Rhabdomyolysis cases and acute kidney injury: a 10-year experience at a regional tertiary centre

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Abstract

Objectives Rhabdomyolysis is defined as a clinical syndrome due to skeletal muscle injury resulting in the release of muscular cell constituents into the circulation. There is a varied spectrum of severity of rhabdomyolysis. The purpose of this study was to analyse the incidence, aetiologies, biochemical variables and complications in patients with rhabdomyolysis at a regional tertiary hospital over a 10-year period.

Methods A retrospective review of patients from May 2006 to May 2016 with a diagnosis of rhabdomyolysis was undertaken. An episode of rhabdomyolysis was defined as acute elevation of serum creatine kinase (CK) value greater than five times the upper limit of normal (>1000U/L). Data extracted included: patient demographics, aetiology of rhabdomyolysis, peak serum CK, renal function, use of kidney replacement therapy (KRT) and death.

Results Of the 631 patients identified, 248 were excluded based on: a diagnosis of myocardial or cerebral infarction; serum CK levels not meeting the criteria for rhabdomyolysis; and duplicated or inadequate data from medical records. A total of 383 patients, 220 males and 163 females, of median age 71 years, were analysed. The leading cause of rhabdomyolysis was prolonged immobility (71.5%). 194 patients (50.7%) developed an acute kidney injury (AKI), 9.9% required KRT, and 7.6% died.

Conclusion In this 10-year review of 383 cases of rhabdomyolysis, the most common cause was prolonged immobility, rhabdomyolysis was more common among males, and half of the patients developed an AKI.

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Introduction

Rhabdomyolysis is a clinical syndrome characterised by muscle injury leading to the disintegration of muscle constituents into the extracellular fluid and systemic circulation. Serum creatine kinase is the most sensitive marker of muscle damage, and there is a varied spectrum of severity of rhabdomyolysis ranging from a subclinical increase in creatine kinase (CK) levels to life-threatening complications such as muscle oedema, electrolyte disturbances and acute kidney injury (AKI) (Bosch et al., 2009; Khan, 2009; Vanholder et al., 2000).

Rhabdomyolysis has a wide range of causes, with ischaemia being a leading cause. Ischaemic injury interferes with oxygen delivery to the cells causing insufficient adenosine triphosphate (ATP) production and subsequent muscle cell necrosis (Efstratiadis et al., 2007; Zutt et al., 2014). Localised causes of ischaemia include thrombosis, embolism, or clamping of vessels during surgery, whereas generalised causes include hypotension and shock (Khan, 2009; Vanholder et al., 2000). Intense muscular exertion results in exhaustion of cellular energy supplies due to increased demand and leads to a disruption of muscle cell membranes. Consequently, this leads to the influx of calcium into the cytoplasm, which eventually results in rhabdomyolysis (Efstratiadis et al., 2007; Khan, 2009). Hypokalaemia, extreme heat, sickle-cell trait, an untrained individual, and pre-exertion fatigue increase the risk of exertional rhabdomyolysis (Khan, 2009).

Toxins and drugs play a part in most cases of rhabdomyolysis, and the myotoxins most commonly implicated in rhabdomyolysis are the statin group of medications (Warren et al., 2002). The proposed mechanisms of statin-induced myopathy and rhabdomyolysis include myocyte membrane dysfunction from decreased cholesterol synthesis and reduced cellular ATP production due to depletion in ubiquinone and mevalonic acid (Efstratiadis et al., 2007; Omar et al., 2001). Alcohol can cause rhabdomyolysis either via direct myotoxicity, electrolyte and acid-base disorders, or prolonged immobilisation from alcohol intoxication (Efstratiadis et al., 2007; Guis et al., 2005).

Neuroleptic malignant syndrome (NMS), a rare but life-threatening condition, is due to dopaminergic blockade secondary to antipsychotic medications and is characterised by massive generation of heat by fever, rigidity and tremor causing muscle breakdown and rhabdomyolysis (Warren et al., 2002). Crush injuries due to trauma are associated with extensive muscle necrosis with leakage of the muscle fibre contents into the systemic circulation leading to rhabdomyolysis (Khan, 2009; Omar et al., 2001).

