Letters to the Editor

Ergotism: an infrequent aetiology of intermittent claudication

Sir,

Ergotism is a rare disorder which usually develops following the chronic intake of ergot derivatives. It involves the peripheral vascular system and the gastrointestinal tract.¹ We describe a patient with symptoms of intermittent claudication which were ascribed to ergotamine abuse.

A 39-year-old woman was admitted to the hospital with an eight-month history of progressive intermittent claudication. During the previous year her usual episodic migraines had been turning into a chronic daily headache. She had been taking 4 mg of ergotamine tartrate rectally each day. No pulses were detected in the lower limbs, and bruits were heard over the femoral arteries. The remainder of the history and clinical examination were unremarkable.

An angiogram showed a marked reduction of the lumen of medium-sized vessels of the extremities (figure 1). Both echocardiographic and carotid ultrasonography–Doppler studies revealed no abnormalities. Routine laboratory assays were normal, including erythrocyte sedimentation rate, immunoglobulins, C3, C4, CH₅₀, fibrinogen, prothrombin time and activated partial thromboplastin time. VDRL, rheumatoid factor, lupus anticoagulant, anticardiolipin, antinuclear and antineutrophil-cytoplasmic antibodies were negative. Ergotamine was discontinued and 30 mg daily of oral nifedipine were given. Within 24 hours, femoral and tibial pulses were present. Six months later she was non-symptomatic and a repeat angiogram demonstrated a normal caliber of the femoral and popliteal arteries (figure 2).

Ergot abuse is a potentially serious condition, generally of iatrogenic origin. Early diagnosis allows an appropriate and successful treatment. It must be considered in young adults with peripheral vascular disease, diarrhoea, vomiting or chronic daily headache. Other vascular sites are less frequently involved.² It may mimic the clinical and radiologic features of an arteritic process.³

Figure 1 Angiogram showing vasospasm in the femoral and popliteal arteries

Figure 2 Arteries of a normal caliber six months after discontinuing the ergotamine tartrate

A direct effect on the arterial smooth muscle via alpha-adrenoceptors leading to vasoconstriction has been proposed as the pathogenic mechanism. Rectal administration makes absorption irregular and favours a toxic effect by avoiding circulation through the liver.¹

Discontinuation of the ergotamine and other aggravating factors (tobacco, caffeine, oral contraceptives, beta-blockers) should be the first therapeutic measures. There are no trials suggesting that a vasodilator provides a better outcome. Intravenous nitroprusside, nitroglycerin and prostaglandins, and oral captopril, prazosin and nifedipine have been administered successfully but on an empiric basis.^{2,3} The use of prophylactic heparin in all cases is advocated by some authors.⁴ Hospital admittance is advisable to monitor the degree of ischaemia. Radiologic abnormalities may still be present, even weeks after the first signs of clinical recovery have been observed.

The most important preventive measure is the correct use of ergot preparations. They should not be prescribed to patients with liver or renal failure, peripheral vascular disease, ischaemic heart disease, hyperthyroidism, Raynaud phenomenon, thrombophlebitis, tobacco use or pregnancy. Migraineurs should be warned not to take more than 4 mg daily and 10 mg weekly of ergotamine tartrate.^{1,2} Ergot does not provide more clinical benefit than placebo in the most common headache in the general population. Other analgesics are thus more suitable for patients with this tension-type headache.⁵

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Candida peritonitis treated with liposomal amphotericin B

Sir,

We would like to report a case of *Candida* peritonitis successfully treated with liposomal amphotericin B.

A 14-year-old boy with severe ulcerative colitis responded initially to medical treatment with coliform enemas, prednisolone, and mesalazine. However, one month later he was admitted with gross rectal bleeding and generalised peritonitis, and underwent total colectomy and ileostomy, leaving the rectal stump. He was treated with cefuroxime and metronidazole, and hydrocortisone was continued. Postoperatively, he developed an obstruction at the ileostomy site which was relieved by a second laparotomy, during which a gastronomy tube was inserted because he had been unable to tolerate a nasogastric tube.

