Case Report

Autopsy Findings and Histopathological Corroboration in A Case of Death due to Hornet Bite

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Abstract

We present a rare case of death due to hornet bite in an adult 64 years old patient who died within 6 hours of admission. There were bites on multiple sites and histopathology of Kidney showed acute tubular necrosis. Liver shows fatty changes periportal fibrosis and portal inflammation. Lungs show intra-alveolar exudation, alveolar wall destruction and inflammatory infiltrates. Heart showed deposition of lipofuschin and brain showed focal oedema with dilatation of vessels. These accidents can occur in places of cohabitation of hornets with humans. People who are allergic to wasp venom are also allergic to hornet stings. In severe cases, allergic individuals may go into anaphylactic shock and die unless treated promptly. Clinical criteria for fatal cases can be formulated from this case and especially susceptible individuals like elderly with co morbid conditions and children should be treated early and extensively to avoid fatal complications.

Key Words: Hornet bite, Fatal, Histopathological corroboration, Allergy, Inflammation

Introduction:

Hornets are the largest eusocial wasps; some species can reach up to 5.5 cm (2.2 in) in length. The true hornets make up the genus Vespa and are distinguished from other vespines by the width of the vertex (part of the head behind the eyes), which is proportionally larger in ***Vespa*** and by the anteriorly rounded gasters (the section of the abdomen behind the wasp waist).

The best known species is the European hornet (Vespa crabro), about 2–3.5 cm in length, widely distributed throughout Europe, Russia, North America and Northeast Asia.

Hornets have stings used to kill prey and defend hives. Hornet stings are more painful to humans than typical wasp stings because hornet venom contains a large amount (pkp, 5%) of acetylcholine. Individual hornets can sting multiple times.

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DOR: 29.12.2014 DOA: 25.06.2015
DOI: 10.5958/0974-0848.2015.00080.9

Unlike honey bees, hornets and wasps do not die after stinging because their stingers are not barbed and are not pulled out of their bodies.The toxicity of hornet stings varies according to hornet species; some deliver just a typical insect sting, while others are among the most venomous known insects. Single hornet stings are not in themselves fatal, except sometimes to allergic victims. Multiple stings by non-European hornets may be fatal because of highly toxic species-specific components of their venom. The stings of the Asian giant hornet (*Vespa mandarinia japonica*) are the most venomous known.

Stinging insects are members of the Order Hymenoptera of class Insecta. In most instances, insect stings are only followed by allergic reactions but sometimes intravascular haemolysis, rhabdomyolysis, thrombocytopenia [9], acute tubular necrosis, acute hepatic injury [9, 10], and myocardial infarction [11] have been reported.

Their venom is a concentrated mixture of complicated active components, such as melittin, apamine, phospholipases, hyaluronidase, acid phosphatase, histamine, and kinin. These have direct and indirect haemolytic effects, neurotoxic and vasoactive properties, which can cause intravascular haemolysis and rhabdomyolysis [4]

Case History:

A 64 years old male was brought for autopsy to the police morgue of Burdwan

Medical College with history of hornet bite. Multiple punctured wounds each measuring 2mm x 2 mm with evidence of blackening induration and oedema of surrounding skin was noticed over following areas of body.

The areas involved were Dorsum of right hand 2x1 inches, Malar area of left side of face 2.5x1 inches, Anterior aspect of left forearm 1.5x1 inches, Posterior aspect of left arm 2x0.5 inches, Dorsum of phalanges of Right index finger 0.8x0.4 inches, Anterior aspect of right leg 2x1.6 inches and Dorsum of right foot 1.4x1inches.

Autopsy Findings:

Huge extravasations of blood and serous fluid were found in underlying areas. Organs were congested all over. Pleura and pericardium show pin point haemorrhages on both sides. Liver showed pin point haemorrhages on under surface of liver.

Kidney showed slight cortico-medullary haemorrhages on both sides. Considering all probable factors Time since death was between 12-36 hours prior to date and time of PM examination.

Histopathological Findings:

On histopathology and haematoxylin eosin staining the tissues under high power (40X) magnification showed following changes. Kidney showed acute tubular necrosis, hyalinisation of glomerular capillary and focal glomeruloscerosis. (Fig. 1 & 2)

Liver showed fatty changes periportal fibrosis and portal inflammation. Lungs showed intra-alveolar exudation, alveolar wall destruction and inflammatory infiltrates. (Fig. 3) Heart showed dilated blood vessels and lipofuschin pigment. Brain showed mild focal oedema and dilated thin walled blood vessels. (Fig. 5)

Discussion:

A death of this nature is sudden and occurs unexpectedly in places where there is cohabitation of humans and hornets. Disturbed hornets are extremely aggressive which can result in fatal consequences from which the victims cannot escape. Hornets are numerous in Indian subcontinent.