AKI is a serious complication of rhabdomyolysis. Renal vasoconstriction, a pathognomonic feature of rhabdomyolysis-induced AKI, is a result of intravascular volume depletion due to fluid sequestration within damaged muscle and consequent activation of the sympathetic nervous system, antidiuretic hormone and the renin-angiotensin system. Other pathophysiologic mechanisms known to contribute to AKI in rhabdomyolysis include direct tubular toxicity mediated by myoglobin-associated oxidative injury and tubular obstruction due to formation of myoglobin casts (Bosch et al., 2009; Warren et al., 2002). While various clinical factors such as serum CK and urine myoglobin have been used to predict the risk of rhabdomyolysis-induced AKI, no single parameter has been established as predictive (Khan, 2009).

Various large and small population studies on rhabdomyolysis have been conducted in the last few decades, but not in regional Australia (Blanco et al., 2002; Gabow et al., 1982; Huynh et al., 2016; McMahon et al., 2013; Melli et al., 2005; Veenstra et al., 1994). University Hospital Geelong is a 406-bed hospital located in Geelong, Victoria. It is the only regional tertiary teaching hospital in Victoria, Australia with approximately 70,000 admissions per year. University Hospital Geelong serves both the local Geelong population of 193,000 and the city of Greater Geelong catchment area of 247,000 people (Population Australia, 2020; City of Greater Geelong, 2019). The purpose of this study was to analyse the incidence, aetiologies, biochemical variables and complications of rhabdomyolysis using retrospective clinical and laboratory data from University Hospital Geelong.

Methods

Utilising relevant International Classification of Diseases 10th Revision (ICD-10) discharge diagnosis codes, we retrospectively reviewed electronic medical records of patients admitted with a diagnosis of rhabdomyolysis over a 10-year period from 1 May 2006 to 30 April 2016 at University Hospital Geelong. All laboratory tests during the study period were performed by a single pathology provider. This study was exempt from ethical approval by the Barwon Health Human Research Ethics Committee.

Data collection

Data collected included patient demographics, aetiology of rhabdomyolysis, details of AKI and mortality, kidney replacement therapy (KRT), intensive care unit (ICU) admission, peak serum CK level, peak serum creatinine and, when available, results of urine myoglobin testing and repeat serum creatinine at 12 months. For patients with more than one admission with rhabdomyolysis, only the first admission was included.

Definitions

Using the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, AKI was defined as an increase in serum creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days or an increase in serum creatinine by ≥26.5mmol/L within 48 hours. The severity

of AKI was then categorised into Stage I (1.5–1.9 times baseline creatinine), Stage II (2.0–2.9 times baseline creatinine) and Stage III (3.0 times baseline creatinine) (Kellum et al., 2012). Pre-hospitalisation serum creatinine or, if not available, the lowest serum creatinine obtained during admission, was used as the baseline value. An episode of rhabdomyolysis was defined as an acute elevation of serum CK value greater than five times the upper limit of normal (>1000U/L). We excluded patients who did not meet this criterion and those with CK elevation secondary to myocardial or cerebral infarction. The aetiology of rhabdomyolysis was classified into one of the following groups – prolonged immobilisation, infection, ischaemic injury, drugs and toxins, excessive physical exertion, hyperthermia, trauma, inherited myopathy, unknown aetiology, and miscellaneous causes. In patients with rhabdomyolysis due to multiple causes, only one causative factor with the closest clinical relationship to the episode of rhabdomyolysis was allocated for categorisation purposes.

Data analysis

Study variables were entered into a spreadsheet (Microsoft Excel, Richmond, Oregon) where all results were collated. Continuous variables were expressed by mean and standard deviation (SD) or median and interquartile range (IQR), while categorical variables were reported as numbers and percentages. Characteristics of patients with CK level >10,000U/L were compared to those with CK level <10,000U/L and the group who developed rhabdomyolysis-associated AKI were compared to the group without AKI. Pearson's chi-squared test was applied to establish the significance of associations between the variables for these groups. Statistical analysis was performed using SPSS software (IBM, New York). A p value of <0.05 was considered statistically significant.

Table 1. Baseline characteristics of patients with rhabdomyolysis

Characteristics	n=383
Age, median (IQR) (years)	71 (50–83)
Sex, male/female (%)	57.4/42.6
Length of hospital stay, median (IQR) (days)	8 (4–16)
Intensive care unit admission, n (%)	115 (30%)
Acute kidney injury, n (%) Stage I Stage II Stage III	194 (50.7%) 88 (23.0%) 51 (13.3%) 55 (14.4%)
Kidney replacement therapy, n (%)	38 (9.9%)
Peak serum creatinine, mean (SD) (µmol/L)	212 (±134)
Peak serum CK, median (IQR) (U/L)	4808 (1994–14275)
Urine myoglobin positive (%)	47.8%
Repeat serum creatinine at 12 months, n (%)	112 (29.2%)
In-hospital mortality, n (%)	29 (7.6%)

CK, creatine kinase; IQR, interquartile range; SD, standard deviation.

