Case Report

Minor Blunt Injury-induced Rhabdomyolysis from a Road Traffic Accident in Nigeria

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Abstract

Rhabdomyolysis, though not a common complication of minor blunt trauma, may result in life-threatening acute kidney injury (AKI). Here is illustrated a case of a young male who sustained minor blunt injuries in a road traffic accident, which he overlooked and presented with features of severe AKI. The patient is a 24-year-old male, who presented with progressive weakness, difficulty in walking, and features of uremia, 14 days after he sustained minor blunt injuries and lacerations in a road traffic accident. Evaluation showed elevated serum creatine kinase, serum myoglobin, and severe azotemia. He was commenced on hemodialysis. He was also commenced on antibiotics, analgesic, and 5% dextrose/saline. He had three sessions of hemodialysis on alternate days. His condition improved remarkably after the first session of dialysis. He was discharged after 18 days on admission. Follow-up in the clinic showed a normal renal function. This case report shows rhabdomyolysis from minor blunt injuries sustained in a road traffic accident and complicated by severe AKI. The patient almost recovered full renal function with management.

Keywords: Acute renal failure, minor blunt injuries, Nigeria, rhabdomyolysis, road traffic accident

Introduction

Rhabdomyolysis is a syndrome that derives from injuries to skeletal muscle.[1] Albeit the myriads nature of these injuries, rhabdomyolysis does not commonly result from common blunt injuries sustained in road traffic accidents.[1]

Injuries to the muscle cell, the sarcolemma, may disrupt many pumps like Na/K-ATPase, which regulates cellular electrochemical gradients.[2] Electrolyte composition is altered following energy depletion that disorganizes the cellular transport mechanisms.[3] Massive proteases and proteolytic enzymes that result from a rise in intracellular calcium, subsequently induce free oxygen radicals' regeneration. In turn, myofilaments are broken down by these substances and enzymes. Membrane phospholipids are also injured causing leakage of intracellular materials into the plasma. Creatine kinase (CK), phosphate, potassium, myoglobin, and urate are some of the materials unleashed into the plasma. Activated neutrophils and fluid infiltrate the muscles.[4] These will incite reperfusion injury and inflammatory cascade, which will cause perpetuation of the muscle breakdown.[5,6]

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In adults, rhabdomyolysis usually manifests with the triad of myalgia, muscle weakness, and dark urine.[7]

The incidence of rhabdomyolysis in Nigeria is not well defined. Myoglobin-induced rhabdomyolysis has not been well documented in Nigeria from the literature search, but worldwide, its incidence is conservatively put at 16%–33%.[8] Severe acute kidney injury (AKI) is a recognized complication of rhabdomyolysis.[9]

Levels of serum CK above 5 times the upper limit of normal establish the diagnosis of rhabdomyolysis.[10] Serum myoglobin may also be considered although this is not specific. Ancillary tests include high potassium levels and elevated lactate

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dehydrogenase.[1] Target treatment in rhabdomyolysis is the treatment of shock and reclamation of renal function.[11]

From the literature search, there was a paucity of reports on rhabdomyolysis from minor road traffic accidents in Nigeria. This has prompted the writing of this case which illustrated a patient with rhabdomyolysis attributable to minor blunt injuries sustained in a road traffic accident, overlooked by the patient.

Case Report

The patient is a 24-year-old male, Igbo by tribe, and an undergraduate, resident in Enugu, Southeast Nigeria. He was referred from the Enugu State University Teaching Hospital staff clinic on account of persistent hiccups of 4 days and confusion of 2 days' duration. The patient was in apparent good health until about 14 days before presentation when he was involved in a road traffic accident, in which he sustained minor blunt injuries, bruises, and lacerations but no fractures. On presentation, he was seen, admitted, and managed by the General Surgical Unit of University of Nigeria Teaching Hospital, Enugu, Nigeria. Four days into the hospital admission in the surgical unit, he developed progressive weakness and worsening of the body pain, and hiccups which became so severe that the patient could not eat without fear of discomfort. As a result of the weakness, he was unable to walk around or bath himself. Two days later, the patient developed nausea and recurrent vomiting, which smothered on until he developed progressive confusion and altered sleep pattern. The patient later developed facial puffiness which was closely followed by bilateral swelling of the legs. The leg swelling progressed to the thigh; he had no associated fever or seizures. His urine volume was noticed to have reduced and a collected urine sample turned dark brown after 6 h. There was no history of hematuria or frothy urine. He had no bleeding from any orifice. There was no history of hypertension or diabetes mellitus. He had no known risk factor for muscular disease from history obtained, and there is no family history of muscular disease.

