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Case Report

Rhabdomyolysis induced acute kidney injury after dengue infection

Pratim Sengupta[1], Debayan Rarhi[1], Atreyee Chaudhuri[1], Kavita Rathore[2]*, Tapas Roy[3], Sourav Sadhukhan[1]

[1]Nephro Care India Limited, Bidhannagar, Kolkata, West Bengal, India
[2]Department of Pharmacology, Ananta Institute of Medical Sciences and Research Center, Rajsamand, Rajasthan, India
[3]Department of Nephrology, ILS Hospital, Dumdum, Kolkata, West Bengal, India

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***Correspondence:**
Dr. Kavita Rathore,
E-mail: rathorekavita466@gmail.com

ABSTRACT

Rhabdomyolysis is the breakdown of striated skeletal muscle tissue, leading to the release of cellular elements such as myoglobin, creatine kinase, aldolase, lactate dehydrogenase, and electrolytes into the blood circulation. It's usually caused by trauma or certain medications, but it can also happen during viral infections like dengue fever, though this is rare and serious. We describe a case of a 33-year-old man with dengue fever who was admitted to the hospital with high fever, nausea, vomiting, and dark urine. Investigations showed high levels of creatine phosphokinase (CPK) and abnormal liver enzymes, suggesting rhabdomyolysis and acute liver injury. Regardless of treatment with fluids and antibiotics, his kidney function got worse, needing hemodialysis. After discharge, he had high blood pressure and required more dialysis sessions. With good care, his kidneys started working better, and the catheter used for dialysis was removed. Rhabdomyolysis from Dengue infection is hard to diagnose and treat. The virus can directly harm muscle cells and cause immune reactions that release harmful substances like myoglobin, which may cause acute kidney injury. Early detection of high CPK levels is crucial to prevent kidney injury. Treatment involves giving fluids carefully to protect the kidneys from myoglobin damage without causing fluid overload, which is critical in helping patients recover. Monitoring CPK levels in Dengue patients is vital to diagnose rhabdomyolysis early and protect the kidneys. This approach can improve outcomes and guide better treatment decisions for patients with this rare but severe complication.

Keywords: Rhabdomyolysis, Dengue, Creatine phosphokinase, Hemodialysis, Liver enzyme, Acute kidney injury

INTRODUCTION

Rhabdomyolysis refers to the breakdown and necrosis of skeletal muscle, releasing intracellular components like myoglobin, creatine kinase (CK), aldolase, and electrolytes into the bloodstream.

Myoglobin causes nephrotoxicity, tubular obstruction, and renal ischemia via vasoconstrictive factors—leading to acute kidney injury (AKI).[1]

This report presents a case of dengue-associated rhabdomyolysis complicated by AKI, acute liver injury, hypoalbuminemia, and coagulopathy.

CASE REPORT

A 33-year-old normoglycemic, normotensive, nonalcoholic, nonsmoker male was admitted to ILS Hospital, Dumdum on 5th November 2023 with 3 days of high-grade fever, nausea, vomiting (2–3 episodes), loose stools (4–5 episodes), dark-colored urine, generalized weakness, and body ache. He had not taken nonsteroidal anti-inflammatory drugs (NSAIDs) or alternative medicine. In the emergency room, his vitals were: blood pressure (BP) 130/80 mmHg, pulse 100/min, temperature 101°F, Oxygen saturation (SpO_2) 94%. He reported decreased urine output for one day and was admitted to the high dependency units (HDU) with suspected rhabdomyolysis and AKI.

Table 1: Haematology and clinical biochemistry of the subject during hospitalization tenure.

