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CASE REPORT – OPEN ACCESS

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Toxic Reaction due to Multiple Wasp Stings in a Child: A Case Report

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ABSTRACT

Background: Poisoning caused by wasp and bee stings is the most common type of poisoning caused by insects in Türkiye. The wasp belongs to the family Vespidae, a subgroup of the class Hymenoptera (membrane-winged insects). While mild symptoms are frequently observed in response to a single wasp sting, multiple wasp stings can cause severe poisoning and toxic reactions.

Case Report: We present a case of a 7-year-old male patient who died after developing rhabdomyolysis, acute kidney injury (AKI), and multiple organ failure as a result of multiple wasp stings.

Conclusion: The case report of our patient revealed that poisoning from multiple wasp stings can show a fatal course in children. When the number of wasp stings is 20–200 and when stings occur in the head-and-neck regions, AKI and mortality are high. Therefore, these patients should be hospitalized in the pediatric intensive care unit and monitored closely for toxic reactions.

Keywords: Bee poising, toxic reaction

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INTRODUCTION

Wasps can attack in swarms for protection when individuals or their nests are disturbed, causing severe poisoning symptoms, such as anaphylaxis, acute kidney injury (AKI), rhabdomyolysis, cardiac dysfunction, coagulation disorder, multiple organ failure (MOF), and shock. This poisoning is caused by the presence of acid phosphatase, lysophospholipase, apamin, mastoparan, hyaluronidase, histamine, antigen 5, and vasoactive peptides in the bee venom (1). Although clinical findings are associated with the number of wasp stings, the strength of the venom, and individual immunity, cases of AKI or MOF in response to a single wasp sting have been reported in the literature (2, 3). The mortality rate of such patients with AKI is 25–50% (3). A series of cases with AKI and MOF as a result of multiple wasp stings have been reported in the literature; however, the number of cases reported in Türkiye is very low (2, 3). Here, we present a case of a 7-year-old male patient who died within 2 days of developing MOF and shock resulting from multiple wasp stings. Our aim is to draw the attention of physicians toward the potential for rapid progression in MOF and mortality caused by wasp stings despite treatment.

CASE REPORT

A 7-year-old male patient was admitted with lethargy; he had fallen into a pit about 3–4 h before admission while playing in the garden of a house the family visited, and his whole body had been covered with wasps (approximately 500–1000 bees according to the family's statement). The patient's medical history indicated that he was previously healthy with no history of recent exposure to other medications or toxic substances. At an external institution, he was treated with an antihistamine and steroids. However, due to an increased tendency to sleep noted during the follow-up, he was admitted to our hospital. His blood pressure was 145/95 mmHg (>99p), heart rate was 132 beats/min, body temperature was 36.5°C, and SpO_2 was 92%. On physical examination, he was found to be conscious, but lethargic. He responded to verbal stimuli by opening his eyes. Lesions were observed across the whole body due to wasp stings, particularly in the head-and-neck regions. He exhibited edema on the lip, preorbital, and neck regions. His hands and feet were cold and the capillary refill time was >2 s. The patient's laboratory findings are summarized in Table 1. The patient developed respiratory failure and hypotension after 4 h of hospitalization and was intubated. Fluid therapy, inotropic support (adrenaline and dopamine), alkalization ($NaHCO_3$ infusion), antihistamine, steroid, and H2 blocker treatment were started by inserting a central venous catheter into the right internal jugular vein. Vitamin K and fresh frozen plasma were administered as coagulation test results were abnormal. Antibiotic therapy was initiated empirically. Liver function was impaired, and ursodeoxycholic acid and N-acetylcysteine infusion was initiated. Insulin infusion and allopurinol were initiated for hyperglycemia and uric acid elevation, respectively. No pathology was observed in the cranial computed tomography of the patient whose abdominal USG was normal. The ejection fraction was 42% on echocardiography and the patient had left ventricular hypertrophy.

