DISSEMINATED INTRAVASCULAR COAGULATION FOLLOWING MULTIPLE BEE STINGS

(A Case Report)

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ABSTRACT

Disseminated intravascular coagulation (DIC) as a toxic effect of multiple bee stings has not been described. A 28 year old male was bitten by a swarm of bees and was hospitalised twelve hours later with generalized bleeding manifestations. He died within three hours of hospitalisation due to uncontrolled bleeding, in spite of blood transfusions and supportive therapy. Detailed laboratory investigations and postmortem histopathological examination showed diagnostic evidence of DIC.

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KEY WORDS : Multiple bee stings; Disseminated intravascular coagulation.

Introduction

Toxic effects due to *Hymenoptera* venoms are not usually seen in man unless there have been many, usually hundreds of stings. Anaphylactic reactions are a commoner cause of death [1,2]. Intravascular haemolysis and rhabdomyolysis as toxic effects of hymenoptera bites are known [1,3]. A case of DIC following multiple bee stings, is reported below.

CASE REPORT

A 28 year old male, was admitted to a service hospital on 13.10.89 after being bitten by a swarm of bees twelve hours earlier. He complained of progressive breathlessness, weakness, pain over both loins, three episodes of haematemesis, each approximately 150-200 ml, and passage of 50 ml of reddish brown urine. After the bites, he had intense pain and swelling all over the body, for which he had received first aid with 1.5 litres IV fluids, injections pheniramine maleate 50 mg IV, adrenaline 0.5 ml (1:1000) diluted in 50 ml distilled water IV, dexamethasone 4 mg IV and morphine 15 mg IV.

On examination, he had air hunger and was pale. BP was 70 mm Hg systolic. Diastolic BP was not recordable. Respiratory rate was 46 per minute and peripheries were cold. There was no cyanosis. Bleeding was seen from multiple venepuncture sites, nose and mouth. Heart sounds were normal. Few crepitations were heard over lung fields, but no rhonchi were heard. Bilateral renal angle tenderness was elicited. Examination of other systems was normal. Nasogastric aspiration produced a

continuous flow of dark red blood, and bladder catheterisation produced 50 ml of bloody urine. He was started on rapid blood and IV fluid transfusions. Supportive therapy was given with intravenous hydrocortisone 200 mg, ranitidine 50 mg, vitamins C and B complex, calcium gluconate and oxygen inhalation. Vitamin K 10 mg IM was also given. Attempts were made to remove stingers yet implanted in the skin. He died within 3 hours of admission in spite of all attempts to resuscitate him.

Investigations showed :- Hb-10 gm%, TLC-18,800/cmm, polymorphs-76%, lymphocytes-21%, monocytes-1% and eosinophils-2%, ESR-9 mm fall 1 hr. Peripheral blood smear showed mild thrombocytopenia with no evidence of haemolysis. Bleeding time - 12 minutes 15 seconds, clotting time-blood did not clot at 45 minutes, prothrombin time-control 13 seconds, test more than 5 minutes, platelet count 80,000/cmm, reticulocyte count 0.8%. Urine- dark brown in colour, acidic, albumin ++, bile salts and bile pigments not detected, Hb-3.5 gm%, numerous RBCs seen. Blood urea 25 mg%, serum creatinine 0.8 mg%, Liver function tests-serum bilirubin : van den Bergh reaction negative, 0.6 mg%, total proteins : 6.9 gm/dl, albumin 4.0 gm/dl, globulin 2.9 gm/dl, A : G = 1.4:1, SGOT 17 and SGPT 15 IU per litre. Blood sugar 76 mg%, X-ray chest suggested bilateral intra-alveolar haemorrhages. ECG was normal.

Post mortem examination showed multiple haemorrhages all over the body, conjunctivae, mouth and nose. Numerous haemorrhagic spots were seen in the gastric and large intestinal mucosa. Blood was present in the peritoneal and pleural spaces, pelvis of both kidneys, and urinary bladder. Blood stained fluid was present in both lungs, and other organs (liver, spleen and brain) were congested.

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Histopathological examination showed salient findings of multiple thrombosis and microhaemorrhages in kidneys, large and small intestines, brain and adrenal glands. Changes suggestive of acute tubular necrosis in the kidneys along with multiple thrombosis and haemorrhages were seen. Final cause of death was disseminated intravascular coagulation.

Tests like fibrin degradation products estimation and PTTK could not be done due to lack of facilities. However, the clinical profile, available laboratory results and postmortem histopathological examinations were diagnostic of DIC.

Discussion

The commonest and most severe *Hymenoptera* stings are caused by members of family Apidae (honey bee, bumble bee etc) and Vespidae (wasps, hornets etc). Allergic reactions to single stings are common, but toxic reactions to many stings are rare [3]. Among the various toxic and allergic reactions to *Hymenoptera* stings, DIC has not been reported. Intravascular haemolysis has been recorded which can also occur in DIC. Majority of deaths due to bee stings are due to allergic manifestations, usually anaphylaxis [2,3].

Known allergic symptoms and signs include tingling, flushing, dizziness, syncope, wheezing, abdominal colic, diarrhoea, tachycardia, visual disturbances, urticaria, angioneurotic oedema, oedema glottis and hypotension. Guillain Barre syndrome, neuralgic amyotrophy, optic neuritis, multiple sclerosis, other demyelinating disorders

and encephalopathy have also been described. Serum sickness may develop later. Known toxic effects of bee stings include vasodilation, hypotension, vomiting, diarrhoea, headache and coma. Other features include epidermal necrosis, intravascular haemolysis, haemoglobinuria, thrombocytopenic purpura, myasthenia gravis, glomerulonephritis and renal failure [3].

Hymenoptera venomous non-allergenic compounds include vasoactive amines like histamine, kinins and specific toxic peptides such as apamine, melittin, and the anti-inflammatory peptide 401 (mast cell degranulating peptide). Other substances are 5-hydroxy-tryptamine, bradykinin, catecholamines and cholinesterases [3]. It is possible that some of these compounds may be the trigger for DIC.

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