S Afr J Surg. 2025;63:52-55. Online first
https://doi.org/10.36303/SAJS.00570

S Afr J Surg
ISSN 038-2361

TRAUMA SURGERY

Urinary pH as a prognosticator of acute kidney injury in crush syndrome

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Background: Crush syndrome arises from community assault when perpetrators of crime within a community are beaten resulting in muscle damage of varying degrees. The insult to the muscles leads to the development of acute kidney injury which may lead to the need for renal replacement therapy, intensive care unit (ICU) admission for cardiovascular and respiratory support, or death. There are various known methods used to prognosticate patients with crush syndrome. The current literature only provides limited information about the role of urinary pH when it comes to risk stratifying patients with crush syndrome, thus the aim of the study is to reveal whether a urinary pH of 6 or less could potentially predict poor outcomes in those who present with this pathology.
Methods: This paper is a quantitative retrospective review of 107 patients who were diagnosed with myorenal syndrome at Ngwelezana Hospital between September 2021 and September 2023.
Results: Sixty out 107 patients developed an acute kidney injury and three patients died. All participants who died had a urine pH of < 6; however, patients who required dialysis or needed ICU admission had urine pH of 5 and 6 showing no correlation between urine pH and adverse outcomes.
Conclusions: Urine pH may not serve as a marker of complications related to acute kidney injury in patients with crush syndrome but may help to prognosticate patients to predict death.
Keywords: trauma, acute traumatic rhabdomyolysis, myorenal syndrome, crush syndrome, acute kidney injury, renal failure, urine pH, urinary pH

Introduction

With the ever-increasing incidence of crime in South African society, citizens often turn to taking the law into their own hands in the form of community justice.[1,2] Extensive soft tissue injury is often inflicted on victims resulting in acute kidney injury due to traumatic rhabdomyolysis. This acute kidney injury is often worsened by a delay in presentation to the hospital while the victim hides in an effort to avoid further assault, thus missing the opportunity for early and aggressive intravenous fluid resuscitation.[3]

Muscular crushing during beatings with sticks and "sjamboks" leads to rhabdomyolysis, the release of myoglobin and intracellular electrolytes into the circulation.[4] Myoglobin deposition in the proximal tubules of the kidneys results in acute kidney injury. Renal replacement therapy and intensive care unit (ICU) admission are often necessary, with significant morbidity and death in some cases.[5]

The intrinsic pathophysiology of myoglobin-induced acute kidney injury involves three mechanisms – tubular obstruction, renal tubular cell necrosis and vasoconstriction. The latter two mechanisms are, at least in part, induced by the formation of ferrihemate when the urine pH is < 6. Accumulation of this compound has a direct toxic effect on renal tubular cells.[6]

Since an acidic urine pH, via the accumulation of ferrihemate, can be expected to exacerbate acute kidney injury, this study aims to assess the potential role that the initial urinary pH from a bedside urine test may play in predicting the severity of acute kidney injury and the need for renal replacement therapy, and how it compares to the previously used predictors, such as serum venous bicarbonate measurements and initial serum urea and creatinine.

Methods

A retrospective analysis was done on all patients over the age of 18 years admitted to Ngwelezana Hospital with a diagnosis of crush syndrome following community assault from August 2021 to September 2023.

Data from patient records were collected into a password protected Microsoft Excel spreadsheet. Data collected included patient demographics, date of injury, the objects used in the beatings, the extent of beatings, date of presentation to hospital and discharge, initial and follow-up results for parameters, such as creatinine kinase, venous bicarbonate, serum urea and creatinine, urinary pH, as well as information about the development of complications along with the interventions for those complications.

An assessment of correlation between urinary pH and severity of crush syndrome was made using venous bicarbonate levels, serum urea, and serum creatinine as a gold standard.

Statistical analysis

Statistical methodology

IBM SPSS version 28 was used to analyse the data. Nonparametric Mann-Whitney U tests were used to compare delay to hospital and other non-normally distributed variables between outcomes. For normally distributed variables, parametric tests such as paired and unpaired t-tests were used. Spearman's rho nonparametric correlation analysis was used to assess relationships between quantitative variables which were skewed. Acute kidney injury was defined by an increase in serum creatinine levels of 26.5 µmol from the upper limit of normal local laboratory values according to the KDIGO clinical practice guidelines.[7] Fisher's exact tests were used to assess associations between categorical variables.

Results

Patient demographics

There were 107 participants included in the study. The mean age was 29 years (SD 8, range 13–47). The participants were mostly male (95%). Younger men are more likely to be admitted for crush syndrome and its related complications.

