HONEY BEE STINGING (APISINATION): WHAT THE MEDICAL AND VETERINARY PROFESSIONALS OUGHT TO KNOW?

Ghulam Muhammad, M. Saqib and S.H. Mallick[1]
Department of Clinical Medicine and Surgery,
Faculty of Veterinary Sciences, University of Agriculture, Faisalabad-38040
[1]Small animal practitioner, Defence, Karachi, 75500

INTRODUCTION

Stinging by honey bees poses a significant life-threatening risk to human and animals. In USA, Hymenoptera (bees, wasps and hornets) are responsible for more human deaths than any other venomous animal (Parrish, 1965). Attacks by honey bees are particularly a problem in wooded hilly areas, groves and park etc., where swarms of bees are found hanging from the boughs of trees, in caves and on the walls of abandoned houses. Cattle, buffaloes, equines, dogs, etc., tethered under the shady trees harboring honey bees hives may receive a fatal assault by these flies. Attack may be provoked by children hurling stones at the hives or when noisy equipments such as tractors, thrashers, lawn mover etc., are operated in areas too close to honeybee hives. In addition, bees when en-route to new colonies may invade stables or yards where in horses, cattle, buffaloes or other animals are housed. This paper is written to improve the competence of the medical professionals and veterinarians in handling the emergencies arising out of honey bee stinging. Wasps and hornets stinging may be treated along the same lines. This subject has not been covered adequately in standard human and veterinary texts.

Types of honey bees

There are five important species of honey bees (Ahmad, 1988; Schmidt and Hassen, 1996).

1. Oriental bee (*Apis cerana*)
2. Rock bee (*Apis dorsata*) commonly found in hilly areas.
3. The little bee (*Apis florea*) commonly known as "Shotee Makhee" in Punjabi.
4. Occidental bee (*Apis mellifera*) found in Western Hemisphere (America, Europe).

Of these varieties, the oriental bee occurs in the Northern and Western hills and foot hills in some parts of the North West Frontier Province, Punjab, Baluchistan and Azad Jammu and Kashmir and the rock bee and little bee in the foot hills, plains and semi-desert areas in all the provinces. The first three species are native to Pakistan. The little bee is the least venomous. The occidental bee was imported from Europe in 1977–78 because of its more efficient honey production. This species can be artificially reared. New colonies of this species are reared in almost all the honey bee producing area of Pakistan. From medical and veterinary standpoint, Oriental and Africanized bees are probably most dangerous.

Composition and properties of bee venom

Studies on the composition and properties of honey bee venom have by and large been limited to that of occidental bee (*Apis mellifera*). Bee venom is transparent, has a sharp smell reminiscent of honey, and a bitter burning taste. Its specific gravity is 1.1313. When tested with litmus paper, it gives an acidic reaction (Ioyrish, 1974). Bee venom is a blend of components including (but not limited to) formic acid, hydrochloric acid, orthophosphoric acid, histamine, dopamine, noradrenaline, tryptophan, sulphur and two major toxic peptides (mellitin and phospholipase A_2). Mellitin which is sometime referred as "direct" hemolysin accounts for 50% of the weight of the dried venom. It has cardiotoxic and cytotoxic properties as well. Phospholipase A_2, a well known allergen constitutes 12% of the weight of the venom. Bee venom also contains magnesium phosphate (0.4%), traces of copper, calcium, several proteins, volatile oils and enzymes (e.g.,hyaluronidase which is a spreading factor) (Ioyrish, 1974; Hermor, 1981; Schmidt and Hassen, 1996). Bee toxin is not decomposed by caustic alkaline solution or sulphuric acid even after 24 hours but its properties are subjected to change, when it is heated for any time with hydrochloric acid or a caustic alkali. Potassium permanganate and other oxidizing agents reduce its activity. Bee venom is extremely heat resistant. Freezing too does not alter its toxic effect when kept dry. Venom retains its toxicity for several years. Bee venom is considered to be one of the strongest antibiotic substances known (Ioyrish, 1974).

