

Traumatic rhabdomyolysis (crush syndrome) in the rural setting

To the Editor: I read with interest the article entitled 'Traumatic rhabdomyolysis (crush syndrome) in the rural setting'.¹ Crush syndrome from sjambok injury is a uniquely southern African experience.² It is unfortunately commonplace, making treatment guidelines essential to prevent the progression of acute kidney injury (AKI) and subsequent need for renal replacement therapy. The advent of the RIFLE and AKIN criteria in the description and risk stratification of AKI provides a framework from which strategies to prevent ongoing injury can be implemented.³ Their use has become commonplace in critical care and should be implemented in the emergency department.

Careful monitoring of fluid balance is essential, and a paper discussing the ATN and RENAL trial results shows that avoiding a positive fluid balance improves renal recovery times.⁴ Therefore I urge caution in trying to force a diuresis with resuscitation fluids if patients present with anuria/oliguria and do not respond to initial fluid therapy as they can be pushed into fluid overload with subsequent need for ventilatory support.

Alkalinisation of the urine with bicarbonate has been challenged as the standard of care. Evidence for this practice is weak; in 2 083 trauma ICU admissions, Velmahos' group failed to show improvement in outcomes despite urinary alkalinisation.⁵

The use of diuretics in AKI does not improve mortality outcomes and the use of renal replacement therapy.⁶ Mannitol has also been implicated as a cause of AKI in head-injured patients and should be used with caution.⁷

At present, measuring serum creatinine and urine output remain the two best indicators of renal function that are easily available to the clinician. These remain our renal biomarkers of choice until the use of newer renal biomarkers, such as neutrophil gelatinase associated lipocalin and cystatin C, becomes commonplace.⁸

Patient therapy must be individualised, with haemodynamic optimisation and careful monitoring of fluid balance, specifically concentrating on urine output. Care must be taken to avoid nephrotoxic agents such as intravenous contrast and aminoglycosides. Early referral for renal replacement therapy is essential in those not responding to conventional fluid therapy.

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Wood replies: Traumatic rhabdomyolysis is often a result of natural disasters such as earthquakes where patients are crushed by debris. However, rhabdomyolysis associated with interpersonal violence such as sjambok injuries and community beatings is endemic to South Africa. Most of these patients present to district or regional hospitals with limited diagnostic capabilities and no renal replacement therapies such as renal dialysis. Our study¹ suggests that early diagnosis of rhabdomyolysis using clinical examination and blood on urine Dipstix as a surrogate marker are critical in preventing ensuing myoglobin-associated acute renal failure. Key to management is early and aggressive fluid management (a target of 200 - 300 ml/h urine output)² to prevent renal tubule damage. A low urine pH augments myoglobin cast sedimentation³ and renal tubular damage. Some guidelines^{2,3} suggest the use of bicarbonate, with a target urine pH >6.5, to reduce this effect. Caution is advised with bicarbonate therapy since hypocalcaemia, hypernatraemia, systemic alkalosis and potential tetany are potential adverse effects. Most guidelines also recommend the use of mannitol in oliguric patients.^{2,3} Theoretical advantages of mannitol include osmotic diuresis, free radical scavenging and stimulating the release of vasodilatory prostaglandins, enhancing glomerular filtration. The use of mannitol and bicarbonate is controversial, with new evidence suggesting that their use is of no benefit in rhabdomyolysis.

One study showed that bicarbonate and mannitol therapy had no advantage in preventing renal failure over saline diuresis alone.⁴ However, the study was not randomised and patients in the mannitol/bicarbonate group had an overall higher average creatine kinase, suggesting a more

severe pathology. The study indicated that patients with a creatine kinase >30 000 U/l may benefit from mannitol/bicarbonate therapy.^{4,5} The use of mannitol and bicarbonate in this setting should be revisited.

In settings with limited or no renal support such as renal dialysis, preventing renal failure in traumatic rhabdomyolysis is critical. Early detection of rhabdomyolysis and fluid therapy is the cornerstone of renal saving. Patients who do not respond to fluid resuscitation and show an increasing trend toward renal failure as indicated by a climbing serum creatinine may need additional treatment strategies when dialysis services are not an option. They should have their intravascular volume monitored, which in most settings is limited to central venous pressure monitoring, before the use of diuretics such as mannitol is considered. Strict monitoring of urine and serum pH when considering the use of bicarbonate is recommended. Our small observational study in a rural regional hospital showed that patients with suspected rhabdomyolysis can be effectively treated using recommended guidelines where resources are not adequate for renal replacement treatments. Serious clinical dilemmas exist when patients don't respond to fluid therapy alone and show worsening renal functions, and where there is no recourse to services such as renal dialysis.

Large prospective randomised controlled trials are required to provide clarity on the most effective treatment strategies in trauma-associated rhabdomyolysis, especially in the resource-challenged areas to which most patients present.

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