FATAL HAEMOLYSIS FROM WASP AND HORNET STING

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Stinging by bees and wasps are perhaps a common occurence and the effects are usually of little consequence. In most cases, a sting by a bee or a wasp causes only a local reaction characterized by pain, redness and slight swelling at the site.

A fatal outcome to the stinging may however result from anaphylactic shock or toxic systemic effects due to the venom itself. The latter effects do not occur until many stings are received coincidentally (Marshall 1957). This is in contrast to the hypersensitive person in whom the final sting results in death. The systemic toxic effects could be haemorrhagic, haemolytic, or neurotoxic. Furthermore, wasps and hornets, unlike bees, are scavengers and are likely to transmit infection along with their venom.

This present communication is a report of two cases of fatal haemolysis with acute tubular necrosis following multiple wasp and hornet stings.

CASE 1

C.Y.C., a four-year-old Chinese girl was apparently well until the afternoon of the 2.12.61 when she was stung by many wasps. She was taken immediately by her parents to the Government outpatient clinic where she was seen by a medical practitioner, treated and sent home as her condition was satisfactory.

During the same night, the patient was noticed by her parents to be breathless. She vomited several times and was unable to retain feeds. There was no haematemesis. She passed only some dark coloured urine during the night. The father noticed that the patient's face was swollen and the next morning it was found that she was jaundiced.

On admission to hospital in the morning, the patient was drowsy, ill-looking and moderately jaundiced. Her breathing was acidotic in nature. She had pitting oedema over the scalp, hands and the eye lids. Twenty-five haemorrhagic sting spots were counted mostly over the head, face and upper limbs. The temperature was 100°F and the pusle rate was 160/minute with a respiration rate of 40/minute. There was no evidence of cardiac failure. The bladder was catheterized and only a small volume of dark coloured urine was obtained.

LABORATORY INVESTIGATION

Blood investigations showed that the patient had a total serum bilirubin—16 mgm %; blood urea = 105 mgm % and alkali reserve = 30 vol. %. The dark coloured urine showed the presence of numerous red blood cells and oxyhaemoglobin on spectroscopy.

PROGRESS AND TREATMENT

The patient was given intravenous 1/6 molar lactate, blood transfusion and hydrocortisone. Progress was poor and she died the same afternoon about twenty-four hours after having been stung.

POST-MORTEM FINDINGS

At necropsy, the interesting feature was the presence of haemorrhage. The heart weighed 60 grams and there were petechial haemorrhages on its surface and in the ascending aorta. The lungs were firm in consistency, oedematous and appeared haemorrhagic. The stomach contained some digested blood. Besides evidence of mucosal haemorrhage in the lower 2 c.m. of the oesophagus, there was in addition, a submucosal haematoma measuring $2 \times \frac{1}{2}$ c.m. The kidneys weighed 90 grams and were bright red in colour. Bladder contained 10 m.l. of blood stained urine.

HISTOLOGY

Skin:

The epidermis showed thinning due to compression of the dead epidermal cells. This thin layer of necrotic epidermis stood out in contrast to the normally thick epidermis. The squamous cells became vacuolated with indistinct cell border and nuclei had lysed and disappeared.

There were some polymorphonuclear and lymphocytic infiltrations of the upper dermis (Fig. 1). In the dermis, some groups of sweat glands underwent necrosis, leaving a gross outline of the glands. These glands did not show any nuclei and the cells had lost their cellular outlines so that syncitial smudgy rings were seen instead (Fig. 2).



Fig. 1. Skin. Haematoxylin and Eosin x 150. The portion of epidermis on the left is thinned due to necrosis. On the right is normal. Note lifting up of the dead epidermis from the corium.



Fig.2. Haematoxylin and Eosin x 150. The sweat glands are necrotic leaving a bare outline. The nuclei are absent and the cytoplasm is reduced to an amorphous mass with no cellular outlines.

Kidney:

The glomeruli were normal. The proximal, distal convoluted and collecting tubules contained a large number of red blood cells and haemoglobin casts.

There was degeneration of distal convoluted tubules, the cells became vacuolated and cell outlines indistinct (Fig. 3). This is a picture of haemoglobinuric (lower nephron) nephrosis.

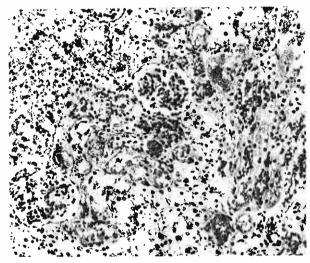


Fig. 3. Haematoxylin and Eosin x 150. Kidney showing normal glomeruli. The proximal and distal convoluted tubules contain red blood cells and haemoglobulin casts. The distal convoluted tubules at the lower left hand corner showed signs of degeneration with disappearance of nuclei.

CASE 2

H.K.H., an eight-year-old Chinese girl was plucking fruits with her two elder brothers at 11.00 a.m. on 2.8.65 when she was stung by a swarm of hornets nesting in another tree. Of the three children, the patient received the most stings, and according to the mother, her head was swarmed with hornets.