Bee stings or injection of apitoxin are conducive to raising immunity not only to bee venom but also to certain infectious diseases. When used correctly, bee

venom is therapeutic and prophylactic remedy affecting the organism as a whole and not just individual organs or particular illness. When bee venom enters the mammalian body, it rallies all the factors of resistance, which explains in part why beekeepers who have worked for many years in a bee garden enjoy good health and live to ripe old ages (Ioyrish, 1974).

PATHOGENESIS AND CLINICAL SIGNS

There are two types of the pathogenic effects of the bee stings (Meerdink, 1983; Cowell *et al*., 1991; Schmidt and Hassen 1996).

Hypersensitivity Reactions

Hypersensitivity reactions occur as a result of one or a few stings and are not related to venom toxicity. An estimated 0.5 to 2% of people are considered hypersensitive to venom of bees, wasps and ants. Hypersensitive individuals may become unconscious within ten minutes of being stung (Hooper, 1991). Cases are on record (very rarely though) where a single bee sting caused the death of a completely healthy person (Ioyrish, 1974). Fatal responses to single hymenopteran stings have likewise been reported in dogs (Cowell *et al*., 1991)

Toxic Envenomation

Stings by a large number of oriental, occidental and Africanized bees cause intoxication independent of hypersensitivity. Symptoms and signs of toxic envenomation include nausea, vomiting, general weakness, hypotension, pulmonary edema, tachycardia, and loss of consciousness. In cases of extreme numbers of stings, death occurs rapidly as a result of the cardiotoxicity of the venom. Delayed manifestations include hematuria, rhabdomyolysis, and acute renal failure. Disseminated intravascular coagulopathy (DIC) has been reported in people and dogs. The clinical syndrome may last for days, during which time-intensive medical support is required.

Toxic envenomation results directly from venom.. In the case of occidental bee, toxic envenomations are not expected to be observed in animals receiving fewer than 2 stings/kg or in people who receive fewer than 100 stings. In adult people, toxic reactions can become evident when the sting number exceeds 300 and are very likely with more than 500 stings. Death is the ultimate result of toxic envenomation. Bee toxin has a selective effect on the nervous system. Cleopatra, (Egyptian queen) who was interested in the effects of poisons, collected every possible kind of poisonous substance, trying to find a poison that would act painlessly. She tested their effect on prisoners who were condemned to death. The poison that caused the least agonizing death, it proved, was wasp venom (bee venom could not be used, as bees were considered sacred. Nevertheless, bee venom is similar in action). A person given an injection of wasp venom lost consciousness, beads of sweat appeared on his face and he died quickly and painlessly. If an attempt was made to arouse the victim from this condition by physical exertions, he resisted as if in a deep sleep (Ioyrish, 1974). People (e.g., beekeepers) when repeatedly stung by bees develop specific antibodies to venom and such people can endure stings without much harm.

TREATMENT

Extraction of the stings together with their poison sacs

It is the most essential and important step in the treatment of honey bee, wasp and hornet stinging (Ioyrish, 1974). The longer the sting remains in the skin, the more poison passes into the body. When a bee has stung a person or an animal (stinging causes death of the honey bee within a couple of days), it instinctively attempts to fly away, but the barb of the sting catches firmly in the skin and the stinging apparatus is torn off with both the poison glands and the poison sac and the ganglion of the abdominal nerve chain, which ensure its automatic innervation and contraction outside the bee's body. As a result venom continues to enter into the body of the victim. Therefore, the sting should be pulled out of the skin as soon as possible. The victim, however, usually uses his/her fingers and thereby presses the entire stock of venom out of the stinging apparatus into the skin. By using fine tweezers it is possible to remove the sting quickly, gently, and carefully, losing hardly any of the poison from the sac. If one can not lay hands on special tweezers, careful removal of the poison bag by scraping out side ways with a blade is recommended. Fingernails can be used alternatively. The most important thing to remember is that sting should not be compressed.

