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Changing spectrum and outcomes of rhabdomyolysis-induced acute kidney injury: A 35 years update form tertiary care unit

Abstract

Background: The skeletal muscle injury and the release of myoglobin into circulation can cause acute kidney injury (AKI), this could be exertional, traumatic, or non-traumatic, non-exertional. We hereby report AKI in association with rhabdomyolysis, its pattern over 35 years among causes, course of illness, management, and outcome.

Methods: This is a retrospective cohort study performed at a tertiary renal care center. From January 1990 to December 2024, case records of all patients who satisfied the KDIGO definition of AKI, developing it after Rhabdomyolysis, were reviewed, and data were collected and analyzed. Rhabdomyolysis was suspected on history and clinical findings and confirmed when Creatine Kinase levels were more than 3 times the maximum reference range. Other causes of AKI were excluded. Results were analyzed on SPSS, and parameters were compared among those who showed complete renal recovery with both; those who died during acute illness or did not reveal complete renal recovery/ lost follow up before 90 days from the start of illness.

Results: A previous report on 25 years of these patients (334 in number) was published. During last 10-year period, another 121 cases of Rhabdomyolysis-associated AKI were seen. Mean age was 28.22±11.22 and 33.59±14.09 years (in the previous 334 and current 121 patients, respectively). We compared causes of rhabdomyolysis, course of illness, and outcome over decades. Most prominent changes among causes were that torture-related rhabdomyolysis was more common from 1990-2009, while paraphenylene diamine poisoning (PPD) causing toxic rhabdomyolysis was very high between 2010 and 2014. The rest of causes show little variations. Among outcome main change in pattern was a higher number of patients (20%) lost follow up after first discharge from hospital during last ten years, as compared to the initial 25 years when most of patients were coming for long-term follow-up. Overall, 92% of patients required renal replacement therapy (94% during initial twenty-five years and 88% during last ten years)

Conclusion: Decline in the number of torture-related rhabdomyolysis, which was more during the initial 20 years with political instability, and then sharp rise in PPD-related toxic rhabdomyolysis during 5 years with emotional instability (as in the majority PPD was ingested for self-harm). Later, extensive public awareness campaigns lowered the number of these cases over the last ten years. With expansion of services and increasing number of patient turnover, we are losing control of effective follow-up.

Keywords: (AKI), Acute Kidney Injury; Rhabdomyolysis, (PPD), Para Phenylene Diamine; Torture, (RRT), Renal Replacement Therapy

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Introduction

The word rhabdomyolysis is derived from the Greek words *rhabdos* (rod-like/striated), *mys* (muscle), and *lysin* (release).[1] It is a syndrome resulted from skeletal muscle injury and release of muscle cell contents in the circulation, these contents could be myoglobin, sarcoplasmic proteins or electrolytes. Myoglobin high levels exceed protein binding with haptoglobin and precipitate in glomerular filtrate, causing obstruction of tubular Lumina, as well as direct ischemic tubular epithelial cell injury and intra-renal vasoconstriction, thus resulting in acute kidney injury (AKI).[2]

The most common causes of rhabdomyolysis are crush injuries secondary to trauma or immobilization, blunt trauma, extreme physical exertion, some specific drugs and poisons, alcohol or other substances Bing, animal toxins, metabolic myopathies, viral illness, and electrolyte disorders.[3]

At molecular levels with damage to sarcolemma sheath there occurs impaired impermeability of calcium and sodium ions, thus Na-K pump and Ca transporter function is affected.[4] Intracellular Ca concentration rises which further disrupts cell structure and more leakage of myoglobin, other proteins and metabolites released in circulation. Vascular permeability increases resulting in interstitial edema and decline in systemic intravascular volume.[5]

Myoglobin when filtered through proximal tubules, tubular epithelial cells breakdown heme into iron, biliverdin and carbon monoxide as a result of catalyzation via heme oxygenase-1.[6] After saturation of heme oxygenase-1 catalyzation capacity, excess iron combines with hydrogen peroxide forming ferric (Fe3) iron. Fe3 causes formation of reactive oxygen species (ROS) and peroxidation of tubular cell membrane lipids.[7]

