Traumatic rhabdomyolysis (crush syndrome) in the rural setting

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Background. Patients with traumatic rhabdomyolysis (crush syndrome)(CS) secondary to community beatings commonly present to a rural emergency department that has limited access to dialysis services. We describe a retrospective study of patients admitted with a diagnosis of CS to the emergency department of a government hospital in rural KwaZulu-Natal, between November 2008 and June 2009.

Objectives. We assessed identification and management of these patients, considering: *(i)* early adverse parameters used to identify poor prognosis, *(ii)* the importance of early recognition, and *(iii)* appropriate management with aggressive fluid therapy and alkaline diuresis to prevent progression to renal failure.

Methods. Diagnosis was based on clinical suspicion and haematuria. Exclusion criteria included a blood creatine kinase level <1 000 U/l on admission. Data captured included demographics, the offending weapon, time of injury and presentation to hospital, and admission laboratory results. Outcome measures included length of time in the resuscitation unit, and subsequent movement to the main ward or dialysis unit, discharge from hospital, or death.

Results. Forty-four patients were included in the study (41 male, 3 female), all presenting within 24 hours of injury: 27 were assaulted with sjamboks or sticks, 43 were discharged to the ward with normal or improving renal function, and 1 patient died.

Conclusions. Serum potassium, creatinine, and creatine kinase levels were important early parameters for assessing CS severity; 43 patients (98%) had a favourable outcome, owing to early recognition and institution of appropriate therapy – vital in the absence of dialysis services.

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Inadequate policing in KwaZulu-Natal (KZN) and an overloaded judicial system has lead to vigilante justice, where local community protection groups administer corporal punishment for criminal behaviour. Their intentions are to injure, but not kill, to serve as a warning to potential perpetrators.[1,2] Beatings frequently result in traumatic rhabdomyolysis (crush syndrome) (CS), which can rapidly cause acute renal failure (ARF) and death. Early identification of CS involves recognition of characteristic signs, including 'tramline' bruising (distinctive lines suggestive of beating with a sjambok), ecchymosis over large muscle groups, and widespread blunt trauma.

Assault injuries are commonly presented to the emergency department of the Ngwelezane (NGW) Hospital – a tertiary referral centre for northern KZN. New departmental clinical guidelines were introduced, with a focus on early identification of patients with CS, their admission to the critical care/resuscitation area, and aggressive management of hypovolaemia and acidosis. We describe

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a retrospective study of CS patients admitted to the hospital between November 2008 and June 2009.

Myoglobin, potassium (K) and creatine kinase (CK) are released from damaged muscle cells. 'Coca Cola' urine discolouration caused by myoglobin is pathognomonic of CS. As an early prognostic marker, testing for the protein in urine is not always possible in a rural setting; however, the presence of blood (haematuria – determined with a dipstick test) serves as a positive indicator for myoglobin. Myoglobin is converted to ferrihaemate in acidotic conditions (urinary pH <5.6),[3] resulting in sludging and mechanical obstruction in the nephrons; urine alkalinisation with sodium bicarbonate (maintaining pH >6.5) significantly reduces this conversion.[4-10] More than 3 times the normal upper limit of CK in the blood (>1 000 U/l) is essential for a diagnosis of CS.[11] Deteriorating renal function can be monitored with serum urea and creatinine measurements, which will rise with increased renal dysfunction.

Tissue damage also causes significant third-spacing and depletion of intravascular volume, requiring early aggressive fluid volume replacement. This is the cornerstone of treatment recommended by the International Society of Nephrology Renal Disaster Relief Taskforce, with a target urine output of >3 ml/kg/h.[6-10,12] Mannitol can be administered to patients who remain oliguric/anuric despite a central venous pressure (CVP) of 15 cm H_2O.[6] A protocol developed in the early 1990s, suggesting aggressive fluids and diuresis, reduced CS-related mortality from 13 - 15% to 2.5%.[13]

Methods

All patients admitted with a diagnosis of CS to the resuscitation unit of NGW Hospital between November 2008 and June 2009 were included in the study; admission medical records were retrospectively analysed. Research ethics approval was obtained from the NGW Hospital Ethics Committee. Diagnosis was based on clinical suspicion without laboratory tests, i.e. 'tramline' bruising, ecchymosis over large muscle groups and widespread blunt trauma, combined with micro- or macroscopic haematuria. Patients without these criteria, or with irretrievable admission notes, were excluded. Exclusion criteria also included CK levels <1 000 U/l in initial blood samples taken on hospital admission.