They form nests close to human dwellings where there is a constant conflict with humans which may result in immense human suffering and even leads to fatalities.

Therefore, utmost priority should be given to recognize hornet stings as an important public health issue and to improve management strategies in hospitals like giving early recognition of symptoms thereby reducing the number of deaths.

Thorough knowledge about possible complications of hornet stings, understanding of venom effects on body homeostasis and testing of different therapeutic regimens that would reverse the deranged physiological processes in the body. In this case study, the patient had similar macroscopic and histopathological findings suggesting an initiation of the same pathophysiological process as a result of envenoming that has progressed to a fatal outcome. In previously reported cases acute pulmonary oedema had been the dominant one detected both clinically and histopathologically that had contributed to their death within a short hospital stay. It is thus obvious that the development of pulmonary oedema can take place in a few hours in the patient.

Our case shows histopathological changes of lungs However, fluid shift as a result of immediate anaphylaxis or anaphylactoid reaction starts very early leading to the development of acute pulmonary oedema followed by a cascade of events such as prolonged hypoxemia, metabolic acidosis and cardiac dysfunction. During the early hours, onset of acute pulmonary oedema was not obvious clinically and detected later in advanced stages when the damage was irreparable.

The immune mechanisms causing the histamine-releasing action of Hymenoptera stings are either Type 1 hypersensitivity that operates through immunoglobulin E-mediated mast cell degranulation leading to anaphylaxis or anaphylactoid reaction where immunoglobulins are not involved [3, 5] or, occasionally, delayed reactions due to Type III hypersensitivity immune response that could cause Arthus reaction and serum sickness.

It is apparent that deaths happening a few hours or days later could be due to different problems which are unpredictable.

Other than lungs and kidneys, other organs did not show significant histopathological changes in these patients. On microscopic examination the myocardium and coronary arteries were normal and what caused the cardiac arrest was not ascertained.

In our case hemosiderin deposition was seen in myocardial muscle fibres. Lessons learnt reiterate that immediate recognition of anaphylaxis, early use of adrenaline, inhaled beta agonist and other measures are crucial for a successful outcome. [12]

Therefore, the positive impact of adrenaline administration in the early stage of Hymenoptera envenoming is clear irrespective of either anaphylactic or anaphylactoid reaction.

Testing for immunological mechanisms is beyond our scope.

In fatal cases one might find insufficient monitoring, missing the onset of organ dysfunctions, delays in early intensive care and organ support and inappropriate therapeutic decisions and medications. Lack of standard management protocols of Hymenoptera envenoming is a global problem which has an amplified effect on victims in resource-poor countries. Therefore, increased vigilance is needed from the time of stinging to detect all the complications and to institute proper management to increase the chances of survival of the victim.

The severity of envenoming and the late complications are related to the number of stings as shown in these cases, but sometimes a single sting could be fatal in a sensitised individual. Hymenoptera stings and envenoming needs more global attention as it appears to be a neglected problem now. [16-18]

Previously various case reports of acute renal failure in patients with Hymenoptera stings have been documented. Initially it was attributed only to tubular necrosis (ATN) either due to shock or pigment nephropathy due to intravascular haemolysis or rhabdomyolysis. [5-7, 12] Three of the five patients with hornet bite (V. orientalis) and renal failure reported by Sakhuja et al had histopathological evidence of ATN. [5] Mejia et al also reported five cases of acute renal failure following African bee stings. [12]

Post-mortem renal biopsy done in one of these patients showed dense proteinaceous casts of collecting tubules and ascending loop of Henley (probably due to shock), arterial nephrosclerosis (due to underlying hypertension) and unexplained membranous glomerulonephritis.

Our case also shows similar findings of acute tubular necrosis. Glomeruloscerosis is however probably a result of long standing premorbid condition likes hypertension.

Rhabdomyolysis and ischemia was thought to be the most probable cause of renal lesions. [12] There were reports of renal failure without any evidence of haemolysis and shock earlier but as renal biopsy was not done, the cause could not be ascertained and it was postulated to be due to the direct toxic effect of the venom. [13]

Sakhuja et al had also found that direct toxic effect cannot be directly excluded. Vikrant et al reported three case of acute renal failure following wasp bite but only two had evidence of intravascular haemolysis. [14]

So obviously there are causes other than ischemic/toxic acute tubular necrosis which are responsible for development of acute renal failure in such patients

Conclusion:

This may help to formulate or postulate clinical criteria for diagnosis in fatal cases of hornet bite especially in cases of elderly and children who form the susceptible population at risk. Premorbid conditions like long standing diabetes, liver and renal diseases also show an increased propensity of fatal outcome and early treatment can help to prevent death in allergic individuals with implementation of crucial treatments such as early use of assisted ventilation, organ support, dialysis, and medications such as adrenaline.