Test	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12	Day 13	Day 14	Day 17	Day 18	Reference range
Liver function test																	
Total protein (g/dl)	4.1	-	-	-	5.1	5	-	5.4	-	5.3	-	-	5.1	-	5.7	-	6.6-8.7
Albumin (g/dl)	2.6	-	-	-	3	2.8	-	2.9	-	2.8	-	-	2.9	-	3.1	-	3.5-5.2
Globulin (g/dl)	1.5	-	-	-	2.1	2.2	-	2.5	-	2.5	-	-	2.2	-	2.6	-	2.3-3.5
SGOT (U/l)	1197	3072			2225	2082	-	-	1530	379	-	-	85	-	33	-	<50
SGPT (U/l)	366	496			253	183	-	-	126	55	-	-	22	-	11	-	<41
Alkaline phosphat-ase (U/l)	40	-	-	-	70	66	-	58	-	53	-	-	51	-	43	-	40-129
GGT (U/l)	71	-	-	-	149	116	-	78	-	54	-	-	37	-	28	-	8-61
Direct bilirubin (mg/dl)		-	-	-	0.5	0.4	-	0.4	-	0.3	-	-	0.3	-	0.3	-	<0.3
Infective parameters																	
CPK (U/l)	>100000	-	-	-	-	>100000	-	-	-	-	-	-	8956.9	-	-	348.6	<190
CRP (mg/l)	57.3	-	-	-	-	26.4	-	-	55.4	41	-	35.5	-	17.6	12.1	-	<6.0
Clinical biochemistry	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Serum creatinine (mg/dl)	2	6.1	7.6	8.1	10.5	9.3	8.6	11	9.6	-	-	10.9	12.5	9.5	10.8	8.9	0.7-1.2
Serum urea (mg/dl)	52	140	139	119	178	109	84	107	101	-	-	120	136	107	127	93	16.6-48.5
Serum sodium (mmol/l)	136	129	130	132	131	134	136	138	-	139	138	135	135	136	135	135	134-145
Serum potass-ium (mmol/l)	3.1	5.2	4.2	3.9	4.1	3.7	3.9	4.4	-	4.3	4.3	4	4.2	3.9	3.9	3.7	3.5-5.5
Serum calcium (mg/dl)	5	-	-	6	6.2	6.4	7	6.2	7	6.9	7.5	-	8.1	-	-	10.7	8.6-10.0
Serum phosph-orous (mg/dl)	10.9	-	-	-	-	5.7	-	-	5.3	-	-	-	5	-	-	5.8	2.5-4.5
Hematology																	
Haemogl-obin (gm/dl)	17.6	-	-	-	-	10.3	9.2	9	9.5	8.7	9.7	10.7	-	9.7	9.2		13-17
WBC ($\times 10^3$ µl)	10.2	-	-	-	-	9.8	11.9	12.2	10.9	8.8	8.6	8.5	-	8.2	5		4-10
Haemato-crit (%)	49.5	42	42.3	37	35.8	29.3	26.6	27.2	28.6	27.6	29.6	31.9	32.1	29.6	28.5		40-50
Platelet ($\times 10^3$ thou/ mm^3)	160	150	95	70	120	145	170	179	209	192	176	187	194	181	195		150-450

Initial investigation revealed: high creatinine phosphokinase (CPK) >100,000 U/l, liver enzymes serum glutamic pyruvic transaminase (SGPT) 1197 U/l, serum glutamic-oxaloacetic transaminase (SGOT) 366 U/l, serum creatinine 2.0 mg/dl, sodium 136 mmol/l, potassium 3.1 mmol/l, haemoglobin 17.6 g/dl, WBC 10.2×10⁹/l, haematocrit 47.7%, platelets 185×10⁹/l (Table 1). Urine routine examination showed 4–6 red blood cells (RBCs), 5–7 pus cells, 2+ protein, 3+ microorganisms; however, cultures showed no growth (Table 2). Serial potassium: 3.1 mmol/l >5.2 mmol/l >4.1 mmol/l. Troponin I was normal; 2D echocardiography: ejection fraction 55%, no regional wall motion abnormalities. Electrocardiogram (ECG) report showing sinus tachycardia with a normal P-QRS-T morphology (Figure 1). The ultrasound reveals that both kidneys are swollen in size, with the right kidney measuring 123.7×56.6 mm and the left kidney 121.1×63.6 mm. There is altered corticomedullary differentiation, and bilateral perirenal fluid is noted. No calculus or hydronephrosis is seen, and the ureters are not dilated. In addition, the liver is enlarged with coarse, hypoechoic parenchyma and a prominent portal vein. The gallbladder is distended with a thickened wall (3.5 mm) and a clear lumen. The pancreas appears bulky and echogenic with smooth margins. The spleen is enlarged (123.6 mm), and the urinary bladder shows a thickened wall (10.4 mm) with an indwelling catheter. The prostate is normal in size with a projecting median lobe. Minimal ascites and dependent bilateral pleural effusion are also present.

After sending blood and urine cultures, aggressive hydration was started due to rising CPK and suspected rhabdomyolysis. Despite 48 hours hydration, creatinine rose to 8.1 mg/dl. A right internal jugular vein catheter was placed, and hemodialysis was initiated. Dengue serology was positive; blood cultures remained negative. He received intravenous antibiotics and nine hemodialysis sessions. CPK levels declined progressively (10000>8956.9>348.6), and liver enzymes improved. Though afebrile and stable, he was discharged with creatinine 8.9 mg/dl and poor urine output, advised to continue dialysis.

Two days later, he returned with shortness of breath and BP 200/100 mmHg. Chest auscultation indicated bilateral basal crepitations. He was re-admitted to HDU, given urgent dialysis and glyceryl trinitrate (GTN) infusion. After two sessions, BP stabilized, urine output improved, and dialysis was withheld. Over 10 days, creatinine declined to 2.3 mg/dl and the internal jugular vein (IJV) catheter was removed. 50 days post-admission, serum creatinine was 1.67 mg/dl (Table 3).

