

## Case Report

# Rhabdomyolysis Following Multiple Wasp Stings: A Case Report

Supun Wedasingha<sup>1,2\*</sup>, Rathnasiri Rajapaksha<sup>1</sup>, Inu Wijerathna<sup>1</sup>, Vasana Mendis<sup>3</sup>, Prasanna Weerawansa<sup>4</sup>

<sup>1</sup>Postgraduate Institute of Medicine, University of Colombo, Sri Lanka.

<sup>2</sup>Department of Pharmacology, Faculty of Medicine & Allied Sciences, Rajarata University of Sri Lanka, Sri Lanka.

<sup>3</sup>Department of Pathology, Faculty of Medicine & Allied Sciences, Rajarata University of Sri Lanka, Sri Lanka.


<sup>4</sup>Department of Medicine, Faculty of Medicine & Allied Sciences, Rajarata University of Sri Lanka, Sri Lanka.

## Abstract

Hymenoptera stings can result in severe systemic reactions, including rhabdomyolysis—a condition characterized by skeletal muscle breakdown. This report describes a case of rhabdomyolysis following multiple wasp stings and underscores the critical role of prompt treatment.

A 63-year-old farmer was admitted to the Emergency Department following multiple wasp stings on the head, face, trunk, and upper limbs. He presented with pain at the sting sites and gastrointestinal symptoms like diarrhoea, but no signs of systemic allergic reactions or previous significant health issues. Initial tests revealed mild liver enzyme elevation, with normal kidney function. By Day 3, the patient exhibited muscle pain and significantly elevated creatine phosphokinase (CK) levels (12,597 U/L), confirming rhabdomyolysis. Aggressive fluid resuscitation, including forced alkaline diuresis, was administered. The patient remained hemodynamically stable, with normal renal function throughout his hospital stay. CPK and liver enzyme levels gradually normalized, and he was discharged after 5 days with advice for continued hydration. Follow-up confirmed recovery without residual complications.

Rhabdomyolysis should be considered in patients with muscle pain following wasp stings. Early diagnosis and aggressive fluid therapy are crucial in preventing severe complications, such as acute kidney injury. Further research is needed to elucidate the mechanisms of wasp venom-induced rhabdomyolysis and refine treatment strategies.

**Copyright** Wedasingha S et al. 2025.  This is an open-access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium provided the original work is properly cited.

**Funding:** None

**Competing interests:** None

**Received:** 23 March 2025

**Accepted:** 01 June 2025

**Published:** 30 June 2025

**\*Corresponding author:** [swnadeeja@gmail.com](mailto:swnadeeja@gmail.com)

 <https://orcid.org/0000-0002-3703-9764>

**Cite this article as:** Wedasingha S et al Rhabdomyolysis following multiple wasp stings: A case report. *Journal of Tropical Health* 2025;1 (2): 104-108. DOI: <http://doi.org/10.4038/joth.v1i2.10>

## Introduction

Hymenoptera stings are one of the frequent environmental hazards in Sri Lanka, accounting for significant mortality and morbidity [1]. Medically significant Hymenoptera are broadly categorised into 3 families: Apidae (honeybees and bumblebees), Vespidae (yellow jackets, hornets, and wasps), and Formicidae (ants) [2]. Wasp stings commonly lead to

local reactions, which are mild and transient [3]. Rarely, wasp stings can lead to serious systemic complications such as acute kidney injury, liver injury, myocardial infarction, and rhabdomyolysis [4].

Rhabdomyolysis is a potentially life-threatening syndrome, which classically presents with muscle pain and tea-coloured urine [5]. Acute kidney injury is the most common systemic complication of

rhabdomyolysis, with an incidence ranging between 10 and 55% [6]. Hymenoptera stings per se can cause acute kidney injury, with [7] or without rhabdomyolysis [8].

Here, we report a case of a patient who developed rhabdomyolysis following multiple wasp stings and successfully managed with early, aggressive fluid resuscitation.