Further studies are needed to understand the pathophysiological mechanisms of pulmonary oedema in Hymenoptera envenoming and to find out appropriate preventive treatments.

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Fig. 1: Acute Tubular Necrosis and Focal Glomeruloscerosis in Kidney HE Stain (40X magnification)

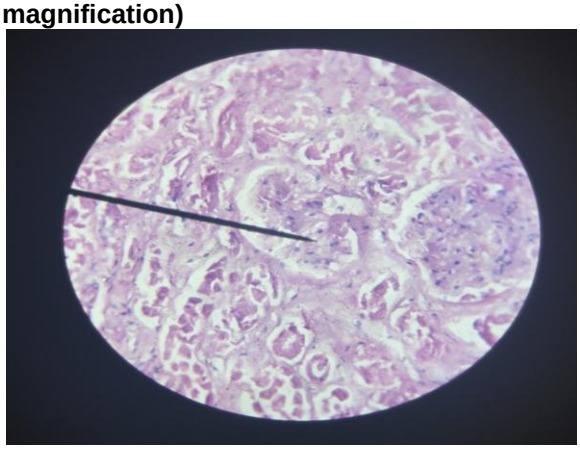

Fig. 2: Peritubular Arterial Wall Thickened and Dilated lumen HE Stain (40X magnification)

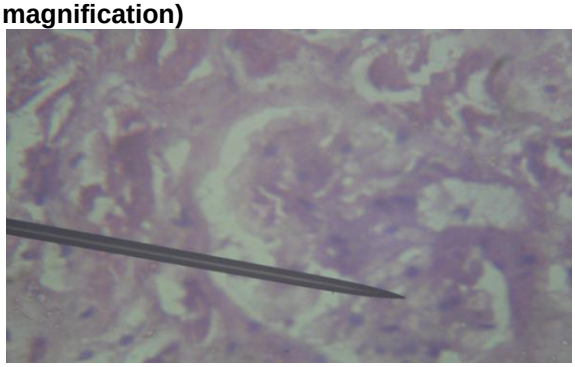

Fig. 3: Lungs Intra-alveolar Exudation, Alveolar wall Destruction and Inflammatory Infiltrates in Lungs HE Stain (40X magnification)

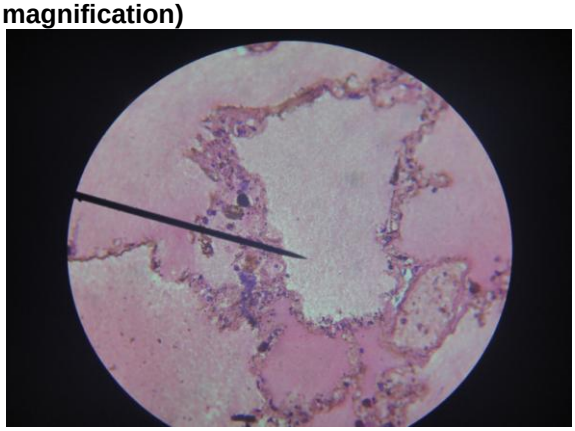

Fig. 4: Intra-alveolar exudation, Alveolar Wall Destruction & Inflammatory Exudate HE Stain (40X magnification)

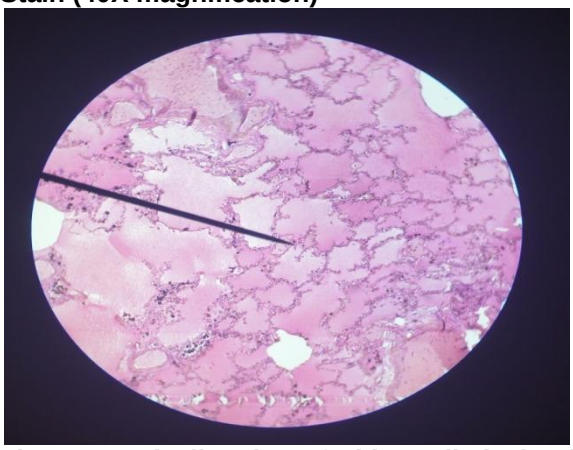

Fig. 5: Focal Dilatation of Thin Walled Blood Vessels of Brain HE Stain (40 X Magnification)