Further down at level of thick ascending limb of loop of Henle, myoglobin precipitates with Tamm-Horsfall protein, these casts

cause obstruction of tubular Lumina at distal tubular cell. With luminal obstruction intra-tubular pressure rises resulting in decline in vascular perfusion and thus glomerular filteration.[8] Experimental micro puncture study in glycerol induced rhabdomyolysis in rats revealed that low intra-tubular pressure failed to flush tubular casts.[9] After rhabdomyolysis, the impaired release of vasodilating nitric oxide also happens in addition to renin angiotensin system mediated vasoconstriction, which occurs in response to hypovolemia and hypotension, as muscle trauma has induced muscular edema.[10]

Presence of myoglobin in tubules can cause apoptosis or necrosis of tubular epithelial cells. Ferro ptosis, a new type of regulated cell death is related to excess iron aggregation in the cell, leading to decreased elimination of lipid peroxides.[11] Also described inadequate free radical scavengers, excessive neutrophil adhesion and cytokine release in association with rhabdomyolysis associated AKI (RAAKI).[12]

Immune mediated mechanisms also studied in RAAKI, as products released from muscle and tubular epithelial cell damage act as immunogenic damage-associated molecular patterns (DAMPs) and activate macrophages and produce inflammatory cytokines. However, the exact role of immune system in RAAKI is still not well understood.

Rhabdomyolysis recognition is centuries old with consumption of birds feed on hemlock plant.[13] Crush injuries during World War II and then major earth quacks reported from different parts of world has reported acute tubular necrosis and pigment casts in renal tubular Lumina on autopsy of patients who acquired crush injuries.[14,15]

From our institution we have previously published our 25-year experience of RAAKI, describing important baseline epidemiological and clinical spectrum of RAAKI in our population.[3] The present study expands upon prior experience and provides if any changing pattern observed in causes of RAAKI over further decade long patient population.

Methods

This is observational retrospective cohort, written in accordance of Strengthening the reporting of Observational Studies in Epidemiology (STROBE) guidelines.[16] The medical records of all the patients developing AKI after Rhabdomyolysis, coming to Sindh Institute of Urology and Transplantation (SIUT), from January 1990 to December 2024 were reviewed. AKI was defined according to Kidney Diseases Improving Global Outcome (KDIGO) guidelines[17] which was applied in retrospect on documented symptoms and clinical findings). Rhabdomyolysis was diagnosed on basis of history and findings of raised Creatine kinase (CK) more than 3 times of upper reference range.

Only AKI patients with Rhabdomyolysis were included, while AKI from other causes or patients with chronic diseases; like chronic kidney disease, long standing hypertension or diabetes mellitus were excluded.

Demographics including age, gender, days of insult (time of first symptom to time of reporting to our institution), clinical symptoms , urine output, and laboratory investigations including hemoglobin, total leucocyte count, platelet count, peripheral film, urea, creatinine, electrolytes, calcium, albumin, lactate dehydrogenase, Creatine phosphokinase, liver function tests, urinalysis, radiological examinations like chest roentgenogram, ultrasound kidneys (in all cases), computerized tomography/magnetic resonance imaging (in selected cases), management with fluid replacement, renal replacement therapies all recorded on Excel sheet for all patients included in study. Renal biopsy was performed in 15 cases, who showed delayed recovery.

Institutional Ethical Review Committee (ERC), granted permission to collect data from case records (SIUT-ERC- 2020/A-216).

Statistical analysis

All analyses were performed using SPSS version 22.0. Continuous variables are presented as mean ± standard deviation (SD), and categorical variables as counts and percentages.