He continued to have gastric drainage, although the ileostomy was functioning well, and he was commenced on total parenteral nutrition. He remained on cefuroxime, metronidazole and hydrocortisone, with the addition of topical antifungals for oral candidiasis. Eight days later, he developed generalised peritonitis, and underwent a further laparotomy. No perforation was found, only small bowel adhesions. In freeing these, the serosa was damaged and a 10-cm length of small bowel resected. Routine histological examination revealed serosal adhesions coated with an inflammatory exudate, with colonies of fungal hyphae and yeast forms. A diagnosis of fungal peritonitis was made, and *Candida albicans* was subsequently isolated from a rectal swab, gastronomy fluid, pus from the laparotomy wound, a catheter specimen of urine and a mouth swab. Blood cultures remained negative.

In view of his precarious state and continuing hypokalaemia, liposomal amphotericin B (AmBisome) was started at a dose of 3 mg/kg/day. During the next three weeks he continued to improve, and he was discharged home on a 10-day course of oral fluconazole, 200 mg daily.

Candida peritonitis is a rare complication of gastrointestinal surgery, with a reported mortality of 34%,¹ and therefore a high index of clinical suspicion is essential. Deep candidal infection has been reported as being more likely to occur in patients with a history of multiple laparotomies, prolonged antibiotic treatment, small bowel operations, parenteral nutrition² or gastronomies.³ The immuno-

suppressive treatment he was on for ulcerative colitis was a significant risk factor for deep-seated fungal infection and, in retrospect, the diagnosis could perhaps have been suspected earlier.

Prompt treatment and early diagnosis is critical, but as this case illustrates, it can be very difficult. Apart from oral candidiasis, there was nothing to raise the suspicion of a fungal peritonitis. Without the third laparotomy, the diagnosis would not have been considered until *Candida* was demonstrated from peripheral sites seven days later.

Serodiagnosis of candidal infection can be difficult. *Candida* antigen detection is specific, and can help distinguish colonised from systemically infected patients, but sensitivity can be low.⁴ Confirmation of systemic candidiasis requires isolation from blood cultures or an otherwise sterile site except urine, or histological evidence of yeast or mycelial forms in tissues from an 'at risk' patient,⁵ which can take time. Amphotericin B is recommended for the treatment of deep *Candida* infections, with fluconazole being a second-line treatment.⁵ Conventional amphotericin B is nephrotoxic and should not be administered in a dosage exceeding 1.5 mg/kg/day. Liposomal amphotericin has a significantly shorter infusion time than conventional amphotericin B and is reported as giving a rise to less systemic toxicity and renal impairment,⁶ enabling the administration of a much higher dosage than with conventional amphotericin. Amphotericin B causes hypokalaemia, and although liposomal amphotericin B has been reported as causing hypokalaemia in up to 18% of cases, our patient tolerated the liposomal amphotericin well, with rapid clinical improvement.

The possibility of candidal peritonitis, although very rare, should be considered in any patient potentially at risk. Empirical antifungal treatment should be considered pending the results of other investigations such as serology and peripheral cultures. We suggest that clinicians consider liposomal amphotericin as a useful alternative to conventional amphotericin B in these patients.

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Follow-up of mycoplasma pneumonia

Sir,
I was interested to read the learning points tabled in Dr Shah's useful paper¹ on adult respiratory distress syndrome due to mycoplasma pneumonia. They did not mention follow-up of this acute condition.

In 1973 my late wife developed respiratory distress shown to be due to mycoplasma pneumonia. It cleared rapidly on treatment and three months later a chest X-ray was normal. A year later another severe respiratory infection led to a further chest X-ray and this showed widespread deposits from primary adenocarcinoma of the lung. This, of course, is anecdotal evidence, but I think you will understand why I personally feel that such a respiratory infection occurring in a previously healthy subject should be followed up very carefully for at least a year or two after the event.

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