of superficial lesions can occur, which together with the presence of regional nodes may suggest an infective or neoplastic process.

Involution has occasionally been described in small lesions, but large lesions invariably persist if left untreated. Although up to 50% of lesions recur after excision or curettage[1,3], and whilst a unique case of microscopic metastasis has been described[7], it is generally felt that a conservative approach is all that is required[1,2,5].

The true nature of this lesion has been unclear for a long time. It is now generally thought to be a true neoplasm composed of proliferating endothelial cells which take on a characteristic plump, epithelioid appearance. The lymphoid and eosinophilic components are considered to be reactive[1,5].

The clinical features of this uncommon lesion are by no means characteristic; indeed Mehregan and Shapiro[6] listed ten separate clinical diagnoses for their series of 14 cases. Surgeons should be aware of this interesting entity to avoid unnecessary treatment.

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Malignant fibrous histiocytoma causing fatal ileal perforation

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Keywords: histiocytoma, intestinal; abdomen, acute; peritonitis

Malignant fibrous histiocytoma (MFH) is a pleomorphic sarcoma of soft tissue occurring predominantly in the extremities and retroperitoneum[1,2]. Primary intestinal MFH is rare; a search of the English literature has revealed only two reported cases[3,4]. Its presentation as an acute abdomen due to bowel perforation has not been previously described. We report a case of fatal bowel perforation due to MFH of the ileum.

Case report

A Negro male, aged 76 years, presented to the Casualty Department with a 5-day history of central abdominal pain, vomiting and fever. The pain, gradual in onset, progressively worsened over the 5-day period. On admission he was an ill-looking, moderately dehydrated man with a pulse of 108/min, blood pressure 140/90 mmHg, temperature 37°C and respiratory rate 24/min. He had generalized abdominal guarding with rebound tenderness, free intraperitoneal fluid, but no palpable mass.

A diagnosis of a perforated viscus was considered and the patient started on ampicillin, gentamicin and metronidazole (Flagyl). At emergency laparotomy there was 1500 ml foul-smelling purulent fluid in the peritoneal cavity and a 7 cm diameter necrotic mass in the terminal ileum, 60 cm from the ileocaecal junction. There was no evidence of liver or node metastases. A total of 30 cm of ileum was resected, including the perforated necrotic mass. After reanastomosing the ileum and appropriate peritoneal toilet, the abdomen was closed with a Penrose drain in the right iliac fossa.

The patient had prolonged postoperative ileus, purulent drainage from the incision and drain sites, and developed a swinging pyrexia on the 9th postoperative day. His condition deteriorated rapidly over the following 24 hours when he developed endotoxic shock and died two days later. Permission for post-mortem was not granted.

Pathology: Multiple sections from the necrotic tumour tissue showed pleomorphic sarcoma composed of large fibroblast-like cells with pronounced nuclear atypia, rounded histiocyte-like cells and pleomorphic giant cells. The giant cells showed prominent

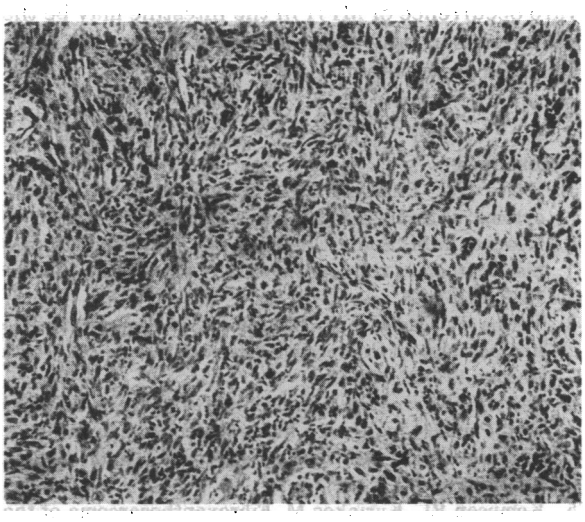

Figure 1. Spindle-shaped cells arranged in characteristic storiform pattern. (H&E × 200; reduced 75%)

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nucleoli and abundant amphophilic staining cytoplasm. Many areas consisted of spindle-shaped cells arranged in characteristic storiform pattern (Figure 1). There were dilated blood vessels surrounded by tumour cells. Extensive areas of necrosis were visible but there was no evidence of epithelial malignancy. Oil red O stains revealed no fat within the tumour cells. PTAH, PAS and mucicarmine stains were also negative. Histologically, it was a typical MFH.

Discussion

MFH, predominantly a tumour of adult life, is uncommon in childhood[1,2]. The tumour typically occurs on the extremities and retroperitoneum. The abdominal tumours may produce signs of increased intra-abdominal pressure, resulting in hernia and abdominal distension[1]. One case of intestinal MFH causing intussusception has been reported[4]. The clinical presentation in our case is unique in that the tumour caused ileal perforation.

MFH is a morphologically heterogeneous neoplasm usually composed of rounded and spindle cells arranged in a storiform pattern accompanied by pleomorphic giant cells and inflammatory cells[1,5]. Because of its variable morphologic pattern, this tumour has often been confused with pleomorphic liposarcoma, rhabdomyosarcoma and anaplastic carcinoma. Our case conformed to the criteria for MFH, there being no elements to confuse it with the entities mentioned above.

Biologically, MFH is a highly malignant sarcoma with a two-year survival of about 50% and a high rate of recurrence and metastases[1,6]. Early and complete removal of the tumour is advised as the best method of improving survival. The biological potential and pathological criteria for prognosis in MFH of the intestine have yet to be determined.

The most controversial aspect of MFH is its histogenesis. Although it has been considered by some investigators to be of histiocytic origin[6,7], others have suggested an origin from a primitive mesenchymal cell[8]. The precise histogenesis of MFH remains uncertain and more sophisticated techniques may be required to characterize them further, particularly in MFH of the intestine.

Our patient developed ileal perforation because of rapid growth and extensive necrosis in the tumour. It might therefore be expected that MFH of the bowel would commonly present with perforation. The very rare occurrence of MFH in the intestine may be the major reason why this complication has not been reported previously.

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Bilateral Achilles tendon rupture simulating peripheral neuropathy: unusual complication of steroid therapy

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Keywords: Achilles tendon rupture, ataxia, steroid therapy, asthma

A history of unsteadiness, with the finding on examination of peripheral weakness and absent ankle jerks, is usually suggestive of a neurological disorder. We report such a patient who on further examination proved to have an unusual orthopaedic problem which is a recognized but rare complication of steroid therapy.

Case report

A 43-year-old housewife suddenly developed unsteadiness of gait, which persisted. She complained of a constant tendency to fall forwards. Her only other symptom was of a vague and not troublesome ache in both ankles. She gave no other history suggestive of neurological disease or of trauma. She had been asthmatic since the age of 7 and had taken short courses of oral steroids for acute exacerbations for many years; in addition she had been taking oral prednisolone 10 mg daily for the twelve months before presentation. She also took terbutaline and slow theophylline orally, and salbutamol and beclomethasone by inhaler. On presentation to her local hospital 9 weeks after the onset of symptoms she was found to have absent ankle jerks, and was referred to our department as a neurological problem.

On examination she was mildly Cushingoid. General examination was otherwise normal. Neurologically, there were no abnormalities of higher functions, cranial nerves or upper limbs. In the lower limbs power was grade 5 (MRC scale) in all muscle groups except for ankle plantar flexion which was grade 3 bilaterally.

There was no muscle wasting or fasciculation. Tone, coordination and sensation were normal, as