# Back pain in childhood

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### **Case history**

A 9 year old girl was referred with a three month history of low back pain which was related to increased physical activity, radiating to both legs. Periodic weakness in both legs was associated with occasional paraesthesiae in both feet. There were no bladder, bowel, or other systemic symptoms. Before the onset of back pain the child had been well, with no history of any trauma.

On examination, she appeared to be of short stature for age, but of normal proportion. On standing there was a hyperlordosis of the thoracolumbar spine. Percussion over the lumbar spine at the L2/L3 level elicited marked tenderness. There was a generalised reduction of all spinal movements. Straight leg raising was 40 degrees bilaterally, with limitation due to low back pain. Neurological examination of the lower limbs was normal. She was afebrile and general systemic examination was normal. Following initial radiographic examination, she was treated in a fibreglass jacket for two months with no relief of symptoms.

## Radiograph of lumbosacral spine

Initial radiographic examination of the lumbosacral spine (fig 1) revealed a grade 1-2 spondylolisthesis at the L5/S1 level with a spondylolytic defect of L5. In addition, there was generalised osteopenia with loss of sharpness of the inferior endplate of L2 and the superior endplate of L3. Although the L2/L3 disc space was well preserved, there was slight beaking of the antero-inferior aspect of the L2 vertebral body.

# **Differential diagnosis**

Back pain in the young requires active investigation. In this age group, the presence of a spondylolysis of L5 with a grade I spondylolisthesis may or may not be symptomatic, and should not be assumed to be the cause of pain. The other radiographic

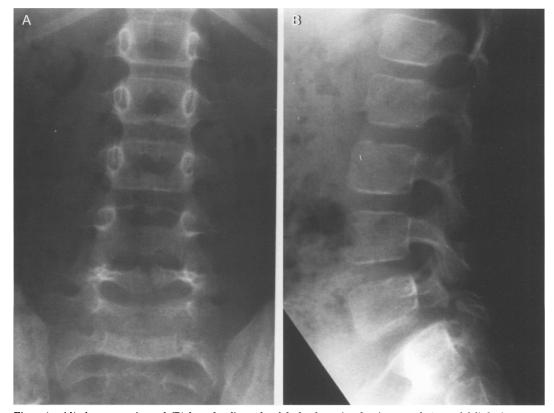


Figure 1 (A) Anteroposterior and (B) lateral radiographs of the lumbar spine showing a grade 1 spondylolisthesis at L5/S1 together with a defect of the pars interarticularis of L5. Note the loss of definition of the inferior and superior endplates of L2 and L3 respectively. Note also the beaking of the L2 vertebral body antero-inferiorly.

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Figure 2 Coned lateral radiograph of the L2/L3 interspace 4 weeks later showing marked resorption of the inferior endplate of L2.

features gave rise to concern that there might be a dual pathology.

Generalised osteopenia in a child may indicate an underlying systemic disease such as metabolic bone disease or osteogenesis imperfecta, or a diffuse infiltrative marrow process such as leukaemia. A poorly defined endplate often indicates an infective discitis, although the preserved disc space in this case was against infection. The "beaking" of the anteroinferior aspect of the body of L2 suggests new bone formation, probably reactive to a local pathological process. The loss of definition of the vertebral end plate is a sign of weakened bone.

Thus, despite the presence of a spondylolysis, the clinical history and examination, together with features on the radiograph, suggest other pathology. Further imaging together with assessment of haematological and biochemical profiles are required.

A full blood count with a differential white cell count was normal, and the ESR was 16 mm/h. Biochemistry including calcium and phosphorus and protein electrophoresis was normal.

A coned view of the L2/L3 level four weeks after the initial radiograph showed marked resorption of the inferior end plate of L2 (fig 2).

A magnetic resonance imaging (MRI) examination of the lower thoracic and lumbar spine revealed abnormal signal within the vertebral bodies of L2, L3, and T12 levels. Intermediate to low signal intensity was seen on the T1 weighted (T1W) images (fig 3) with increased signal on T2 weighted (T2W) images (fig 4). The inferior endplate of L2 was ill defined but the disc space and signal were well preserved. Following intravenous gadolinium, the areas of abnormal marrow signal were enhanced to become isointense with marrow fat on T1 weighting (fig 3B).

An MRI study of the cervical and thoracic spine showed similar foci of abnormal signal intensity in the bodies of C5, T2, and T8 (fig 4), raising the possibility of eosinophilic granuloma. At the T7/T8 level there was loss of disc height and slight irregularity of the superior endplate of T8. No fluid signal within the disc was apparent. No abnormality of the paravertebral soft tissues was seen at any of these other levels.

In view of the multiplicity of findings within the spine, a whole body technetium bone scan was carried out (fig 5). This showed areas of



Figure 3 (A) Sagittal T1W MR image showing decreased signal intensity in the bodies of L2 and L3, and posteroinferiorly in the body of T12. (B) Sagittal T1W MR image after intravenous gadolinium shows relative enhancement in the bodies of T12, L2, and L3.

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Figure 4 Sagittal T2W study of the lumbar (A) and cervico-thoracic spine (B) showing increased signal intensity in the bodies of C5, T2, T8 (arrow), T12, L2, and L3.

increased activity within the spine corresponding to the approximate levels of T8 and L2 vertebral bodies, and also at the lumbosacral junction. The increased activity at the lumbosacral junction was attributable to the spondylolysis and the areas of increased activity at T8 and L2 corresponded to high signal foci seen on the MR images. Activity in the cervical and upper thoracic region appeared within normal limits. Increased activity was also present at the left ankle, the left foot, and the proximal right humerus. These areas were asymptomatic.

