Scientific Research Publishing

Case Reports in Clinical Medicine, 2025, 14(8), 440-452
https://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

# Successful Use of HA 330 II Extracorporeal Therapy in a Case of Multi-Organ Dysfunction Syndrome Following Multiple Hornet Stings

## —A Case Report of Successful Bridging Therapy to Recovery

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**How to cite this paper:** Anandhakumar, A., Ganapathy, G., Ganapathy, S., Ganapathy, N. and Govindan, T. (2025) Successful Use of HA 330 II Extracorporeal Therapy in a Case of Multi-Organ Dysfunction Syndrome Following Multiple Hornet Stings. *Case Reports in Clinical Medicine*, **14**, 440-452.
https://doi.org/10.4236/crcm.2025.148055

**Received:** July 10, 2025
**Accepted:** August 10, 2025
**Published:** August 13, 2025

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## Abstract

Hornet stings can result in severe local tissue injury, anaphylactic shock, and systemic inflammatory response syndrome (SIRS), potentially progressing to multi-organ dysfunction syndrome (MODS). We report a case of a 39-year-old gentleman who developed MODS following multiple hornet stings. His clinical course was complicated by the development of acute kidney injury, toxic hepatitis, disseminated intravascular coagulation (DIC), and cardiovascular instability. Laboratory findings revealed hyperkalaemia, metabolic acidosis, thrombocytopenia, hyperbilirubinemia, and elevated inflammatory markers. Initial management was focused on fluid resuscitation, haemodynamic support using inotropes, and initiation of sustained low-efficiency dialysis (SLED). Due to progressive worsening of acute kidney injury associated with haemodynamic instability, Continuous Veno-Venous Haemodiafiltration (CVVHDF) was initiated. The addition of plasmapheresis and HA 330 II haemoperfusion therapy led to marked clinical improvement with expedited weaning from supportive therapies, and the patient recovered without long-term complications. This case highlights the importance of early initiation of plasmapheresis in the management of MODS following hornet envenomation. Furthermore, the addition of haemoperfusion therapy facilitated the removal of cytokines, which contributed to improved clinical outcomes. Early institution of plasmapheresis and haemoperfusion may reduce morbidity and mortality associated with severe systemic hornet envenomation.

## Keywords

Multiple Hornet Sting-Induced Multi-Organ Dysfunction Syndrome,

Continuous Renal Replacement Therapy, Haemoperfusion Adsorbent 330 II Extracorporeal Therapy

## 1. Introduction

Arthropod stings are generally known to cause mild local reactions. Though, not infrequently, they cause systemic symptoms that can include an urticarial rash and, in some rare cases, anaphylactic shock. Occasionally, they cause serum sickness, and in some rare cases, unusual neurological symptoms have been reported [1]. Bees, wasps, and hornets are common insects that are associated with clinically significant stings. Hornet stings can result in severe local tissue injury, anaphylactic shock, and systemic inflammatory response syndrome (SIRS), potentially progressing to multi-organ dysfunction syndrome (MODS). We report a case of a 39-year-old man who developed MODS following multiple hornet stings. Initial management was focused on fluid resuscitation and haemodynamic support using inotropes. Due to progressive worsening of MODS, management escalated to SLED, plasmapheresis, CVVHDF, and finally HA 330 II haemoperfusion, resulting in a rapid decline in inflammatory markers and full recovery. This case highlights the importance of early initiation of plasmapheresis in the management of MODS following hornet envenomation. Furthermore, the addition of haemoperfusion therapy can bridge critically ill envenomation patients to organ recovery. Early institution of plasmapheresis and haemoperfusion may reduce morbidity and mortality associated with severe systemic hornet envenomation.

We obtained informed consent from the patient and approval from the ethics committee at Dhanvantri Critical Care Center, Erode, Tamil Nadu, India, prior to publication of this case report.

