24. CLINICAL MANIFESTATIONS OF SNAKE BITE BY *VIPERA XANTHINA PALESTINAE* (WERNER) AND THEIR PATHOPHYSIOLOGICAL BASIS

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*Vipera xanthina palestinae* is practically the only poisonous snake in Israel. Bites caused by other snakes, including *Échis colorata* (Guenther), have been extremely rare.

The actual annual incidence of snake bites in our country is not known, as no compulsory notification of cases is required. As a rough estimate, I would assume the incidence to be from 150 to 250 cases per year. For the last 25 years I have under my personal observation 21 cases of viper bite admitted to two hospitals. Of this material I have investigated 15 severe cases for the clinical manifestations which correspond, as a matter of fact, to the natural course of poisoning, as no specific antivenin was given. Specific antiserum produced by Institut Pasteur, Paris, has been on the market since 1956 only. The treatment used was mainly anti-shock treatment.

Soon after having been bitten, the victim experiences local pain (15)*. Shortly afterwards he becomes overwhelmed by weakness (11) and restlessness (10). 15 to 30 minutes after the accident the patients vomits repeatedly (14), perspires profusely and complains of abdominal pains (14) and diarrhoea (12). Sometimes the diarrhoea lasts several hours and the faeces becomes sanguinolent.

At this stage the victim usually reaches the hospital. He is pale, restless and covered by cold perspiration. Two patients were admitted in a state of clouded consciousness. Physical examination revealed in 11 of the 15 patients a conspicuous swelling of the tongue, regardless of the localization of the bite. Sometimes the speech becomes slurred. In 7 out of 15 patients angioneurotic oedema (Quincke) of the lips was observed and one (not included in the present series) lost consciousness for several hours, apparently because of oedema of the brain.

Seven of the patients had hypotension on admission and in 7 others the blood pressure could not be obtained. The pulse was usually weak, rapid or imperceptible. Rarely bradycardia was found. Fang marks could be detected in 11 patients, and they were usually not characteristic.

The affected extremity is often swollen on admission. Later the oedema advance centripetally, reaching the most proximal joint in 4 to 5 days. Often the oedema crosses the trunk and spreads up and down on the opposite side.

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*Number in parenthesis indicate the number of cases out of the 15 investigated ones, which manifest the symptoms.*
Comparing the circumference of the affected extremity which that of the normal one, one can find differences of as much as 14 cm (at the proximal part of the thigh!).

Several hours after the bite had been inflicted, the swollen skin reddens in many places, representing subcutaneous haemorrhages and/or ascending lymphangitis. Later, blisters make their appearance differing in size and colour, containing plasma or blood. Usually they abound when the swelling becomes conspicuous. Some of them contain as much as 30-80 ml of fluid. On one occasion we removed 250 ml of blood from a single blister. Laboratory investigations often reveal signs of haemoconcentration, haemoglobin reaching 16-20 Gm% (11). Severe leucocytosis was found in 8 patients (15,000-34,000/cumm) and a slight one in others. In two cases of the series, thrombocytopenia (10,000; 90,000) was observed, and in two others fibrinolysis occurred. Half of the patients had albuminuria, disappearing subsequently.

PATHOGENESIS AND PATHOLOGICAL PHYSIOLOGY

According to the literature, the viper venom, having a molecular weight higher than 20,000 is absorbed from the site of the bite through the lymph vessels into the circulation. The slow flow of the lymph enables the inactivation of a certain quantity of the venom "en route". On the other hand, a "spreading factor" possessing hyaluronidase-like activity favours the absorption of the venom. There is some clinical evidence supporting these assumptions. One can see for instance, lymphangitis and/or haemorrhages advancing along lymph to the regional lymph nodes. On post mortem examination there is sometimes a peculiar distribution of haemorrhages in the urinary bladder or in the uterus: they remain on one side of the mid-line.

Even small quantities of venom entering the circulation may cause generalized anaphylactoid reaction manifested by gastro-intestinal disturbances (vomiting, abdominal pains, diarrhoea), peripheral shock and angioneurotic oedema (type Quincke) of tongue, lips, glottis, brain, etc. Obviously, greater quantities of venom cause a more severe reaction. There is some experimental evidence suggesting that release of histamin from the tissues, initiated by the venom, might be the pathogenetic mechanism of this anaphylactoid reaction.

The primary anaphylactoid shock, is left untreated, passes readily over to secondary shock, owing to transudation of plasma and bleeding into the tissues. Proteolytic enzymes — called haemorrhagins — in the venom increase the permeability of small vessels resulting in extravasation of fluid. Contraction of the blood volume perpetuates the failure of the circulation (shock). In addition, bleeding into vital organs (heart, lung, brain) may kill the patient.

The morbidity is also prolonged due to severe secondary anaemia. We have never encountered massive haemolysis causing anaemia. Complications in kidney function — shock kidney — occurred very rarely indeed. Because of the increased permeability of the small vessels, toxic substances may be resorbed from the intestines, causing liver damage.

No cases of disturbances in the function of the central nervous system were observed which could be ascribed to neurotoxic factors in the venom. Clouded consciousness, coma, restlessness, occurred together with manifestations of the anaphylactoid syndrome and cleared by treatment with gluco-corticoid hormones,
affirming — in this way — their anaphylactoid origin. In some cases there were
disturbances in peripheral nerves located near to the site of the bite, apparently
caused by direct local action of the venom. In a recent case transient aphasia
occurred with positive Babinsky’s sign, probably due to haemorrhage.

In a patient treated by us and in another one of a nearby hospital, radiculitis
occurred as a part of a severe serum reaction.

Accordingly, we suggest the following essential treatment:

1. First-aid: complete immobilization of the affected extremity.
2. Adequate quantities of specific serum injected intravenously as early as pos-
sible.
3. Anti-shock treatment for shock and haemorrhages.
4. Hydrocortison for the anaphylactoid syndrome.

Treatment carried out as suggested has changed completely the clinical course,
as well as the prognosis of viper bite.

LITERATURE


DISCUSSION

E. R. Trethewie: “If the anaphylactoid shock is due to histamine one might
expect this effect to be less in children, because the amount of histamine in tissue
in children is less. Histamine release is proportional to the histamine content of
tissue.”

P. Efrati: “Nothing to answer.”

H. I. Bicher: “Did the speaker consider a rôle for the neurotoxin, described
in this venom, in the pathogenesis of his syndrome?”

P. Efrati: “The neurotoxic action of the venom was revealed only in mice
poisoned by multiple lethal doses of venom and protected by antiserum. In human
beings such a dose would cause death before neurotoxic symptoms could appear.”