The primary outcome was categorized into three groups: complete renal recovery, death, non- recovery (which includes both partial recovery and persistent requirement of renal replacement therapy or lost follow up before 90 days of insult). P value calculated using Pearson Chi-Square test while comparing complete renal recovery vs death and complete renal recovery vs non-recovery. Fisher's Exact test was applied when number of patients found <5 in any comparing parameter.

Results

Data from all AKI cases; for different causes was collected for 35 years, and from this pool of all cause AKI, RA-AKI separated out. Patients' details from January 1990- to December 2014 (25 years) were previously analyzed and published. Current study adds patients' information brought between January 2015 to December 2024 (ten years), 121 patients with AKI along with rhabdomyolysis were dealt at this institution. Here we also compare any difference in pattern of causes and outcome over 3.5 decades. Among current population 110 (90.9%) were males and 11(9.09%) were female. Presenting symptoms varied from muscle aches, loin pain, nausea, vomiting, altered sensorium, change in color of urine, decline in urine output (<400/24 hours in 81 and <100/24 hours in 11). While signs varied from multiple bruises, crush injury, fracture/ s, bullet marks, tender muscles, fever, shortness of breath etc. Demographics and laboratory results on day of admission to this institution are given in Table 1.

Table 1 Demography and initial laboratory results (n=121)

Parameter	Mean ±SD	Range
Age (years)	33.59±14.09	18-71
Duration from first symptom to reaching this institution (Days)	8.43±4.60	Jan-28
Hemoglobin (11.5-15.4 G/dl)	11.02±2.35	5.4-15.4
WCC (4-11 x103/µl)	14.51±7.58	1.8-49.54
Platelet (150-400 x103/µl)	225.81±119.34	9-621 <150K=37 >450=7
Urea (15-39 mg/dl)	247.42±110.75	79-606
Creatinine (0.5-1.2 mg/dl)	11.08± 5.57	1.4-39.8
Na (135-145 mmol/L)	132.40±10.18	103-172
K (3.5-5.0 mmol/L)	5.45±1.18	2.7-8.9
HCO3 (22-30 mmol/L)	15.94±5.61	4-35
LDH (140-280 U/L)	1823.79±2020.85	184-13022
CK (30-170 U/L)	25179.10±58779.008	1062-544800
Calcium (8.5-10.2 mg/dL)	7.67±1.45	4.8-13
Albumin (3.5-5.5 g/DL)	3.03±0.62	1.5-5.5
SGOT /AST (8-45 U/L)	689.88±1212.47	13-7806
SGPT/ ALT (7-56 U/L)	469.34±848.73	9-7230

WCC, White Cells Count; Na, Sodium, K, Potassium; HCO3, Bicarbonate; CK, Creatine Kinase; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; Reference range in parenthesis

Rhabdomyolysis divided in traumatic, non-traumatic exertional and non-traumatic non-exertional and cause for all these groups are given

in Table 2, while Table 3 provides comparison among contribution in each group seen at this institution over a span of 3.5 decades, past 25 years' data taken from our previous published study. Renal biopsy was performed only in those who showed delay in recovery they were 15 patients and findings were acute tubular necrosis in 3, acute tubular necrosis with pigment cast in 11, acute tubulo-interstitial nephritis with pigment cast in one patient. Majority of patients were in state of advanced uremia, 88.42% requiring renal replacement on arrival, intermittent hemodialysis in 105 and sustained low-efficiency dialysis (SLED) in 2. Rest of 13 patients managed conservatively with fluids. One person was sick enough died before starting renal replacement therapy (RRT). In our past experience RRT on arrival was required in 94% population with RAAKI.

