

Rheumatology 2000;39:1290–1292

Aseptic psoas pyomyositis and erosive discitis in a case of calcium pyrophosphate crystal deposition disease

SIR, Iliopsoas abscess and pyomyositis are rare clinical entities. Except for a few cases of carcinoma or focal myositis mimicking abscesses [1, 2], they are infectious. The concurrence of spondylodiscitis and bilateral psoas abscesses is virtually pathognomonic of an infectious process, *Staphylococcus*, *Brucella* and *Mycobacterium tuberculosis* being the most likely organisms. We report a case of calcium pyrophosphate crystal deposition (CPPD) disease presenting with erosive spondylodiscitis associated with an aseptic inflammatory reaction of both psoas muscles which mimicked such an infectious process.

A 76-yr-old woman was admitted for exacerbation of chronic low back pain with leg weakness and repetitive falls. There was a history of diabetes with discrete polyneuropathy, chronic venous insufficiency, hypertension and mild chronic renal insufficiency. She had CPPD disease with radiological chondrocalcinosis at the wrists, knees and lumbar spine. Two years previously, a L2–L3 erosive discitis associated with a herniated disc had been treated conservatively.

On admission, she complained of some morning stiffness and occasional night sweats and fever, but denied any weight loss, recent surgical procedures and urogenital or gastrointestinal problems. Examination demonstrated severe limitation of lumbar spine mobility with muscle spasms and tenderness of the paralumbar region. There was no sign of sacroiliac joint involvement. Light touch sensation was diminished over the anterior thigh, and both knee and ankle jerks were absent. There was no fever, cutaneous lesions, abdominal tenderness, signs of cardiac involvement or any other signs of an infectious process. The psoas sign was absent on both sides. The white cell count was 12 000/mm³ and the erythrocyte sedimentation rate 75 mm/h. There was mild inflammatory anaemia and routine laboratory tests were normal except for a moderate increase in creatinaemia. Spine radiographs demonstrated multiple discopathies from D9 to L4 and a previously known increased density of the body of L2.

After 3 weeks of standard conservative therapy consisting of analgesia, rest and non-steroidal anti-inflammatory drugs (NSAIDs), she presented an exacerbation of the weakness of the quadriceps muscle. A magnetic resonance imaging (MRI) study demonstrated spondylodiscitis of the L2–L3 space infiltrating the vertebral bodies from L1 to L3. An image compatible with an abscess or intradural sequester as well as cellulitis and myositis of both psoas muscles with enhanced gadolinium uptake evoking abscesses were further demonstrated (Fig. 1).

Blood cultures, a Mantoux skin test and brucella serology were negative. A percutaneous CT-guided needle biopsy was performed. No organism was demonstrated on direct examination, and cultures for both



FIG. 1. MRI of the lumbar spine. Gadolinium-enhanced T₁-weighted images. (A) Sagittal section showing L2–L3 erosive discitis with narrowing and inflammation of the disc space with fluid (arrow) and marrow oedema extending from L1 to L3. (B) Coronal section showing discitis with intradural free fragment or abscess (arrow) and target lesion of the psoas muscle with an irregular thickened wall of increased signal surrounding a central hyposignal (arrowheads) evoking an abscess.

aerobic and anaerobic bacteria and for *Mycobacterium*, brucella and fungi remained negative. Histology revealed only fibro-adipose and chronic inflammatory tissues. Because of the strong suspicion of an infectious origin, an L2 hemilaminectomy with psoas drainage was performed. At no time before, during or after operation were antibiotics administered. Tissue samples were sent for culture and histology. No organism was demonstrated on direct examination and the cultures remained negative. Histology demonstrated necrotic cartilaginous and osseous tissues, with a severe lymphoplasmocytic inflammatory reaction. Some giant cells, but no granulomas, were observed (Fig. 2). The post-operative course was favourable without antibiotic treatment, with the disappearance of pain and neurological deficits. The patient was discharged after 1 month and at 6 months still had no complaints.

