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CASE REPORT

Juvenile spondylodiscitis : the value of magnetic resonance imaging A report of two cases

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Two boys presenting with reluctance to sit straight and stand were diagnosed with spondylodiscitis of the lumbar spine. After confirmation of the diagnosis on plain radiographs, computed tomography and magnetic resonance imaging, they were successfully treated with antibiotics and in one case a lumbar orthosis. The use of magnetic resonance imaging is discussed and compared to the other radiological techniques. Magnetic resonance imaging seems to be the most sensitive and specific imaging technique used in the diagnostic process of spondylodiscitis. Computed tomography and technetium bone scan both play a specific part in the process of diagnosis and follow-up.

INTRODUCTION

Juvenile spondylodiscitis is an infection of the intervertebral disc destroying the adjacent vertebral endplates ; it makes up for 2% of all juvenile bone infections (*4*). The pathology and location are probably facilitated by the typical involution of the vascularisation of the immature intervertebral disc in children, thus creating a stasis and even an inversion of the blood flow (*1, 3, 5, 7*). Depending on the systemic presentation of the disease and the severity of the bony destruction, a differentiation is made between pure discitis, without bony destruction, spondylodiscitis and vertebral osteomyelitis with massive bone destruction and severe systemic repercussions (*4*).

Two age groups show a high incidence of the disease : 0.5 to 4 and 10 to 14 years (*1, 3, 7*). Symptoms are generally non-specific and are determined by the age of the child and the level of the lesion, which is mainly lumbar (*6, 9*).

The use of several diagnostic techniques and the various options in the treatment of spondylodiscitis are still a subject of discussion.

CASE 1

A Caucasian boy, aged 13 months, was seen at the out-patient clinic because of whinging and crying for one week, diarrhoea for two days and reluctance to sit straight and stand. From his recent history we withheld an episode of fever one month earlier and twice a fall on his back during the last month. At clinical examination, mobilising the child's lumbar region seemed to evoke pain. The

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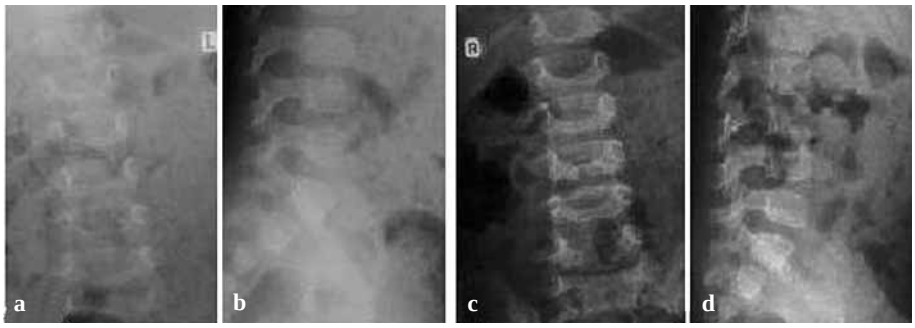

Fig. 1. — a-b. Initial radiographs : narrowing of the intervertebral space L3-L4 and irregular borders of the adjacent vertebral endplates ; c-d. Radiographs after one year show a slight dextroconvex scoliosis and reduced lumbar lordosis.

