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A Case Report of Anaphylactic Shock Due to Hornet Sting with Multiple Organ Dysfunction Complications

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Abstract

BACKGROUND: Hornet venom has been recognized to induce lethal systemic effects such as anaphylaxis shock, bronchospasm, acute kidney or liver injury, disseminated intravascular coagulation (DIC), cardiovascular collapse, and even death. It consists of several enzymes and polypeptides with low molecular weights. It has been reported to contain the enzymes phospholipase A2, hyaluronidase, phosphomonoesterase acid ester, α-D-glucosidase, lysophospholipase, α-galactosidase, α-acetylamino-deoxsiglucosidase, and arylamidase. The venom will cause localized or systemic inflammation, an anticoagulant effect that may be fatal.

CASE REPORT: Herewith, a survival case report suffered from DIC with multi-organ failure following severe anaphylaxis shock post a hundred or massive Hornet sting.

CONCLUSION: We have reported a case of anaphylactic shock after a massive hornet sting with DIC that presented symptoms of shock, AKI, ALI, and multi-site bleeding. Although the mortality rate for similar cases is quite high, the outcome will be better with comprehensive and adequate treatment.

Introduction

The spectrum of allergic reactions due to wasp (Hornet) sting belonging to the ordo Hymenoptera can vary from mild to fatal symptoms. In the majority of the cases, the sting causes localized erythema, edema, and pain. In some cases, the sting can trigger a Type I hypersensitivity reaction mediated by immunoglobulin E (IgE), with manifestations of urticaria, angioedema, laryngeal edema/bronchospasm, cardiovascular symptoms of hypotension, myocardial depression, anaphylactic shock, and gastrointestinal symptoms [1].

Allergic reactions can be induced by only one single sting, and the more stings, the worse the prognosis because multiple stings can lead to inoculation of a higher amount of venom [2]. Insect toxins from the ordo Hymenoptera (bees and wasps) account for 14% of cases of anaphylactic reactions, which are the second most common cause after food allergies (33–34%) [3]. Bees have a thorny stinger that will remain on the victim's body, while wasps do not have a thorny stinger, so they can sting multiple times. Massive envenomation can be lethal in previously non-allergic individuals. The estimated lethal dose from a wasp sting is 20 stings/kg body weight in most mammals. However, the anaphylactic reaction to a Hymenoptera sting is not dose-dependent and does not depend on the number of stings [4]. Hymenoptera toxin generally consists of protein. An anaphylactic reaction can occur within 10 min after the sting. The diagnosis is made from the history of the sting and the symptoms that relevant to the anaphylactic reaction. The ability to recognize severe cases early with intensive management of complications will increase the patient's survival rate.

Case Illustration

A 43-year-old male was referred from a private hospital with the main problems shock after sting with a thousand wasps. He was cutting trees when the wasps sting his face, trunk, and extremities around 3 h before his admission. He immediately felt pain, reddish, and whole-body swollen. It was followed by choking and difficulty for breathing. At the first hospital, his blood pressure (BP) was 80/60 mmHg, pulse rate (PR) 112 beats per min, respiration rate (RR) 22 times/min,

and afebrile. He was treated with 2 liters of normal saline, epinephrine 0.3 ccs intramuscular for 3 times, diphenhydramine 10 mg intravenous every 8 h, and methylprednisolone 125 mg intravenous every 12 h. He has reported no pre-existing diseases. Since there was no improvement, and the swelling getting worse he was referred to Sanglah Hospital (Figure 1).