### Case report

A 63-year-old previously healthy farmer was admitted to the Emergency Treatment Unit about 1 hour after multiple wasp stings. He was attacked on the head, face, upper part of the trunk and both upper limbs by more than 50 wasps while he was walking on a road. On admission, he complained of pain at the sites of the sting. He had three episodes of watery, loose stools without associated vomiting or abdominal pain. On examination, he was afebrile and was conscious and rational. No stingers were seen at sites of the sting as would be expected following wasp stings, and there were circumscribed punctuated lesions typical for wasp stings. He did not have cutaneous manifestations suggestive of an allergic reaction, and he was not dehydrated. His blood pressure was 130/90 mmHg, and his heart rate was regular at 76 beats per minute. His respiratory rate was 18 cycles per minute, oxygen saturation was 100% on ambient air, and his lungs were clear during auscultation. He had no chest pain, shortness of breath, palpitations or faintishness. He did not have a recent history of heavy exertion or any febrile illnesses. He was not on any routine medications. There was no history suggestive of abuse of alcohol or illicit drugs. He did not have a significant history of allergies or atopy, and there was no previous history of wasp stings. The culprit insect was confirmed to be a wasp after the patient was shown a picture of it. Initial laboratory tests revealed derangement of liver enzymes with markedly elevated serum aspartate aminotransferase (AST) and alanine transaminase (ALT) levels (Table 1).

After 48 hours from the wasp sting, the patient complained of generalised muscle pain. On examination, there was significant generalised muscle tenderness without weakness. He had neither oliguria nor hematuria. He did not complain of any chest pain or shortness of breath. Plasma AST and ALT had further risen to 636 U/L and 143 U/L, respectively. However, serum bilirubin levels and alkaline phosphate

levels were normal. The coagulation profile was normal. Serum LDH levels were elevated at 1329 U/L: there was no red cell fragmentation seen in the blood picture. Urine microscopic examination revealed 1+ protein and occasional red blood cells. Urine myoglobin sent three days after the admission was negative. Serum creatinine was normal, and there was no acidosis in venous blood gas analysis. Serum electrolytes, including corrected calcium and phosphate levels, were within normal range. The electrocardiogram revealed sinus rhythm without any ST-T changes. However, high-sensitive troponin was elevated (highest troponin value 285 ng/L). The possibility of skeletal muscle damage leading to rhabdomyolysis following a wasp sting was raised. Serum creatine phosphokinase (CPK) was significantly elevated at 12,579 U/L (The upper limit of normal 195 U/L).

The patient was given adequate hydration with oral fluids as well as intravenous fluids (normal salines of an average of 2.5- 3.0 l/24 h) following regular assessment of hydration status (including twice daily ultrasonic bedside assessment of the inferior vena cava) and strict input and output monitoring was maintained. Liver and renal function tests were monitored daily. In anticipation of probable acute kidney injury, forced alkaline diuresis with intravenous sodium bicarbonate solution was also undertaken once a diagnosis of rhabdomyolysis was made.

Throughout the hospital admission, the patient remained hemodynamically stable, with adequate urine output and normal serum creatinine levels. The patient was discharged after 5 days of inpatient management and was advised to continue taking adequate fluids even after discharge. During day 12 post-sting, the patient's AST and ALT levels, as well as CPK levels, had returned to normal. The thyroid profile was normal, and the ultrasound scan of the abdomen did not show any focal lesions.

### Discussion

Here, we report a previously healthy adult who developed rhabdomyolysis following multiple wasp stings without subsequent development of acute kidney injury. Prompt diagnosis, early aggressive fluid therapy including forced alkaline diuresis, strict input-output monitoring and regular monitoring of renal function helped prevent the development of acute kidney injury and thus led to a favourable outcome.