Data concerning age, sex, offending weapon, time of assault, and time of presentation to hospital were collected. Admission test results were recorded, including degree of haematuria, urinary pH, serum pH, and serum levels of CK, K, urea, creatinine and amylase. CVP line insertion and the amount of bicarbonate, mannitol and furosemide administered was also documented. Outcome measures included the length of time spent in the resuscitation unit, and subsequent movement to the main ward or dialysis unit, discharge from hospital, or death.

Results and discussion

Forty-four patients were included in the study (41 male, 3 female); average age (mean) was 27 years (range 14 - 53). All patients presented within 24 hours of injury; 20 within 6 hours, 11 within 6 - 12 hours, 6 within 12 - 24 hours, and timing unknown in 7 patients. Similar numbers of patients had suffered beatings with sticks, metal bars, and sjamboks; 2 were beaten with fists, and 4 were injured with an unknown weapon. Recorded outcome measures are summarised in Table I.

Two patients had 'Coca Cola' urine pathognomonic of rhabdomyolysis, and 29 showed haemoglobin (Hb^{4+}) on urine dipsticks, indicative of myoglobinuria. A urinary pH of 5 was recorded in 37 patients on admission; intravenous sodium bicarbonate was administered.

A variety of studies give weight to different predictors of ARF development in initial blood tests; however, urea, creatinine, K, and CK are universally cited.[14,15] Three of the 5 patients who needed prolonged resuscitation (>48 hours) had raised serum creatinine, suggesting that this is a significant indicator of CS severity (Table II).

High urea concentrations were reported in initial blood samples of 8 patients, 1 of whom required prolonged resuscitation. Blood urea can be an indicator of intravascular depletion, but also of recent protein intake. Urea levels in initial samples of 18 patients were lower than the normal range, possibly indicating a poverty-related low-protein diet. Urea was therefore a weak prognostic marker of renal compromise.

All hyperkalaemic patients (5) had elevated CK (>1 725 U/l); a strong correlation between serum K and CK has been documented.[12,15] It may be significant that 3 of the hyperkalaemic patients had some degree of renal failure on admission. Presentation within 24 hours of injury coincides with the time when the risk of hyperkalaemia complications is highest. Hyperkalaemia is thought

Table I. Outcome measures

Outcome		Number of patients (*N*)
Time spent in resuscitation unit (h)	0 – 24	23
	24 – 48	16
	48+	5
Patient outcome	Discharged from hospital	4
	Main ward	38
	Dialysis unit	1
	Death	1

Table II. Details of patients staying >48 hours in the resuscitation unit

Patient	Urine dipstick test	Urine pH	CK nl (38 - 174 U/l)	K nl (3.6 - 5 mEq/l)	Creatinine nl (53 - 115 µmol/l)	Urea nl (3.5 - 6.4 mmol/l)	Patient outcome
1	Hb^{4+}	5	*	2.94	355	14.6	Ward
2	Coca-Cola	5	Too high	nl	nl	nl	Ward
3		5	*	5.9	nl	nl	Ward
4	Hb^{4+}	5	15 685	5.7	200	nl	Dialysis
5	Hb^{4+}	5	1 822	6.3	134	nl	Death

*Not measured.
nl = normal range.

to be the major cause of mortality in CS, and the most important indicator of the need for dialysis,[12] including a strong correlation with creatinine levels.[16]

Interestingly, 3 patients were hypokalaemic on admission; the patient with the lowest K level (2.94 mEq/l) remained in the resuscitation unit for more than 48 hours, and was admitted with a blood creatinine level of 355 µmol/l (Table II). A high rate of hypokalaemia (as opposed to hyperkalaemia) was documented after the Bingöl earthquake in Turkey in 2003: K replacement was necessary in resuscitation efforts; the reason for this is unknown.[17]

The lower limit of serum CK to exclude CS ranges in the literature from 520 - 1 000 U/l.[16,18] In a study of 2 083 ICU trauma admissions, the lowest CK level linked to renal failure was 5 000 U/l; 19% of those with CK above this concentration developed renal failure.[18] Elevated CK levels consistently relate to the need for dialysis.[15,18]

The main cause of morbidity in this study was ARF secondary to CS, and the subsequent need for dialysis. Of 44 patients at risk of CS, 43 (98%) were discharged from the resuscitation unit with normal or improving renal function. In contrast to studies describing earthquake victims where prolonged entrapment is characteristic, all patients presented within 24 hours of injury, which gives the advantage of early intervention, provided that the condition has manifested. This emphasises that clinical suspicion is of key importance in a busy casualty department run by junior staff. Simple tests with urine dipsticks should not be underestimated; this useful bedside test is widely available and easy to use.