Results

Patient characteristics

A total of 631 patients were identified during the study period. Of these, 248 patients were excluded based on: a diagnosis of myocardial or cerebral infarction (9); serum CK not fulfilling the criteria for rhabdomyolysis (209); repeated admissions (9); and insufficient clinical information (21). 383 patients therefore met the case definition of rhabdomyolysis. The median age was 71 years (IQR 50–83), with 220 (57%) males. The median length of hospital stay was 8 days (IQR 4–16) and 115 patients (30%), required ICU admission. The median peak serum CK was 4808U/L (IQR 1994–14275). Of the 67 available urine myoglobin results in established rhabdomyolysis cases, nearly half were positive. 56.3% of patients with positive urine myoglobin developed AKI. The mean peak serum creatinine was 212µmol/L (±134). Serum creatinine results at 12 months were available for 112 patients (29.2%). 38 patients (9.9%) required KRT. The inpatient mortality rate was 7.6% (Table 1).

Causes of rhabdomyolysis

Prolonged immobilisation was the most common cause of rhabdomyolysis (71.5%), with 62.8% of cases related to a fall (Table 2). Infection was the second leading cause among 23 patients, with the majority due to respiratory tract infection (43.5%). The ischaemic injury category included 12 patients with lower limb ischaemia and two with abdominal compartment syndrome. Among drug and toxin-induced cases, statins were the predominant culprit among all myotoxins. Among cases due to excessive physical activity, 15 were due to strenuous exercise and one was due to amphetamine-induced psychosis and hyperactivity. Hyperthermia manifested by NMS and heat stress were each identified in five cases. Most cases of trauma-induced rhabdomyolysis (77.8%) were due to crush injury. In the inherited myopathy group, there were two cases of Duchenne muscular dystrophy and one with McCardle disease. 12 cases were categorised as miscellaneous, including patients with seizures, hypothermia, critical myopathy, ruptured aortic aneurysm, and spontaneous limb haematoma. No cause was identified in eight patients (2.1%). Patients with rhabdomyolysis due to prolonged immobilisation were older compared to the other patient groups and mean CK values were highest in the miscellaneous group. Patients with rhabdomyolysis due to infection had the highest mortality.

AKI and serum CK values

The incidence of AKI was 50.7%, which was classified into Stage I (88 patients, 45.4%), Stage II (51 patients, 26.2%) and Stage III (55 patients, 28.4%) (Table 1). The patients with the highest rate of AKI were those cases of rhabdomyolysis caused by hyperthermia. Characteristics of patients with AKI were compared with those without AKI (Table 3). The median

age of the group with AKI was older with longer length of stay and more patients in this group required ICU admission. The mortality rate was also higher in those with AKI compared with the non-AKI group.

A total of 259 patients had a CK level below 10,000U/L, while 124 patients had a CK level above 10,000U/L. To assess the effects of CK concentration on morbidity and mortality, the group of patients with CK level >10,000U/L were compared to