**Table 1:** Summary of laboratory investigations of the patient

	Normal range	Day 1	Day 3	Day 4	Day 5
Hemoglobin (grams/dL)	11-17	13.8	13.2	12.2	11.0
Platelet count (10 <sup>9</sup> /L)	150-400	462	273	195	268
White cell count (10 <sup>9</sup> /L)	4.00-10.00	21.46	29.10	16.45	14.15
Neutrophil count (10 <sup>9</sup> /L)	2.00-7.00× 10 <sup>3</sup>	20.25	25.23	14.26	7.97
Lymphocyte count (10 <sup>9</sup> /L)	1.00-3.00× 10 <sup>3</sup>	0.64	2.97	1.71	4.63
Eosinophil count (10 <sup>9</sup> /L)	1.00-3.00× 10 <sup>3</sup>	0.03	0.01	0.05	0.31
Aspartate transaminase (U/L)	15-37	467	636	609	207
Alanine transaminase (U/L)	30-65	67	143	175	163
Creatine phosphokinase (CPK)	≤ 195 U/L		12579		2660
Alkaline phosphatase (U/L)	46-116			72	75
Gamma GT (U/L)	7-50			55	60
Total bilirubin (μmol/L)	5-21			10.7	8.9
Direct bilirubin (μmol/L)	0.0-3.4			3.9	3.5
Indirect bilirubin (μmol/L)	5-21				
Albumin (g/L)	35-45				
PT-INR	??		0.863	26	0.81
Sodium (mEq/L)	136-149	136		135	137
Potassium (mEq/L)	3.6- 5.2	4.5		3.5	3
blood urea (mmol/L)	2.5- 6.4	6.62		3.1	
Serum creatinine (micromoles/L)	60-120	81		62.7	63
C reactive protein (milligram/L)	<5	88.6			
High sensitive troponin (ng/L)	0-0- 53		285	179	
Corrected calcium (mmol/L)	2.1-2.6			2.25	
Phosphorous (mmol/L)	0.7- 1.45			0.69	
LDH (U/L)	225- 450			1329	
Venous pH	7.35-7.45			7.40	7.476
Venous HCO <sub>3</sub> (mmol/L)	22-26			26	27.2
Venous pCO <sub>2</sub> mmHg	35-45			42	36.5
Venous pO <sub>2</sub> mmHg	75-100			20	32.4
Serum lactate (mmol/L)	??			1.4	1.1
Urine pH	??			7.0	
Urine red cells	??		Occasional		
Urine casts	??		Nil		
Urine crystals	??		Nil		
Urine for myoglobin	absent			Negative	

LDH; Lactate dehydrogenase, PT-INR; Prothrombin time-international normalised ratio

Rhabdomyolysis is a potentially fatal condition characterised by the breakdown of skeletal muscle fibres and subsequent release of intracellular muscle components, including myoglobin, CPK, AST, aldolase, and lactate dehydrogenase, as well as electrolytes, into the bloodstream and extracellular space [9]. The clinical manifestations can vary widely, ranging from an asymptomatic state with elevated serum creatine kinase levels to severe, systemic effects. Tea-coloured urine, muscle tenderness, swelling, stiffness, cramping and muscle weakness are some of the classic manifestations of rhabdomyolysis [5]. Myoglobinuria, acute renal failure, compartment syndrome and disseminated intravascular coagulation are among the major life-threatening complications associated with rhabdomyolysis [5,9]. The commonest cause of rhabdomyolysis is direct traumatic injury. It

can also occur due to drugs, toxins, infections, muscle ischemia, electrolyte disturbances, metabolic disorders, genetic disorders, vigorous exercise or prolonged bed rest, and temperature-induced states such as neuroleptic malignant syndrome and malignant hyperthermia [9]. Hymenoptera envenomation is one of the less frequent causes of rhabdomyolysis [10].

Wasp envenomation is known to cause systemic hyper-inflammatory reaction. Several studies have shown elevated cytokine levels, including Interleukin-2 (IL-2), IL-6, IL-8, IL-10, IL-17, Interferon-γ (IFN-γ), and tumour necrosis factor-α (TNF-α) following wasp stings [11]. Rhabdomyolysis is thought to result from skeletal muscle cell membrane disruption by PLA1 and melittin in wasp venom [12]. Mastoparan can induce myonecrosis, apoptosis and activation of cytokines [13].

The diagnosis of rhabdomyolysis is primarily based on clinical findings and laboratory investigations. An elevated plasma CPK level is considered the most sensitive laboratory finding indicative of muscle injury [5]. A creatine kinase concentration 5 times the upper limit of the normal reference range (i.e., 1,000 IU/L) is commonly used to diagnose rhabdomyolysis. CPK level is generally considered to correlate with the likelihood of developing acute kidney injury, and a concentration greater than 5,000 IU/L is associated with the development of kidney damage [9]. In this case, the CPK level of the patient is markedly elevated, thus confirming the diagnosis of rhabdomyolysis. Elevated serum myoglobin and myoglobinuria are also sometimes taken as diagnostic parameters for rhabdomyolysis. However, their sensitivity and specificity can be confounded by many factors [5]. CPK has a half-life of 1.5 days, whereas myoglobin has a half-life of 2- 4 hours [9]. Therefore, serum myoglobin typically rises before a rise in CPK and declines more rapidly than CPK [5]. Moreover, urine myoglobin will show red blood cell positivity on urine dipstick testing because the orthotoluidine portion of the dipstick can turn blue in the presence of myoglobin [9]. Therefore, serum or urine myoglobin levels are neither sensitive nor specific enough to make a diagnosis of rhabdomyolysis [5]. In this case, serum myoglobin levels were not readily available. Even the urine myoglobin levels sent after three days of admission came as negative, which would be expected with the half-life of myoglobin. Even without serum or urine myoglobin levels, rhabdomyolysis could be reliably diagnosed with CPK levels. Being vigilant about the possibility of rhabdomyolysis and noting the

derangement of liver enzymes facilitated a prompt diagnosis.