## 2. Case Report

Mr R, a 39-year-old male who is a coconut plucker, was admitted to our hospital with a history of multiple hornet stings when he accidentally disturbed a nest on the tree. He was reportedly stung by approximately 100 hornets, following which he felt intense pain, dizziness, and generalised abdominal discomfort, which led him to lose the grip he had held around the tree. Consequently, he fell from an approximate height of 25 metres (the height of the tree), landed on a haystack, and sustained an injury to his left upper limb. There was no history of head strike, loss of consciousness, or injury to the neck, torso, or lower limbs. Local villagers transported him to a regional public hospital within 2 hours of the injury, where he received basic supportive care (analgesia and intravenous fluids). Due to resource constraints at the centre, he was transferred to our hospital 24 h later for further management.

## 3. Presentation

On arrival at our ED, the patient was conscious, alert, oriented, afebrile, clinically hypovolemic, and mildly tachycardic at 118/min, and normotensive with a BP of 110/70. The primary trauma survey was largely unremarkable. A superficial abrasion over the ventral aspect of the left forearm was noted during the secondary survey, and this was sustained when he fell from the tree immediately after the hornet sting. There was no evidence of other injuries sustained from the fall. Multiple blisters, associated with evidence of central necrosis of the surrounding skin at sting sites, were noted throughout his torso and limbs (Figure 1).

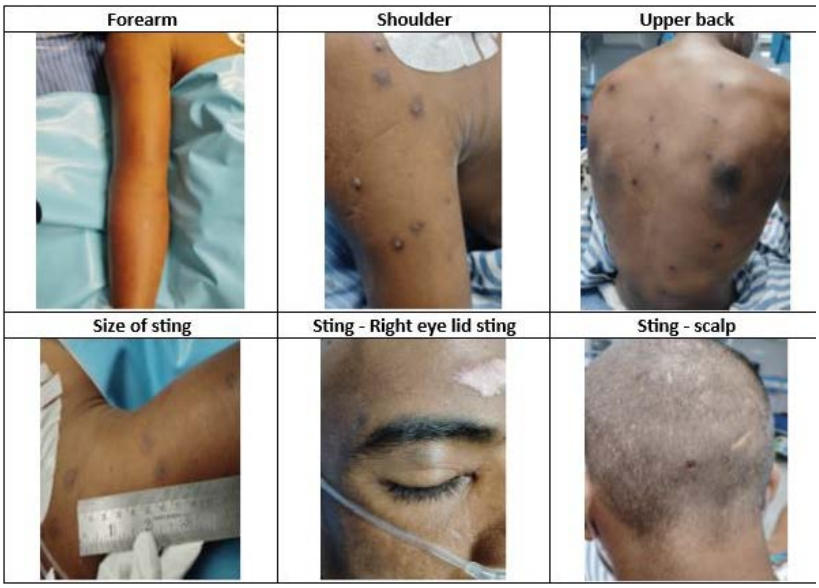


Figure 1. Hornet sting sites—various parts of the body.

Mr R denied any previous history of wasp, bee, or hornet stings. He had no significant past medical history, no known comorbid conditions, was not on any prescription medications, and denied any use of illicit substances or non-prescription drugs. He was able to identify the insect as a "hornet," also known as "malaikolavi" (in the local language), based on its appearance, size, and characteristics of the nest and its location on the tree (Figure 2).

Figure 2. Hornet (involved in this case).

### 3.1. Early Investigation & Management

Initial management included supportive care with fluid rehydration and provision of analgesia. A blood gas analysis revealed hyperkalaemia (K+ – 6.0 mmol/L) with partially compensated metabolic acidosis (pH – 7.22, $HCO_3^-$ 13 mmol/L, BE – 14 mmol/L, $PCO_2$ – 32 mm Hg). Hb was 180 gm/L, and lactate was 4.1 mmol/L. ECG revealed mild sinus tachycardia and hyperacute T waves. Hyperkalaemia was treated with nebulised salbutamol and 10 ml of 10% calcium gluconate intravenously, with a good response. Subsequent blood gas analysis showed a reduction in potassium to 4.7. Complete blood count revealed Hb 18 gm/dl, leucocytosis 22.65 × $10^9$, and mild thrombocytopenia 90 × $10^9$/L; coagulation profile revealed mild coagulopathy (INR 1.4). Due to signs of evolving sepsis, the patient was commenced on meropenem. Further investigations revealed evidence of acute kidney injury with creatinine 5.4 mg/dl (ref: 0.7 - 1.3 mg/dl), urea 161 mg/dl (ref: 5-20 mg/dl), mild hepatic injury (ALT 559 U/L), and elevated serum bilirubin 34 mg/dl (ref: < 1.2 mg/dl). However, the hepatitis serology panel was negative. Abdominal ultrasound revealed the liver was of normal echogenicity and echotexture, and an unremarkable portal vein and biliary tract.