Local treatment

Once stings alongwith their poison bags have been removed, the wound should be treated locally. To that end followings have been recommended:

(a). Cream containing calendula, surgical spirit, and vaseline or lanolin. The combination of calendula and spirit soon relieves the pain and the unpleasant burning sensation (Ioyrish, 1974).

(b). Australian workers (Henderson and Easton, 1980 reported that stingose (Hamilton Labs), a preparation of 20 per cent aluminum sulphate and 1.1 per cent surfactant, is a wide acting and effective treatment for

bees, wasps, hornets, mosquitoes, sandflies and ants, as well as marine stingers including jellyfish, bluebottles, catfish and stinging plants.

(c). Potassium permanganate ($KMNO_4$) and other oxidizing agents reduces the activity of the bee, wasp and snake venom (Ioyrish, 1974). Therefore, $KMNO_4$ can be applied when cream/medicaments especially formulated for bee/wasp stinging are not available.

d) Local application of an antihistamine lotion (e.g., Caladryl, Parke-Davis) and local anaesthetic gel (e.g., Xylocaine gel, Barrett Hodgson) may be of value. Local application of weak ammonia and sodium bicarbonate likewise usually proves beneficial (Hungerford, 1990).

1. Antihistamine/Corticosteroids

Inject epinephrine (adrenaline) immediately by subcutaneous route. Intoxication and anaphylaxis may occur simultaneously or may be difficult to distinguish. To counteract the effect of venom-induced histamine release, give both H_1 and H_2 blockers I.V (e.g diphenhydramine at 1–2 mg/kg twice a day and cimetidine at 5–10 mg/kg thrice a day. Also give corticosteroids at higher than general dosage level (Cowell *et al*., 1991; Schmidt and Hassen, 1996).

2. Supportive and other treatment measures

a) Fluid and electrolytes or blood replacement if there is hypovolemic shock, renal or gastrointestinal blood loss (Cowell *et al*., 1991; Schmidt and Hassen, 1996).

b) Urinary alkalization (by sodium bicarbonate) if there is severe rhabdomyolysis (Schmidt and Hassen, 1996).

c) Valium if there are convulsions (Hungerford, 1990).

d) Tracheotomy/ventilatory support particularly if there is asphyxia due to multiple stings around the nares or throat.

REFERENCES

Ahmad, R., 1988. Beekeeping in Pakistan: Present status and economic importance. *Progressive farming* (Pakistan Agriculture Research Council) 8 (2): 32–37.

Cowell, A.K., R.L. Cowell, R.D. Tyler and M.A., Nieves, 1991. Severe systemic reactions to *Hymenoptera* stings in three dogs. J. Am. Vet. Med. Assoc., 198 (6): 1014–1016.

Henderson, D. and R.G. Easton, 1980. Med. J. Aust., (2): 146–150.

Hermor, H.R., 1981. Social Insects Vol. II. Academic Press New York. pp: 157–159.

Hooper, T., 1991. Guide to Bees and Honey. 3rd Ed. Blandford - an imprint of Cassel Publication, London. Pp: 63–64.

Hungerford, T.G., 1990. Hugerford's Diseases Of Livestock. 9th Ed. McGrawHill Book Co. Sydney.

Ioyrish, N., 1974. Bees and People (English translation from Russian). Mir Publisher, Moscow.

Meerdink, G.L., 1983. Bites and stings of the venomous animals. In: Kirk, R.W. (ed). Current Veterinary Therapy. VIII. Small Animal Practice. W.B. Saunders Co. Philadelphia. Pp: 155–159.

Parrish, H.M., 1965. Analysis of 460 fatalities from venomous animals in the United States. Am. J. Med. Sci. 245: 129–241.

Schmidt, J.O. and L.B. Hassen, 1996. When Africanized bees attack: what you and your client should know. *Vet. Med.* Oct. 1996. pp: 923–928.