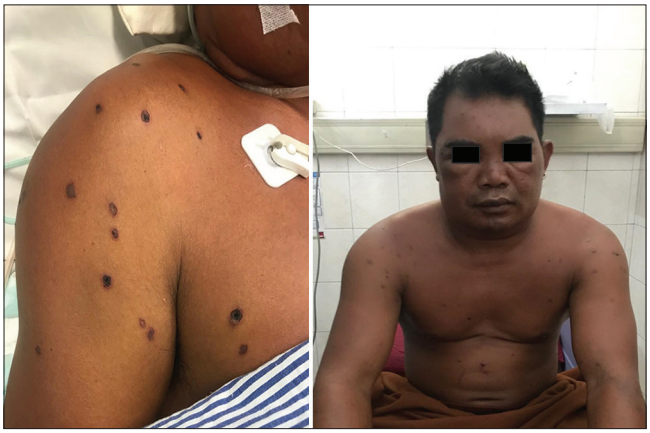

Figure 1: Hornet stings lesions and angioedema on the patient

At admission, the patient was fully alert, generalized edema, normal BP 120/80 mmHg, PR 84 beat/min, regular, normal RR 20 times/min, body temperature 36°C, and pain scale 4/10, body weight 78 kg, and height 171 cm, and urine production was 1500 ccs/24 h. On examination of the eyes, there was no anemic and icteric, but there was angioneurotic edema on both eyelid and mouth. No abnormality was detected in lungs and heart examination as well as abdominal examination. The extremities were also normal. There were hyperpigmented macula with discrete distribution on the chest, trunk, and extremities with various size.

Laboratory investigation showed a white blood cell level of 13.4 × 10^9/L, a hemoglobin level of 17.6 g/L, and a platelet count of 42.6 × 10^9/L. The LFT result of aspartate aminotransferase (AST) was 6045.1 IU/L, of alanine aminotransferase (ALT) was 909.3 IU/L, albumin level was 3.7 g/dL, and globulin level was 2.3 g/dL. The results of IgM-anti HAV, HBsAg, and anti-HCV were non-reactive. Hemostatic function in this patient showed elevated D-dimer levels, and high fibrinogen, accompanied by prolonged PPT, INR, and activated partial thromboplastin time (APTT). Urea and creatinine level was 119.6 mg/dL and 10.96 mg/dL, respectively. The total IgE level was 1366 IU/mL. Blood gas analysis of admission revealed compensated metabolic acidosis. Anterior-posterior chest X-ray examination on October 2, 2017, revealed a cardiothoracic ratio: 49%, no infiltrates or nodules were seen, bronchovascular patterns were normal, with the impression: that heart and lung showed no abnormalities. Ultrasound examination of the upper and lower abdomen on October 12, 2017, showed gastritis and there were no signs of cirrhosis, portal hypertension, or splenomegaly. The liver, gall bladder, pancreas, spleen, right and left kidney, bladder, and prostate did not show any abnormalities.

Based on the anamnesis, physical examination, and further investigation, the patient was diagnosed with anaphylactic shock et causa massive hornet sting with laryngeal edema, acute liver injury, acute kidney injury Stage III et causa suspicion prerenal, and suspected disseminated intravascular coagulation (DIC). The patient was given therapy in the form of an infusion of 0.9% NaCl 30 drops per minute, dietary 2500 kcal/day, injection of diphenhydramine 10 mg intravenously, hydrocortisone 100 mg every 8 h intravenously, omeprazole 40 mg every 12 h intravenously, metoclopramide 10 mg every 8 h, and sucralfate 15 cc every 8 h intraorally, hepatoprotection 1 tablet every 12 h, and paracetamol 750 mg every 8 h orally. The patient also receives platelet transfusion (TC) until the platelet level is more than 20.000.

During treatment, the patient experienced bloody vomiting accompanied by blackish stools and red urine. It was followed by a decrease in consciousness, and a worsening of kidney function. The patient was treated in the ICU for 6 days. He underwent hemodialysis every day. His condition was getting better and the treatments were continued in the normal ward. He was discharged after 5 days of treatment in the ward. because his clinical and laboratory conditions had improved.

Discussion

Anaphylaxis is a severe and fatal systemic allergic reaction that may be occurring suddenly after contact with an allergenic substance. It is a serious allergic reaction that occurs quickly and can cause death. The criteria for the diagnosis of anaphylaxis are stated in the Sampson criteria, as shown in Table 1. In this case, the patient complained of swelling of

Table 1: Criteria for the diagnosis of anaphylaxis [5]