Sixty participants (56%) already had acute kidney injury at initial presentation, and this number increased to 63% at 24 hours post presentation. The number of patients who had an acute kidney injury on presentation can be explained by either the average 1-day delay prior to hospital presentation and possibly missing the opportunity for renal tubular myoglobin clearance during fluid resuscitation as soon as possible following injury, or the subsequent dehydration and shock that may take place as a result of trauma of this nature. It may also be possible that these patients had pre-existing kidney disease; however, the majority of patients in this study were young without pre-existing medical conditions. A total of 31% required dialysis. There were three deaths (3%). Complications of crush syndrome are common due to various factors, and they manifest themselves as the development and worsening of acute kidney injury, the need for renal replacement therapy and admission into the ICU.

The median delay to hospital presentation was 1 day (IQR 0–10); 36% of participants presented on the same day while 41% had 1-day delay. It is common for patients to present one day after they have been assaulted.

Factors significantly associated with the need for renal replacement (Table I) included delay to hospital presentation, lower venous bicarbonate levels, and higher serum urea and creatinine levels at the time of admission. Although dialysed patients had higher serum creatine kinase levels, the correlation was not statistically significant. Those who received dialysis remained in hospital for a median of 14 days (IQR 7–18). There was no association between patient age and need for dialysis ($p = 0.341$). In keeping with previous data, the current study revealed that a delay to hospital presentation, decreased initial venous bicarbonate values, and increased initial serum creatinine values are risk predictors of the need for renal replacement therapy. A delay to hospital presentation was associated with a greater number of days on renal replacement therapy and a longer overall hospital stay.

The results of this study further validate the work of Muckart, Skinner, Buitendag and coworkers as far as the use of venous bicarbonate levels as a risk predictor is concerned.[8-10] The data shows that patients who ended up needing renal replacement therapy or ICU admission had lower initial venous bicarbonate levels. The higher the initial venous bicarbonate level, the less likely the patient was to complicate and the shorter the length of their hospital stay.

There was a strong correlation between patients with a higher initial creatinine and creatine kinase levels and the likelihood for complications, such as the need for renal replacement therapy, respiratory and cardiovascular dysfunction requiring organ support and death.

There was a weak positive correlation between the delay to hospital and days on haemodialysis (Spearman's rho = 0.342, $p < 0.001$). This meant that the longer the delay to hospital, the longer the duration on dialysis. There was no association between delay to hospital and death. One can go a step further to interpret this by saying that the more delayed interventions are, the more likely the outcome will be poor.

Although there were a total of 107 patients in this study, only 97 participants with initial urinary pH values, 28 (29%) had a urinary pH < 6. The data for the remaining 10 patients who did not have urine pH values were not included where the statistical analysis of the urine pH was concerned but rather in the analysis of other parameters, such as delay to hospital presentation, serum bicarbonate, urea and creatinine levels. Some patients may not have had their urine pH values charted due to the fact that, routinely, urinalysis would be done to assess for haematuria to indirectly give an indication of the amount of myoglobin in the urine. Initial urine pH is not a priority for physicians referring from district hospitals in the periphery. There was a significant association between urinary pH < 6 and death ($p = 0.023$), since 100% of the participants who died were in this category while none of those with urinary pH ≥ 6 died; this was not compared to a control group. However, there was no significant association between urinary pH < 6 and severity of acute kidney injury or the need for renal replacement therapy.

In-hospital complication rates, including renal replacement therapy, ICU admission for pulmonary oedema,

Table I: Associations with need for renal replacement therapy

	Dialysis – no Median (IQR)	Dialysis – yes Median (IQR)	*p*-value
Delay to hospital (days)	1 (0–10)	2 (1–3)	< 0.001
Creatine kinase (U/L)	4 323 (2307–8019)	7 732 (3801–18139)	0.071
Venous bicarbonate (mmol/L)	23.6 (20–15.8)	18.6 (15.6–21.3)	< 0.001
Urea (mmol/L)	4.95 (3.80–6.80)	12 (8.40–23.70)	< 0.001
Creatinine (µmol/L)	99 (77–141)	247 (186–635)	< 0.001
Days in hospital	3 (1–5)	14 (7–18)	< 0.001

Table II: Associations with in-hospital complications

	No complications Median (IQR)	Any in-hospital complications Median (IQR)	*p*-value
Venous bicarbonate (mmol/L)	23.9 (21.3–25.7)	18.5 (15.6–21.3)	< 0.001
Creatine kinase (U/L)	3 799 (2262–7231)	7 816 (3830–18139)	0.029
Creatinine (µmol/L)	96 (76–120)	241 (159–632)	< 0.001