The primary goal in managing rhabdomyolysis is to prevent acute kidney injury by maintaining adequate renal perfusion and promoting the clearance of myoglobin. This is achieved through early, aggressive intravenous fluid therapy. In this case, the patient was treated with intravenous fluids, as well as forced alkaline diuresis, even without any renal impairment. The patient maintained an adequate urine output throughout the hospital stay, with no impairment of renal function noted. This aggressive fluid resuscitation protocol ultimately led to a gradual normalization of CPK levels. Additionally, monitoring and correction of electrolyte imbalances, particularly hyperkalemia, are crucial components of management.

### Conclusion

This case report highlights the importance of recognizing rhabdomyolysis as a potential complication following wasp envenomation. Early recognition and aggressive fluid therapy are the key to preventing the development of further complications, particularly acute kidney injury. When a patient complains of muscle pain and weakness following wasp stings, clinicians need to consider rhabdomyolysis as one of the primary differential diagnoses. Future research should focus on better understanding the pathophysiological mechanisms of rhabdomyolysis and the development of targeted therapeutic modalities.

### Consent

Informed written consent was obtained from the patient for the publication of this case report.

## References

1. Ralapanawa DMPUK, Kularatne SAM. A case of Kounis syndrome after a hornet sting and literature review. *BMC Res Notes*. 2014;7:1-5. doi:10.1186/1756-0500-7-867.
2. Witharana EWRA. Hymenoptera (wasps, bees and ants) envenomation; a neglected tropical disorder in Sri Lanka. *J Ceylon Coll Physicians*. 2023;54:4-11. doi:10.4038/jccp.v54i1.7992.
3. Gong J, Yuan H, Gao Z, Hu F. Wasp venom and acute kidney injury: The mechanisms and therapeutic role of renal replacement therapy. *Toxicon*. 2019;163:1-7. doi:10.1016/j.toxicon.2019.03.008.
4. Radhakrishnan H. Acute kidney injury and rhabdomyolysis due to multiple wasp stings. *Indian J Crit Care Med*. 2014;18:470-2. doi:10.4103/0972-5229.136079.
5. Zutt R, van der Kooi AJ, Linthorst GE, Wanders RJA, de Visser M. Rhabdomyolysis: Review of the literature. *Neuromuscul Disord*. 2014;24:651-9. doi:10.1016/j.nmd.2014.05.005.

6. Chavez LO, Leon M, Einav S, Varon J. Beyond muscle destruction: A systematic review of rhabdomyolysis for clinical practice. *Crit Care*. 2016;20:135. doi:10.1186/s13054-016-1314-5.
7. Natarajan G, Jeyachandran D, Thanigachalam D, Sakthirajan R, Balasubramaniyan T, Kurien AA. Myoglobinuric renal failure after a wasp attack. *Kidney Int*. 2016;89:725. doi:10.1016/j.kint.2015.12.013.
8. Zhang R, Meleg-Smith S, Batuman V. Acute tubulointerstitial nephritis after wasp stings. *Am J Kidney Dis*. 2001;38:1-5. doi:10.1053/ajkd.2001.29289.
9. Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: Pathogenesis, diagnosis, and treatment. *Ochsner J*. 2015;15:58-69.
10. Mingomataj EÇ, Bakiri AH, Ibranjic A, Sturm GJ. Unusual reactions to hymenoptera stings: What should we keep in mind? *Clin Rev Allergy Immunol*. 2014;47:91-9. doi:10.1007/s12016-014-8434-y.
11. Ruwanpathirana P, Priyankara D. Clinical manifestations of wasp stings: a case report and a review of literature. *Trop Med Health*. 2022;50:38. doi:10.1186/s41182-022-00475-8.
12. Constantino K, Pawlukiewicz AJ, Spear L. A case report on rhabdomyolysis after multiple bee stings. *Cureus*. 2020;12:e9501. doi:10.7759/cureus.9501.
13. Rocha T, Soares de Barros LL, Fontana K, Monson de Souza B, Palma MS, Alice da Cruz-Höfling M. Inflammation and apoptosis induced by mastoparan Polybia-MPII on skeletal muscle. *Toxicon*. 2010;55:1213-21. doi:10.1016/j.toxicon.2009.12.005.