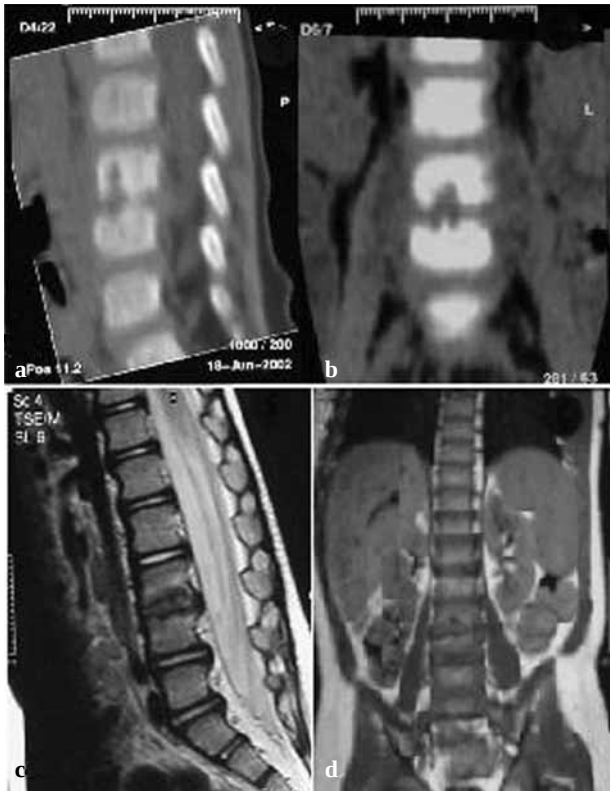

Fig. 2. — a-b. sagittal and frontal CT image, showing the extent of the bony destruction at L3-L4 ; c. MRI-T2 sagittal image, showing a hypointense signal at the L3-L4 intervertebral disk space 6 months after diagnosis ; d. MRI-T1 frontal image, showing little difference in signal intensity between the vertebrae.

boy was having a fever of 38.3°C, but further clinical examination showed no possible entrance portal. The erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) were elevated, while investigation of the urine revealed a non significant culture of *Enterococcus Faecalis*. Blood and faeces cultures were negative.

Plain radiographs of the lumbar spine showed signs of a spondylodiscitis at L3-L4 (fig 1a and b). This was confirmed by computed tomography (fig 2a and b). A technetium 99 m bone scan was highly positive. The tuberculin skin test was negative, pleading against a tuberculous spondylodiscitis. Finally a magnetic resonance imaging scan (fig 2c and d) confirmed the extended bone destruction as seen on the CT scan and revealed no damage to the surrounding soft tissue.

Treatment initially consisted of intravenous (IV) cefotaxim and flucloxacillin at respectively 3×250 mg/day and 3×200 mg/day. After confirmation of the diagnosis by MRI, doses were increased to respectively 4×500 and 4×200 mg/day. CRP and ESR dropped immediately. After one week of therapy the fever also disappeared. Because of persistent pain and reluctance to sit, a lumbar brace was applied with an obvious antalgic effect : the child immediately started to stand and sit straight again.

After three weeks of favourable evolution, the child developed a pustular rash on his torso and limbs, combined with a fever up to 39.7°C and oedema. His body weight increased with 10% in just three days. The suspicion of an allergic vasculitis was confirmed by high blood levels of CRP, Lactate Dehydrogenase (LDH) and eosinophilic leukocytes, combined with leuko- and thrombocytopenia. This is a known side effect of the long-term use of cefotaxim and flucloxacillin. Therapy

was switched to clindamycine 3 × 100 mg/day, cetirizinedihydrochloride, 2 × 10 mg solumedrol and 5mg furosemide IV per day. The beneficial effect was immediate. After a total of 4 weeks of IV therapy, antibiotic therapy was continued orally for another 4 weeks.

After a total of 8 weeks of antibiotic treatment and 4 months of brace support, the boy was clinically considered cured and showed no more signs of illness or functional disability. Radiographs taken after 1 year (fig 1c and d), showed a slight dextroconvex scoliosis and lumbar kyphosis of no functional significance.

CASE 2

An 18-month-old Caucasian boy presented with difficulties to walk and sit straight for more than six weeks. Mobilisation of the pelvis seemed painful. During the first weeks, the focus of pain was thought to be in the hip, but earlier examinations in another clinical orthopaedic practice did not reveal any obvious clinical signs or radiological abnormalities at the lower limbs ; a total body technetium bone scan was also negative. The temperature of the child was normal, though he had slightly elevated CRP and ESR.