Table III: Correlations of duration of dialysis and length of hospital stay with blood parameters

			Venous bicarbonate	Creatine kinase	Creatinine
Spearman's rho	**Days on HD**	Correlation coefficient	-0.485	0.229	0.638
		Sig. (2-tailed)	< 0.001	0.048	< 0.001
	Days in hospital	Correlation coefficient	-0.443	0.422	0.732
		Sig. (2-tailed)	< 0.001	< 0.001	< 0.001

Table IV: Associations between blood parameters and initial urine pH

	Initial pH ≥ 6 Median (IQR)	Initial pH < 6 Median (IQR)	*p*-value
Creatine kinase (U/L)	5 912 (2554–8778)	5 849 (3097–16642)	0.787
Venous bicarbonate (mmol/L)	22.2 (18.6–25)	21.3 (17.5–24)	0.739
Urea (mmol/L)	6.4 (4.1–9.85)	7.45 (5.55–12.3)	0.084
Creatinine (µmol/L)	114 (83–206)	153 (99–257)	0.090

haemodynamic instability and hospital-acquired respiratory infections and death, were significantly higher in patients with lower venous bicarbonate values, higher creatine kinase values and higher creatinine values. The complications that were taken into account were the need for renal replacement therapy, ICU admission and death (Table II).

Venous bicarbonate levels correlated negatively with days on dialysis (rho = -0.485) and with days in hospital (rho = -0.443). This means that as venous bicarbonate values decreased, days on haemodialysis and days in hospital increased amongst participants (Table III). In other literature, complications of crush syndrome started to occur at venous bicarbonate levels < 16 whereas this study shows that complications occurred at initial venous bicarbonates as high as 18; this may be explained by metabolic compensatory mechanisms which may have taken place during the delay to hospital presentation.

There was a weak positive correlation between creatinine kinase and days on renal replacement therapy (rho = 0.229) which was statistically significant (p = 0.048). There was a moderate positive correlation between days in hospital and creatinine kinase values (rho = 0.422) which was also statistically significant. Therefore, in general, as the creatinine kinase value increased, so did the days on renal replacement therapy and days in hospital (Table III).

Venous bicarbonate, creatinine kinase, urea, and creatinine values were not different between those participants with initial urinary pH < 6 and those with higher urinary pH. Urine pH did not correlate with other previously used markers of severity for acute kidney injury (Table IV).

Discussion

Community assault resulting in crush syndrome is an ongoing issue that plagues South African society, adding a significant burden to already stretched healthcare services.[1-3]

There are multiple established methods of risk-stratifying patients with crush syndrome to assess severity of the acute kidney injury and predict the need for renal replacement therapy.[7,11,12] Currently the RIFLE (risk, impairment, failure) system, the Acute Kidney Injury Network and the KDIGO (kidney disease, improving global outcomes) scoring system are being used to risk-stratify patients according to urine output, serum urea, creatinine and estimated glomerular filtration rate.[7,12] Venous bicarbonate levels have been studied and validated as an accessible risk stratification tool to predict the severity of acute kidney injury and the need for renal replacement therapy.[8-10,13]

Although this study specifically aims to draw conclusions about whether a urinary pH < 6 correlates with the severity of crush syndrome specifically measured by the extent of acute kidney injury, this study managed to confirm and validate what is already known in the literature with regards to risk-stratifying patients who have crush syndrome.

Physiologically, a lower urine pH which points to renal tubular acidosis leads to a greater accumulation of myoglobin within the renal tubules, an increased number of myoglobin casts which cause the obstruction of the renal tubules and later acute kidney injury.[14] This points to the rationale of this paper's aims. It can be said that urine pH is a surrogate marker for renal tubular function.

In the literature that is currently available regarding the correlation of a more acidic urine pH with acute kidney injury, namely a case series by Mehandru et al.[14] of 32 patients with acute kidney injury secondary to rhabdomyolysis by any mechanism, there was a significant correlation between a lower urine pH and the development of acute kidney injury. Mehandru et al.[14] showed that most patients who had a urine pH < 6.5 subsequently sustained an acute renal injury while there was no renal affectation in patients who had a urine pH > 7 independent of the creatinine kinase levels.

This paper's results did not substantiate the aim of this paper. The only conclusion that can be drawn from the data pertaining to urine pH is that all the patients who died had a more acidic urine pH of < 6 and that those who had a pH

> 6 did not die. This paper did not reveal any significant correlation between a urine pH < 6 and the severity of acute kidney injury. There are various reasons why this may be the case – the measurement of urine pH is dependent on an individual's subjectivity and one's own interpretation of colour, 10 of the 107 patients did not have their urine pH measured which might have led to skewing of the data, and lastly other factors that were not taken into account which may have contributed to the urine having a lower acidity, such as starvation, the amount of intravenous fluids received on presentation, as well has the haemodynamic status of the patient.

