SHORT REPORTS

✓ Syngamus in a West Indian

With extensive international travel and the presence of a large immigrant population in England, it is important for doctors to be aware of the possibility of rare causes for common complaints in patients from overseas.

Case report

In March 1975 a 34-year-old businesswoman from Dominica, West Indies, was admitted to the Princess Margaret Hospital, Dominica, for investigation of a chronic cough. She had travelled extensively between the Windward and Leeward group of Caribbean Islands. Three months before admission to hospital she developed an irritating cough, associated with a "tight" sensation in her chest and productive of thick mucoid sputum. The symptoms became more severe and were accompanied by episodic "wheezing" spells. On occasions during the course of her illness she claimed to have coughed up "live worms." There was no associated chest pain, fever, haemoptysis, or weight loss.

She was an obese apprehensive black woman who had a blood pressure of 150/80 mm Hg, pulse rate of 90 beats/minute, and no fever. There was no clinical evidence of bronchospasm. The heart and other systems were normal. Haemoglobin concentration was 12 gm/dl. The leucocyte count was $6 \times 10^9/l$ with 4% eosinophils. The erythrocyte sedimentation rate was 20 mm in one hour. The chest radiograph was normal. Repeated stool microscopy was negative for parasites. Examination of one of the sputum specimens expectorated after a severe bout of coughing showed a pair of copulating syngamus worms producing a characteristic Y shape (see figure) together about 20 mm in length.

She recovered completely after treatment with thiabendazole.

Syngamus showing Y-shaped appearance of the permanent copulatory position, and buccal capsules for attachment to host.

Comment

The genus *Syngamus* (gapeworms) commonly produce "the gapes" in birds and domestic fowl, a disease characterised by spasms of dyspnoea and ending in death from asphyxia due to disease of the trachea and bronchi.

Human infection is rare. The first reported case in English literature appeared in 1913,¹ and since then only a few isolated cases have been documented, particularly from the Caribbean area.^{2,3} As recently as 1974, Basden *et al*⁴ described for the first time gapeworm infestation in a patient from Britain, and noted that the patient, a white doctor, had visited the West Indies on holidays in 1970.

Syngamus infection may be difficult to diagnose even when the organism is producing symptoms, and most reports have highlighted the unexpected manner in which the diagnosis was made. St John *et al*⁵ in 1929 found syngamus ova in the sputum; Basden *et al* aspirated the worm on bronchoscopy; and in our patient we identified the adult copulating parasites in the sputum after a severe bout of coughing.

At the University Hospital in Jamaica no case of gapeworms has

yet been recorded in man, perhaps because of a low general level of suspicion. This case report is intended to alert doctors dealing with people from the West Indies and elsewhere to considering and excluding this unusual parasite when presented with respiratory problems defying more conventional diagnoses.

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Paraplegia as a result of lightning injury

Neurological complications of lightning injury were reported as early as 1934.¹ We report a patient with paraplegia resulting from lightning injury.

Case report

A 25-year-old man was struck by lightning while climbing a mountain. He was unconscious for 30 minutes. He presented at hospital with electrical flash burns over his occiput and neck, across the shoulders, round the right side of the chest, and down the left leg. He also had frostbite of his fingers. The initial neurological examination was normal. Thirty-six hours later he developed complete paralysis of both legs without bladder or bowel disturbance. The upper limbs were neurologically normal and the cranial nerves were intact. He had a pyramidal lesion affecting both legs, with pronounced ankle and patellar clonus and bilateral extensor plantar response. There was profound weakness of hip, knee, and ankle flexors. The abdominal reflexes were absent. There was some loss of pin-prick sensation below the groin and definite loss of vibration in the lower limbs together with some impairment of joint and position sense. There was considerable weakness of the erector spinae muscles. Investigations showed haemoglobin 12.5 g/dl and white cell count $16.8 \times 10^9/l$ ($16\,800/mm^3$) with a pronounced shift to left. Sequential multiple analysis results (SMA 12) were within normal limits. Radiographs of the skull, cervical spine, and right ankle were normal: radiographs of hands and feet showed much soft tissue swelling.

The burns of the occiput and left ankle were treated with skin grafts. Six months after the initial injury there was still some weakness of both legs, particularly of the hip and knee flexors. Ankle clonus persisted but touch, vibration, and joint and position sense had returned to normal.

Comment

Lightning injuries are uncommon in Britain. About 12 people annually are struck.² Contrary to popular belief, the injuries do not always result in death. Complications are cutaneous burns, loss of consciousness, loss of memory, and subdural and subarachnoid haemorrhage.³ Loss of consciousness with retrograde amnesia and a reversible flaccid paralysis, thought to be peripheral in origin, are encountered most commonly.¹ Cerebral oedema, generalised convulsions, and hemiplegia have all been reported, as well as extra

pyramidal signs.⁴ Delayed myelopathy has been described as a complication of severe electrical burns.⁴ Spastic paraplegia⁵ and quadriplegia have been recorded secondary to electrical injury but not after lightning injury. We ascribe the delay of 36 hours before the onset of symptoms in our patient to oedema of the spinal cord.

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Amanita phalloides poisoning treated by early charcoal haemoperfusion

Treatment of *Amanita phalloides* poisoning remains disappointing. Prednisone, thioctic acid, vitamin C, penicillin, cytochrome-C, and haemodialysis alone or in combination have been used with variable success.¹⁻² Perfusion of blood over coated activated charcoal has also been tried in a few cases, but always after the onset of acute liver failure.³⁻⁴ We have used charcoal haemoperfusion as early as possible after the ingestion of *A phalloides* to try to fix the toxins before they caused hepatic or renal damage.