Anaphylaxis is highly likely when any one of the following three criteria is fulfilled:
1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g., generalized hives, pruritus or flushing, and swollen lips-tongue-uvula) and at least one of the following: a. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced PEF, and hypoxemia) b. Reduced BP or associated symptoms of end-organ dysfunction (e.g., hypotonia (collapse], syncope, and incontinence)
2. Two or more of the following that occur rapidly after exposure; to a likely allergen for that patient (minutes to several hours): a. Involvement of the skin-mucosal tissue (e.g., generalized hives, itch-flush, and swollen lips-tongue-uvula) b. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced PEF, and hypoxemia) c. Reduced BP or associated symptoms (e.g., hypotonia [collapse], syncope, and incontinence) d. Persistent gastrointestinal symptoms (e.g., crampy abdominal pain and vomiting)
3. Reduced BP after exposure to known allergen for that patient (minutes to several hours): a. Infants and children: low systolic BP (age specific) or greater than 30% decrease in systolic BP b. Adults: systolic BP of less than 90 mm Hg or greater than 30% decrease from that person's baseline

BP: Blood pressure

the whole body and lips, accompanied by respiratory problems such as shortness of breath. On examination, it was found that the patient had low BP (80/60 mmHg). This was in accordance with the anaphylaxis diagnostic criteria.

Fatal anaphylaxis cases due to hymenoptera sting were reported at 33.9% following drug/contrast allergy with the most postmortem findings being signs of asthma/bronchospasm such as mucous plugging or pulmonary hyperinflation [6]. Meanwhile, in Sweden, mortality from severe anaphylactic reactions because of wasp stings was 19 cases, compared to only 1 case due to bee stings and no mortality because of snake bites [7]. Hymenoptera toxin consists of a mixture of biologically active ingredients such as enzymes (phospholipase and hyaluronidase), peptides (melittin, apamin, mastoparans, and bombolitins), and low molecular weight components (biogenic amines, acetylcholine, carbohydrates, lipids, and free amino acids) [3]. Melittin, a pathogenic substance, is a neurotoxin that can cause hemolysis and mast cell degranulation. Other researchers reported specifically the content of wasp toxins such as histamine, dopamine, serotonin, mellitin, hyaluronidase, apamin, and phospholipase A [8]. Characteristics of wasp toxin are a toxin that is myotoxic, hemolytic, neurotoxic, hepatotoxic, nephrotoxic, and vasodilator [9]. Fatal stings can occur because of a sting on the head-and-neck area due to laryngeal edema, hypotension, and bronchoconstriction within 1 h after the sting. Wasp toxin is reported to cause multi-organ failure [7], acute liver injury [10], acute kidney injury and rhabdomyolysis [11], pulmonary edema [12], [13], and several other fatal conditions. In patients with near-fatal cases of anaphylaxis, the administration of epinephrine is one of the main therapies in the management of anaphylaxis. Epinephrine, 0.01 mg/kg (maximum dose 0.5 mg) administered intramuscularly every 5–15 min is the recommended dose for symptom control and maintenance of BP. Intramuscular administration of epinephrine reaches peak plasma concentrations higher than subcutaneous administration [5]. However, in some cases, intravenous administration of epinephrine may also be considered in patients during surgery. In this case, the initial therapy of 0.03 ml of epinephrine intramuscularly was also given to this patient. Vasopressors such as noradrenaline, vasopressin, or metaraminol may be given to prevent vasodilation if epinephrine and fluid resuscitation fail to maintain a systolic BP > 90 mmHg. Antihistamines (H1 and H2 agonists) have a slower effect than epinephrine, have little effect on BP, and are second-line therapy for anaphylaxis. Antihistamines are used for symptomatic treatment of urticaria-angioedema and pruritus. Administration of diphenhydramine intravenously or intramuscularly (or orally for mild symptoms) can be given 25–50 mg for adults and 1 mg/kg body weight for children. The administration of steroids is ineffective in managing the acute phase because of the slow onset of action. When given intravenously, corticosteroids should be equivalent to 1–2 mg/kg of methylprednisolone [5]. In this case, the patient was given hydrocortisone 100 mg every 12 h intravenously.