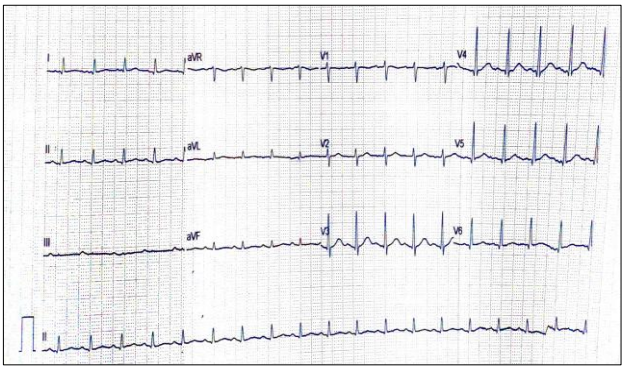

Figure 1: Electrocardiogram showing normal rhythm except sinus tachycardia on day 2 of hospitalization.

Table 2: Urine regular and microscopic examination during hospitalization tenure.

Examination	Day 1	Day 22	Day 23	Reference range
Urine red blood cells (RBC)	4-6	-	10-15	Negative
Urine pus cell (/hpf)	5-7	-	8-10	0-5
Urine protein	2+	-	2+	Negative
Urine culture	No growth of uropathogenic	No growth of uropathogenic	No growth of uropathogenic	No growth of uropathogenic
Microorganism	3+	No growth	2+	Negative
Troponin1 high sensitive (ng/l)	97	-	-	<15

Table 3: Haematology and clinical biochemistry of the subject during re-hospitalization tenure.

Test	Day 1	Day 3	Day 5	Day 6	Day 8	Day 9	Day 10	Day 11	Follow up visit after 1 month	Reference range
Haemoglobin (Hb) (gm/dl)	8.8	8.6	9	8.7	-	-	-	-	8.8	13-17
WBC (×10³ µl)	7.7	10.3	7.2	6.1	-	-	-	-	7.7	4-10
Haematocrit (%)	-	26.5	28.2	26.4	-	-	-	-	-	40-50
Platelets (10^3/mm^3)	10.7	182	193	191	-	-	-	-	213	150-450
CRP (mg/l)	-	53	139.5	-	-	32	-	-	-	<6.0
Urea (mg/dl)	34	34	59	89	111	-	111	107	34	16.6-48.5
Creatinine (mg/dl)	1.64	1.64	5.8	7.7	9.3	-	7.8	6.3	1.64	0.7-1.2

DISCUSSION

Dengue haemorrhagic fever (DHF) is a severe complication of dengue infection, accompanied by fever, thrombocytopenia, haemorrhage, and plasma leak.[2] AKI in dengue is typically associated with hypotension, shock, haemolysis, and sepsis, though rare cases of acute glomerulonephritis and rhabdomyolysis-induced AKI requiring dialysis have also been reported.[3,4]

Myositis and rhabdomyolysis in dengue remain under-investigated. Malheiros et al identified inflammation and necrosis in muscle biopsies from dengue patients with myalgia, also noted transient muscle dysfunction and raised creatine kinase (CK) during a dengue outbreak.[5,6] These indicate that muscle injury in dengue is likely more common than previously recognized. Proposed mechanisms include direct viral invasion and immune-mediated muscle damage.

Rhabdomyolysis leads to AKI in 5–30% of cases, primarily via renal vasoconstriction, tubular injury, and obstruction.[7] Myoglobin, though not directly nephrotoxic, can precipitate renal injury when combined with other urinary proteins under acidic conditions. Inflammatory responses involving innate immune cells also contribute to renal damage.[8] Diagnosis of rhabdomyolysis requires elevated serum CPK and urinary myoglobin, though precise quantification remains challenging due to limitations in current assays.[9]

Management hinges on early, aggressive hydration, tailored to the severity of rhabdomyolysis and renal function.[10] However, in dengue, excessive fluid administration may worsen plasma leakage and increase mortality risk, necessitating careful fluid balance.[11]

Few cases showed rhabdomyolysis without AKI or muscle symptoms without renal issues, suggesting that rhabdomyolysis alone may be insufficient for AKI development without contributing factors like hypovolemia, acidosis, and aciduria.[12]

AKI has also been documented in dengue in the absence of rhabdomyolysis, potentially due to hemodynamic instability, glomerular damage, or direct viral cytopathy.[13]

CONCLUSION

In our case, the patient presented with fever, myalgia, dark urine, elevated CPK, and subsequent AKI, without other known causes of rhabdomyolysis. This constellation strongly supports dengue-associated rhabdomyolysis as the cause of AKI. Given the under-recognition of this complication, routine CK monitoring in severe dengue is recommended for early detection and prevention of renal failure.

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