Cantharidin poisoning with neurological complications

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Summary

Two cases of cantharidin poisoning are described in which the patients developed the well-known complications of acute renal failure and pseudopolycythaemia. In addition both developed a Guillain-Barré type of flaccid paralysis but spontaneous reversal occurred. Cantharidin poisoning may be responsible for some of the unexplained neurological presentations in Black South Africans.

Cantharidin poisoning is relatively common in South Africa because of the widespread use of cantharides by Black herbalists. Cantharidin is a crystalline substance obtained from beetles, approximately 165 species of which have been described in this country. The insects are known as 'blister beetles' and as the 'Spanish fly' (Lyctus vesicatoria), probably the most familiar, although in South Africa the commonest is the 'sergeant beetle' (Mylabris ocultata). Cantharidin is a very potent irritant often used by herbalists as an abortifacient in females and as an aphrodisiac in males.

The acute gastro-intestinal and urinary tract symptoms are well known, as are the acute renal failure and pseudopolycythaemia that may follow in some patients. Since neurological complications have not previously been documented, 2 patients who both developed the classic picture as well as a Guillain-Barré-like syndrome are presented.

Case report

Case 1

A 33-year-old Black man drank a herbalist’s potion containing cantharidin 0,08 g/100 ml for vague back pains; he had previously been well. He was admitted to hospital 4 days later with the typical picture of cantharidin poisoning, namely vomiting and diarrhoea followed by generalized body pains, headache, anorexia and a decreased urine output. Macroscopically, haematuria and melena were absent. The patient also complained of grittiness of the eyes and complete loss of taste. At this stage no other cranial nerves were involved and his motor system was normal. On the 6th day he complained of numbness in his hands and feet and examination showed a ‘glove and stocking’ peripheral neuropathy to light touch, pinprick and temperature. Proprioception and vibration sense were initially spared. The ankle jerks were depressed and the reflex plantar responses were elicited. In the upper limbs the tendon reflexes were brisk but symmetrical. The following day progressive distal weakness developed in the lower limbs and the knee reflexes became depressed. A diagnosis of acute Guillain-Barré syndrome was then considered. Ten days after ingestion of the poison the patient developed bilateral lower motor neuron lesions of the facial nerves, on the right side first. He also had difficulty in swallowing but never developed respiratory problems. The weakness spread proximally in the lower limbs, the upper limbs became weaker distally and he was unable to walk. There was evidence of a partial ophthalmoplegia affecting the 3rd, 4th and 6th cranial nerves bilaterally.

Three weeks after admission the patient began to improve slowly but steadily, initially with resolution of the facial palsy and return of the pupils, unequally at first, to normal. Improvement of the weakness and sensory loss followed.

Investigations included a lumbar puncture which showed a normal pressure, 4 lymphocytes, 4 polymorphonuclear leucocytes, 2880 erythrocytes, protein 1,28 g/l, glucose 4,5 mmol/l and chloride 131 mmo1/l. No organisms were isolated. A Paul-Bunnell (Monospot) test, screening for porphyrins, tests for hepatitis A and B antigens and viral studies were all negative. There was no vitamin or iron deficiency. The total creatine kinase level was raised to 1052 U/l (normal 0 - 90 U/l) and the creatine kinase MB fraction was 34 U/l (normal 0 - 16 U/l). The serum aldolase level was normal. Electromyography of the left extensor digitorum brevis revealed no denervation activity but a very reduced interference pattern on volitional activity. The nerve conduction of the left peroneal muscle showed prolonged distal latency and a markedly slowed conduction velocity.

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indicative of a peripheral neuropathy on a demyelinating basis which was compatible with the diagnosis of Guillain-Barré syndrome.

Case 2
A 21-year-old Black man drank half a cup of cantharidin (0.04 g/100 ml). This was given to him by a herbalist for constipation (not the same herbalist as in the first case). On admission, the patient had diarrhoea, vomiting, abdominal pain, generalized body pains, anorexia and lethargy. He also had oliguria, macroscopic haematuria, melena and loss of taste, but no dysuria or priapism. The blood pressure was 130/80 mmHg and the pulse rate 56/min, and his temperature was 37°C. Hydration was good, the conjunctivae were injected and the pupils were equal and reacted to light. The abdomen showed moderate epigastric tenderness but no guarding, and also bilateral renal angle tenderness. The patient’s urine contained numerous white blood cells, a few red blood cells and hyaline granular casts; protein was present (3+). The patient’s haemoglobin concentration and packed cell volume were also raised, with a concomitant drop in the reticulocyte and leucocyte counts, but there was no evidence of haemolysis. He developed a left-sided lower motor neuron lesion of the facial nerve about 2 weeks after drinking the poison. The lesion improved a week later. There was also a mild sensory peripheral neuropathy in both lower limbs which did not progress. The cerebrospinal fluid contained 1 polymorphonuclear leucocyte, no lymphocytes, 300 red blood cells, protein 1 g/l, glucose 3.5 mmol/l and chloride 124 mmol/l. The patient was discharged after 4 weeks, having almost fully recovered.

Comment
It is well known that cantharidin intoxication can cause renal failure due to acute tubular necrosis.1 Pseudopolythaeinemia has also been described.2–4 However, no mention has been made of concomitant thrombocytopenia or leucopenia. The finding of fixed dilated pupils has previously been noted,5 but the development of a Guillain-Barré-like syndrome with reversible cranial nerve palsies and peripheral neuropathy has not been described. The relationship between cantharidin and neurological complications may be immunological, but this is purely speculative. It is felt that doctors should be made aware of the neurological complications which may possibly develop some time after intoxication with cantharidin when the well-known manifestations have disappeared.

REFERENCES

Uterine hyperstimulation and rupture after induction of labour with prostaglandin E2
Case reports
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Summary
Three patients are described in whom hyperstimulation of the uterus followed induction of labour with prostaglandin E2 tablets administered either orally or vaginally. All 3 patients were grand multiparas. Two patients sustained a ruptured uterus. It is concluded that the dangers of inducing labour in grand multiparas by means of this technique are no less than those associated with amniotomy and infusion of oxytocin. Recommendations concerning dosage schedules are made.


There has been a rapid increase in the use of prostaglandin E2 (PGE2) tablets administered either orally or vaginally to induce labour in patients near term. Most obstetricians using this technique recognize the advantages as regards ease of administration and supervision, while some consider the risk of overstimulation of the uterus to be minimal.1 The case reports below draw attention to the dangers of hyperstimulation of the uterus after the administration of PGE2 (either orally or vaginally), particularly in women of high parity.