In general, the mechanism of anaphylaxis in one or some wasp stings is a Type I hypersensitivity reaction in which IgE plays a key role. IgE-specific antibodies directly counteract the components of the toxin by activating mast cells and basophilic granulocytes, leading to the release of mediators that can lead to acute manifestations of the disease. This reaction usually appears after 10 to 30 min after the sting, but it can also occur shorter or later. This is also in accordance with the case, where the total IgE result was very high that is 1366. In multiple or massive hornet stings, it can cause a direct effect of the toxin on cell tissue damage, causing severe complications such as intravascular hemolysis, rhabdomyolysis, DIC, cardiovascular disorders, liver damage, renal failure, and hemorrhagic pancreatitis. In addition, the mechanism of anaphylaxis in hornet stings is complement activation where the production of anaphylatoxin C3a was found to increase after the hornet-sting challenge test in eight individuals with anaphylactic reactions due to the previous hornet stings [13]. In individuals who did not show an anaphylactic reaction, C3a levels did not increase, C3a levels increased slightly in three individuals who showed mild anaphylactic symptoms, and C3a levels greatly increased in four individuals who showed severe anaphylactic symptoms.

AKI is defined as a sudden decline in kidney function. This condition can occur due to various conditions, including specific kidney disease (acute nephritis, glomerulonephritis, and renal vasculitis), non-specific conditions (ischemia, toxins), and extrarenal pathology (prerenal azotemia and postrenal obstructive nephropathy). Two criteria that define AKI depending on serum creatinine and urine production (RIFLE and AKIN) have been validated. A comparison of RIFLE and AKIN criteria is shown in Table 2. In this case report, a patient with the increased renal function was found where the serum creatinine level had reached 10.96 mg/dl so the patient was said to have Stage 3 AKI with prerenal suspicion caused by the toxic effects of hornet stung. The mechanism of AKI in hornet stings is a direct mechanism of toxin nephrotoxicity, the shock that occurs triggers acute tubular necrosis (ATN), and nephropathy due to hemoglobinuria and myoglobinuria. The pathological features of AKI due to hornet stings can include degeneration and necrosis of renal tubular epithelial cells. The prevalence of AKI after hornet stings was 84.5%. AKI in hornet stings is often associated with shock, ATN, acute interstitial necrosis (IAIN), thrombotic microangiopathy, or acute cortical necrosis as demonstrated by postmortem examination. Treatment of AKI can be hemodialysis, hemofiltration, or peritoneal dialysis. Plasmapheresis or exchange TC may be an option in life-threatening multi-organ failure. There was no difference in mortality between continuous venovenous

Table 2: Diagnostic Criteria of AKI based on AKIN and RIFLE [14]

AKI stage	Urine production	RIFLE	
Creatinine serum		Category	Creatinine serum or GFR
Stadium 1: Increase in serum creatinine of≥0.3 mg/dl or≥1.5 or 2 times baseline	≤0.5 ml/kg/h within 6 h	Risk	Increase in serum creatinine 1.5 times baseline or decrease GFR > 25%
Stadium 2: Increase in serum creatinine of≥2 mg/dl to 3 times baseline	≤0.5 ml/kg/h within 12 h	Injury	Increase in serum creatinine 2 times baseline or decrease GFR > 50%
Stadium 3: Increase in serum creatinine of≥4,0 mg/dl or≥3 times baseline	≤0.3 ml/kg/h for 24 h or anuria for 12 h	Failure	Increase in serum creatinine 3 times baseline, or serum creatinine > 4 mg/dl or decrease GFR > 75%
		Loss	Persistent acute renal failure (loss of kidney function > 4 weeks)
		End stage kidney disease	ESRD > 3 months

AKI: Acute kidney injury.

hemofiltration (CVVH), intermittent hemodialysis, and CVVH with plasma exchange. Recovery from renal function generally takes 1–3 months. Several cases were reported become chronic kidney disease [19].