Table 2 Causes of Rhabdomyolysis during last ten-year population (n=121)

Traumatic	Non-traumatic exertional	Non-traumatic, non-exertional
RTA = 17	Prolonged exercise = 27	Poison PPD =3
Crush Injury = 2	Recurrent convulsions =14	Alcohol or other substance Bing = 4
Blunt trauma / torture = 17		Infections = 29
Fire arm injury = 2		Drugs = 5
		I/M injection =1

RTA, Road Traffic Accidents; PPD, Para Phenylene Diamine; I/M, Intra Muscular

Table 3 Pattern over 3.5 decades at same institution

Category of Rhabdomyolysis	1990-1999 N=67	2000-2004 N= 35	2005-2009 N=52	2010-2014 N= 180	2015-2024 N=121	Total 35 years N=455
Traumatic	42 (62)	20 (57)	32 (62)	32 (18)	38 (31)	164 (36)
Non-traumatic exertional	21 (31)	10 (28)	13 (52)	9 (5)	41 (34)	94 (21)
Non-traumatic, non-exertional	4 (6)	5 (14)	7 (13)	139 (77)	42 (35)	197 (43)

% in parenthesis (25 year results from previous article)[3]

Statistical significance of different parameters calculated among those who showed complete renal recovery vs those who died during acute phase of illness, it was found that need for mechanical ventilation, severe acidosis (venous bicarb <10 mmol/L) showed significance to high mortality. Chi-Square test or in parameters when number of patients was <5 Fisher's Exact test was applied. (Table 4) Similar parameters also compared among those who showed complete renal recovery vs those who survived but either have not shown complete renal recovery till 90 days of insult or lost follow up before 90 days, statistically no parameter was found significant, elevated International Normalized Ratio (INR) was seen more frequently in renal recovery group while none had in non-recovery group. (Table 5) Hypocalcemia was seen in almost all except two patients, with a median value of 7.64 mg/DL.

Table 4 Parameters differences among recovered and died patients

Parameters	Recovered (n=74)	Died (n=17)	P value*
Oliguria	68 (92)	14 (82)	0.235
Anuria	7 (9)	2 (12)	0.774
Thrombocytopenia	20 (27)	5 (29)	0.094
Elevated INR (≥1.2)	7 (9)	8 (47)	0.019
Hyperkalemia	43 (58)	12 (71)	0.343
Hypocalcemia	9 (12)	5 (29)	0.675
Hypoalbuminemia	13 (18)	9 (53)	0.412
Raised Serum Bilirubin	17 (23)	7 (41)	0.102
Raised liver enzymes	64 (86)	15 (88)	0.939
Severe Acidosis (venous bicarb <10 mmol/L)	8 (11)	5 (29)	0.035
Acidosis (venous bicarb <22 mmol/L)	63 (85)	14 (82)	0.807
Hemo Dialysis requirement	64 (86)	16 (94)	0.384
Mechanical ventilator requirement	2 (3)	11(65)	<0.001

*Chi-square test / Fisher's exact (applied where numbers were <5), Percentages in parenthesis.

Table 5 Parameters differences among recovered and non-Recovered patients

Parameters	Recovered (n=74)	Non-Recovered (n=30)	P value*
Oliguria	68 (92)	24 (80)	0.085
Anuria	7 (9)	2 (7)	0.646
Thrombocytopenia	20 (27)	8 (27)	0.97
Elevated INR (≥1.2)	7 (9)	0	0.043
Hyperkalemia	43 (58)	13 (43)	0.171
Raised Serum Bilirubin	17 (23)	5 (17)	0.853
Raised liver enzymes	64 (86)	19 (63)	0.114
Severe Acidosis (venous bicarb <10 mmol/L)	8 (11)	1 (3)	0.234
Acidosis (venous bicarb <22 mmol/L)	63 (85)	25 (83)	0.89
Hemo Dialysis requirement	64 (86)	27 (90)	0.624
Mechanical ventilator requirement	2 (3)	0	0.363

*Chi-square test / Fisher's exact (applied where numbers were <5), Percentages in parenthesis.

Outcome in patients dealt over last ten years was complete renal recovery in 61%, death during acute phase of illness in 14%, partial or no recovery in 5%, and lost to follow up were 20 % (Figure 1). Whereas, previous 25 years' patient population showed renal recovery in 70%, death in 16 %, partial or no recovery in 11% and lost follow up in 3% (Figure 2).

