



MASSIVE RHABDOMYOLYSIS WITH CREATINE KINASE >600,000 U/L IN PCR-CONFIRMED CHIKUNGUNYA INFECTION: A CASE REPORT

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ABSTRACT Chikungunya infection rarely causes severe rhabdomyolysis, and extreme creatine kinase (CK) elevations are seldom reported. In this study, a 32-year-old previously healthy male presented with fever, generalized myalgia, rash, and dark-colored urine. Laboratory evaluation revealed massive rhabdomyolysis with CK 606,000 U/L. Multiplex PCR confirmed chikungunya infection, while other infectious causes, including dengue, leptospirosis, and scrub typhus, were negative. To our knowledge, this represents one of the highest reported CK levels in a PCR-confirmed chikungunya-associated rhabdomyolysis case from India. The patient was managed with early aggressive intravenous hydration and urine alkalinization. Extensive evaluation excluded traumatic, drug-related, metabolic, and autoimmune causes of rhabdomyolysis. Serial monitoring demonstrated a rapid decline in CK levels with preservation of renal function and complete clinical recovery. This case highlights that chikungunya infection may present with extreme rhabdomyolysis. Early recognition and aggressive fluid therapy may prevent acute kidney injury even in cases with very high CK levels.

KEYWORDS : Chikungunya, Rhabdomyolysis, Creatine Kinase, Viral Myositis, Acute Kidney Injury, Arboviral Infections

BACKGROUND

Rhabdomyolysis is a potentially life-threatening syndrome resulting from skeletal muscle breakdown with the release of intracellular components such as creatine kinase (CK), myoglobin, potassium, and phosphate into the circulation [1]. Severe cases may result in electrolyte imbalance, metabolic disturbances, and acute kidney injury (AKI) [1, 2]. Viral infections are increasingly recognized as important non-traumatic causes of rhabdomyolysis [3]. Chikungunya virus, an arbovirus transmitted by *Aedes* mosquitoes, typically presents with fever, rash, and severe arthralgia [4]. Myalgia is common; however, severe myositis and rhabdomyolysis remain rare complications [4, 5]. Only a limited number of cases describing chikungunya-associated rhabdomyolysis have been reported in the literature, and extreme CK elevations are rare [4, 5].

Case Description

A 32-year-old previously healthy male IT professional presented with five days of fever, generalized myalgia, fatigue, and erythematous rash over the limbs and trunk. He also reported dark-colored urine prior to admission. Two weeks earlier, he experienced a short episode of sore throat with fever that resolved with symptomatic treatment. One week before the illness, he had traveled to Kerala. A detailed drug and social history was obtained; the patient specifically denied use of statins or other lipid-lowering agents and had no history of alcohol consumption, both of which are recognized causes of elevated serum creatine kinase and transaminases. There was no history of trauma, seizures, excessive physical exertion, or use of other myotoxic drugs.

Initial laboratory investigations at an outside hospital showed hemoglobin 13.3 g/dL, leukocyte count 7,850/mm³, platelets 2.06 lakh/mm³, ALT 352 U/L, and creatinine 1.21 mg/dL. Serological tests for dengue, leptospirosis, and scrub typhus were negative. The patient was started empirically on doxycycline and ceftriaxone. CT abdomen incidentally revealed a small non-obstructive renal calculus.

On admission to our center, he was conscious and hemodynamically stable with pulse 101/min, blood pressure 130/70 mmHg, oxygen saturation 98% on room air, and temperature 99.7°F. Examination revealed diffuse maculopapular rash over the extremities, sparing the palms and soles (Figure 1). Systemic examination was otherwise unremarkable.

Repeat investigations showed hemoglobin 11.9 g/dL, leukocyte count 8,900/mm³, platelets 2.2 lakh/mm³, sodium 134 mEq/L, potassium 5.5 mEq/L, elevated AST and ALT, and mild creatinine elevation. Urinalysis demonstrated proteinuria and hematuria with positive urine myoglobin. The presence of myoglobinuria with minimal red blood cells supported pigmenturia secondary to rhabdomyolysis.



