Abnormal Electrocardiogram After Adder Bite

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Adder bites are occasionally reported in this country. Their effects are often thought to be mild (Manson-Bahr, 1957) and cardiac complications are infrequently observed. Walker (1945) collected 50 case reports of adder bites from doctors in England and Wales, which included 7 deaths. The mode of death in most of these was stated to be circulatory collapse, and electrocardiograms were not mentioned. Askanas (1959) described a case of snake bite in a 50-year-old patient who developed a posterior myocardial infarct. This was attributed to coronary artery thrombosis occurring during severe hypotension as a result of the bite. Brown and Dewar (1965) postulated a similar mechanism for myocardial damage occurring in their case of adder bite. Reid *et al.* (1963) described extensive and fluctuating T wave inversions in a patient bitten by the Malayan pit viper (*Ancistrodon rhodostoma*). We are prompted to report another case in which a temporarily abnormal electrocardiogram followed adder bite.

Case Report

On June 6, 1965, a schoolboy, aged 14, was on the Cumberland moors when he found a snake lying in the centre of a patch of burned heather. He thought at first that it was dead but it moved sluggishly and he caught it by picking it up behind the head. It made no attempt to escape. He showed it to his parents and took it home, nursing it on the seat of the car between his thighs. On arrival at his home he put it in a glass case which had been an aquarium, decorated the case with heather, added a glass lid, and fed the snake with snails. Periodically he took it out and allowed it to crawl on the lawn. It was handled by his brother aged 4 and his sister aged 10. On the following day he removed the lid from the glass case, placing his right hand on its edge. The snake now appeared quite alert and made a dart for his thumb. He withdrew his hand rapidly, shaking the snake off. There were two points of blood at the base of the right thumb. His father squeezed the digit above the wound, his mother sucked it, and he was brought to the hospital, arriving there 25 minutes later. There were

two fang wounds at the base of the right thumb near the web. There was oedema of the right hand, forearm, and right side of the face. The temperature was 38°C. (101°F.). The initial treatment included the incision of the wound and administration of hydrocortisone intramuscularly. In the meantime the snake was identified as a common adder (*Vipera berus*). The initial electrocardiogram was taken after arrival in the ward. 10 ml. antivenomous serum (Pasteur Institute) was given in the manner recommended by Morton (1960), together with immobilization and cooling of the right arm. No further treatment was given apart from prophylactic penicillin. The subsequent findings were the development of two blisters in the vicinity of each wound and the enlargement of cervical and axillary lymph nodes. These took four days to subside.

Chest x-ray film, blood urea, serum electrolytes, and erythrocyte sedimentation were normal. SGOT was 22 S.F. units/ml. 24 hours after the snake bite. The white blood cell count was 11,000/c.mm. and initial serum bilirubin 1.1 mg./100 ml. Liver function tests apart from bilirubin were normal. Coombs test was negative. Serum bilirubin fell to 0.4 mg./100 ml. within a week. The electrocardiogram before the administration of antivenomous serum showed dimpling of T wave in V3, a biphasic T wave in V4, and flattening of T wave in V5. By the end of the first week these changed into a frank T wave inversion in V4 and flattening of T wave in V5 (Fig. 1). There were associated T wave changes in leads II, III, and aVF, varying from flattening or inversion to biphasic configuration and a slurring of QRS in lead aVL (Fig. 2). Serial electrocardiograms showed minor abnormalities of T waves even at the end of two weeks, but these had reverted to normal on follow-up a month later.

Discussion

Hypothetically the T wave inversion in this case could be due to (a) coincidental heart disease, (b) disturbance of serum electrolytes, (c) drugs used in the treatment, (d) the effect of fear or fright, and (e) the effect of the venom. Our patient had no clinical evidence of any other cardiac disease and a chest x-ray film showed a heart of normal size and outline. Serum electrolytes were normal and the

FIG. 1.—Serial electrocardiograms (leads V2-V5) during the six weeks following adder bite. All T waves in V4 before 23.7.65 are abnormal. The T wave abnormalities are maximal 7 days after the bite.

changes in the cardiogram were present before either penicillin or antivenin was given, and persisted for more than two weeks. Mainzer and Krause (1940) have shown that T wave flattening or inversion can occur with fear but the changes are transient. Therefore it would be reasonable to assume that the changes described above were due to the venom.

Accepting that the T wave inversions were due to the venom, does it act (a) by producing coronary

FIG. 2.—Unipolar and bipolar limb leads taken on the 7th day after the adder bite showing slight but definite conduction delay compared with similar leads obtained on the 16th day in which this has virtually disappeared.

ischaemia and even thrombosis, resulting from fall of blood pressure, (b) by promoting venous thrombosis with possible pulmonary embolism, (c) by causing thrombosis or subintimal haemorrhage in the coronary vessels, (d) by causing pericarditis, (e) by causing hypoglycaemia, or (f) by direct effect on the myocardium?