### 3.2. Clinical Course in Hospital

The patient was initially managed in the ED and was transferred to the intensive care unit for close monitoring and ongoing management. He developed central chest and epigastric discomfort. An ECG showed normal sinus rhythm, isoelectric ST segments, and no evidence of conduction abnormalities. He was treated with antacids and proton pump inhibitors, with some relief. He was haemodynamically stable, though bedside echocardiography revealed evidence of myocardial dysfunction with anterior wall hypokinesia, which was associated with a mild reduction in ejection fraction (EF 51%), and the corresponding Troponin I was 0.5 ng/ml (ref: < 0.04 ng/ml); serial electrocardiographs were unremarkable. Epigastric pain was initially thought to be gastritis; however, in view of persistent symptoms, pancreatic enzymes were tested, which revealed evidence of mild pancreatitis (amylase 1374 IU/L, lipase 550 IU/L). Inflammatory markers were elevated (CRP 118 mg/L, procalcitonin 20 ng/ml (ref: < 0.05 ng/ml), and IL-6 was 1648 pg/ml (ref: 1 - 5 pg/ml). A short course of methyl prednisolone [oral loading dose 100 mg followed by 50 mg 6-hourly for 2 days and tapered over 5 days] was administered in view of evolving SIRS. Methyl prednisolone was administered to treat the allergic reaction secondary to multiple stings, and it was tapered over a period of 5 days.

Coagulopathy was corrected using one unit of whole blood. One unit of single-donor platelet was transfused as per local practice. Right common femoral vein CVC catheter and left radial artery cannula were placed for haemodynamic monitoring. 16Fr Foley's catheterization was performed for close monitoring of

urine output. The patient developed oliguric renal impairment with rising creatinine (5.4 mg/dl). Point-of-care renal ultrasound demonstrated a high resistive index in both kidneys [Right 0.81, Left 0.79], which indicated evolving renal injury.

Despite crystalloid fluid resuscitation, he developed oliguric renal injury, which was associated with worsening metabolic acidosis; his urine colour was tea-coloured (**Figure 3**). The patient was commenced on Sustained Low-Efficiency Dialysis (SLED) at a blood flow of 250 ml/min and a dialysate flow of 300 ml/min. In view of the progression of SIRS into multiorgan dysfunction, therapeutic plasmapheresis was commenced using the Spectra Optia Apheresis System (TERUMOBCT) for 2 h 30 min. Six units of fresh frozen plasma and three units of 20% human albumin, mixed in 500 ml of 0.9% normal saline, were utilised during plasmapheresis. The patient developed multifactorial shock over the subsequent 24 h, which was managed with crystalloid fluid resuscitation and 100 ml of 20% human albumin, followed by escalation to inotropic support using dobutamine at 6.6 mcg/kg/min; concurrently, in view of haemodynamic instability, SLED was transitioned to continuous renal replacement therapy with a Prismaflex M100 in CVVHDF mode. Renal ultrasound demonstrated a loss of cortico-medullary differentiation of both kidneys. Once haemodynamic stability was achieved, blood flow in CVVHDF was increased from 100 to 120 ml/min, and an HA 330 II haemoperfusion cartridge (**Figure 4**) was incorporated into the Prismaflex M100 to facilitate enhanced elimination of toxins and inflammatory mediators, as the HA 330 II resin cartridge is known to absorb most inflammatory mediators and cytokines, including IL-1, IL-6, IL-8, and TNF-α. His clinical course was complicated by the development of new hypoxia ($SpO_2$ 90%; $PaO_2$ on ABG at 28% $FiO_2$ was 67 mmHg) and mild anaemia (Hb 11 g/dl). There was no clinical evidence of a septic focus. Point-of-care ultrasound (POCUS) revealed a dilated IVC with a collapsibility index of 18%, and lung ultrasound demonstrated bilaterally scattered B lines and pleural effusion suggestive of systemic capillary leak syndrome (SCLS). Chest X-ray demonstrated diffuse, mild ground-glass opacification consistent with ARDS, associated with bilateral moderate-volume pleural effusion; however, there was no evidence of consolidation. These findings, along with echocardiographic features of mild LV anterior wall hypokinesia and ongoing haemodynamic instability, led to the diagnosis of myocardial dysfunction due to toxic myocarditis. The corresponding troponin I was 1.2 ng/ml (ref: < 0.04 ng/ml). Oxygen therapy was titrated to achieve an SpO2 > 95%, and the flow rate on CVVHDF was gradually increased to 500 ml/h; blood flow was increased from 120 to 150 ml/min for a period of 8 hours, during which a total of 1,500 ml of fluid was removed. His oxygen requirement reduced, and he demonstrated haemodynamic stability with a BP of 130/80 mm Hg on a low dose (2.5 mcg/kg/min) of dobutamine.