Figure 1: Showing Diffuse Maculopapular Rash Over Extremities

Creatine kinase was markedly elevated at 606,000 U/L (Figure 2). Multiplex PCR testing confirmed chikungunya infection. An extensive inflammatory myopathy profile including Mi-2 α , Mi-2 β , MDA5, NXP2, TIF1 γ , SAE1, Ku, PM-Scl100, Jo-1, PM-Scl75, SRP, PL-7, PL-12, EJ, OJ, and Ro-52 antibodies was negative.

TEST NAME	RESULT	REFERENCE INTERVAL	UNITS
CK (CPK); CREATINE KINASE - SERUM (UV acc. to IFCC liquid)	606000 *	0-125 yrs: 39-308 U/L	U/L
Comments : (Note : Kindly correlate the values with clinical findings)			
CK (CPK): CREATINE KINASE - SERUM VALUE IS CRITICAL.			
* END OF REPORT *			
Report Status: Final			

Figure 2: Creatine Kinase Report on Day 1 of Our Case

Additional evaluation to exclude alternative etiologies included ANA, anti-dsDNA, and ANCA (MPO and PR3) testing, all of which were negative, with normal serum complement C3 (120 mg/dL) and C4 (36.7 mg/dL), arguing against systemic lupus erythematosus, ANCA-associated vasculitis, or complement-mediated microangiopathy. Dengue serology (IgM and NS1 antigen) was negative, and dengue RNA was not detected on the multiplex PCR panel that confirmed chikungunya viremia. Peripheral blood smear for malarial parasites (QBC) was negative, and blood culture (BCID panel) showed no bacterial growth, excluding malaria and bacteremia/sepsis as contributors. Twenty-four-hour urine porphyrin quantification was within normal limits (68 mg/24h; reference 50–200), excluding acute porphyria as a cause of the myalgia and pigmenturia.

Antibiotics were discontinued after confirmation of viral etiology. The patient was managed with aggressive intravenous isotonic fluid resuscitation and urine alkalinization using bicarbonate infusion [1, 6]. Due to extremely elevated CK levels, the patient was closely monitored in the critical care unit with serial laboratory evaluations.

Serial laboratory monitoring demonstrated a progressive decline in CK levels (Table 2). Renal function improved from (serum creatinine 1.2

mg/dL to 0.8 mg/dL) and remained normal throughout hospitalization. Fever subsided, urine color normalized (Figures 3–5), and the patient showed steady clinical improvement. He was discharged in stable condition and remained asymptomatic on follow-up.



Figure 3: Dark Coloured Urine in Rhabdomyolysis (Day 2)



Figure 4: Cola Coloured (Day 3)

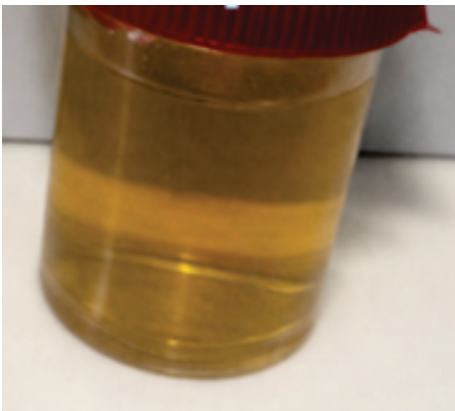


Figure 5: Normal Colour of the Urine During Discharge (Day 7)

Table – 2 Clinical and Biochemical Trends Across Hospitalization and Follow-Up

Hospital Day	Date	CK (U/L)	Creatinine (mg/dL)	AST (U/L)	ALT (U/L)	Potassium (mEq/L)	Urine pH
Day 1	31/12/2025	606,000	1.21	—	357	5.5	6.0

Day 2	01/01/2026	436,500	1.31	—	—	4.5	5.0 → 8.0*
Day 3	02/01/2026	162,200	1.31	2,552	312	4.6	9.0
Day 4	03/01/2026	36,143	1.12	1,460	265	4.1	8.5 → 8.0*
Day 5	04/01/2026	7,876	—	—	—	—	8.0
Day 6	05/01/2026	3,892	1.18	413	157	3.9	7.5 → 7.0*
Day 7	06/01/2026	3,356	—	—	—	—	—
Follow-up	07/01/2026	2,646	1.08	276	—	4.1	7.0
Follow-up	09/01/2026	1,791	1.05	206	—	4.7	7.5
Follow-up	16/01/2026	376	1.15	48	—	4.7	6.5
Reference Range		39–308	0.70–1.20	10–50	10–50	3.5–5.1	4.5–8.0

Note: Empty cells (—) indicate parameters not evaluated on that specific timeline date.