Table 1. Laboratory findings

Laboratory	Normal range	Hospitalization	1st day	2nd day
Complete blood count				
Hb g/dL	(11–14)	13.5	10.4	12
Hct%	(32–42)	39	31	34
MCVfL	(75–95)	75	77.6	79
RDW	(8–18)	17.2	18.1	19.6
WBC/mm^3	(8–14)	12.340	11.800	20.800
PLT/mm^3	(150.000–400.000)	471.000	397.000	196.000
Acute phase reactants				
CRP mg/L	(<5)	2	2	4.6
Procalcitonin ng/mL	(<0.07)	16	–	
Coagulation parameters				
PT sec	(11–15)	15.3	19.7	30.5
aPTT sec	(22–36)	Prolonged	65.7	33.8
INR	(0.85–1.2)	1.2	1.6	2.5
D-Dimer µg/L	(0–500)	–	–	1110
Biochemical parameters				
Glucose mg/dL	(60–99)	301	486	130
Na mmol/L	(130–150)	140	145	149
K mmol/L	(3.5–5.5)	3.5	6.2	4.4
Cl mmol/L	(98–107)	109	113	108
BUN mg/dL	(9–22)	10	16	15
Creat mg/dL	(0.46–0.76)	0.7	1.8	1.5
Ca mg/dL	(8.8–10.8)	8.8	8.1	8.1
P mg/dL	(4–6.1)	7.6	9.4	4.4
ALP U/L	(<500)		561	339
AST U/L	(9–80)	37.628	33.876	6766
ALT U/L	(6–46)	13.927	13.456	4384
LDH U/L	(145–300)	34.579	26.493	8611
GGT U/L	(7–50)		278	113
CK U/L	(30–200)	219	2569	10628
T. Bil mg/dL	(0.2–1.2)		3.5	5.4
D. Bil mg/dL	(0.11–0.43)		1.4	3.4
Albumin mg/dL	(3.8–5.4)	4.3	3.6	3.3
Mg mg/dL	(1.7–2.1)	2.5	2.8	2.0
Uric Acid mg/dL	(2–5.8)	11.6	13.4	4.8
Ammonia nmol/L	(40–80)			272
Ferritin ng/mL	(15–120)			>2000
Cardiac enzymes				
CK-MB U/L	(<24)	189	430	429
Troponin I ng/L	(0–034.2)	49.7	215	3771
Complete urinalysis				
PH		5.5	6	
Density		1030	1015	
Glucose		–	+	
Protein			+	
Urobilinogen		–	–	
Blood		+++	+++	
Microscopy		1–3 leukocytes, epithelia	2–4 erythrocytes	
Blood gas				
PH		7.29	7.06	7.21
PCO_2 mmHg		37	54	52
PO_2 mmHg		128	52	44
HCO_3 mmol/L		15.8	12.8	18.6
Base deficit mmol/L		-11.5	-13.4	-6.2
Lactate mmol/L		5.1	6.9	12.1
Cranial tomography		Normal		
Abdominal ultrasonography			Normal	
Echocardiography			Left ventricular hypertrophy	Left ventricular systolic dysfunction

Hb: Hemoglobin; Htc: Hematocrit; MCV: Mean corpuscular volume; RDW: Red cell distribution width; WBC: White blood cell; PLT: Platelet; CRP: C reactive protein; PT: Prothrombin time; aPTT: Activated partial thromboplastin time; INR: International normalized ratio; Na: Sodium; K: Potassium; Cl: Chlorine; BUN: Blood urea nitrogen; Ca: Calcium; P: Phosphorus; ALP: Alkaline phosphatase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; LDH: Lactate dehydrogenase; CK: Creatine kinase; GGT: Gamma glutamyl transferase; Mg: Magnesium

Therapeutic plasma exchange (TPE) was performed after 8 h of hospitalization when the patient was first thought to be exhibiting a toxic reaction. Milrinone, noradrenaline, and hydrocortisone were added, but hypotension, oliguria, metabolic acidosis, and electrolyte disturbances continued, and continuous venovenous hemodialysis (CVVHD) was initiated after TPE. However, hypotension continued. Terlipressin was added to the treatment. The antibiotics were changed to vancomycin and meropenem (at the renal failure dose). A second TPE was performed after CVVHD; however, laboratory investigations continued to show MOF and rhabdomyolysis at 36 h of hospitalization. The ejection fraction was 30% as observed by control echocardiography. ECMO was scheduled based on persistent hypotension, circulatory disorder, and shock findings. However, the patient experienced cardiac arrest at 42 h of hospitalization, was unresponsive to cardiopulmonary resuscitation, and died.