Table 2. Causation of rhabdomyolysis and relevant patient characteristics

Aetiology	No. of patients (n) (%)	AKI (n) (%)	Median age (years)	Median CK (U/L)	ICU admission (n) (%)	Mortality (n) (%)
Prolonged immobility	274 (71.5%)	134 (48.9%)	79	3501	63 (23%)	18 (6.6%)
Fall	172 (44.9%)					
Alcohol intoxication	29 (7.6%)					
Unknown cause	24 (6.2%)					
Vasovagal	18 (4.7%)					
Opioid overdose	11 (2.9%)					
Hypoglycaemia	8 (2.1%)					
Benzodiazepine overdose	7 (1.8%)					
Cardiac arrhythmia	5 (1.3%)					
Infection	23 (6.0%)	15 (65.2%)	58	4942	18 (78.3%)	6 (26.1%)
Pneumonia	10 (2.6%)					
Sepsis of unknown origin	4 (1.0%)					
Necrotising fasciitis	3 (0.7%)					
Urosepsis	2 (0.5%)					
Tetanus	1 (0.3%)					
Pyomyositis	1 (0.3%)					
Leptospirosis	1 (0.3%)					
Cholangitis	1 (0.3%)					
Excessive physical exertion	16 (4.2%)	2 (12.5%)	31.5	27901	2 (12.5%)	0 (0%)
Strenuous exercise	15 (3.9%)					
Amphetamine-induced hyperactivity	1 (0.3%)					
Ischaemia	14 (3.7%)	9 (64.3%)	71	23009	12 (85.7%)	2 (14.3%)
Lower limb ischaemia	12 (3.2%)					
Abdominal compartment syndrome	2 (0.5%)					
Drugs and toxins	14 (3.7%)	11 (78.6%)	63.5	12510	6 (42.9%)	2 (14.3%)
Atorvastatin	5 (1.3%)					
Rosuvastatin	4 (1.1%)					
Simvastatin	1 (0.26%)					
Simvastatin and diltiazem	1 (0.26%)					
Clenbuterol	1 (0.26%)					
Suxamethonium	1 (0.26%)					
Snakebite	1 (0.26%)					
Hyperthermia	10 (2.6%)	8 (80%)	47	14108	2 (20%)	0 (0%)
Neuroleptic malignant syndrome	5 (1.3%)					
Heat stress	5 (1.3%)					
Trauma	9 (2.3%)	3 (33.3%)	30	3649	2 (22.2%)	0 (0%)
Crush injury	7 (1.8%)					
Knee dislocation	1 (0.2%)					
Elbow dislocation	1 (0.2%)					
Inherited myopathy	3 (0.8%)	1 (33.3%)	5	14645	1 (33.3%)	0 (0%)
Duchenne muscular dystrophy	2 (0.5%)					
McCardle disease	1 (0.3%)					
Unknown cause	8 (2.1%)	4 (50%)	56.5	10459	3 (37.5%)	1 (12.5%)
Miscellaneous	12 (3.1%)	7 (58.3%)	33.5	18331	6 (50%)	0 (0%)
Seizure	4 (1.0%)					
Hypothermia	4 (1.0%)					
Critical myopathy	2 (0.5%)					
Ruptured aortic aneurysm	1 (0.3%)					
Spontaneous limb haematoma	1 (0.3%)					

AKI, acute kidney injury; CK, creatine kinase; ICU, intensive care unit.

those with CK level <10,000U/L (Table 4). A higher incidence of AKI and a higher proportion of patients requiring ICU admission and KRT were noted in the group with serum CK exceeding 10,000 U/L. However, there was no difference in mortality.

Discussion

This study describes the spectrum of clinical and biochemical findings among 383 patients with rhabdomyolysis and analyses the factors predictive of morbidity and mortality compared with what is available in the literature. Similar to the literature, our study had a predominant male distribution; however, the median age of our patients was older than those in other studies (Huynh et al., 2016; Melli et al., 2005; Veenstra et al., 1994). In our study, prolonged immobilisation was the most common cause of rhabdomyolysis, while in other studies, drugs and toxins predominated among causes of rhabdomyolysis (Gabow et al., 1982; Koffler et al., 1976; Melli et al., 2005). In a large series of 2371 patients, trauma was the leading aetiology, while in other studies, sepsis and ischaemia have been reported to be the most frequently associated clinical conditions (Blanco et al., 2002; McMahon et al., 2013; Veenstra et al., 1994). These differences may reflect the different case ascertainment and inclusion criteria of each study. For example, some studies only analysed patients in a single aetiologic category, while others focused on rhabdomyolysis-associated AKI, which could have influenced the frequency of aetiologies (Blanco et al., 2002; Eneas et al., 1979; Huynh et al., 2016; Koffler et al., 1976). Our study, however, included patients meeting the aforementioned diagnostic criteria for rhabdomyolysis irrespective of causation and renal function.

Rhabdomyolysis attributable to prolonged immobilisation is well described. Constant and prolonged compression of blood supply to one or more limbs causes intracompartmental pressure high enough to cause obstruction of blood flow, leading to myocyte injury and tissue swelling, which in turn further raises the intracompartmental pressure (Efstratiadis et al., 2007; Gabow et al., 1982; Penn et al., 1972; Szewczyk et al., 1998). Falls were the cause most strongly associated with prolonged immobility in our study, and patients with prolonged immobility as a cause of rhabdomyolysis in this study were older given that the risk of falling increases with age (Deandrea et al., 2010; Nitz & Low Choy, 2008).