*Arrows (→) denote chronological variations captured during intra-day serial monitoring, reflecting target urinary alkalization progression.

DISCUSSION

Rhabdomyolysis is a potentially life-threatening syndrome resulting from the breakdown of skeletal muscle fibers, leading to the release of intracellular contents — including creatine kinase (CK), myoglobin, potassium, and phosphate — into the systemic circulation. The clinical spectrum ranges from an asymptomatic elevation in serum CK to a life-threatening condition associated with acute kidney injury (AKI), electrolyte disturbances, disseminated intravascular coagulation, and multi-organ failure [1]. The classic triad of myalgia, muscle weakness, and cola-colored urine is present in fewer than 10% of patients, making early clinical recognition challenging [1, 2]. Serum CK remains the most sensitive and reliable biochemical marker, with levels typically exceeding five times the upper limit of normal on presentation, and peaking between 24 and 72 hours [2, 3].

Viral infections are an increasingly recognized but under-reported cause of non-traumatic rhabdomyolysis. Influenza A and B are the most commonly implicated viruses, though a wide range of pathogens — including dengue, COVID-19, coxsackievirus, Epstein-Barr virus, and enteroviruses — have also been described [1, 4] (Table 1). Acute viral myositis complicated by rhabdomyolysis is uncommon, and progression to severe AKI is even rarer [4]. In adults, the most frequent causes overall remain exertion, crush injury, alcohol and drug abuse, and statin use, with viral etiologies contributing a smaller but clinically significant proportion [3]. The pathophysiological mechanisms by which viral infection leads to muscle injury are not fully understood but are thought to involve direct viral invasion of myocytes, immune-mediated inflammatory myositis, cytokine-mediated muscle necrosis, and microvascular dysfunction leading to muscle ischemia [1, 4]. Irrespective of etiology, the final common pathway involves disruption of intracellular ion homeostasis, failure of Na⁺/K⁺ and Ca²⁺ ATPase pumps, pathological calcium influx, sustained myofibrillar contraction, and ultimately necrosis of the muscle fiber [1].

Table – 1 Reported High Creatine Phosphokinase (CPK) Levels in Severe Rhabdomyolysis

Study / Report	Etiology	Peak CPK (U/L)	AKI	Dialysis	Outcome
Weber et al. [3]	Coxsackie virus	>1,300,000	Yes	Yes	Recovery
Influenza cases	Influenza A	>400,000	Variable	Variable	Variable
COVID reports	COVID-19	>1,000,000	Rare	Rare	Recovery
Dengue reports	Dengue	200,000–500,000	Sometimes	Sometimes	Recovery
Exertional rhabdomyolysis	Trauma/exertion	100,000–300,000+	Common	Sometimes	Recovery
Drug-induced cases	Statins/toxins	50,000–200,000	Variable	Rare	Recovery

Present case	Chikungunya	606,000	No	No	Full recovery
Baddam et al.	Viral flu	880,300	Yes	No	Recovery
Elfert et al.	Chikungunya	>2,000	Yes	No	Recovery

AKI: Acute kidney injury; CPK: Creatine phosphokinase.

Chikungunya virus, an *Aedes* mosquito-transmitted arbovirus, is well recognized for causing fever, debilitating polyarthralgia, rash, and fatigue. Although myalgia is a common feature of acute chikungunya infection, severe myositis and rhabdomyolysis remain rare complications. Most previously reported cases of chikungunya-associated rhabdomyolysis from India and other endemic regions have described CK elevations generally below 50,000 U/L, often with concurrent AKI. A 2019 case report by Elfert et al. similarly highlighted rhabdomyolysis as an atypical but serious complication of chikungunya [5]. The present case, with a peak CK of 606,000 U/L confirmed by multiplex PCR, underscores that extreme biochemical severity is possible in this infection. To the best of our knowledge, we performed a comprehensive literature search of PubMed/MEDLINE, Scopus, Google Scholar, Crossref, and other indexed databases using combinations of the keywords “India”, “chikungunya”, “rhabdomyolysis”, “creatine kinase”, “creatine phosphokinase”, “CK”, and “CPK”. We did not identify any published case report from India describing chikungunya-associated rhabdomyolysis with a peak serum creatine kinase (CPK/CK) level exceeding that observed in our patient. Therefore, this case appears to represent the highest reported CPK value in a published Indian case of chikungunya-associated rhabdomyolysis to date.