DISCUSSION

Multiple wasp stings cause serious poisoning and toxic reactions. Wasp venom exerts a toxic effect by various mechanisms. Components of the venom, such as acid phosphatase, lysophospholipase, apamin, mastoparan, hyaluronidase, antigen 5, and vasoactive peptides (thromboxane, leukotriene, serotonin, and histamine), can have hepatotoxic, cardiotoxic, neurotoxic, nephrotoxic, hemolytic, myotoxic, and vasodilator effects. Moreover, the venom initiates the inflammatory process by increasing the release of inflammatory cytokines, such as IL6, TNF, IL-1, and IL-8 (4, 5). Among the possible toxic reactions, AKI and rhabdomyolysis are the most frequently reported, and the mortality rate in cases with AKI is 25–50% (5).

The number of wasp stings, the site of the sting, the strength of the venom, age, gender, accompanying skin findings, the time between the sting and the onset of symptoms, and individual immunity are all related to the severity of clinical findings. When the number of wasp stings is 20–200 and stings occur in the head-and-neck regions, AKI and mortality are high (4). Ambarsari et al. (1) have presented two cases in which AKI developed after 30 wasp stings and MOF developed after 80 bee stings. The family of our patient reported that he was attacked by approximately 500–1000 wasps, and physical examination indicated that the stings mainly occurred in the upper body.

There is no specific treatment for toxic reactions that develop after wasp stings, as there is no antidote. In addition to steroid and antihistamine treatment, the standard treatment includes hydration and correction of hypovolemia to prevent renal ischemia, alkalization with $NaHCO_3$ (especially in patients with rhabdomyolysis), and renal replacement therapy to remove the wasp venom from the circulation. In addition to supportive treatments, peritoneal dialysis (PD), CVVHD, continuous venovenous hemodiafiltration (CVVHDF), or intermittent HD can be initiated in patients with oliguria, metabolic acidosis, and severe electrolyte disorders (6). Ambarsari et al. (1) employed intermittent HD in one patient showing only AKI and employed CVVHD in another patient with MOF, obtaining positive results in both cases. Since wasp venom consists of medium- and high-molecular-weight proteins, toxins, myoglobin, and various inflammatory cytokines are expected to be removed more effectively by CVVHD/CVVHDF than by intermittent HD or PD (5–7). Another preferred treatment method, especially in patients with hemolysis and rhabdomyolysis, is TPE. This method is very effective in removing the toxin and toxin-induced cytokines (5–7). Various studies have suggested that the combination of TPE and CVVHD/CVVHDF is more effective than TPE treatment alone (5, 6, 8). Wang et al. (5) treated a patient who developed MOF after 80–90 wasp stings with TPE and CVVHD. The previous studies have emphasized that patients with more than 20 wasp stings must be monitored for AKI and that TPE and renal replacement therapies initiated in the early period are life-saving (7–10). Our patient was treated with TPD and CVVHDF in the early period, along with supportive treatments, but was unresponsive. This was attributed to the high venom load in the patient due to the high number of wasp stings.

CONCLUSION

Our findings demonstrate that poisoning from multiple wasp stings can result in fatalities in children. Patients should be hospitalized in the pediatric intensive care unit and monitored closely for rhabdomyolysis, AKI, and MOF. Renal replacement therapy and TPD should be considered in the early period. We also want to increase the awareness of the issue in the general population and emphasize the implementation of proper precautions.

Informed Consent: Written informed consent was obtained from the patient's family for the publication of the case report.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – YÖ; Design – MÖ, YÖ; Supervision – MÖ; Literature Search – YÖ; Writing – MÖ, YÖ; Critical Reviews – MÖ.

Conflict of Interest: The authors have no conflict of interest to declare.

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