**Figure 3.** Tea-coloured urine.

**Figure 4.** Mr R undergoing CVVHDF with an HA 330 II haemoperfusion cartridge.

**High-resolution CT: Chest**

Key findings:

Bilateral pleural effusion is seen with passive collapse of the basal segment.

2) Interstitial septal thickening with superimposed ground glass opacities in the apico-posterior segment of the left upper lobe

**CT: Abdomen**

Key findings:

1) Bilateral bulky kidneys with perinephric fat stranding – acute kidney injury.

2) Bilateral non-obstructive renal calculi

3) Small haemorrhagic cyst in the left kidney

4) Mild ascites

5) Mild bilateral pleural effusion is noted, with collapse of both lower lobes.

6) The pancreas appeared normal in size and attenuation. No evidence of calculus/calcification. The peripancreatic fat planes appeared normal.

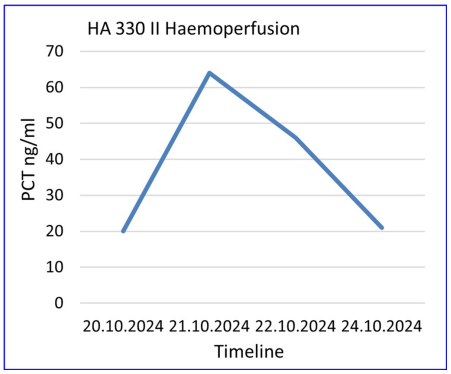


**Figure 5.** Serum procalcitonin (ng/ml)—[The *arrow indicates the time at which HA* 330 *II haemoperfusion was commenced*].