A notable and clinically instructive aspect of this case is the absence of worsening AKI despite markedly elevated CK levels. AKI develops in approximately 10–40% of patients with rhabdomyolysis and represents its most serious acute complication [1]. The nephrotoxicity of rhabdomyolysis is primarily driven by myoglobin-induced tubular obstruction, direct renal tubular toxicity, and renal hypoperfusion due to hypovolemia [1, 3]. In the landmark case series by Dadana et al. (2024), a 30-year-old African American male with recurrent rhabdomyolysis and a world-record CK of 4,570,630 U/L similarly avoided AKI, attributed to early and aggressive intravenous fluid resuscitation [6]. Likewise, Baddam et al. (2025) described a case of viral myositis with CPK of 880,300 U/L that progressed to AKI and required hemodialysis, illustrating that even lower CK levels than those seen in our patient can cause significant renal injury when fluid resuscitation is delayed or insufficient [2]. Together, these cases highlight that the absolute CK value alone does not reliably predict the development or severity of AKI; rather, the timing and adequacy of fluid resuscitation, baseline hydration status, and metabolic derangements are critical determinants [2, 6]. Risk-stratification tools such as the McMahon Score can further aid clinicians in deciding when to escalate renal support. This validated model incorporates age, sex, etiology of rhabdomyolysis, and admission values of creatinine, calcium, phosphate, and CK to estimate the risk of acute kidney injury requiring renal replacement therapy or in-hospital mortality, with a score ≥ 6 indicating a “not low risk” category warranting close renal monitoring [7]. Applying this score to our patient’s admission parameters (age 32 years, male sex, serum creatinine 1.21 mg/dL, calcium 8.4 mg/dL, phosphate 3.1 mg/dL, bicarbonate 20 mEq/L, CK 606,000 U/L, and a non-classical etiology) yielded a McMahon Score of 5, placing him in the low-risk category despite the extreme CK elevation. This is notable because the score does not scale linearly with CK beyond the 40,000 U/L threshold, and it correctly anticipated our patient’s ultimately benign renal course. This observation suggests that the McMahon Score (refer to Table 3) may retain discriminative value even in cases of extreme levels of CK, and supports incorporating such scores at admission, alongside serial CK and renal function trends, to identify patients likely to benefit from early, aggressive renoprotective therapy before renal function begins to deteriorate.

Table – 3 The McMahon Risk Prediction Score for Rhabdomyolysis

Variable	Clinical Stratification	Points
Demographics		
Age	≤ 50 years	0
	51–70 years	1.5
	71–80 years	2.5
	> 80 years	3.0
Gender	Male	0

	Female	1.0
Etiology		
Primary Cause	Low-risk (Exertion, seizure, syncope, statin, myositis)	0
	High-risk (Trauma, crush injury, ischemia, infection, overdose)	3.0
Admission Laboratories		
Serum Creatinine	< 1.4 mg/dL	0
	1.4–2.2 mg/dL	1.5
	> 2.2 mg/dL	3.0
Serum Calcium	≥ 7.5 mg/dL	0
	< 7.5 mg/dL	2.0
Serum Phosphate	< 4.0 mg/dL	0
	4.0–5.4 mg/dL	1.5
	> 5.4 mg/dL	3.0
Serum Bicarbonate	≥ 19 mEq/L	0
	< 19 mEq/L	2.0
Initial/Peak CK	$\leq 40,000$ U/L	0
	$> 40,000$ U/L	2.0

Note: The McMahon Score is calculated on hospital admission for rhabdomyolysis patients. A score of ≥ 6 identifies patients at high risk of developing severe acute kidney injury (AKI) requiring renal replacement therapy (RRT) or in-hospital death (positive predictive value $> 50\%$, negative predictive value $\sim 97\%$).

