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TRANSIENT COAGULOPATHY DUE TO WASP STING: A CASE REPORT

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Abstract—Background: Insect venom anaphylaxis is a potentially life-threatening disorder. Transient coagulopathy in insect venom anaphylaxis is a rare phenomenon. **Case Report:** A 41-year-old man presented to the Emergency Department (ED) with hypotension after a run in a park. History and examination revealed signs of anaphylactic shock. A deranged coagulation profile with a normal platelet count led to the diagnosis of wasp sting anaphylaxis. **Why Should an Emergency Physician Be Aware of This?:** Transient deranged coagulation profile with a normal platelet count may arise from insect venom anaphylaxis. This specific finding may aid the emergency physician in making a diagnosis of anaphylactic shock in an otherwise healthy patient presenting with shock with no apparent cause. © 2016 Published by Elsevier Inc.

Keywords—anaphylaxis; coagulopathy; insect venom; shock

INTRODUCTION

Insect venom-induced allergic reaction is potentially life threatening. Insect stings can cause severe anaphylaxis, leading to cardiovascular collapse and death. The mortality rate is estimated at 1–5% in different countries (1). Severe cases of allergic reaction are known to be associated with disseminated intravascular coagulation (2). Transient coagulopathy, however, is a rare phenomenon. We report a case of transient deranged coagulation profile with a normal platelet count in a patient who had a wasp sting. To our knowledge, only four other cases have been reported before (3,4).

CASE REPORT

A 41-year-old Chinese man presented with acute-onset giddiness and transient loss of consciousness after jogging for about 20 min in a park. Clinically, he was afebrile, diaphoretic, and flushed. He was hypotensive (with a blood pressure of 72/30 mm Hg) and tachycardic (heart rate of 135 beats/min). There was no obvious urticaria or angioedema. Cardiorespiratory examination was unremarkable. Electrocardiogram showed sinus tachycardia. A presumptive diagnosis of anaphylactic shock was made based on the rapid onset of symptoms and absence of fever to suggest sepsis or heat stroke. He was treated with intramuscular adrenaline 0.3 mg, intravenous diphenhydramine 25 mg, and intravenous hydrocortisone 200 mg. He was resuscitated with 500 mL 0.9% normal saline bolus. His state of consciousness and blood pressure improved 15 min after the administration of adrenaline. Further detailed questioning revealed that he was possibly stung by an insect on his neck 5 min prior to the syncopal event. He reported a prior history of urticaria and angioedema after a bee sting. At that time, he was only treated symptomatically, with no further follow-up. Initial laboratory investigations were essentially normal except for grossly deranged coagulation profile (Table 1). In view of the cardiovascular instability at first presentation, he was admitted for further monitoring. His blood pressure remained stable without inotropic support. The next day, his coagulation profile normalized without intervention. Subsequent allergy testing was positive for white-faced hornet, yellow jacket

Table 1. Coagulation Profile of Patient on Day of Admission and Day After Admission

Laboratory Test	Unit	Normal Range	D0	D1
PT	Seconds	11.7–14.0	16.5	16.3
aPTT	Seconds	25.0–36.0	>120.0	30.1
TCT	Seconds	15.0–18.0	>100.0	38.1
Fibrinogen	g/L	1.8–4.5	1.2	–
D-dimers	Ug/mL	<0.40	1.15	–
Platelet count	$\times 10^9/L$	170–420	219	167

D0 = day of admission; D1 = day after admission; PT = prothrombin time; aPTT = activated partial thromboplastin time; TCT = thrombin clotting time.

venom, paper wasp, and yellow hornet. He was discharged well, with counseling for anaphylaxis and adrenaline autoinjector training.

DISCUSSION

This case was a diagnostic challenge to the emergency physician. The patient presented with hypotension with no apparent cause. Differential diagnoses included anaphylactic shock, sepsis, heat stroke, and dehydration. In the absence of fever, sepsis and heat stroke were excluded. Dehydration was a possibility but it was unlikely to cause such a low blood pressure. Hence, a presumptive diagnosis of anaphylactic shock was made. The initial investigations revealed deranged coagulation profile with no other abnormalities. It was only after the history of possible insect sting provided later that led to the definitive diagnosis of anaphylactic shock.

The patient had two unusual laboratory results (Table 1). The first was an unrecordable activated partial thromboplastin time (aPTT). The second was a low fibrinogen level in the presence of a normal platelet count and only slight increase in D-dimer. These findings were similar in previously reported cases of insect venom anaphylaxis (4).

According to published diagnostic criteria, disseminated intravascular coagulation (DIC) cannot be totally

ruled out (5). However, even if DIC was present, it was only mild and cannot explain the above laboratory results. Dilution is also not the causal mechanism (Table 2).

Unrecordable aPTT can be explained by the pathophysiology of anaphylaxis. In anaphylaxis, immunoglobulin E-mediated reaction activates mast cells and basophils. These cells release multiple mediators, some of which have direct effect on the coagulation and fibrinolysis pathways. Heparin, which is one of the mediators released by mast cells, functions as an anticoagulant by binding to antithrombin, inhibiting its activity and thus explaining the abnormal aPTT (4).

Tryptase, which is also released by mast cells when activated, would explain the prolonged thrombin clotting time and low fibrinogen level (6,7). Tryptase is a protease with many substrates. In particular, tryptase works on fibrinogen by cleaving its alpha and beta chains. This results in inhibition of thrombin-initiated clot formation as cleaving of the alpha and beta chains of fibrinogen removes its thrombin cleavage site and a critical polymerization site from the beta chain. Tryptase also has an effect on the fibrinolytic pathway as it activates single-chain urinary-type plasminogen activator (scu-PA or urokinase) (7). This results in plasminogen being converted to plasmin, which breaks down fibrinogen and other coagulation factors.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

An emergency physician should be aware that anaphylactic shock may give rise to transient deranged coagulation profile with a normal platelet count. This laboratory finding is useful when there is diagnostic uncertainty in an otherwise healthy patient who presents with shock without an apparent cause.

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Table 2. Comparison of Coagulation Abnormalities Due to Dilution, Disseminated Intravascular Coagulation, and Patient's Results

Laboratory Test	Unit	Patient Results	Dilution	Disseminated Intravascular Coagulation
PT	Seconds	Raised	Raised	Raised
aPTT	Seconds	Raised	Raised	Raised
TCT	Seconds	Raised	Raised	Raised
Fibrinogen	g/L	Reduced	Reduced	Reduced
D-dimers	Ug/mL	Raised	Reduced	Raised
Platelet count	$\times 10^9/L$	Normal	Reduced	Reduced

PT = prothrombin time; aPTT = activated partial thromboplastin time; TCT = thrombin clotting time.