Letters to the Editor

Bot-fly bite

Sir,

Increased foreign travel has exposed many European travellers to 'tropical' diseases with which Western doctors are unfamiliar, but which may nevertheless present in routine clinical practice. We describe such a case, in a patient who had recently returned from a holiday in Belize.

A 37 year old man presented with a one month history of abscesses on his upper arm and back which developed at the site of two insect bites sustained 5 weeks earlier, shortly before his return. The abscesses discharged small amounts of blood-stained pus, from which Staphylococcus aureus was cultured. Although his symptoms initially responded to a course of erythromycin, one week before admission he developed increasing pain and swelling and the blood-stained discharge recurred. On examination he had two tender, bluish, indurated subcutaneous lesions 2–3 cm in diameter, each with a central punctum from which a small amount of dark blood-stained fluid could be expressed. The surrounding skin was mildly erythematous, although there was no accompanying leucocytosis.

At operation, each lesion was found to contain a single writhing larva approximately 1.5 x 0.7 cm in size, buried within the subcutaneous fat (Figure 1). These were extracted and the underlying cavities curetted and lightly packed with an iodine-soaked wick: 2 weeks later, the wounds had healed completely. The larvae were subsequently identified as those of the human bot-fly Dermatobia hominis, an insect with a fascinating, if somewhat gruesome, life-cycle. Dermatobia hominis is found widely in tropical South and Central America, and like the African Tumbu fly, it is a 'myiasis' fly whose larvae can only survive in the living tissues of a warm-blooded vertebrate.1,2 The adult female bot-fly captures a jungle mosquito or other biting insect in flight and glues eggs to the underbelly of her captive prey before releasing it. When the mosquito settles on a human host, bot-fly eggs are deposited on the skin, typically in the region of a small scratch or bruise;3 the maggots which hatch burrow subcutaneously, leaving a narrow tail-segment containing respiratory spiracles exposed to atmospheric air through a small punctum in the skin.4 The larvae may remain in situ for several months and can result in severe ulceration, often with secondary infection.3,5 Once mature, larvae erupt through the hosts' skin and drop to the ground, where the puparium is formed, which finally releases the adult fly.1

Surgery is not usually indicated for these parasites. It is enough to immerse the affected part in either oil or water in an attempt to asphyxiate the larva, which can be extracted as it struggles to the skin surface. Other practitioners enlarge the skin opening with mosquito forceps and extrude the larva by applying slight pressure. In the days when horses were a common form of transport in these islands and before insecticides were widely used, the horse bot-flies Gasterophilus intestinalis, G. nasalis and G. haemorrhoidalis, would cause a similar syndrome in man in the United Kingdom, which is well described in medical textbooks published at the end of the last century.

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References

Primary tuberculous appendicitis

Sir,

Singh et al.1 discussed 17 cases of tuberculosis of the appendix. We would like to report a case of primary tuberculous appendicitis in a Caucasian man confirmed histologically postlaparotomy.

Figure 1 Dermatobia hominis larva (scale 0–2 cm). Ventral aspect, showing mouthparts (left) and tail segment (right).
A 39 year old epileptic, non-smoking, Caucasian male presented with abdominal pain, pyrexia and generalized abdominal tenderness. He had had episodes of diarrhoea during the week prior to his admission. Investigations including chest X-ray were unremarkable apart from a leucocytosis of 13.2 x 10⁹/l and plasma albumin of 25 g/l.

A working diagnosis of acute appendicitis was made, and at laparotomy, an inflamed retro-caecal appendix was removed. The ileum was normal and no Meckel’s diverticulum was detected. Histology revealed well-developed granulomatous appendicitis with widespread epithelial granuloma becoming confluent in the sub-mucosa and mucosa. Scattered acid-fast bacilli were present. The serosa showed only mild chronic inflammation with no granulomas.

In retrospect he admitted to being unwell for the past year with abdominal pain, night sweats and a weight loss of 7 kg. On review after 4 months of drug treatment he was asymptomatic.

This case illustrates many features of a disease rarely encountered in Great Britain. Weight loss, abdominal pain, diarrhoea and fever are typical symptoms of gastrointestinal tuberculosis, although the differential diagnosis should primarily include lymphoma, Crohn’s and Yersinia enterocolitis. It is usual to find a normal chest X-ray and no signs of tuberculosis elsewhere in the body.²

Three types of clinical presentation of tuberculous appendicitis have been described. The most common is chronic disease with mild to moderate intermittent right iliac fossa pain indistinguishable from ileocaecal tuberculosis, acute obstructive appendicitis as in our patient, and a latent type discovered incidentally.¹

It is interesting that the patient had presented 9 months earlier with a non-traumatic right knee effusion. This was thought to be Rieter’s syndrome and was successfully treated with non-steroidal anti-inflammatory drugs. However, it is probable that he had a peripheral arthritis which is a well-described manifestation of tuberculosis,¹ the mechanism of which is poorly understood, although an immune complex arthritis has been suggested.⁴ A year after his initial illness no other symptoms of arthritis have occurred.

In the UK, isolated tuberculous appendicitis in a Caucasian is certainly unusual and according to our search has not been documented before. It seems that tuberculosis has replaced syphilis as the great mimicker of other diseases, and the diagnosis should be actively sought in view of ease of treatment with modern drug regimes.

References


An unusual cause of biliary peritonitis

Sir,

Stone disease leading to cystic ducts obstruction and inflammation remains the commonest cause of gallbladder perforation. We report an unusual cause of gallbladder perforation leading to biliary peritonitis resulting from common bile duct obstruction by an periampullary tumour.

A 40 year old man presented with pain, fever and fluctuating jaundice for 3 months and abdominal distension for 2 days. Examination revealed extreme cachexia, deep jaundice and signs of systemic toxicity. The abdomen was distended, diffusely tender and showed signs of peritonitis. Flank tap revealed bile. An emergency laparotomy revealed generalized bile peritonitis with a perforation at the gallbladder neck. The gallbladder itself was thin walled and inflamed with no stones or sludge; the common bile duct was dilated up to its lower end. Repair of the perforation, cholecystostomy and peritoneal toilet were performed. In the post-operative period, the patient had persistent hypotension and uncontrolled arrhythmias secondary to tight mitral stenosis that had been previously asymptomatic. The patient recovered after an emergency closed mitral valotomy performed on the day after the abdominal surgery. A side viewing endoscopy was later performed to look for the cause of obstruction at the lower end of the common bile duct. It revealed a large periampullary adenocarcinoma. At a second laparotomy, the tumour was found to be infiltrating the head of pancreas and the portal vein and was locally unresectable. Cholecystectomy (because of the very low insertion of the cystic duct), Roux-en-y choledochojejunostomy, gastrojejunostomy and jejunojejunostomy were performed. Biopsy of the gallbladder showed features suggestive of resolving acute cholecystitis.

Cystic duct obstruction, acute inflammation, ischaemia and necrosis of the gallbladder wall form a recognized sequence in the genesis of gallbladder perforation due to stone disease. Infection and hyperconcentrated bile are the other predisposing factors.¹ Carcinoma of the head of pancreas and periampullary region have rarely been associated with gallbladder perforation.² We consider that gallbladder perforation in the present case occurred due to overdistension of the gallbladder in an obstructed biliary tree, already weakened by the damaging action of infected and hyperconcentrated bile. Timely interventions, first for biliary peritonitis and secondly for a tight mitral valve stenosis, salvaged the patient from an otherwise hopeless situation.

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