In our patient, early recognition and prompt initiation of aggressive isotonic fluid resuscitation likely played the principal role in preventing tubular myoglobin deposition and preserving renal function despite the degree of muscle injury. Urine alkalization with sodium bicarbonate was administered as an adjunctive measure; however, current evidence indicates that isotonic fluid resuscitation remains the cornerstone of renal protection in rhabdomyolysis, while the incremental benefit of bicarbonate therapy over saline alone remains controversial and unproven in randomized trials [2, 3]. We therefore attribute the favorable renal outcome primarily to early and adequate volume resuscitation rather than to alkalization per se. Several additional patient- and treatment-related factors likely contributed to the improvement of renal function in our patient despite the extreme CK elevation. These include early clinical presentation and prompt evaluation, allowing timely hydration before significant tubular myoglobin deposition could occur; preserved intravascular volume and absence of hypotension throughout the clinical course; the patient’s relatively young age (32 years) with no pre-existing renal or vascular comorbidity; and the absence of concurrent nephrotoxic exposures such as NSAIDs or contrast agents. Together, these factors likely mitigated the risk of myoglobin-induced tubular injury despite a CK level exceeding 600,000 U/L, reinforcing the concept that the timing and adequacy of resuscitation — rather than the absolute CK value — are the principal determinants of renal outcome.

The cornerstone of management in rhabdomyolysis is aggressive intravenous fluid resuscitation with isotonic saline, targeting a urine output of 200–300 mL/h [2, 3]. Fluid rates of 1–2 L/h may be necessary in severe cases to counteract hypovolemia and promote myoglobin clearance. Urine alkalization with sodium bicarbonate, aiming for a urine pH greater than 6.5, is employed to minimize the nephrotoxic effects of myoglobinuria and prevent intratubular cast formation, though evidence supporting improved clinical outcomes over saline alone remains mixed [2, 3]. Electrolyte derangements — particularly hyperkalemia, hypocalcemia, and hyperphosphatemia — require close monitoring and prompt correction to prevent fatal cardiac arrhythmias [3, 4]. The role of mannitol as an osmotic diuretic remains controversial, with no well-established benefit demonstrated in controlled studies [2]. In cases where AKI progresses despite these measures, renal replacement therapy, including continuous venovenous hemofiltration or intermittent hemodialysis, may be required, as demonstrated in the case by Baddam et al., where the patient required four weeks of dialysis before renal function recovered [2]. In contrast, our patient’s renal function remained preserved throughout hospitalization, consistent with the favorable outcomes achievable with timely intervention.

The diagnosis of chikungunya-associated rhabdomyolysis in this patient was supported by a convergence of clinical, temporal, and laboratory evidence: (i) multiplex PCR confirmation of chikungunya viremia; (ii) a compatible clinical presentation of fever, rash, and arthralgia; (iii) a clear temporal relationship between symptom onset

and travel to an endemic region; (iv) systematic exclusion of alternative infectious causes (dengue, leptospirosis, scrub typhus, malaria, bacteremia) and non-infectious causes (traumatic, drug-induced, metabolic, and autoimmune); and (v) a comprehensively negative autoimmune and inflammatory myopathy evaluation. This convergence of evidence supports a causal, rather than coincidental, association between chikungunya infection and the observed rhabdomyolysis. Diagnosing viral myositis as the etiology of rhabdomyolysis requires a systematic exclusion of more common causes. In our patient, an extensive workup excluded traumatic, drug-induced, autoimmune, inflammatory, metabolic, and other infectious etiologies. The inflammatory myopathy panel, including Mi-2, MDA5, NXP2, TIF1 γ , SAE1, Jo-1, PL-7, PL-12, and SRP antibodies, was entirely negative, effectively ruling out autoimmune myositis. Systemic autoimmune disease was further excluded by negative ANA, anti-dsDNA, and ANCA (MPO and PR3) testing with normal complement C3 and C4 levels. Malaria, bacteremia, and acute porphyria were excluded by a negative peripheral smear for malarial parasites, sterile blood cultures, and normal 24-hour urine porphyrin quantification, respectively. Thyroid function tests were normal, excluding hypothyroidism-associated myopathy. Urine drug screen was negative. The patient had no prior statin use, no significant exertional history, and no trauma sufficient to explain the severity of CK elevation. Multiplex PCR positivity for chikungunya, combined with the clinical context of recent travel to an endemic region (Kerala), febrile illness, rash, and arthralgia in the absence of all alternative explanations, established the viral etiology. This case also raises the possibility that an unrecognized predisposing factor, such as a subclinical metabolic myopathy or individual genetic susceptibility, may have amplified the degree of muscle injury, as has been proposed in similarly extreme CK elevations of apparent viral origin [3, 6]. Muscle biopsy, which could have provided histopathological confirmation of myofiber necrosis and inflammatory infiltrates characteristic of viral myositis, was not performed, representing a recognized limitation.