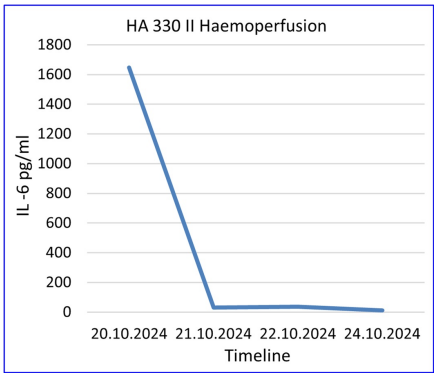


**Figure 6.** Serum IL-6. (pg/ml)—[*The arrow indicates the time at which HA* 330 *II haemoperfusion was commenced*].

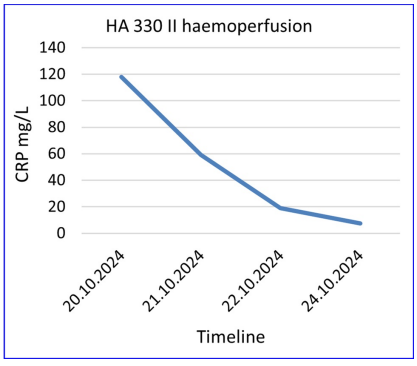


**Figure 7.** Serum CRP (mg/L)—[*The arrow indicates the time at which HA.* 330 *II haemoperfusion was commenced*].

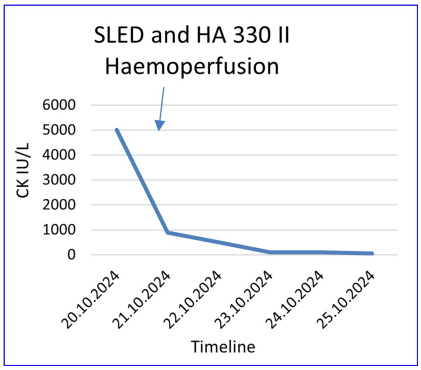


**Figure 8.** Serum CK (IU/L)—*Arrow indicates the time at which SLED and HA* 330 *II haemoperfusion were commenced.*

## 4. Discussion

Hymenoptera, often referred to as stinging wasps, represent an exceptionally diverse group of arthropods encompassing more than 70,000 species [2]. Broadly, this group is categorized into two main families: Apidae (bees) and Vespidae (wasps and hornets). A key distinction lies in the sting apparatus; dominant hornet species found globally exhibit variations in their size, structure, and stinging mechanisms. These differences contribute to distinct sting profiles and, consequently, diverse clinical effects in victims. Beyond internal mechanisms, there are observable morphological differences that assist in distinguishing between these two families. For example, bee stingers typically detach and remain embedded in the victim's skin, whereas wasp stingers do not. This means bees can sting only once, while wasps and hornets are capable of multiple stings, increasing the risk of significant envenomation.

Hymenopteran stings are a common reason for visits to local health facilities, with victims typically presenting with localized pain and allergic reactions. Often, victims can identify whether they were stung by a bee, wasp, or hornet based on the insect's appearance and its nest. For instance, a retrospective analysis of 114 hymenopteran sting cases in Thailand revealed that bees were responsible for 48% and hornets for 14% of documented incidents [3]. Despite the prevalence of bee stings, the ability of wasps and hornets to inflict multiple stings poses a greater risk of severe envenomation.

In the Indian subcontinent, *Vespa affinis*, *Vespa orientalis*, *Vespa mandarinia*, and *Vespa velutina* are some of the common hornet species. In Tamil Nadu, India, the Asian giant hornet (*Vespa mandarinia*) and Asian hornet (*Vespa velutina*) are particularly abundant in peri-urban and rural villages, especially those close to agricultural and bushlands. While many of these stings typically result in mild local reactions, often managed by victims using simple analgesics and traditional local remedies, the potential for repeated stings from wasps and hornets highlights the need for vigilance against severe envenomation. Beyond mild local reactions, Hymenopteran stings can frequently induce more significant systemic symptoms, such as urticarial rash, and, in severe instances, anaphylactic shock, prompting

presentation to local health facilities. Notably, Hymenopteran stings are associated with up to 14% of anaphylactic shock cases [4]. Rarer complications, like delayed hypersensitivity reactions (e.g., serum sickness) and unusual neurological events, have been reported [1]. Clinical predictors of the severity of Hymenopteran stings are scarcely covered in the existing literature, largely confined to case reports. For example, in a small series of 15 patients, Yanagawa *et al*. observed that fatalities were linked to an average sting count exceeding 59 ± 12. They further postulated that the presence of cutaneous haemorrhage or necrosis could signify progression to multiorgan injury [5]. Their report details a case of Multi-Organ Dysfunction Syndrome (MODS) resulting from multiple hornet stings and highlights the therapeutic utility of CVVHDF and resin-based haemoperfusion in this clinical setting.