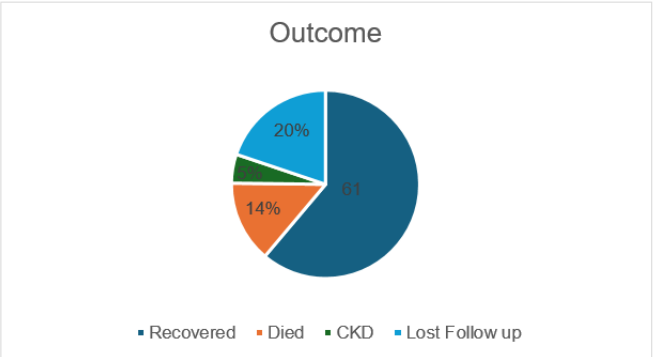

Figure 1 Outcome in population over last decade (n=121).

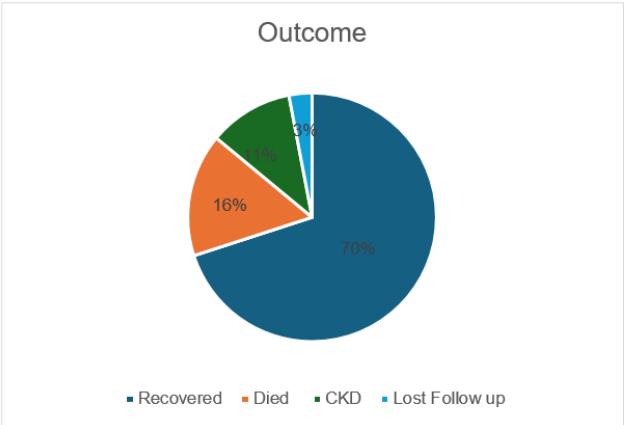

Figure 2 Outcome in population over initial 25 years (n=334).

Discussion

During the 2010–2015 period, our center observed a sharp increase in cases of severe acute kidney injury requiring renal replacement therapy, secondary to toxic rhabdomyolysis from paraphenylene diamine (PPD) poisoning. We documented this trend in our previous published studies,[3,18] which mirrored concurrent nationwide reports of rising PPD toxicity from other medical centers,[19,20] After 2015 our experience of RA-AKI secondary to toxic rhabdomyolysis after PPD poisoning has almost disappeared and taken over by infections in non-traumatic, non-exertional group. One remarkable finding with PPD poisoning was enormously high CK levels, in some cases it was more than half million unit (maximum level reached to 544800 U). Similar observation was also reported in a prospective study on PPD poisoning published from neighboring country India.[21] PPD poisoning also affects liver along with kidneys, though reported liver recovery is faster than kidney.[18]

Traumatic rhabdomyolysis has shown decline in number over last 1.5 decades (Table3), previously with political instability in town there were many patients brought with RA-AKI after physical torture.[3,22]

Elevated INR were found significantly more frequently in those who died, these were mainly the group of patients who had road traffic accidents, with crush injury and element of sepsis. Thromboplastin release from damaged cells and giving rise picture like disseminated intravascular coagulation has also been reported by Lee etal.[23]

Several studies identify metabolic acidosis as predictive of AKI in rhabdomyolysis, as risk of AKI increases with low bicarb levels.[24] In our experience majority of our patients had low bicarb levels, in some it was severe enough to <10 in venous blood. We found severe acidosis significantly different among patients who survived and showed renal recovery in comparison to those who died. Whereas, acidosis or even severe acidosis had no significant different among patients who survived and showed renal recovery in comparison to those who did not show complete renal recovery.

Hyperkalemia is well known and widely described feature associated with RA-AKI, we have seen it irrespective of effect on outcome. Other published studies have also reported hyperkalemia in their RA-AKI population, sometimes this may necessitate need for emergency dialysis.[25,26]

Hyperuricemia may be present due to the release of nucleosides from damaged muscle cells, as these nucleosides convert into purines and then in uric acid.[27] In our studied population as a limitation of being a retrospective study we had serum uric acid level available in very few patients and it was difficult to compare this parameter in different categories of out studied population.