This case carries important clinical implications for physicians practicing in regions where chikungunya is endemic, including large parts of India and Southeast Asia. Chikungunya should be included in the differential diagnosis of unexplained febrile rhabdomyolysis, particularly in patients presenting with arthralgia and rash following travel to or residence in endemic areas. Clinicians should maintain a high index of suspicion and measure serum CK early in such presentations, even in the absence of the classic triad of myalgia, weakness, and dark urine, which is present in fewer than 10% of cases [1, 2]. Furthermore, this case reinforces the principle that the severity of CK elevation should not be the sole criterion for anticipating renal failure — early aggressive fluid therapy, vigilant electrolyte monitoring, and prompt correction of metabolic derangements are the decisive factors in determining renal outcomes [2, 3, 4]. Limitations of this report include the absence of a muscle biopsy and the lack of quantitative serum myoglobin measurements.

CONCLUSION

This case illustrates an unusually severe manifestation of chikungunya infection presenting with massive rhabdomyolysis and extremely elevated creatine kinase levels. Early recognition and aggressive hydration likely contributed to the preservation of renal function despite severe muscle injury.

Learning Points

1. Chikungunya infection can rarely cause severe rhabdomyolysis.
2. Creatine kinase levels may exceed 600,000 U/L even without renal failure.
3. Early aggressive intravenous hydration is critical to prevent acute kidney failure.
4. Viral infections should be considered in patients with unexplained rhabdomyolysis in endemic regions.

REFERENCES

- [1] Torres, P. A., Helmstetter, J. A., Kaye, A. M., and Kaye, A. D. (2015), "Rhabdomyolysis: pathogenesis, diagnosis, and treatment." *Ochsner J*, 15(1), 58-69. PMID: 25829882; PMCID: PMC4365849.
- [2] Baddam, S., Thiriveedi, M., and Haley, H. (2025), "High Creatine Kinase Levels in Viral Myositis: A Case of Rhabdomyolysis-Induced Renal Failure." *American Journal of Case Reports*, 26, e946551. <https://doi.org/10.12659/ajcr.946551>
- [3] Weber, M., Goss, W., Hoffer, C., Ogunsulire, J., and Schafer, F. (2024), "Rhabdomyolysis of Infectious Etiology with Creatine Kinase Above One Million: A Case Report." *American Journal of Case Reports*, 26, e946364. <https://doi.org/10.12659/ajcr.946364>
- [4] De Silva, A. P., and Prabagar, K. (2026), "Chikungunya-associated rhabdomyolysis: a case series of three patients from Sri Lanka." *Transactions of The Royal Society of Tropical Medicine and Hygiene*. <https://doi.org/10.1093/trstmh/trag030>

- [5] Elfert, K. A., Abdelwahed, M., and Chi, G. (2019), "Chikungunya Virus Infection-related Rhabdomyolysis: A Case Report." *Cureus*, 11(2), e4036. <https://doi.org/10.7759/cureus.4036>
- [6] Dadana, S., Kondapalli, A., and Jadav, R. S. (2024), "Unusually high creatine kinase in a case of rhabdomyolysis without acute kidney injury: a case report." *AME Case Rep*, 8, 41. doi: 10.21037/acr-23-172. PMID: 38711898; PMCID: PMC11070983.
- [7] McMahon, G. M., Zeng, X., and Waikar, S. S. (2013), "A Risk Prediction Score for Kidney Failure or Mortality in Rhabdomyolysis." *JAMA Intern Med*, 173(19), 1821-1827. doi:10.1001/jamainternmed.2013.9774