Hymenoptera venom contains a complex mixture of enzymes (phospholipase and hyaluronidase), peptides (melittin), biogenic amines, and amino acids. These toxins can induce various systemic effects, including hepatic injury, kidney injury, myolysis, haemolysis, and vasodilatory shock [4]. *In vitro* studies by Neuman *et al*. using rat hepatocytes demonstrated that hornet venom extract contains hepatotoxins capable of causing transaminitis [6]. Similarly, Tsai *et al*. reported a case of hornet sting-associated toxic hepatitis and coagulopathy that developed four hours post-exposure and resolved with supportive care over four days [7]. In a retrospective analysis of Hymenopteran sting cases at the Ramathibodi Poison Centre, Thailand, Srisuwarn *et al*. identified that more than 10 stings, dark-coloured urine, and cardiorespiratory collapse were associated with adverse outcomes [3]. Mr R reported over 100 stings and developed severe acute kidney injury (AKI) within the first 24 hours, with subsequent progression to MODS. In a case series of 11 patients with wasp stings, Dhanapriya *et al*. found that all developed rhabdomyolytic AKI, with three exhibiting features of acute interstitial nephritis (AIN) or acute tubular necrosis (ATN) [8]. They concluded that early initiation of corticosteroid therapy and commencement of dialysis in patients who present with AKI were associated with improved renal recovery. Prevalence of AKI following hornet stings is high (84.5%), predominantly associated with rhabdomyolysis [8]. Mr R presented with oliguric renal failure with metabolic acidosis; hence, haemodialysis in SLED mode was initiated promptly. He also received a course of methylprednisolone to initially treat the allergic reaction secondary to stings and as an adjunct measure to treat evolving septic shock. It was weaned over a period of 5 days. Annane *et al*. conducted a systematic review of the use of corticosteroids in the treatment of severe sepsis and septic shock in adult patients and found that a subgroup of 12 trials investigating low-dose corticosteroid treatment suggested a favourable effect on all-cause mortality [9]. According to these findings, corticosteroids should be considered at a daily dose of 200 to 300 mg of hydrocortisone (equivalent to 40 to 60 mg of methylprednisolone) as an intravenous bolus or continuous infusion. Although evidence is not particularly robust, the authors suggested that treatment should be given at full dose for at least 100

hours and only in adults with vasopressor-dependent septic shock.

Reports suggest a lethal dose for Hymenopteran stings to be over 20 stings/kg [10] [11]; however, this generally applies to Native American and European species, which possess significantly less potent venom compared to their Asian counterparts. Liu *et al*.'s research highlighted the severe toxicity of Asian species, reporting a 37% mortality rate in patients with more than 30 stings, escalating to 74% for those sustaining over 100 stings [12]. Drawing from these critical observations, the authors formulated a Wasp Sting Severity (WSS) score (**Table 1**). This score utilizes several key parameters: the number of stings, presence of tea-coloured urine, and levels of LDH and total bilirubin. A WSS score exceeding 3 was identified as a strong predictor of severe illness, warranting consideration for therapeutic plasma exchange (TPE). Mr R had a WSS score of 9 as he had been stung; 100 hornets, developed oliguric renal impairment with evidence of tea-coloured urine (**Figure 3**), and his bilirubin level was 34 mg/dl (581 μmol/L).

**Table 1.** Wasp sting severity score (WSS).

| Assigned points | 1 | 2 | 3 |
|---|---|---|---|
| Tea coloured urine | | | Positive |
| Number of stings | 15 - 29 | 30 - 49 | >50 |
| LDH (U/L) | 400 - 699 | 700 - 999 | >1000 |
| Total Bilirubin (μmol/L) | 30 - 49 | 50 - 79 | >80 |

*Each point represents approximately a 20% higher probability of developing severe illness requiring blood purification. Liu et al.*

The therapeutic efficacy of TPE is thought to stem from its ability to enhance the clearance of venom components, secondary toxic agents, and various inflammatory mediators [13]. Mr R presented with a constellation of severe complications: acute kidney injury; acute toxic hepatitis, evidenced by elevated transaminitis and hyperbilirubinemia; acute pancreatitis, indicated by raised lipase and amylase levels; and toxic rhabdomyolysis, characterized by elevated CK (5000 IU/L) and dark-coloured urine. These were further complicated by the development of myocardial dysfunction (reduced ejection fraction and elevated troponin I) and SCLS, which was associated with significantly raised inflammatory markers, including C-reactive protein, procalcitonin, and IL-6. The prompt initiation of plasmapheresis, followed by CVVHDF augmented by HA 330 II haemoperfusion, resulted in enhanced clearance of inflammatory markers along with uremic toxins, which ultimately led to the patient's clinical recovery from multiorgan dysfunction (**Figure 5-8**). In patients with severe sepsis, Zhao *et al*. demonstrated that HA 330 II haemoperfusion was associated with improved haemodynamics, due to a possible anti-inflammatory effect by the removal of inflammatory cytokines, and the improvement of organ dysfunction, including overall clinical outcome [14]. At our centre, we regularly use the HA 330 II haemoperfusion device for patients exhibiting signs of MODS, resulting in favourable clinical outcomes (unpublished data).