It is worth mentioning that hypotension as a proponent of acute kidney injury and the need for renal replacement therapy was not explored even though this factor alone could have had a significant influence on renal function.

The weaknesses of this study include limited numbers and a single-centre focus. Multi-centre data may allow for stronger conclusions to be drawn and provide more generalisability.

Further research on admission urinary pH in this setting may provide a valuable additional tool for risk stratification, especially in resource-limited settings.

Conclusion

This paper reveals that urinary pH may not be useful as a predictor of the severity of acute kidney injury in patients with crush syndrome, although it is known that urine pH can indirectly measure renal tubular acidosis with subsequent acute kidney injury caused by myoglobin deposition in the renal tubules. The latter in turn gives rise to the complications of crush syndrome, leading to worse patient outcomes and an added expense to public health.

Conflict of interest

The author declares no conflict of interest.

Funding source

No funding was required.

Ethical approval

Ethical approval was granted by the Biomedical Research Ethics Committee of the University of KwaZulu-Natal (BCA027/19).

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REFERENCES

1. Governance, public safety and justice survey victims of crime 2018/19. Available from: http://www.statssa.gov.za/publications/P0341/GPSJS_VOCS2018_19.pdf. Accessed 20 October 2022.
2. Rosedale KJ, Wood DL. Traumatic rhabdomyolysis (crush syndrome) in the rural setting. S Afr Med J. 2012;102(5):271. https://doi.org/10.7196/SAMJ.5802.
3. Proctor M, Carter N, Barker P. Community assault - the cost of rough justice. S Afr Med J. 2009;99(3):160-1.
4. Smith WA, Hardcastle TC. A crushing experience - the spectrum and outcome of soft tissue injury and myonephropathic syndrome at an urban South African University Hospital. Afr J Em Med. 2011;1(1):17-24. https://doi.org/10.1016/j.afjem.2011.04.002.
5. Bywaters EG, Beall D. Crush injuries with impairment of renal function. Br Med J. 1941;1(4185):427-32. https://doi.org/10.1136/bmj.1.4185.427.
6. Efstratiadis G, Voulgaridou A, Nikiforou D, et al. Rhabdomyolysis updated. Hippokratia. 2007;11(3):129-37.
7. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephr Clin Pract. 2012;120(4):c179-c184. https://doi.org/10.1159/000339789.
8. Muckart DJ, Moodley M, Naidu AG, Reddy AD, Meineke KR. Prediction of acute renal failure following soft-tissue injury using the venous bicarbonate concentration. J Trauma. 1992;33(6):813-7. https://doi.org/10.1097/00005373-199212000-00003.
9. Skinner DL, Laing GL, Bruce J, Biccard B, Muckart DJJ. Validating the utilisation of venous bicarbonate as a predictor of acute kidney injury in crush syndrome from sjambok injuries. S Afr Med J. 2017;107(5):446-50. https://doi.org/10.7196/SAMJ.2017.v107i5.12213.
10. Buitendag JJP, Patel MQ, Variawa S, et al. Venous bicarbonate and creatine kinase as diagnostic and prognostic tools in the setting of acute traumatic rhabdomyolysis. S Afr Med J. 2021;111(4):333-7. https://doi.org/10.7196/SAMJ.2021.v111i4.14915.
11. Delaney KA, Givens ML, Vohra RB. Use of rifle criteria to predict the severity and prognosis of acute kidney injury in emergency department patients with rhabdomyolysis. J Em Med. 2012;42(5):521-8. https://doi.org/10.1016/j.jemermed.2011.03.008.
12. McMahon GM, Zeng X, Waikar SS. A risk prediction score for kidney failure or mortality in rhabdomyolysis. JAMA Int Med. 2013;173(19):1821. https://doi.org/10.1001/jamainternmed.2013.9774.
13. Baeza-Trinidad R, Brea-Hernando A, Morera-Rodriguez S, et al. Creatinine as predictor value of mortality and acute kidney injury in rhabdomyolysis. Intern Med J. 2015;45(11):1173-8. https://doi.org/10.1111/imj.12815.
14. Mehandru SK, Kaur S, Masud A, et al. Significance of urine pH in predicting renal outcomes for patients with rhabdomyolysis. J Clin Rev Case Rep. 2023;8(8):149-56.