

letters to the editor

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Star fruit intoxication with acute kidney injury

Editor – I read with great interest the article by Mike Jones (*Clin Med* June 2012 pp 287–9) on recognising acute kidney injury (AKI). I would like to highlight a less common but important cause of AKI associated with star fruit nephrotoxicity.

Star fruit (*Averrhoa carambola*) is a popular fruit in tropical and subtropical countries, and its consumption is high in Asia, central America and tropical west Africa. Over the years, multiple case series have reported its nephro- and neurotoxicity in chronic kidney disease patients and, more recently, even in people with normal renal function.¹ This potentially fatal condition can be easily missed unless such history of star fruit intake is specifically sought, especially in patients with AKI with no apparent etiology.

The amount of fruit ingested which causes toxicity can be as low as approximately 25 ml, or half a fruit. However, its association with severity of symptoms is poor.² In patients with impaired renal function, a large amount of star fruit ingestion on an empty stomach appears to be a risk factor for toxicity. Onset of symptoms is within a few hours, commonly with intractable hiccups, vomiting and insomnia. In cases of moderate to severe intoxication, neuropsychiatric manifestations such as psychomotor agitation, mental confusion and seizure can occur, which may progress rapidly to coma and refractory status epilepticus, resulting in death.^{1–3}

Star fruit nephrotoxicity is believed to be due to its high oxalate content, which could cause acute obstructive oxalate nephropathy, as found on renal biopsy of affected patients.¹ Experimental study in rats has

suggested that oxalate crystals may provoke AKI by inducing apoptosis of renal epithelial cells.⁴ Prompt treatment with intensified haemodialysis and haemoperfusion, close monitoring and supportive care has been proposed as an effective therapeutic approach. Peritoneal dialysis is ineffective in this condition, especially when there is neurological involvement.² Interestingly, it was reported that intoxicated patients with initial normal renal function had recovered without any dialysis given,¹ which suggests that baseline renal function is a prognostic factor of renal recovery.

In conclusion, star fruit intoxication may be life-threatening and this diagnosis should be considered in patients with unexplained AKI, especially if associated with neuropsychiatric symptoms. Emergency renal replacement therapy may be required for the management of this potentially treatable condition.

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The case for the physician assistant

Editor – During more than 20 years working as a physician in the United States, I have interacted with physician assistants in a wide range of clinical settings. Accordingly, I read with great interest Ross and colleagues' excellent discussion regarding the potential future role of the physician assistant in the NHS (*Clin Med* June 2012 pp 200–6).

For many years I had the privilege of training and then supervising a number of physician assistants on an interventional cardiology team. These physician assistants provided the long-term stability and organisational skills needed to support the smooth running of an often frantic service. In addition, they were key in teaching junior cardiology residents the basics of pre- and post-procedural care of patients undergoing invasive cardiac testing. I have observed a similar clinical utility of physician assistants in other specialist services, such as cardiac surgery and urology.

I would, however, like to offer a word of caution regarding the considerable potential for the misuse (and abuse) of physician assistants by individual physicians. In the fee-for-service model prevalent in the United States, physicians may charge, at physician rates, for services provided by the physician assistant, as long as the physician supervises that service. Unfortunately, in my experience, this supervision is often scanty and superficial, or even non-existent. This is particularly seen in the private practice setting, where physicians employ multiple physician assistants to work directly for them. While such practices may considerably increase the earnings of the physician, they frequently cause distress to the physician assistants who are trained to look for consistent and meaningful direction from their physician.

Consequently, I would urge those educators, physicians and administrators who will be responsible for expanding the number of physician assistants in the NHS, to be very clear in defining the extent and depth of physician supervision. Vigilant and thoughtful supervision of the supervisors themselves is strongly recommended.

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Fluid assessment is critical in acute kidney injury (AKI)

Editor – Muniraju and colleagues highlight the need for education around recognition and definition of acute kidney injury (AKI) at junior doctor level (*Clin Med* June 2012 pp216–21), supporting Ali and Lewington's findings that trainee doctors were frequently unable to define AKI.¹ However, this phenomenon is not restricted to junior doctors. In a survey of 159 doctors in the West Midlands, only 30% of general physician consultants and registrars (n=39) were able to correctly define AKI. In our qualitative analysis of perceived learning needs in AKI, doctors at all levels asked the question 'how much fluid should we give in AKI?' This implies a belief that fluid requirements are static, when in fact it is a dynamic process requiring repeated clinical assessment.

As such, the importance of correctly determining the amount of fluid required to resuscitate the patient with AKI is critical. Junior doctors are often the first people to assess such patients and may prescribe inadequate volumes.² Our survey showed that foundation year (FY) doctors assessed

the jugular venous pulse (JVP) and postural blood pressure (BP) less frequently than did nephrologists (JVP 55% vs 100%, χ^2 p=0.0008; postural BP 6.7% vs 41%, χ^2 p=0.0014). Even nephrologists did not agree on the best method of determining volume status.

Although the evidence base for individual clinical skills in determining intravascular fluid is sparse, there is negligible harm in performing a comprehensive clinical examination compared with invasive monitoring such as measuring the central venous pressure. By using just skin temperature and JVP, shock can be correctly differentiated between septic, cardiogenic and hypovolemic causes in 76% of cases.³ Hence by performing a comprehensive assessment it is likely that this accuracy could be improved.

In our study, 18 separate terms were used to define the routine performed, ranging from 'fluid assessment' and 'volume status' to 'hydration status' and 'circulation'. When there is such variation in terminology it is unsurprising that doctors at all levels miss vital clinical skills that can add a great deal to the practical management of patients with AKI.

We suggest that a standardised routine of fluid assessment be taught and assessed at undergraduate level, on a par with the 'cardiovascular examination' routine that stu-

dents hone throughout medical school. FY1 doctors would be equipped with a robust tool to correctly prescribe fluids that would be applicable to patients under any discipline, with any diagnosis.

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