The advice of a general practitioner was sought and as the patient's condition was good, she was given antitetanus toxoid, aspirins and an antihistamine. As advised, the patient was again seen by the practitioner the same afternoon and as her general condition was good, she was sent home.

The patient was apparently well until the next morning 3.8.65, when the mother noticed that she was drowsy, jaundiced, pale and had no appetite. She also passed a small quantity of reddish urine. The general practitioner saw the patient again and referred her for admission but because of the indecision of the parents for hospitalization, the patient was not admitted till the afternoon of the same day.

On admission, the patient was markedly jaundiced, pale, drowsy and mildly dehydrated. She was, however, able to answer questions with the appropriate gestures. She had a temperature of 99.4°F and a pulse and respiration rate of 120/minute and 35/minute respectively. The blood pressure was 110/70. Over twenty hornet stings situated mostly over the head and neck region were counted on the patient-each bite measured about 1/4 inch in diameter and was surrounded by a raised carcinate erythematous margin with a central area of necrosis, both hands were markedly swollen. The cardiovascular, respiratory and central nervous systems were normal. The liver was one finger breadth below the costal margin and not tender. The kidneys were not palpable.

LABORATORY INVESTIGATION

The patient had a haemoglobin of 11 gm %, a reticulocyte count of 1 % and a total white cell count of 21,700 with a differential count of polymorphonucleocytes 85 %, lymphocytes 10 %, monocytes 5 %, and eosinophil 0 %. The platelet count was 340,000. The bleeding and clotting times were within normal but the prothrombin time was 21 seconds against a control of 17 seconds. No Heinz bodies were detected in the red blood cells.

The one minute serum bilirubin was 7.1 mgm % and the total serum bilirubin was 12 mgm %. The peripheral blood showed the presence of oxyhaemoglobin and the methaemoglobin was 0.17 gm %. The blood urea was 104 mgm %. The catheterized bladder revealed the presence of 45 ml of reddish urine which was positive for oxyhaemoglobin and red blood cells. The cerebrospinal fluid obtained was clear.

TREATMENT AND PROGRESS

The patient was given intramuscular and intravenous hydrocortisone, blood transfusion and 5% dextrose solution to correct the dehydration. However the patient died suddenly of cardiac arrest at 3.00 a.m. on 4.8.65 (40 hours after the stinging). A post-mortem intracardiac serum potassium, after half an hour's attempt at external cardiac massage, was estimated at 14.4 mEq/L.

SKIN BIOPSY

No post-mortem was obtained. The skin showed areas of coagulative necrosis in the epidermis with vacuolated cells, pyknotic nuclei and indistinct cell border. There was infiltration of polymorphonuclear leukocytes and lymphocytes (Fig. 4). The sweat glands were also necrotic and an identical picture was seen to the previous, case.



Fig. 4. Haematoxylin and Eosin x 75. The left half of epidermis show coagulation necrosis and cellular outlines are still present. However, all the nuclei are pkynotic.

DISCUSSION

The insects which produce allergic phenomenon may be divided into three groups according to whether they sensitize the human host (1) by innocently, by scales or dust shed from wings or body (*e.g.* butterflies); (2) by injection of venom through the sting situated in the rear extremity (*e.g.* bees and wasps); (3) by installation of salivary secretion through the mouth parts (*e.g.* mosquitoes).

The Hymenoptera family may be divided into two groups consisting of the honey bee and the wasp. Hornets, are included in the latter group. This family of insects threaten man and animals by their stinging mechanism situated in the rear end of the animal which comprises an elaborate, offensive and defensive apparatus formed by the adaptation of the ovipositor. Through its hollow double lancet is injected the venom—a highly complex mixture of chemical ingredients. Jacques and Schachter (1954) found that wasp venom contains hyaluronidase in addition to three active muscle stimulants—histamine— 5-hydroxytryptamine and a substance which resembles bradykinin. The high concentration of histamine and 5-hydroxytryptamine account for some of the features of the skin reaction following wasp sting. However, wasp venom itself is also able occasionally to release histamine as in anaphylaxis. The large amounts of hyaluronidase the venom contains would enhance the diffusion of toxic substances in the skin. Schenken et al (1953) record that the venom of bees contain three toxic factors—(1) a haemolytic factor known as a phosphatidase; (2) a

Many of the deaths from bee or wasp sting appear to be due to anaphylaxis from foreign protein injected with the venom. In the case of the bee, the implanted barb and the pollen carried have been indicated as anaphylactic agents. The presenting features of these two cases was not one of anaphylaxis, as they were seen to be well a few hours after the stinging, but of haemolytic anaemia.

neurotoxic factor which paralyses adjacent nerve

endings to cause localized oedema; (3) hista-

Irvine (1962) described a case of fatal haemolysis in a girl aged three years who was stung by several hundred bees. Many of the sting sites were urticariae with a petechiae centre and often with vesicle or pustule at the point of entry. On both these two cases were counted not more than thirty sting sites. These were sited mostly on the head, upper chest and arms and were about 1/4 inch in diameter. They were seen to be gangrenous. Histology showed evidence of necrosis of cellular elements of the skin appendages.