Physical examination revealed an acutely ill-looking young male. He was in respiratory distress, was afebrile (temperature 37.1°C), not pale, and not anicteric. There was no significant peripheral lymphadenopathy. He had bilateral pitting lower limb edema. Asterixis was present.

The significant observations on examination of the systems were that the patient was drowsy with flapping tremor and bilateral pitting lower limbs and facial edema. He had a pulse rate of 102 beats/min which was of small volume, with a blood pressure of 88/56 mmHg and respiratory rate of 34 cycles/min. Four lacerations and bruises, all dressed; they were neat and not offensive. He had a bilateral basal crepitation.

The assessment of AKI was made.

The following investigations were carried out: urinalysis, urine microscopy culture and sensitivity (MCS), full blood count (FBC), prothrombin time, activated partial thromboplastin time, serum electrolytes, urea, and creatinine (SEUC), serum calcium, serum phosphate, serum uric acid, fasting blood sugar, serum protein, serum CK, serum myoglobin, electrocardiogram (ECG), chest X-ray, and renal ultrasound scan. The results are shown in Tables 1-4.

A diagnosis of rhabdomyolysis complicated by AKI was made.

Hemodialysis was commenced for the patient, intravenous fluid 5% dextrose/saline 1 L 8 hourly for 72 h, ciprofloxacin 200 mg 12 hourly for 72 h, and later converted to oral ciprofloxacin 500 mg 12 hourly for 7 days. The patient was also on tramadol 50 mg 12 hourly for 5 days. He regained full consciousness after the first session of hemodialysis. However, he received two further sessions of hemodialysis. His edema resolved within 15 days. The results are shown in Tables 1-4. He was discharged after 18 days on admission. His first clinic check-up after 2 weeks showed that he had no edema and had normal BP. Laboratory evaluation at day 32 showed FBC, SEUC, urinalysis, serum CK, and serum myoglobin, and ECG results displayed in Tables 1-4. He was stable and doing well. Further follow-up of this patient continued but was not captured, as this report was written 2 weeks after his discharge from the hospital.

Table 1: Biochemical investigation results of the patient

Investigation	At presentation	5 days	32 days
Sodium	137 mmol/l	138 mmol/l	141 mmol/l
Potassium	8.9 mmol/l	4.7 mmol/l	5.1 mmol/l
Bicarbonate	24 mmol/l	23 mmol/l	27 mmol/l
Urea	34 mmol/l	31 mmol/l	18 mmol/l
Creatinine	1202 µmol/l	424 µmol/l	230 µmol/l
Chloride	103 mmol/l	101 mmol/l	
Serum calcium		3.1 mmol/l	2.6 mmol/l
Serum phosphate		1.1 mmol/l	1.0 mmol/l
Serum protein	7.2 g/dl		
Serum albumin	4.1 g/dl		
Serum globulin	3.1 g/dl		
Serum uric acid	426 µg/l		295 µg/l
Serum creatine kinase	60,000 units/l		10,000 units/l
Serum myoglobin	102 mg/dl		56 mg/dl

Table 2: Urinalyses investigation results of the patients

Urinalysis	At presentation	5 days	32 days
Specific gravity	1.020	1.020	1.020
pH	6.0	6.5	6.0
Protein	Nil	Nil	Nil
Sugar	Nil	Nil	Nil
Blood	++	Nil	Nil
Hemoglobin	++	Nil	Nil
RBC	0/hpf	0/hpf	0/hpf
Pus cells	0-1/hpf	0-1/hpf	0-1/hpf
Casts	Nil	Nil	Nil
Urine culture	No growth	No growth	No growth

No growth – Yielded no bacterial growth; Hb – Hemoglobin; RBC – Red blood cell

Discussion

The incidence of rhabdomyolysis is not well defined in Nigeria but seems to be low.[12] This index patient was the third case in our unit in 6 years suggesting that rhabdomyolysis is rare in this environment.