The mechanism of acute liver injury in hornet stings is unclear. On anatomical pathology examination can be found diffuse fatty degeneration, as well as hepatocyte necrosis which may be caused by direct toxic effects on hepatocytes. In cerebral edema caused by hornet stings, focal necrosis of the cerebral cortex, degeneration, necrosis of nerve cells, and proliferation of glial cells were found. In the study by Xie *et al*., liver injury from bee stings was defined as elevated ALT or serum glutamic-pyruvic transaminase (SGPT). A total of 30.1% of patients with hornet stings had ALT levels >40 IU/L and 44.8% of patients with AST levels >40 IU/L which indicated liver injury or no other organ dysfunction. Transaminase levels, especially AST, are not an accurate marker of rhabdomyolysis. More stings are said to play a role in increasing both serum glutamic-oxaloacetic transaminase and SGPT. Of the total 25 hornet sting patients who had serious injuries, 16 were toxin-specific IgE positive for at least one of the seven common hornet toxins (class 2) caused by allergic reactions, of which 14 had hepatic dysfunction. This study also stated that every hornet sting patient was treated with glucocorticoids and antihistamines. Tests performed for patients who experience multiple stings within 14 days of a sting include toxin-specific IgE, lymphocyte subsets, and various inflammatory mediators. The patient in this case reported with more than 100 stings had very high AST and ALT values on September 13, 2018, with an AST level of 6045.1 U/L and ALT of 909.3 U/L. The patient was treated with a hepatoprotector as supportive therapy for a hepatocellular injury. The patient improved within 1 week after being given anaphylactic therapy and supportive therapy.

DIC is a pathological syndrome as a result of various severe diseases. This condition is characterized by systemic activation of the coagulation pathway, which can lead to fibrin clot formation leading to organ failure with consumption of platelets and coagulation factors leading to hemorrhagic manifestations. This serious condition can damage the microvasculature and can cause organ dysfunction. Laboratory tests used to diagnose and evaluate patients with DIC are needed to know the changes in hemostatic function. Hemostatic function tests, such as prothrombin time (PT), APTT, or platelet count, can provide important evidence of consumption and activation of coagulation factors. Another thing to note is the calculation of fibrin degradation products and D-dimers. The diagnostic criteria for DIC, such as ISTH scoring, are shown in Table 3. These cases meet the diagnostic criteria for DIC; based on an examination, the patient has gastrointestinal bleeding accompanied by low platelet levels that have reached 8.3 g/dL. The examination of the coagulation parameter showed an increase in D-dimer and fibrinogen accompanied by prolonged PT. DIC can occur because all components of the toxin, especially phospholipase A2 (PLA2) in hornets are similar to other hymenoptera toxins such as bees. Based on the guidelines of the British Journal of Haematology [16], the management of DIC, in general, is therapy for the underlying disease condition. Low levels of platelets and coagulation factors increase the risk of bleeding. Blood TC is not the main therapy, it is only indicated patients with active bleeding, patients undergoing invasive procedures, and in patients at risk of bleeding complications. In general, platelet TCs are given to patients who are bleeding and whose platelet levels are <50 × 109/L. In patients without bleeding, a lower platelet level of 10–20 × 109/L is an indication of a platelet TC [16]. Similar to the case, where there was gastrointestinal bleeding in this patient accompanied by a low platelet level of 42,000, platelet TCs were also given. At autopsy, acute pulmonary edema, AKI, acute liver failure, cerebral edema, and cardiac dysfunction were found in multiple hornet stings [8]. The most of the massive hornet sting cases end in death, although there are case reports reporting success in treating massive hornet stings with multiple organ dysfunctions and AKI [15].

Table 3: ISTH scoring diagnostic system for DIC [16]

Platelet count (> 100×10^9/L = 0, < 100×10^9/L = 1, < 50×10^9/L = 2)
Elevated levels of a fibrin-related marker (e.g., D-dimer, fibrin degradation products) (no increase = 0, moderate increase = 2, and severe increase = 3)
Prolonged PT (< 3 s = 0, > 3 but < 6 s = 1, > 6 s = 2)
Fibrinogen level (> 1 g/L = 0, < 1 g/L+1)

Score:
≥5: Compatible with overt DIC: repeat score daily.
< 5: Suggestive non-overt DIC: repeat score day 1–2.

The same manifestation was reported in fatal cases due to massive hornet stings resulting in death [17], [18]. The mechanism of DIC is thought to be affected by high hornet toxin because levels of Mellitin

and PLA2 act as anticoagulants and interfere with coagulation function. Comprehensive and adequate treatment supports the successful management of fatal cases that were reported in other case reports [15]

Summary

We have reported a case of anaphylactic shock after a massive hornet sting with DIC that presented symptoms of shock, AKI, ALI, and multi-site bleeding. Although the mortality rate for similar cases is quite high, the outcome will be better with comprehensive and adequate treatment.

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