(a) *T Wave Inversion due to Coronary Ischaemia or Thrombosis Resulting from fall of Blood Pressure.* The fall of blood pressure following a snake bite may result from the action of bradykinin, a polypeptide formed by the action of snake venom on plasma globulin (Rocha e Silva, Beraldo, and Rosenfeld, 1949). This could favour coronary thrombosis resulting in changes in the cardiogram (Askanas, 1959; Brown and Dewar, 1965). The erythrocyte sedimentation rate and SGOT were normal in our case; moreover there was no fall of blood pressure and this would make even temporary myocardial ischaemia unlikely.

(b) *T Wave Inversion due to Venous Thrombosis and Pulmonary Embolism.* This appears to be a logical possibility in view of the fact that adder bite venom is known to produce extensive venous thrombosis (Arneil and Maclaurin, 1961). The rapid onset of electrocardiographic change, however, is unlikely to be explained by this hypothesis. The distribution of T wave inversions was not that of pulmonary embolism, and the swelling of the right arm was thought to be due to lymphangitis rather than venous thrombosis.

(c) *T Wave Inversion due to Thrombosis or Subintimal Haemorrhage in Coronary Blood Vessels.* Some venoms, such as that of Russell's viper, contain both coagulants with a predominantly local

effect and anticoagulants (haemorrhagins) with more systemic action. This could result in thrombosis and/or subintimal haemorrhage in the coronary arteries resulting in their occlusion. Such an explanation, however, is untenable in our case as discussed under (a).

(d) *T Wave Inversion due to Pericarditis.* There was no pericardial friction rub, the changes were too early, and the ST segment configuration was not that of pericarditis.

(e) *T Wave Inversion due to Hypoglycaemic Effect of Venom.* Certain snake venoms, e.g. black snake (*Walterinesii Eegypted*) toxin, have been shown to produce hypoglycaemia (Mohamed and Zaki, 1959). These authors have advanced the hypothesis that the inhibition of suprarenal cortex and the stimulation of parasympathetic system, produced by the snake toxin, lead to hypoglycaemia. No blood sugar estimations were done in our patient and we are not aware of any blood sugar studies in relation to adder bite.

(f) *T Wave Inversion by Direct Effect of Venom on Myocardium.* In the absence of any fall of blood pressure or any evidence of myocardial infarction, pulmonary embolism, or pericarditis, one must examine the likelihood of a direct effect of the venom on the myocardium. Kellaway and Trethewie (1940) could produce T wave inversion, AV block, and eventual ventricular fibrillation by the intravenous administration of cobra venom to a cat. Our evidence for suggesting that the abnormalities are due to direct toxic effect of the venom is mainly derived by a process of exclusion. However, slight generalized delay in conduction as shown by the difference in the duration of QRS complexes in the earlier electrocardiograms compared with the last one on 23.7.65 (Fig. 2) suggests a direct toxic effect of the venom on the myocardium.

Summary

A case is described of a boy who had an abnormal electrocardiogram following an adder bite. The abnormality was present before the administration of antivenomous serum and persisted for more than two weeks. Arguments are presented for this being due to a direct toxic effect of adder venom on the myocardium.

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References

- Arneil, G. C., and MacLaurin, J. C. (1961). Case of adder-bite with thrombosis of the saphenous vein. *Brit. med. J.*, 1, 1587.
- Askanas, Z. (1959). Myocardial infarct in shock produced by snake bite. (In Polish.) *Pol. Tyg. lek.*, 14, 1528.
- Brown, R., and Dewar, H. A. (1965). Heart damage following adder-bite in England. *Brit. Heart J.*, 27, 144.
- Kellaway, C. H., and Trethewie, E. R. (1940). The liberation of adenyil compounds from perfused organs by cobra venom. *Aust. J. exp. Biol. med. Sci.*, 18, 63.
- Mainzer, F., and Krause, M. (1940). The influence of fear on the electrocardiogram. *Brit. Heart J.*, 2, 221.
- Manson-Bahr, P. (1957). Snake-bite. *Brit. med. J.*, 2, 468.
- Mohamed, A. H., and Zaki, O. (1959). The effect of the black snake toxin on blood glucose levels. *Indian J. med. Res.*, 47, 522.
- Morton, T. C. (1960). Adder-bites in Cornwall. *Brit. med. J.*, 2, 373.
- Reid, H. A., Thean, P. C., Chan, K. E., and Baharom, A. R. (1963). Clinical effects of bites by Malayan viper (*Ancistrodon rhodostoma*). *Lancet*, 1, 617.
- Rocha e Silva, M., Beraldo, W. T., and Rosenfeld, G. (1949). Bradykinin, a hypotensive and smooth muscle stimulating factor released from plasma globulin by snake venoms and by trypsin. *Amer. J. Physiol.*, 156, 261.
- Walker, C. W. (1945). Notes on adder-bite (England and Wales). *Brit. med. J.*, 2, 13.