Day (1962) found in the literature seven cases of a total of ten fatal bee and wasp sting with necropsy findings of damage to the central nervous system. His own case was an adult who died nine hours after stinging by a swarm of "yellow jackets"—death resulted from haemorrhagic cerebral infarction. Day believes that the powerful hypotensive and smooth muscle stimulating action of bradykinin from the sting sites in the neck could conceivably produce jugular venous sinus, produce a haemorrhagic cortical necrosis. The cerebrospinal fluid of Case 2 was clear and there was no evidence of cerebral haemorrhage in Case 1 inspite of the stinging in the neck region, but at necropsy evidence of haemorrhage was found in the lungs, oesophagus, pericardium and ascending aorta.

There are several interesting and significant features about the two cases described in this paper. There was an interval of several hours between the stinging and the onset of systemic toxic effects during which the patients were apparently well. Both the cases were seen by doctors soon after the stinging and were found to be in satisfactory condition. In both cases the systemic toxic effects appeared within 24 hours after the stinging, thus it seems advisable that all cases of multiple wasp or hornet stings should be observed in hospital for at least 24 to 48 hours. The cases of wasp or bee sting with systemic toxic side effects as reported by James and Walker (1952), Irvine (1962), Day (1962), all had several hundred stings. In these two cases of ours, a mere 25-30 stings were sufficient to cause fatal toxic systemic effects.

There was clinical evidence of haemolysis as well as haemorrhage in both the patients. Evidence of haemolysis was revealed by pallor, jaundice, raised serum bilirubin level, presence of oxyhaemoglobin and methaemalbumin in the blood and spectroscopic evidence of haemoglobin in the urine. The finding of numerous red blood cells in the urine of both patients suggest the presence of haemorrhage. At necropsy of Case 1, there were haemorrhages in the lungs, gastrointestinal tract, pericardium and ascending aorta.

The cause of death in both cases was probably due to acute tubular necrosis and not from internal haemorrhage. Histology of renal tissue from Case 1 showed evidence of acute renal tubular necrosis (Fig. 3). Both cases had oliguria and raised blood urea on admission. The post-mortem serum potassium of 14.4mEq/L found in Case 2 was high compared against similar non-haemolytic, non-renal tubular necrosis fatalities of 8 mEq/L.

James and Walker (1952) attributed the probability of the temporary renal failure in their eighteen months old child after 477 wasp stings to a pre-renal origin—due to the generalized oedema which must have diminished the renal blood supply considerably. Irvine (1962) found acute tubular necrosis in his case of fatal haemolytic bee-stinging and inspite of two renal dialysis, the patient died on the eighteenth day.

mine.

Thus, besides observing all multiple bee or wasp stings in hospital, it is advisable to treat such cases of haemolysis with evidence of acute tubular necrosis with repeated renal dialysis when indicated.

SUMMARY

- 1. Two cases of fatal haemolysis resulting from wasp and hornet sting are reported. The feature to note is the apparent good health of the patients a few hours after the stinging until systemic poisoning—haemolysis and haemorrhage—developed.
- 2. Case 1 showed evidence of acute tubular necrosis and Case 2 (no autopsy done) probably died of the same condition.
- 3. It is advisable therefore to observe all cases of multiple bee or wasp stings in hospital and to institute renal dialysis when indicated.

REFERENCES

- 1. Benson, R.L. and Semenov. H. (1930): "Allergy in its relation to bee-sting", J. Allergy. 1, 105-116.
- Benson, R.L. (1939): "Diagnosis of hypersensitiveness to bee and to mosquito with report on successful specific treatment", Arch. Intern. Med. 64, 1,306-1,327.
- 3. Day, M.J. (1962): "Death due to cerebral infarction after wasp sting", Arch. Neuro. 7, 184.
- 4. Dyke, S.C. (1941): "Wasp sting as an immediate cause of death", Lancet 2, 307-308.
- Irvine, R.O.H. (1962): "The twin coil artificial kidney in the treatment of acute oliguric renal failure", New Zealand Med. J. 61, 184-190.
- 6. James, E.S. and Walker, W.G. (1952): "ACTH in the treatment of wasp stings", Canada Med. Ass. J. 67, 50.
- 7. Jacques, R. and Schachter, M. (1954): "The presence of histamine, 5-hydroxytryptamine and a potent slow contracting substance in wasp venom," Brit. J. Pharm. 9, 53-58.
- 8. Marshall, T.K. (1957): "Wasp and bee stings", Practitioner. 178, 712.
- 9. Schenken, J.R.; Tamisiea, J.; Winter, F.D. (1953), "Hypersensitivity to bee sting. Report of a fatal case and review of literature", Am. J. Clin. Path. 23: 1,216-1,221.