A radiograph of the left ankle (fig 6A) showed a lucency within the fibular metaphysis. No radiographic abnormality was visible in the right shoulder. MRI of the left ankle showed marked abnormal signal intensity in the distal fibula involving the metaphysis, the growth plate, and the epiphysis (fig 6B), with a similar appearance in the metaphysis of the distal tibia (fig 6C). An unsuspected area of involvement of the medial aspect of the navicular bone was also evident.

A limited MR study of the proximal right humerus showed a diffuse marked increase in signal in the metadiaphyseal region on a coronal short-tau inversion recovery (STIR) image consistent with diffuse oedema (fig 7).

Needle biopsy of the body of L2 was done. Culture of a specimen failed to grow any organisms. Histological examination showed widespread replacement of the marrow by oedematous fibrous tissue containing numerous plasma cells and lymphocytes and occasional polymorphs. Some reactive new bone formation was also present. The findings were considered to be consistent with chronic infection.

#### Diagnosis

The presence of multiple symptomatic and asymptomatic bony lesions, the classic radiographic signs, and the biopsy appearances suggested a diagnosis of chronic recurrent multifocal osteomyelitis (CRMO), notwithstanding the absence of microbial growth.



Figure 7 Coronal MR STIR image of the right shoulder showing diffusely increased signal in the metadiaphysis of the proximal humerus.

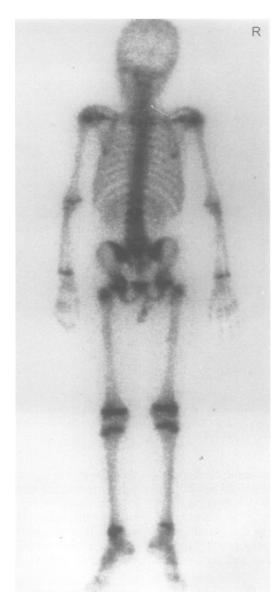


Figure 5 Whole body isotope bone scan showing areas of increased uptake in the thoracic and lumbar spine at the levels of T8, T12, L2, and L3 and at the lumbosacral junction. Increased activity at the left ankle and right shoulder are also present.

#### Discussion

Chronic recurrent multifocal osteomyelitis was first described by Giedion et al in 1972 as a subacute and chronic symmetrical osteomyelitis.<sup>1</sup> Although it has been described as representing a variety of chronic osteomyelitis, certain features suggest that it is a specific entity with well defined clinicopathological features. King et al<sup>2</sup> indicated set criteria for the diagnosis of CRMO: (1) multifocal (two or more) bone lesions, clinically or radiographically diagnosed; (2) a prolonged course (over six months) characterised by varying activity of disease and with most patients being healthy between recurrent episodes of pain, swelling, and tenderness; (3) lack of response to antimicrobial treatment given for at least one month.

This is an uncommon disorder of unknown aetiology, more common in girls, and occurring in childhood and adolescence. Clinically, the presentation is frequently of pain, and joint swelling when a peripheral joint is involved, but systemic manifestations such as fever and malaise are atypical. Laboratory investigations are usually unhelpful. Initially, radiographs may be normal but later metaphyseal lucencies followed by a variable sclerotic reaction may develop. Should metaphyseal lucencies be present initially then the diagnosis is more readily made. The diagnosis is ultimately one of exclusion and a bone biopsy is almost always necessary in order to rule out a neoplastic marrow infiltrative process. Histology may show a polymorphonuclear infiltrate early in the disease course, but more commonly lymphocytic and plasma cell infiltrates with varying degrees of fibrosis predominate.<sup>2-4</sup> Culture for microorganisms is usually disappointing. Antibiotic treatment is frequently unhelpful, even if prolonged, although published reports document some response to antibiotic treatment in some cases.4 Non-steroidal anti-inflammatory drugs may, however, provide symptomatic relief while the disease runs its course<sup>1 2 4-7</sup> and steroids have been used in some cases.<sup>1</sup><sup>2</sup>

Joint involvement is chronic and often recurrent and relapsing. Commonly the patients present with clinical evidence of inflammation in a single bone or joint. With time other sites of involvement usually become clinically mani-Each site of involvement fest. may independently show repeated episodes of exacerbation and remission. Although the long term prognosis is good, radiographs may show residual sclerosis and widened metaphyses long after the disease has become quiescent,<sup>5</sup> and there have been reports of premature epiphyseal fusion<sup>8</sup> and progressive kyphosis secondary to vertebral body collapse.4 Associations have been described, particularly with palmo-plantar pustulosis.4 9 Children developing CRMO have been found to have a normal cellular and humoral immunity and the condition is quite distinct from chronic granulomatous disease of childhood which, however, must also be considered in the differential diagnosis.

Scintigraphy is still the method of choice for identifying other asymptomatic foci within the skeleton. However, MRI of the abnormal scintigraphic sites is indicated to reveal the full range of morphological characteristics.<sup>7 10</sup> The present case shows that MRI may also detect lesions without scintigraphic changes. In this regard, however, MRI is not a feasible replacement for scintigraphy.

In conclusion, it is important to consider the diagnosis of CRMO in a young person and to recognise its essentially benign nature. Antibiotic treatment, in the absence of a cultured organism, is unlikely to be helpful. Perhaps the most important purpose of identifying this condition is to reassure the parents that in the majority of cases this is a benign condition, characterised by exacerbations and remissions, which will ultimately settle with no lasting aftereffects.

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Figure 6 (A) AP radiograph of the left ankle showing a lucency in the fibular metaphysis. (B) Coronal MR STIR sequence of the left fibula showing increased signal in the fibular metaphysis and epiphyseal region adjacent to the growth plate.  $\$  Coronal MR STIR image of the left tibia demonstrating increased signal focally in the metaphysis.

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