The mechanisms of infection-induced rhabdomyolysis include direct muscle invasion, production of myotoxins and cytokine-mediated muscle proteolysis (Guis et al., 2005; Khan, 2009; Knochel, 1993). Among patients with infection causing rhabdomyolysis, microorganisms were isolated in 52.2% of cases. Similar to the findings of Betrosian et al. (1999), our study has shown a predominance of gram-positive organisms, specifically *Staphylococcus aureus*, which is one of the most common rhabdomyolysis-associated gram-positive bacteria (Betrosian et al., 1999). However, other studies on infection-related rhabdomyolysis reported a majority of gram-negative pathogens (Blanco et al., 2002; Kumar et al., 2009; Singh & Scheld, 1996). Statins, especially atorvastatin and rosuvastatin, were the most commonly implicated myotoxins leading to rhabdomyolysis in our study. This is likely because they have longer elimination half-lives exposing patients to higher risk of adverse effects and, secondly, they are two of the most frequently prescribed lipid-lowering agents in Australia (Schachter, 2005; NPS MedicineWise, 2016). The likelihood of rhabdomyolysis increases greatly when statins are co-administered with medications that interfere with their cytochrome P450 3A4 (CYP3A4) metabolic pathway, as

Table 3. Characteristics of patients with rhabdomyolysis with and without acute kidney injury

Characteristics	Non-AKI, n=189	AKI, n=194	P-value
Age, median (IQR) (years)	71 (43–82)	72 (52–84)	0.047
Mortality, n (%)	6 (3.2%)	23 (11.9%)	0.001
CK, median (IQR) (U/L)	4190 (1763–10620)	5785 (2169–18057)	0.088
ICU, n (%)	37 (19.6%)	78 (40.2%)	<0.001
LOS, median (IQR) (days)	7 (4–13)	11 (5–19)	0.001

AKI, acute kidney injury; IQR, interquartile range; CK, creatine kinase; ICU, intensive care unit; LOS, length of stay.

Table 4. Comparison of clinical characteristics between serum CK <10,000U/L and serum CK >10,000U/L

Characteristics	CK <10,000U/L (n=259)	CK >10,000U/L (n=124)	P-value
Age, median (IQR) (years)	79 (60–85)	58 (35–72)	<0.001
AKI, n (%)	122 (47.1%)	72 (58.1%)	0.088
ICU admission, n (%)	52 (20.1%)	63 (50.8%)	<0.001
KRT, n (%)	10 (3.9%)	28 (22.6%)	<0.001
Mortality, n (%)	20 (7.7%)	9 (7.3%)	0.76

AKI, acute kidney injury; CK, creatine kinase; ICU, intensive care unit; IQR, interquartile range; KRT, kidney replacement therapy.

demonstrated in one of our patients who received simvastatin concurrently with diltiazem, a CYP3A4 inhibitor (Ogu & Maxa, 2000; Omar et al., 2001).

Our study also included cases of rhabdomyolysis secondary to snake venom, clenbuterol and suxamethonium, all of which have been previously reported (Efstratiadis et al., 2007; Grimmer et al., 2016; Le Puura et al., 2000; McKishnie et al., 1983; Warren et al., 2002). All patients in our study with alcohol-related rhabdomyolysis presented following prolonged immobilisation, which is in keeping with other studies (Efstratiadis et al., 2007; Guis et al., 2005). While inherited muscular disorders represent a very small percentage of cases of rhabdomyolysis overall, patients with recurrent rhabdomyolysis warrant further investigation for an underlying metabolic myopathy or muscular dystrophy (Lahoria & Milone, 2016; Tein et al., 1990; Tonin et al., 1990).

In our study, the incidence of AKI was 50.7%, at the upper limit of reported rates of 13–50% (Bosch et al., 2009; McMahon et al., 2013; Melli et al., 2005; Ward, 1988). The higher incidence of AKI in our study is potentially due to the older patient population, as the elderly are particularly susceptible to the development of AKI (Abdel-Kader & Palevsky, 2009; Chao et al., 2014). However, the actual incidences of AKI in rhabdomyolysis in previous studies may be difficult to determine owing to the differences in patient selection and the use of non-uniform definitions. In our study, patients with hyperthermia-induced rhabdomyolysis had the highest rate of AKI. In contrast, sepsis, ischaemia, illicit drugs and alcohol were among causes most strongly associated with the development of AKI in previous studies (Melli et al., 2005; Veenstra et al., 1994; Ward, 1988). In addition, we did not find a statistically significant association between peak serum CK and AKI. Only loose correlations have previously been found (Bosch et al., 2009; Melli et al., 2005; Ward, 1988). This suggests that factors other than peak serum CK such as older age, toxins, sepsis or other risk factors for AKI such as dehydration could predispose to an increased risk of developing AKI in rhabdomyolysis.