Patients, methods, and results

Four men and four women, aged 16 to 55, were admitted to the emergency room. All had eaten mushrooms 16 hours earlier. Seven had eaten more than three—a supposedly lethal quantity. They all had severe gastrointestinal symptoms. There were no clinical or biochemical signs of hepatic failure (serum alanine transaminase (serum ALT; SGPT) concentration <40 IU/l, blood ammonia <45 µg/100 ml (32 µmol/l)). The mushrooms remaining after the meal were identified as *A phalloides*. All the patients except the one who had eaten only a small amount were treated by haemoperfusion. In three patients this was started immediately. In the remainder it was begun a few hours after an initial dialysis. All patients were given penicillin 1 000 000 U hourly and vitamin C 1 g six-hourly intravenously. Each haemoperfusion lasted three hours, and they were continued daily until there was clinical and biochemical improvement. One patient had five haemoperfusions, one had four, three had three each, and two patients had two each.

There were no technical problems. Platelet counts dropped during most of the perfusions to a mean of 80% of the starting value. The lowest count observed after one hour of haemoperfusion was $76 \times 10^9/l$ ($76\,000/mm^3$). White blood cell counts did not change. The clinical course in each case was uneventful. Vomiting and diarrhoea subsided within 24 to 36 hours. No clinical signs of liver failure were observed, even in the two patients with high ALT concentrations. Four of the patients left hospital after three days. The gastrointestinal symptoms in the patient who was not haemoperfused were more severe and lasted longer than those in any of the others. Serum ALT concentrations responded in two different ways (table). In two patients they rose to 1920 and 310 IU/l on the third day. In the remainder there were only slight and transient rises. Changes in aspartate transaminase (AST; SGOT) concentrations closely paralleled those of ALT. In contrast, in all eight patients there were sharp changes in blood urea nitrogen (BUN) and blood ammonia concentrations. BUN fell from 21.5 ± 1.1 mg/100 ml (7.6 ± 0.4 mmol/l) to reach its lowest level of 3.5 ± 0.8 mg/100 ml (1.2 ± 0.2 mmol/l) (mean $\pm$ SEM) on day four, while blood ammonia concentrations rose from 34.9 ± 2.9 µg/100 ml (25.9 ± 2.1 µmol/l) to 56 ± 3.9 µg/100 ml (42.1 ± 2.7 µmol/l). These changes, suggestive of impaired hepatic synthesis activity,

Serum alanine transaminase (serum ALT; SGPT) concentrations (IU/l) in eight patients with mushroom (*A phalloides*) poisoning

Day No	Case No:								Mean $\pm$ SEM
	1	2	3	4	5	6	7	8	
1	51	15			10		5		
2	179	180	33	8	18	7	6	8	55 ± 27
3	1920	312	17	24	18	6	6	8	289 ± 236
4	990	142	14	5	9	5	4	4	147 ± 122
5	600	105	31	10	10	3	6	19	98 ± 73
6	460	76	47	29	10	8	12	11	82 ± 55
7	360	65	48	33	8	14	14	4	68 ± 42
8	265		32			30	13		
9	155								
10	105	40	22		30	27	14		40 ± 14
11	60	20							

were confirmed by a fall in serum cholesterol to 116 ± 11 mg/100 ml (3.0 ± 0.2 mmol/l) and a prolonged thromboplastin time (quick level 55% of normal value) four days after ingestion. The following serum concentrations remained unchanged: alkaline phosphatase, bilirubin, lactic and pyruvic acid, fibrinogen, coagulation factors V, VII, and X, calcium, and creatinine. Follow-up examination two months later showed no sequelae in any of the patients.

Comment

Since there was no way of measuring the dose of toxin we could not know whether it was lethal. But all except one patient had eaten more than three mushrooms (>50 g), which is generally accepted as a lethal dose. The wife of one patient (case 2), who had also taken the meal, was treated in another hospital by exchange transfusion and peritoneal dialysis. She was in hepatic coma for eight days, with serum ALT concentrations of 4000 IU/l, and remained in hospital for more than three months. Her husband, who had eaten the same quantity and was treated by early haemoperfusion, left hospital after six days. Seeger and Bartels have since reported that in-vitro charcoal haemoperfusion removes alpha-amanitine from water and protein solutions.⁵ Our cases provide strong circumstantial evidence that charcoal can effectively remove toxins from the blood even 24 hours after eating *A phalloides*. Further clinical trials of charcoal haemoperfusion seem justified.

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Effect of 24,25-dihydroxycholecalciferol on calcium absorption in proximal small intestine in uraemia

In patients who have undergone nephrectomy and patients with chronic renal failure the available 25-hydroxycholecalciferol is preferentially converted to 24R,25-dihydroxycholecalciferol (24R, 25(OH)₂D₃), the biological activity of which is uncertain, rather than to 1α,25-dihydroxycholecalciferol (1,25-(OH)₂D₃), the most potent hormonally active form of vitamin D₃. Despite the generally suppressed production of both 1,25-(OH)₂D₃ and 24R,25-(OH)₂D₃, which is reflected by, respectively, their absence¹ or low plasma