Wasp toxin is well documented to cause a spectrum of systemic effects, including myotoxicity, haemolysis, neurotoxicity, hepatic injury, nephrotoxicity, and multi-organ dysfunction syndrome [4]. Among these, myocarditis is a relatively uncommon but significant complication of wasp stings. Anandan *et al*. illustrated this with a case of Kounis syndrome, a type of allergic angina, in a patient who suffered a non-STEMI, elevated troponin, and echocardiographic findings of anterior wall hypokinesia (ejection fraction 40%) after a hymenopteran sting [15]. Despite these findings, angiography showed normal coronary arteries, leading to the hypothesis that the injury stemmed from coronary vasospasm—a process linked to vasoactive amine release triggered by arachidonic acid cascade activation following allergen exposure, often termed "cardiac anaphylaxis" [16] [17]. Mr R developed myocardial dysfunction, characterized by chest discomfort, raised troponin, regional hypokinesia, and haemodynamic instability, which progressed to circulatory shock, requiring inotropic support. This further highlights the risk of developing myocarditis due to a hornet sting.

Treatment for SIRS secondary to Hymenopteran stings primarily involves supportive care and anti-inflammatory agents. In cases of renal injury, extracorporeal support such as haemodialysis may be necessary. While haemodialysis effectively clears uremic substances and maintains fluid, electrolyte, and acid–base balance, the removal of venom proteins and circulating inflammatory mediators is crucial for restoring homeostasis and protecting vital organ function. CVVHDF is known to remove various plasma cytokines in septic patients [18]. This suggests its potential utility in managing critically ill patients within the SIRS-Multi-Organ Dysfunction Syndrome (MODS) spectrum. Wang Han-min *et al*. published a successful case of wasp sting-associated multiorgan failure managed with a combination of therapeutic plasma exchange and CVVHDF, resulting in complete recovery [19]. Our patient mirrored a similar clinical course with development of AKI within 24 h after being stung by hornets and progressed to develop multiorgan failure, which responded well to early commencement of CVVHDF augmented by HA 330 II haemoperfusion therapy.

## 5. Conclusion

This is a case report of a patient who developed multiorgan failure following multiple hornet stings. The patient received standard symptomatic treatment, corticosteroid therapy, haemodynamic support, blood product transfusion, SLED, TPE, and CVVHDF, which resulted in complete recovery. This case highlights the importance of early initiation of renal replacement therapy and plasmapheresis in the management of SIRS–MODS following hornet envenomation. Furthermore, the addition of HA 330 II therapy facilitated the removal of cytokines and endotoxins, contributing to improved clinical outcomes. Early initiation of plasmapheresis and haemoperfusion may reduce morbidity and mortality associated with severe systemic hornet envenomation. Future research should build upon existing knowledge in the role of early initiation of haemoperfusion in hornet en-

venomation and delve into the implications of these technological advancements.

## Acknowledgements

The authors would like to acknowledge and thank Mr Rohan Thiru [Medical Student, Year 3, University of Adelaide, South Australia] for his assistance with preparing and editing this manuscript.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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## Abbreviations and Acronyms

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|---|---|
| HA 330 II | Disposable Haemoperfusion Cartridge |
| SIRS | Systemic Inflammatory Response Syndrome |
| MODS | Multi-Organ Dysfunction Syndrome |
| SLED | Sustained Low-Efficiency Dialysis |
| CVVHDF | Continuous Veno-Venous Haemodiafiltration |
| TPE | Therapeutic Plasma Exchange |
| SCLS | Systemic Capillary Leak Syndrome |
| CRP | C-reactive protein |
| PCT | Procalcitonin |
| INR | International Normalised Ratio |