Reported mortality rates in rhabdomyolysis vary widely from 3.4–32% depending on the study population, causative factors and comorbidities (Bosch et al., 2009). In accordance with the findings of previous studies, we found a higher mortality illustrated among patients with AKI compared to patients without AKI. In this study, urine myoglobin in established cases of rhabdomyolysis were positive in 48% of cases tested, while a previous study showed a lower rate of 19% positive, indicating the low sensitivity of this test in rhabdomyolysis (McMahon et al., 2013; Melli et al., 2005; Veenstra et al., 1994; Ward, 1988). Although myoglobin is the true pathogenic factor in rhabdomyolysis-induced AKI, it is infrequently measured directly in plasma or urine due a variety of reasons. The rise in serum myoglobin precedes the rise in CK and myoglobin is rapidly and unpredictably eliminated by hepatic metabolism. As a result, serum and urine levels may have normalised by the time of hospital presentation. Therefore, the absence of myoglobinuria does not exclude the diagnosis of rhabdomyolysis (Melli et al., 2005; Gabow et al., 1982).

Aggressive intravenous rehydration is considered the mainstay of therapy in preventing AKI in rhabdomyolysis. Fluid resuscitation expands the intravascular volume which induces diuresis and clearance of toxins, resulting in the reduction in cast formation and obstruction of proximal tubules (Efstratiadis et al., 2007; Khan, 2009). Nursing care for patients with rhabdomyolysis includes sequential monitoring of vital signs as well as urine output to guide fluid resuscitation and to identify oliguria or anuria. In patients with heart disease, pre-existing renal disease or advanced age, close monitoring for signs and symptoms of fluid overload is essential. The use of indwelling urinary catheter allows accurate hourly measurement of urine output. Serum CK and electrolytes must be monitored closely, and any life-threatening electrolyte disturbances such as hyperkalaemia should be promptly addressed. (Criddle, 2003; Khan, 2009). Cardiac monitoring with the involvement of coronary care nurses is required in those with cardiac arrhythmia secondary to hyperkalaemia. Patients with severe rhabdomyolysis associated with severe organ dysfunction or metabolic derangements often require intensive care. KRT is indicated in patients with AKI associated with intractable hyperkalaemia, uremic encephalopathy or pericarditis, severe metabolic acidosis or refractory fluid overload, necessitating input from renal and intensive care nurses. Other nursing interventions include monitoring of compartment pressures in injured extremities, treatment of hyperthermia or hypothermia, pain assessment, provision of antibiotic therapy and administration of antivenom as indicated by each specific cause (Criddle, 2003; Efstratiadis et al., 2007; Khan, 2009). Therefore, nursing staff, irrespective of their specialties, play an instrumental role in the management of this potentially life-threatening condition.

Comparing another large Australian study on rhabdomyolysis carried out in a metropolitan healthcare service, in which 528 patients with CK levels >1000U/L were included, similar patient characteristics and findings were noted – median age in the seventh decade, male preponderance, almost half of patients were found to have AKI using the 2012 KDIGO criteria. Pressure injury was the most common cause, followed by drugs and toxins. Inpatient mortality rate 8.9%, slightly higher than our study of 7.6% (Lim et al., 2020). To the best of our knowledge, our study is the first on rhabdomyolysis conducted in regional tertiary healthcare centre in Australia. Further studies involving Australian cohorts would be informative.

The limitations of this study include that it was retrospective, with case finding reliant on medical diagnostic codes which may be inaccurate. Despite this, we believe that our review of the medical records of all cases identified by coding was sufficient to ensure that cases not fulfilling criteria for rhabdomyolysis were excluded. We acknowledge that volume status and urine output, which are important clinical parameters of kidney failure, were not all comprehensively recorded in cases of rhabdomyolysis-associated AKI. Finally, this study involved patients from a single regional tertiary referral centre, hence the results may not be generalisable to other healthcare centres in different geographical locations.

Conclusion

We describe the patient characteristics, aetiologies and outcomes of rhabdomyolysis in a cohort of patients in a tertiary care hospital over a 10-year period. In our study, rhabdomyolysis was more common among males and the most common cause was prolonged immobilisation. Half of our patients acquired an AKI, 9.9% required KRT and 7.6% died during their inpatient stay, indicating that rhabdomyolysis is associated with moderately high rates of morbidity and mortality. The degree of CK elevation has a weak correlation with the development of AKI.

Conflict of interest

The authors declare no conflicts of interest.

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