Eventually, careful examination of the back revealed pressure tenderness in the lower lumbar region and painful straight leg raising ; there was no neurological deficit. Radiographs confirmed the suspicion of a lumbar spondylodiscitis at L5-S1 with destruction of the vertebral endplates. This diagnosis was confirmed on CT ; MRI revealed an associated ventral epidural abscess (fig 3a and d).

The boy was admitted to the hospital and received intravenous flucloxacillin**,** 4 × 500 mg a day during a period of 25 days. After one week the pain disappeared and the child recovered slowly, regaining his ability to walk. CRP and ESR decreased within a few weeks. The antibiotic treatment was continued orally for another six weeks and the child recovered without any complications. The epidural abscess had almost totally disappeared and the vertebrae were healing on a second MRI taken two months after the first, thus confirming a favourable evolution (fig 3e and f).

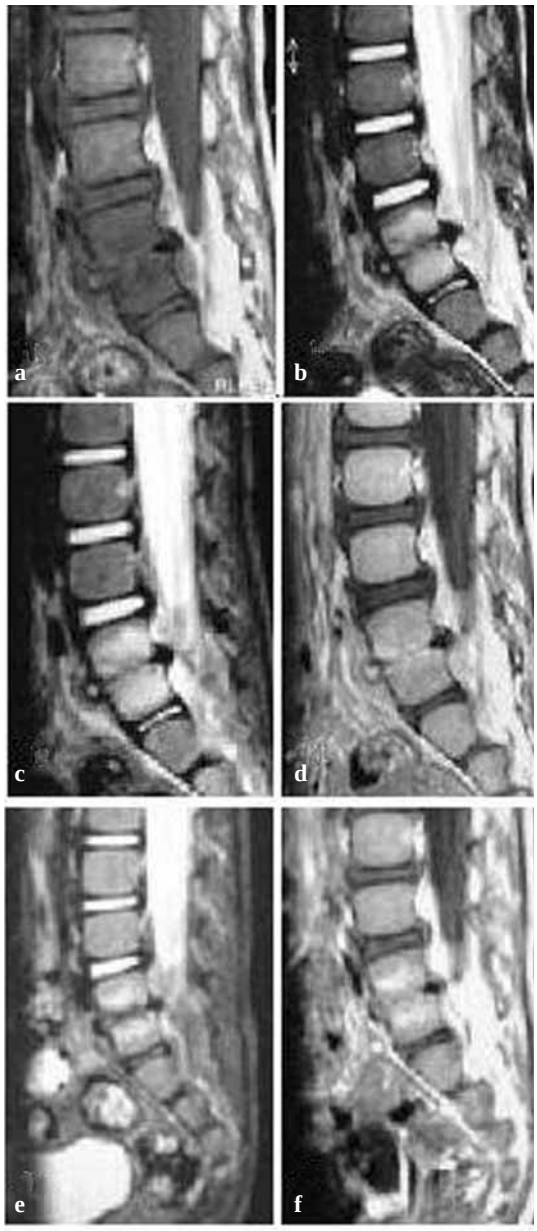

Fig. 3. — a-d. Respectively MRI T1 TSE 512, T2 TSE 512, STIR Long TE and T1 Gadolinium DOTA sagittal images taken one month after presentation of the patient, showing oedema and slight collapse of the L5 and S1 vertebrae and destruction of the L5-S1 vertebral end-plates. Subtotal destruction of the intervertebral disk L5-S1 in combination with a ventral epidural abscess and an anterovertebral subligamentous abscess at the level of L5-S1 ; e-f. MRI STIR Long TE and T1 Gadolinium DOTA sagittal image taken 3 months after presentation of the patient, showing oedema and hyperaemia of the L5-S1 vertebrae and a slightly advanced bony destruction ; limited mass effect of the residual fibrotic intervertebral disc on the anterior and posterior ligaments ; no paravertebral abscess formation visible.

