

Acute Kidney Injury

Q Our radiology department is discussing use of IV magnesium for diabetic patients to “protect them from kidney injury.” Is this a standard of care now?

Magnesium, the fourth most abundant cation in the body, plays an important physiologic role. Balance is maintained by renal regulation of magnesium reabsorption, and deficiency occurs when there is increased renal excretion initiated by osmotic diuresis. Clinical manifestations of deficiency include cardiac arrhythmias, neuromuscular hyperexcitability, and biochemical abnormalities of hypocalcaemia and hypokalemia.

Diabetes is one of the leading causes of magnesium deficiency, with incidence ranging from 25% to 39%.¹ Fluctuations in serum magnesium concentrations are directly correlated with fasting blood glucose, A1C levels, albumin excretion, and the duration of diabetes. It has been postulated that magnesium depletion, via its effect on inositol transport, is pathogenic in the progression of diabetic complications.

Contrast-induced acute kidney injury (CI-AKI) is a potentially adverse consequence of percutaneous coronary interventions (PCI), particularly in diabetic patients. It results in significant morbidity

and mortality and adds to the costs of diagnostic and interventional cardiology procedures. Intravenous (IV) agents used during radiologic imaging are notorious for causing acute kidney injury in diabetic patients. Preprocedural hydration and discontinuation of all nephrotoxic medications have proven beneficial in protecting these patients from CI-AKI.

A recent prospective, randomized, open-label clinical trial looked at the effect of administering IV magnesium prior to PCI.² The control group underwent standard preprocedural hydration and discontinuation of nephrotoxic medications. The study group added IV magnesium to the standard protocol.

In this single-center study, 26.6% of patients in the control group and 14.5% in the study group sustained CI-AKI, a statistically significant result ($P = .01$). Neither group experienced mortality or required dialysis.

Although not considered standard of care at this time, prophylactic use of IV magnesium (pending pre-op labs), along with the recognized benefit of preprocedural hydration and discontinuation of nephrotoxic medications, can be supported in primary PCI patients. Your radiology department is on the cutting edge of protecting these very high-risk patients. —DLC

Q I've heard a lot of talk about all the kidney problems that the sugarcane workers in Central America have. Does anyone know why this is happening?

The unusually high rates of chronic kidney disease (CKD) among sugarcane workers in Central America have been a subject of great interest since National Public Radio (NPR) aired a special on this topic.³ There has been a rising epidemic of CKD in otherwise healthy male farm workers (ages 20 to 50), particularly those who harvest sugarcane.^{4,5} It has been hypothesized that recurrent episodes of acute kidney injury (AKI)—related to dehydration, volume depletion, pollutants, and rhabdomyolysis with inflammatory stress—are the underlying cause.⁵

Sugarcane harvesters typically work nine-hour days, six days per week, in extremely high temperatures and while wearing heavy, hot clothing. Each worker cuts approximately 10 tons of sugarcane daily, since they are paid based on cutting volume. Workers drink between five and 10 L of water during their shifts.

TABLE Acute Kidney Injury Criteria

AKI is defined as any of the following:

Increase in serum creatinine ≥ 0.3 mg/dL within 48 h

Increase in serum creatinine ≥ 1.5 x baseline within 7 d

Urine volume < 0.5 mL/kg/h x 6 h

Source: KDIGO.⁶

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Santos et al designed a study to prospectively examine the effects of burnt sugarcane harvesting on renal function in healthy male farm workers. Twenty-eight men (ages 19 to 39) with no CKD risk factors (diabetes, smoking, obesity, hypertension, illicit drug or alcohol use) were followed for eight months from preharvest to postharvest. Blood samples were collected at the beginning and at the end of the workday and preharvest and postharvest season.⁵

Preseason lab values were normal in all 28 men. But post-season, all workers had elevated creatinine levels, with five meeting the criteria for AKI (see Table at left).^{5,6}

Santos and colleagues identified potential causes for AKI in this population. These included

- Dehydration and volume depletion (episodes of tachycardia, increased urine density, lower urinary/serum sodium, higher hematocrit)
- Rhabdomyolysis (increased creatine kinase at the end of each workday)
- Systemic inflammation (increased white blood count, neutrophils, lymphocytes, and

monocytes during the workday—possibly indicative of an inflammatory burst)

- Other factors (burning of the sugarcane releasing unknown nephrotoxic substances; unreported NSAIDs use)⁵

Compared to workers who showed early signs of CKD, those who developed frank AKI were more likely to have hyponatremia. Recommendations to reduce the problem include consumption of water/salt hydrating drinks, use of appropriate clothing, work-hour limitations, and changes to payment structures (ie, from a volume system to an hourly or daily system). Furthermore, education on the need to avoid alcohol, illicit drugs, and NSAIDs during the harvest season should help to decrease incidence of AKI among these workers. —ECE **CR**

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The National Kidney Foundation Council of Advanced Practitioners' (NKF-CAP) mission is to

serve as an advisory resource for the NKF, nurse practitioners, physician assistants, clinical nurse specialists, and the community in advancing the care, treatment, and education of patients with kidney disease and their families. CAP is an advocate for professional development, research, and health policies that impact the delivery of patient care and professional practice. For more information on NKF-CAP, visit www.kidney.org/CAP

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