An elevated serum lactate dehydrogenase (LDH) levels is also a known feature associated with both hemolysis and rhabdomyolysis,[27] we have seen high LDH levels in majority of our patients with

considerably high mean value, which is more than six times higher than maximum normal range.

As a result of calcium phosphate preposition in injured muscles, and decreased bone responsiveness to parathyroid hormone, up to two third of rhabdomyolysis patients show hypocalcemia.[28] We have seen hypocalcemia in almost all of our patient with one or two exceptions.

Renal replacement therapy during initial 25 years was required in 94% while 88.4 % during last ten years, which differs markedly from a study published from northern part of country reported 300 RAAKI patients received over 15 months' duration in emergency of a teaching hospital, AKI was advance enough to require dialysis in 26.6% of these patients.[26] Being tertiary renal care we receive majority of patients in advanced renal failure while above mentioned emergency of teaching hospital were handling all patients as first hand.

Working at a facility which provides all health care free of cost in a country where health care facilities and health budget is poor for a common person, we always find more and more patients coming to this institution. With increasing number of new patients coming and rapid turnover, we are now facing lost to long term follow up on them. During our 25 years published report there were 3% while during last ten years this number jumped up to 20%. Other published studies when discuss in term of outcome they mainly focus on complete renal recovery or death during acute illness and it is hard to find what numbers they see as lost long term follow up.[26] One published from neighboring country India, reported mean follow up of six months.[25]

Nielsen et al did not find high mortality with very high baseline CK levels, though risk of AKI and need for RRT was higher with high baseline CK levels.[29] We have also seen very high levels of CK especially in patients with PPD poisoning, but mortality was not high in these patients.

The mainstay of RA-AKI prevention is early and aggressive volume administration with crystalloid fluids. Aim is to maintain or enhances kidney perfusion, and minimizing ischemic injury. These measures increase the urine flow rate thus limit intra-tubular cast formation.[30] In current study we received patients where AKI was already set in, parenteral fluids were either given at their primary care center or in patients who reached in early stage of exposure to insult causing rhabdomyolysis. Thus 13 (11%) of our patients were only treated with parenteral fluids, 10 were among those who showed complete renal recovery while 3 were either showed partial recovery at 90 days or lost follow up before labelling CKD. The study done at northern part of country in emergency of a teaching hospital have shown higher number of patients treated and responded to parenteral fluids, reason being they were primary care center. In their report 62% patients with rhabdomyolysis recovered from AKI after early fluid resuscitation.[26]

Limitations; ours was retrospective study with probability of some missing data. Being a tertiary renal care we only received these patients when they were in established/ advanced AKI. Though urine for pigmented granular cast and for brown supernatant was observed and documented during initial part of study, in later years' only urine findings of dipstick was available from records. There is also lacking quantitative proteinuria measurement in majority. Heptoglobin levels were also lacking in our studied population.

Conclusion

RA-AKI remains a significant contributor to the overall AKI burden. Early detection and aggressive fluid resuscitation can substantially reduce the need for RRT, an expensive modality that is often unavailable in state-run hospitals. Over the past 3.5 decades, the etiology of rhabdomyolysis in our experience has shifted dramatically. During the first 20 years, a period marked by local political instability, torture-related cases were highly prevalent. This was followed by a sharp, 5-to-6-year surge in toxic rhabdomyolysis caused by PPD ingestion, predominantly driven by self-harm. Fortunately, extensive public awareness campaigns over the last decade have successfully reduced PPD related admissions. However, due to recent service expansions and rising patient volumes, maintaining effective long-term follow-up has become increasingly challenging.

Authors contribution

RN: Conceptualization, data collection, manuscript writing. KA: Literature Search, Critical Review. MA: Data collection.

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Conflicts of interest

The authors declare no conflicts of interest.

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