Table I. — Differential diagnosis of spondylodiscitis (*5*)

1. Trauma	
2. Hip pathology	hip dysplasia, transient synovitis
3. Spinal pathology	spondylolysis, spondylolisthesis, disk herniation
4. Infections	urinary, gastro intestinal, respiratory, arthritis, osteomyelitis, spondylodiscitis, tuberculous spondylodiscitis
5. Tumoral pathology	

Table II. — Comparison of sensitivity (*2, 4, 8, 10*)

	Sensitivity in case of spondylodiscitis	Sensitivity in case of an associated abscess
X-Ray	60-85%	25%
CT	89-92%	69%
Bone Scan	72-95%	/
MRI	92-100%	85%

Radiographs at 4 months after diagnosis showed healing of the vertebral endplates and no evolution to spontaneous fusion.

DISCUSSION

Juvenile spondylodiscitis is a condition with vague and non-specific symptoms. The investigating physician should always consider a spinal pathology when a child cries persistently during diaper change or when it refuses to sit straight or stand. Sometimes there is a localised tenderness in the affected region, but very often clinical investigation only reveals pain during mobilisation of the pelvis. The differential diagnosis should consider a variety of pathologies (table I). The result of CRP and ESR are mostly helpful in confirming the infectious origin of the disease, although their values can occasionally be normal.

Statistics show that the diagnosis of spondylodiscitis is usually made 1 tot 28 days after the presentation of the patient. When MRI is not used in the process of diagnosis, it takes an average of 16.6 days before the pathology is discovered. On the other hand, where MRI is used, it takes only 7.6 days to make the diagnosis (*1*). Unlike the bone scan, MRI can provide sufficient evidence to diagnose a spondylodiscitis even when there is little or no bony destruction, but a positive bone scan is helpful to focus the MRI imaging and thus increase its accuracy.

In both cases the pathology was first discovered on plain radiographs, although they are the diagnostic tool with the lowest sensitivity (table II) and although the latent phase is known to be extended. It may take up to 8 weeks before the radiographs show any signs of the pathology (*3, 5, 9*) and usually the sagittal images present the best view (*4, 9*).

MRI seems to have the highest sensitivity (table II) and specificity (83 to 97%) in case of spondylodiscitis (*1, 2, 4, 10*), especially when medullary compression or abscess formation are present, as in 37% of the cases (*4*). The gadolinium contrast images increase the accuracy of the investigation even more and are crucial in the exclusion of tumoral pathology and the evaluation of soft tissue damage (*1, 5*).

CT is most useful in the evaluation of the extent of bone destruction at the primary investigation (4), and in the course of the further follow-up of the patient. Wirtz *et al* (*10*) proved that CT images can show signs of osseous consolidation and regression of inflammation as soon as 5 to 6 weeks after initiating the antibiotic treatment. The alternation of the

Table III. — Change in signal intensity on MRI during the inflammatory process (*10*)

	NMR T1	NMR T2
Progressive inflammation	Hypo-intense signal	Hyper-intense signal
Regressive inflammation	Hyper-intense signal	Hypo-intense signal
Healing phase	Normalisation of signal intensity	

signal intensity on T1 and T2 MRI images also allows us to differentiate between progression, regression and healing of spondylodiscitis (table III), but the signs of regression seem to appear later on MRI, after an average of 12 weeks (*10*). According to these findings, the evolution of CT images corresponds better with the decrease of CRP, which is the most reliable parameter for the severity of the inflammation.

ESR and clinical symptoms are the essential parameters during the healing phase of a spondylodiscitis. In most cases, the pathology will disappear without sequels, but fusion of the involved vertebrae is seen in 0 to 44% of the cases (*1, 6*). Scoliosis and kyphosis or decreased lumbar lordosis also prevail but functional restrictions are rare (*1, 4, 9*).

To summarize, we could say that MRI plays a very important role not only in the process of diagnosis and differential diagnosis of juvenile spondylodiscitis, but also in the initial evaluation of soft tissue damage. CT and radiographs are sufficient and less expensive to evaluate the bony deformations during the healing phase.

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