Conclusion

Traumatic CS has previously been related to a high incidence of ARF and mortality, especially when dialysis is not readily available. Favourable outcomes of CS cases in this study can be attributed to early recognition by simple means, and skilful management in the ordinary casualty department described.

Clinical signs and screening with urine dipsticks aid identification of at-risk patients. Blood tests to determine K, creatinine and CK concentrations are important for prognostication, alerting the clinician to the severity of rhabdomyolysis and guiding appropriate treatment. Measuring CK is a cheap and reliable means of aiding CS diagnosis, and must be encouraged in all suspected cases. Elevated K and creatinine may suggest impending renal compromise, while urea may be less indicative in a population with a low-protein diet. In all cases, forced alkaline diuresis is indicated.

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References

1. Mistry D. Falling Crime, Rising Fear: 2003 National Victims of Crime Survey. Pretoria: Institute for Security Studies. SA Crime Quarterly 2004;8:17-24.
2. Proctor M, Carter N, Barker P. Community assault – the cost of rough justice. S Afr Med J 2009;99(3):160-161.
3. Forrest M, Southern D, Neal M, et al. Anaesthetic Trauma And Critical Care (ATACC) Course Manual Ed. 6.2. Warrington, UK: ATACC, 2006;209-215.
4. Better OS. The crush syndrome revisited (1940-1990). Nephron 1990;55(2):97-103.
5. Michaelson M. Crush injury and crush syndrome. World J Surg 1992;16(5):889-903.
6. Sever MS, Vanholder R, Lameire N. Management of crush-related injuries after disasters. N Engl J Med 2006;354(10):1052-1063.
7. Better OS. Rescue and salvage of casualties suffering from the crush syndrome after mass disasters. Mil Med 1999;164(5):366-369.
8. Abassi ZA, Hoffman A, Better OS. Acute renal failure complicating muscle crush injury. Semin Nephrol 1998;18(5):558-565.
9. Better OS, Rubinstein I. Management of shock and acute renal failure in casualties suffering from the crush syndrome. Ren Fail 1997;19(5):647-653.
10. Malinoski DJ, Slate MS, Mullins RJ. Crush injury and rhabdomyolysis. Crit Care Clin 2004;20(1):171-192.
11. Ward MM. Factors predictive of acute renal failure in rhabdomyolysis. Arch Intern Med 1988;148(7):1553-1557.
12. Better OS, Rubinstein I, Winaver JM, Knochel JP. Mannitol therapy revisited (1940- 1997). Kidney Int 1997;52(4):886-894.
13. Knottenbelt JD. Traumatic rhabdomyolysis from severe beating — experience of volume diuresis in 200 patients. J Trauma 1994;37(2):214-219.
14. Ensari C, Tüfekçioglu O, Ayli D, et al. Response to delayed fluid therapy in crush syndrome. Nephron 2002;92(4):941-943.
15. Erek E, Sever MS, Serdengeçti K, et al. An overview of morbidity and mortality in patients with acute renal failure due to crush syndrome: the Marmara earthquake experience. Nephrol Dial Transplant 2002;17(1):33-40.
16. Fernandez WG, Hung O, Bruno GR, et al. Factors predictive of acute renal failure and need for hemodialysis among ED patients with rhabdomyolysis. Am J Emerg Med 2005;23(1):1-7.
17. Gunal AI, Celiker H, Dogukan A, et al. Early and vigorous fluid resuscitation prevents acute renal failure in the crush victims of catastrophic earthquakes. J Am Soc Nephrol 2004;15(7):1862-1867.
18. Brown CV, Rhee P, Chan L, et al. Preventing renal failure in patients with rhabdomyolysis: do bicarbonate and mannitol make a difference? J Trauma 2004;56(6):1191-1196.

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