reflexia and bilateral extensor plantar reflexes. Power in the legs was grade 2 and remained so during the clinical phase of tetanus, with gradual recovery subsequently. No sensory or sphincteric abnormality was observed during the illness.

The patient was given 20 000 IU antitetanus serum intramuscularly after a sensitisation test. Injections of diazepam, chlorpromazine, and phenobarbitone were used to control the spasms, with adequate parenteral and oral feeding. He made an uneventful recovery from tetanus, and about one month after onset plain radiography of the thoracolumbar spine, cerebrospinal fluid examination (including a Venereal Disease Research Laboratory test), and myelography showed nothing abnormal. He still had the facial palsy but could stand with support. Hyperreflexia and extensor plantar responses were still evident. At follow up two months after discharge the facial palsy was barely discernible and he could walk unaided. Mild hyperreflexia with equivocal plantar responses were elicited. Follow up at one year showed no neurological deficit.

Comment

This patient showed the characteristic features of tetanus, and 12 days before onset he had injured his foot. In view of the paraplegia lathyrism was considered; lathyrism is common in this region.[1] The patient denied eating seeds of *L sativus*, there was no lathyrism in the family, the paraplegia resolved, there was a facial nerve palsy, and he had tetanus—all going against the diagnosis of lathyrism.

Clostridium tetani produces two toxins, tetanospasmin and tetanolysin. Tetanospasmin interferes with inhibition within the central nervous system and produces the excessive motor activity which characterises the disease. It may also produce some of the autonomic dysfunction, especially the sympathetic overactivity,[2] which may occur in severe cases. Paralysis, however, is unusual unless the wound is on the face, when cranial nerve palsies, notably of the ipsilateral facial and oculomotor nerves, may occur. Paraplegia may occur if the tetanic spasms cause compression fracture of the spine, but our investigations eliminated this and other compressive causes of paraplegia. It appears that the paraplegia is an unusual effect of tetanus as described by Bahemuka.[3] The facial nerve palsy must also be attributed to tetanus, though it is unusual for this to occur when the wound is not on the face.

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Unusual strychnine poisoning and its treatment: report of eight cases

Strychnine is now used rarely and poisoning is unusual. Intoxication is usually accidental in children or suicidal in adults.[1] Treatment is directed towards control of convulsions and prevention of asphyxia, either by a short-acting barbiturate and muscle relaxant combined with artificial ventilation[2] or by the more recently successful use of intravenous diazepam alone.[3 4] We describe eight cases of unusual strychnine poisoning, three of which were treated with intravenous diazepam and four by artificial ventilation.

Case reports

Eight young adults sniffed quantities of strychnine in the mistaken belief that it was cocaine. All developed toxic symptoms within 30 minutes and were admitted to two Dublin hospitals.

Five patients were admitted to St Laurence's Hospital. The man who had taken the largest quantity developed convulsions within five minutes of inhalation. These became sustained, and he was in cardiopulmonary arrest on admission. Asystole was converted to ventricular fibrillation by intravenous injection of 2 ml of 1:1000 adrenaline and 10 ml 10% calcium gluconogalactogluconate. Cardioversion resulted in a supraventricular tachycardia, which was converted to sinus rhythm by 10 mg sotalol given intravenously. Spontaneous respiration was not restored, and artificial ventilation was instituted after muscle relaxation by suxamethonium. Urine analysis showed large amounts of strychnine.[5] He remained deeply comatose but was free of convulsions. Nine hours after admission he had a fatal cardiac arrest.

Three of the four remaining patients complained of pain and stiffness in the paravertebral muscles within 30 minutes of inhalation. Convulsions characterised by risus sardonicus, opisthotonus, flexor spasm of upper limbs, and extensor spasm of lower limbs, occurred with minimal sensory stimuli. They were treated in a quiet dark room. Diazepam 10 mg intravenously controlled convulsions and was repeated one hour later in two patients in whom convulsions recurred. Diazepam was continued in an oral dose of 5 mg six hourly, and the patients remained free of convulsions. Hyperaesthesia and hyperreflexia with associated paravertebral myalgia persisted for 48 to 72 hours. Diazepam was discontinued 72 hours after admission. Serum creatine phosphokinase activity was raised in all patients (mean 1980 IU/l; normal < 130 IU/l), and estimation of isoenzymes showed raised muscle-type (MM) creatine kinase activity, indicating damage to skeletal muscle during the convulsions. Urine analysis showed moderate quantities of strychnine.

The remaining patient was only mildly affected. He complained of paravertebral pain and stiffness. Tendon reflexes were brisk but no convulsions occurred, and specific treatment was not prescribed. Urine analysis showed traces of strychnine.

Three patients were admitted to the Charitable Infirmary. All developed symptoms within 30 minutes of inhaling strychnine and were having intermittent convulsions on admission. Each was given intravenous diazepam 10 mg, phenoperidine 2 mg, and suxamethonium 100 mg. Artificial ventilation using Cape Waine ventilators was instituted and was continued using pancuronium for muscle relaxation and phenoperidine and diazepam for sedation. Convulsions tended to recur as muscle tone recovered but were readily controlled with pancuronium. Urine analysis showed moderate amounts of strychnine in each case. After 36 hours, when strychnine was no longer detected in the urine, artificial ventilation was discontinued. No further convulsions occurred. All patients had raised serum creatine phosphokinase activity (mean 2797 IU/l; normal < 170 IU/l). Physical examination showed no abnormality after 48 hours.

Comment

This report highlights a mode of strychnine poisoning not previously described. Strychnine is now rarely used but may be stocked by some retail pharmacies, so this type of accident might possibly recur. It is not known how these patients acquired the strychnine, which was inhaled apparently in mistake for cocaine at a party.

Apart from the patient in whom poisoning was fatal and the most minimally affected patient, who recovered without specific treatment, the remaining six patients were comparable in terms of degree of intoxication and urinary strychnine concentrations. In these patients two different therapeutic regimens were used with equal success. Treatment with intravenous diazepam in a quiet darkened room has the advantage of being simpler than artificial ventilation, but if diazepam alone fails to control convulsions artificial ventilation should be instituted.

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