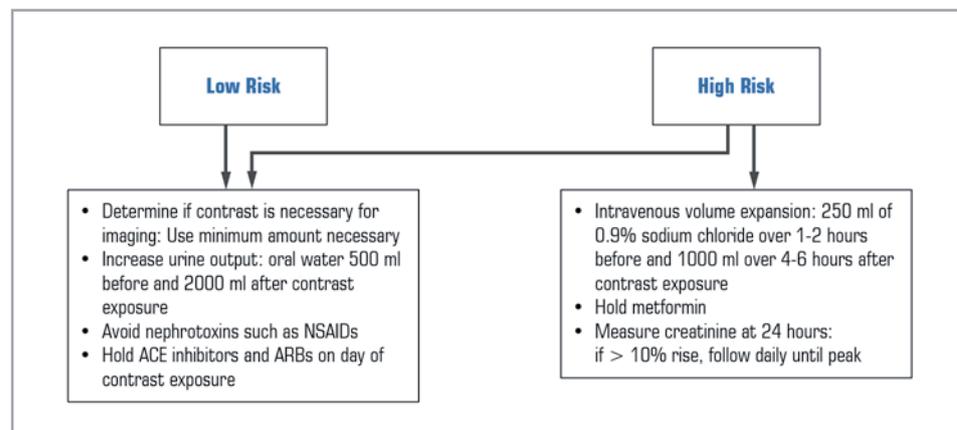
nus removal of contrast [9]. Hemodialysis immediately after contrast administration has also been proposed [10]. While many of these maneuvers do decrease the amount of contrast administered, the evidence that the incidence of AKI is also reduced is not compelling.

3. PROVIDE ADEQUATE FLUID INTAKE

There are many strategies to minimize the risk of PA-AKI, but the only consensus involves the use of fluids to induce a high urine output. There are many uncertainties about this approach, which have been studied in high-risk patients. Although intravenous fluids have been most often recommended, there is increasing evidence that oral fluids may be equally efficacious [11]. For intravenous fluids, isotonic saline and isotonic bicarbonate have been most often compared and there doesn't seem to be any difference in efficacy [12]. The timing of fluid administration has been less well studied but in general, the longer the administration the

Figure 2. Algorithm for managing patients undergoing exposure to contrast media.

ACE inhibitors = angiotensin-converting-enzyme inhibitors; ARBs = angiotensin receptor blockers; NSAIDs = nonsteroidal anti-inflammatory drugs



better the outcomes [13]. The mechanism of benefit with fluid administration is unknown, but a growing body of evidence suggests that at least part of the benefit lies in inducing a high urine flow rate. This may dilute out the contrast in the nephron and decrease the contrast contact time with the renal tubular epithelium. Additionally, high urine output seems to increase blood flow in the medulla, the most sensitive part of the kidney with respect to ischemia [14]. An algorithm for managing patients is shown in Figure 2.

CONTROVERSIES

As mentioned above, there are many areas that are hotly debated. One is whether we have grossly overestimated the impact of contrast on renal injury. Certainly, in the cardiology space, other sources of injury may

be present such as hemodynamic effects and atheromatous embolic disease. While these are less of a problem in patients undergoing outpatient CT exams, no difference in the incidence of AKI between patients receiving contrast-enhanced CT and non-contrast CT has been reported [15]. These two groups have been propensity matched on the reasons for the imaging study.

CONCLUSION

Post-angiography AKI continues to be a concern and much has been learned over the past 60 years. A reasonable approach is outlined in Figure 2. Central to preparing all patients for exposure to contrast is the induction of a high urine output. High-risk patients should have an assessment of renal function in the 72 hours post-exposure to ensure that AKI has not occurred.

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Conflicts of Interests

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