Myocardial infarction after probable black mamba envenomation

A case report

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Summary

A probable direct toxic effect of elapid venom on the myocardium as evidenced by transient ECG changes and transiently elevated cardiac enzyme levels is described. The patient made good recovery with no residual cardiac damage.


Of all snakes indigenous to South Africa, the black mamba (Dendroaspis polyplepis polyplepis) is probably the most dangerous. The lethal nature of the venom is demonstrated by the 100% mortality in the 7 cases reported by Visser and Chapman. Recent reports have documented survival with adequate use of antivenom and respiratory support.

A patient who, although he developed cardiopulmonary problems with myocardial damage, survived probable black mamba envenomation is reported.

Case report

A 24-year-old black man was admitted to the Casualty Department of King Edward VIII Hospital, Durban, on 5 July 1985. The accompanying person reported that the patient had been bitten by a snake ½ hours earlier (at 14h00) while walking along the river bank. A tourniquet had been applied immediately and he was brought to hospital.

On examination he was in respiratory distress, frothing at the nose and mouth, conscious but unable to speak. He was not cyanosed or shocked. The pulse rate was 120/min and the blood pressure 110/70 mmHg. He was given polyvalent antivenom 80 ml together with hydrocortisone intravenously in the Casualty Department and admitted to the Emergency Unit where he vomited when suctioned. Seven hours later he was found to be flaccid, cold and sweating and at 21h10 developed cardiorespiratory arrest. He was intubated, resuscitated and transferred to the Respiratory Department and admitted to the Emergency Unit where he vomited and was intubated. Seven hours later he was found to be flaccid, cold and sweating and at 21h10 developed cardiorespiratory arrest. He was intubated, resuscitated and transferred to the Respiratory Intensive Care Unit (ICU) where he was immediately put on to a ventilator.

In the ICU the patient was found to be unconscious with a Glasgow coma scale rating of 4. The blood pressure was 130/100 mmHg and the pulse rate 112/min, regular. Two fang marks were present on the left forearm, which was slightly swollen. The chest was clear and heart sounds were normal. He had brisk reflexes but the plantar response was flexor. Fasciculation was present. Approximately 20 hours after admission there was a drop in blood pressure to 64/44 mmHg and ST-segment elevation with Q waves appeared in the precordial leads (V1-6) of the ECG. The blood pressure was stabilised at 120/80 mmHg after a fluid infusion. Two days later the patient developed sinus bradycardia at 45/min. This and two further episodes responded to intravenous atropine. The ECG still showed mild ST-segment elevation. No ventricular arrhythmias were noted on ECG monitoring in the ICU. On the 5th day the ST-segment elevation had subsided and the patient's condition was thereafter stable.

Initial ward urinalysis was normal but subsequently glycosuria was detected (blood glucose level 29.6 mmol/l). Upon stopping the 10% dextrose infusion the blood glucose level fell to 10.4 mmol/l. The serum creatinine level was 1052 U/l, y-glutamyl transferase 47 U/l and aspartate aminotransferase 591 U/l. The creatine kinase level fell to 182 U/l the next day. The serum sodium level was 132 mmol/l, serum potassium 3.7 mmol/l, serum chloride 97 mmol/l, serum bicarbonate 19 mmol/l, serum urea 7.0 mmol/l serum creatine 168 µmol/l and serum urate 0.46 mmol/l. Upon rehydration the serum creatinine level fell to 88 mmol/l and the urate level to 0.29 mmol/l. The haemoglobin value also fell from 17.8 to 13.3 g/l and the white cell count from 34.5 x 10⁹/l on admission to 15.4 x 10⁹/l 2 days later.

Attempts to wean the patient off the ventilator were initially unsuccessful, necessitating tracheostomy. He was extubated 7 days after admission and discharged on the 13th day with full recovery of power and function.

Definitive identification of the snake was not possible. It was thought to be a black mamba by the group of people accompanying the patient as well as by the patient himself. He was able to identify the snake immediately on being shown the Visser chart, pointing out that it was 2.5 - 3 m long and greyish black in colour. He also said that he had been bitten twice by the snake at the same site in quick succession as his arm brushed the boughs of a tree overhanging the river bank.

Discussion

The point made by Harvey² needs to be re-emphasised: neurotoxicity with envenomation by elapid snakes is a medical emergency that must be managed by elective intubation and ventilation if necessary. In this instance the patient was fortunately observed in the emergency admissions ward and resuscitated after a cardiorespiratory arrest. In the 7 cases of elapid bites recorded by Blaylock³ neurotoxicity was manifest within 10 minutes in 5 patients. It may, however, result in respiratory arrest several hours later and weakness may persist for several days, as in our patient. It is likely that further administration of antivenom soon after admission may have reduced the degree and the duration of paralysis.

Chest pain was documented with elapid bites in 2 out of 7 cases reported by Blaylock and in a medical colleague treated by Saunders.⁴ There is no record of the ECGs in these cases. Cardiotoxicity manifest by a thready pulse, arrhythmia or hypotension has not been documented with South African elapid snake bites. Furthermore, myocardial infarction has been described only with bites by the viper⁵ and the adder,⁶ the basis in these instances being a tissue toxin, directly affecting the myocardium. Prolonged hypotension following shock as well as hyperfibrinogenaemia with coronary thrombosis have been proposed as additional factors in these instances.
Subarachnoid haemorrhage

A report of 2 cases

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Summary

Two patients admitted to the medical intensive care unit with subarachnoid haemorrhage, one with the provisional diagnosis of 'sick sinus syndrome' the other as having an acute myocardial infarction complicated by ventricular tachycardia, are described and the arrhythmias associated with this condition are reviewed.

Case reports

Case 1

A 34-year-old man was admitted to the medical intensive care unit (ICU) of J. G. Strijdom Hospital, Johannesburg, suffering a syncopal episode while exercising. The only other relevant history was a similar syncopal episode, 6 months previously, while driving a motor car.

On examination he was disorientated and had a blood pressure of 100/70 mmHg and a pulse rate of 30/min. No neck stiffness was present and there were no localising neurological signs. The rest of the physical examination was normal.

An ECG showed sinus arrest with a slow nodal rhythm of 30/min (Fig. 1a). After the intravenous administration of atropine 0,5 mg the heart reverted to a sinus rhythm. A further episode of bradycardia was treated similarly.

Approximately 8 hours after admission he complained of a severe generalised headache. Clinical examination revealed meningism and mild bilateral 6th nerve palsies. Fundoscopy was normal. At lumbar puncture the cerebrospinal fluid (CSF) pressure was 340 mmH₂O and the fluid was grossly bloodstained.

A four-vessel cerebral angiogram 7 days later demonstrated diffuse arterial spasm and an anterior communicating artery aneurysm. The aneurysm was successfully ligated.

Case 2

A 70-year-old woman was admitted to a general medical ward with a 1-day history of confusion, headache, nausea, vomiting and loose stools. Clinical examination revealed a temperature of 38°C, dehydration and terminal neck stiffness. The latter was attributed to osteo-arthritis. An ECG on admission showed pathological Q waves in leads II, III and aVF compatible with the history of a myocardial infarction 7 years previously.

In the ward she developed sustained haemodynamically stable monomorphic ventricular tachycardia, which was electrically cardio-