Iliopsoas abscess and pyomyositis are rare clinical entities, but they are nearly always infectious. Over the past century, the cause of this disease has changed. *Mycobacterium tuberculosis* was responsible for most cases initially, but after *M. tuberculosis* had been controlled as a pathogen the predominant cause became secondary iliopsoas abscess by direct extension of an adjacent infection [3]. Gastrointestinal tract diseases, particularly Crohn's disease, became the most common cause of psoas abscess [3]. Nowadays, primary iliopsoas abscess is increasingly related to intravenous drug abuse and the large number of HIV-infected patients [4]. Primary iliopsoas abscess occurs primarily in young males, but secondary abscesses are observed in a somewhat older age group. The local anatomy of the psoas muscle explains the various causes of secondary abscesses, as this muscle is in contact with digestive, urogenital and osseous structures [5]. The source of more than 80% of secondary iliopsoas abscesses is a gastrointestinal tract perforation [resulting from inflammatory bowel disease (in particular severe Crohn's disease), appendicitis, diverticulitis, carcinoma, or others] or a primary

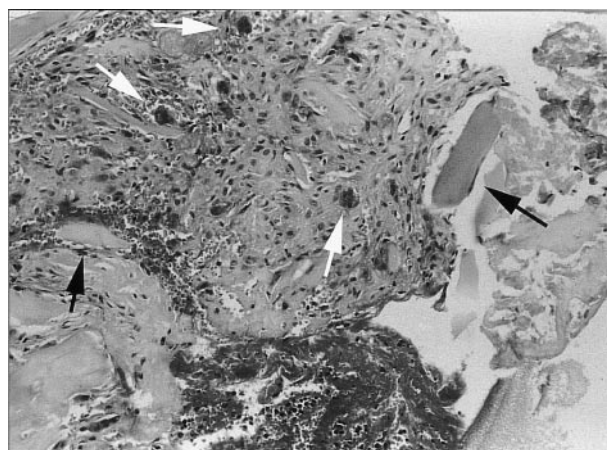


FIG. 2. Section of a surgical biopsy stained with haematoxylin and eosin, showing non-specific inflammatory reaction with lymphoplasmocytic reaction, necrotic cartilage (black arrows) and giant cells without granuloma formation (white arrows).

bone infection within the spine, the ileum or the sacroiliac joints, as initially appeared to be the case in our patient [3, 5, 6].

Clinical diagnosis of chondrocalcinosis can be very difficult, as signs and symptoms are not specific and can mimic simple osteoarthritis and also rheumatoid arthritis. Most cases involve the pelvis and the extremities, but spine involvement is well recognized [7–10]. In most cases, chondrocalcinosis of the spine will be asymptomatic, with observation of silent radiographic disc degeneration. Rarely, acute and chronic chondrocalcinosis may be symptomatic and produce destructive lesions of the vertebral bodies very similar to those observed with infections or ankylosing spondylitis. Erosions may then proceed to secondary instability and pseudospondylolisthesis. Such spondylitis complicates CPPD disease in about 5–7.5% of cases [8, 11]. Myelopathy, with medullar compression secondary to nodular deposition of CPPD crystals in the ligamentum flavum, has also been reported [12]. Finally, involvement of the nucleus pulposus with calcification has been demonstrated at autopsy as well as at surgery. It can produce an intraspinal mass with acute radiculopathy, as seen in our case [7].

Radiographically, asymptomatic calcification of the intervertebral disc is observed in up to 30% of patients with systemic chondrocalcinosis, frequently at the L2–L3 level [10]. MRI studies of CPPD involvement of the spine have not been performed to our knowledge, but there should be a low signal intensity on all sequences, a finding consistent with calcification, as opposed to infectious spondylodiscitis, which has a low signal on T₁-weighted images but a hypersignal on T₂-weighted images [13]. Definitive diagnosis is usually made by demonstration of the crystals, but they can be absent in destructive or erosive lesions [14]. Rest and NSAIDs are the mainstay of treatment, but decompression surgery has been necessary in selected cases with neurological deficit [15].

Thus, while crystal-related diseases are known to be a cause of erosive discitis, and had actually been recognized 2 yr previously in our patient, the association of myositis and fasciitis of both psoas muscles with erosive discitis was regarded as pathognomonic of an infectious process. All cultures, even from surgical biopsies, and serologies were negative, arguing against such an infectious process. Biopsies demonstrated an inflammatory infiltrate with giant cells but without granulomas. No calcium pyrophosphate crystals were demonstrated, but this description is compatible with previous observations in peripheral destructive CPPD arthropathy [14]. The aseptic causation is further supported by the favourable evolution without antibiotic treatment.

To our knowledge, this is the first observation of pseudo-psoas abscesses associated with erosive discitis in a case of CPPD disease. Now that imaging techniques such as CT and MRI are becoming increasingly prevalent, we believe it to be important to recognize that CPPD spine disease can be accompanied by a severe inflammatory reaction of the psoas muscles which can

mimic abscess images. This should avoid unnecessary, costly and aggressive investigations to exclude an infectious process.

We would like to thank Dr Guillou, Department of Pathology, for the histological photography and Dr Landry, Department of Radiology, for the MRI examination.

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Accepted 11 April 2000

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