LETTER TO JCP

Chronic osteomyelitis mimicking sarcoma
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This report describes a rare case of chronic osteomyelitis in a 60 year old man mimicking a soft tissue sarcoma. Chronic osteomyelitis is an infrequent cause of a soft tissue mass and is usually diagnosed clinically by a combination of radiology and microbiology. Rarely, COM can mimic a primary bony neoplasm, but this is the first reported case where it mimicked a soft tissue sarcoma. The clinical, radiological, and histological appearances of this case will be discussed.

Chronic osteomyelitis (COM) is an infrequent cause of a soft tissue mass and is usually diagnosed clinically by a combination of radiology and microbiology. Rarely, COM can mimic a primary bony neoplasm. However, it is extremely rare for osteomyelitis to mimic a soft tissue sarcoma. This case report outlines the clinical, radiological, and histological appearances of such a case.

CASE REPORT
A 60 year old man presented with a rapidly enlarging, painless mass in the left side of the anterior chest wall. He was otherwise healthy and had no previous medical or surgical history. Initial treatment by his general practitioner included a course of oral co-amoxiclav, which had no effect, and he was referred for further evaluation. Clinical examination showed a 10 cm round mass centred on/above the left fifth rib. It was non-tender and firm without evidence of inflammation. Blood tests showed a mildly increased erythrocyte sedimentation rate (11 and 40 mm/hour on two separate occasions) and white blood cell counts within normal limits (7.0–8.2 × 10⁹/litre on several occasions during the investigation). The initial radiological workup included x ray of the thorax, which was normal, and ultrasound of the affected area, which showed generalised thickening of the pectoralis muscle and thickening of the costal cartilage, but no definite focal soft tissue abnormality, and a magnetic resonance imaging (MRI) scan was recommended. This showed swelling and oedema of the subcutaneous fat and anterior thoracic wall musculature, in addition to small pleural effusions (fig 1). This was interpreted as probably being inflammatory in nature. For further evaluation, a surgical biopsy was carried out of the soft tissue. The biopsy showed skeletal muscle infiltrated by spindle cells scattered in abundant collagenous stroma (fig 2). Focally, the muscle showed atrophic changes. No mitoses were identified and the spindle cells displayed bland nuclei similar to myofibroblasts. Immunohistochemically, the cells showed positivity for vimentin. Staining for cytokeratins (AE 1/3 and CAM 5.2 antibodies), S100 protein, HMB45, smooth muscle actin, smooth muscle specific actin, CD34, common leucocyte antigen, and desmin was negative. The suggested pathological differential diagnosis at this stage was of a non-neoplastic myofibroblastic process. However, in view of the clinical data further biopsies were suggested to ensure the lesion was fully sampled.

“The biopsy showed skeletal muscle infiltrated by spindle cells scattered in abundant collagenous stroma with occasional lymphocytes”

Figure 1 Initial magnetic resonance imaging scan of chest wall lesion showing subcutaneous and anterior thoracic wall oedema and bilateral pleural effusions.

Figure 2 Light microscopic photograph (original magnification, ×200). Initial biopsy shows degenerating skeletal muscle with bland spindle cells scattered in abundant collagenous stroma.

Abbreviations: COM, chronic osteomyelitis; MRI, magnetic resonance imaging.
The lesion continued to enlarge rapidly over the next two months and a second MRI scan was carried out five weeks after the first (fig 3). It showed a significant increase in involvement. The pattern was more mass like and extending into the thoracic cavity, abutting the pericardium, a pericardial effusion. The bilateral pleural effusions were not changed.

On the basis of the rapid growth it was decided to excise the lesion by a local resection including a segment of the anterior chest wall with parts of ribs two to four extending to the sternum. At surgery, the lesion was abutting but only superficially attached to the pericardium. The defect was reconstructed with a sandwich of Marlex mesh and methacrylate cement. A pectoralis flap was used and the skin was closed.

The surgical pathological specimen consisted of a 10 cm resection of part of the chest wall, including the chest wall lesion with three ribs and intercostal soft tissue. The parietal pleural surface was smooth and a cross section of the soft tissues showed a 4.5 × 4 × 3 cm ill defined process involving intercostal musculature. A cross section of one of the ribs revealed on gross inspection several areas of pus-like material, up to 0.5 cm in size, but no cortical bone destruction (fig 4).

On light microscopy, the soft tissue showed similar features to the original biopsy, namely an infiltrative spindle cell process with scanty scattered inflammatory cells and collagen deposition. Cross sections of bone were examined and, corresponding to the pale areas noted grossly, a dense mixed acute and chronic inflammatory infiltrate with abundant plasma cells was noted (figs 5,6). In addition, there were foci of necrotic bone and pronounced osteoblastic activity around viable bone. A histological diagnosis of osteomyelitis with a striking myofibroblastic reaction was made. Special stains for microorganisms (periodic acid Schiff, Ziehl-Neelsen, Grocott, and Gram) were all negative and no material had been submitted for culture.

After surgery the patient made a speedy recovery and was discharged after five days.

**DISCUSSION**

This case report is important because it highlights a case of a clinically aggressive soft tissue mass caused by osteomyelitis. The rapid growth of this mass mimicked the behaviour of a soft tissue sarcoma.

Numerous spindle cell proliferations can mimic sarcomas: spindle cell carcinomas, spindle cell melanomas, “pseudosarcomas” such as nodular fasciitis, fibromatosis, inflammatory...
myofibroblastic tumour, and inflammatory processes (for example, mycobacterial spindle cell pseudotumour) can all give rise to lesions that mimic a sarcoma. In the case of bone destruction, COM can be confused with primary bone tumours, but extremely rarely with soft tissue sarcomas. Review of the literature showed no similar cases of osteomyelitis mimicking a soft tissue sarcoma.

"The presentation described in our case report appears to be unique."

In our case, the diagnosis of osteomyelitis with an associated spindle cell soft tissue mass was made only after extensive surgery. This diagnosis was not made preoperatively because it was felt the soft tissue mass involved bone as a secondary event rather than being a primary bone lesion. Osteomyelitic foci were not seen on chest X rays or MRI scans, and computed tomography scans that might have shown more bone details were not performed. Histologically, the biopsy of this lesion revealed a myofibroblastic proliferation with only occasional lymphocytes, and no features of osteomyelitis were present.

In conclusion, COM is a well known source of confusion with primary neoplasms of bone. However, it is an extremely rare mimic of primary soft tissue sarcoma and the presentation described in our case report appears to be unique.

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