The causes of rhabdomyolysis include crush injuries, muscle compression, infections, and others.[13] There was no crush injury or muscle compression injuries in this index patient. Rhabdomyolysis could be accounted for by the blunt trauma; he sustained in the road traffic accident.

In rhabdomyolysis, muscle damage disrupts sarcolemma microstructures and electrolyte cellular transport mechanisms and alters electrolyte composition.[3] Our index patient had elevated serum CK, elevated serum myoglobulin, hyperkalemia, and hypocalcemia.[10]

Myalgia, weakness, and dark urine usually characterize rhabdomyolysis.[11] This patient under discussion had progressive severe weakness impairing ambulation. He also had dark urine, which is one of the signs of rhabdomyolysis.

Severe renal failure and disseminated intravascular coagulation are challenging outcomes of rhabdomyolysis.[14] Of note, this patient had severe acute renal failure; however, there were no features of intravascular coagulation.

Table 3: Investigation results

Investigation	At presentation	5 days	32 days
Full blood count			
PCV	34%		39%
ERS	50 mm/1st h		12 mm/1st h
Hb	11.3 g/dl		13.0 g/dl
WBC	13,300/dl		7200/dl
Neutrophils	73%		50%
Lymphocytes	27%		49%
Monocytes	0%		0%
Eosinophils	0%		1%
Platelets	233×109/l		
Prothrombin time	Normal		
Activated partial	Normal		
Thromboplastin time	Normal		

PCV – Packed cell volume; ESR – Erythrocyte sedimentation rate; Hb – Hemoglobin; WBC – White blood cell

Rhabdomyolysis accounts for 5%–25% of all adult cases of acute renal failure.[8] Nonetheless, myoglobin-induced acute renal failure, though not common, may warrant hemodialysis, albeit in short term. Our index patient had three sessions of hemodialysis with very good outcomes.

Some hereditary enzyme myopathies may potentiate rhabdomyolysis in the events of trauma.[15] Our index patient was a male but did not have a family history of muscle disease. However, he had trauma-associated rhabdomyolysis. Although drugs such as steroids, quinine, and antihistamines are known causes of rhabdomyolysis, the history of incriminating drugs was not obtained in our patients.

The diagnosis of rhabdomyolysis was based on compatible history of severe weakness and muscle pain with antecedent blunt trauma from a road traffic accident, dark urine, microscopic hematuria noted in the urinalysis, elevated serum creatine levels above 5 times the upper limit of normal,[10] elevated serum myoglobin levels, ancillary positive tests to hyperkalemia, neutrophil leukocytosis, hypocalcemia, and hypophosphatemia.[10] In addition, acute renal failure requiring dialysis was a complication of the rhabdomyolysis.[9]

The thrust of management is to correct cardiovascular insufficiency and address renal function impairment. Our patient received 3 L of 5% dextrose/saline/24 h. Hemofiltration is effective in removing large molecules like myoglobin from the bloodstream.[7] However, hemodialysis is the mainstay of renal replacement therapy in this condition. Our patient had three sessions of hemodialysis with correction of hyperkalemia. Most patients who have sustained renal impairment due to rhabdomyolysis fully recover their renal function.[2,16,17] A near similar picture was observed in this patient who had a progressive appreciation of the renal function.

Conclusion

This case report shows rhabdomyolysis from minor blunt injuries sustained in a road traffic accident and complicated by severe AKI. The patient almost recovered full renal function with management.

Informed consent

Written informed consent was obtained from this patient.

Table 4: Investigation results

Investigations	At presentation	5 days	32 days
Viral screens			
HIV	Negative		
HBsAg	Negative		
HCV	Negative		
ECG	Showed peaking of T-waves and broad QRS	Sinus rhythm, normal T-waves and QRS	Sinus rhythm, normal T-waves and QRS
Abdominal ultrasound scan	Showed normal size kidneys, with normal echotexture and normal corticomedullary differentiation		

HIV – Human immunodeficiency virus; HBsAg – Hepatitis B surface antigen; HCV – Hepatitis C virus; ECG – Electrocardiogram

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given consent for his clinical information to be reported in this journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity will not be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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