The investigation and management of back pain in children

P J C Davis,[1] H J Williams[2]

[1] Department of Rheumatology, Birmingham Children's Hospital, Birmingham, UK; [2] Department of Radiology Birmingham Children's Hospital, Birmingham, UK

Correspondence to:
Dr H Williams, Departments of Radiology Birmingham Children's Hospital, Steelhouse Lane, Birmingham B4 6NH, UK; helen.williams@bch.nhs.uk

Accepted 19 February 2008

ABSTRACT

Back pain in children and adolescents is probably much less common than in adults, but its true incidence is unknown. Although back pain has traditionally been considered a rare and often sinister presentation in the paediatric age group, recent literature now suggests that a relatively high number of children do experience back pain, but only a small proportion seek medical attention. For the majority of children with back pain no underlying cause is identified, but some require investigation to exclude serious underlying pathology. Laboratory and imaging investigations should be targeted towards those with "red flag" symptoms and signs. Imaging studies, particularly MRI, have an important role in diagnosis of underlying pathology such as infection or malignancy.

INTRODUCTION

Back pain in children and adolescents is probably much less common than in adults, but its true incidence is unknown. One of the problems in determining the incidence and prevalence of back pain is the way in which it is defined. The majority of studies look at *low* back pain (LBP), often without defining the term, rather than *any* back pain. LBP may be variously defined as low back pain with no apparent clinical cause, non-specific pain or non-organic pain. It is also used as a general descriptive term for any type of back pain. The term mechanical back pain is also confusing as this may refer to pain without a pathological underlying cause, but conversely is also used to describe conditions arising from overuse or trauma such as muscle strain, intervertebral disc prolapse or even spondylolysis.

Although back pain has traditionally been considered a rare and often sinister presentation in the paediatric age group, recent literature now suggests that a relatively high number of children do experience back pain, but only a small proportion seek medical attention.[1] However, of those that do present to medical professionals, there is a larger proportion of pathology, especially in the pre-pubertal age group.

When assessing children and adolescents with back pain, it is important to consider lifestyle, psychological and social factors, because the presence of spinal pain does not necessarily equate with spinal disease. It is also essential to consider serious underlying pathology, and perform the most appropriate investigations to exclude these causes promptly, without over-investigating those patients (usually adolescents) in whom non-specific musculoskeletal pain is the cause. Imaging studies, particularly MRI, have an important role in diagnosis of underlying pathology such as infection or malignancy.

This article aims to provide a framework for assessing back pain in children and adolescents, as well as describing the presentation, imaging features and brief management of some of the underlying conditions that can cause back pain.

HISTORY

A thorough history and examination should guide the clinician as to whom to investigate and which investigations to perform. The nature, site of the pain and any radiation, plus exacerbating or relieving factors should be determined; for example, pain exacerbated by exercise and relieved by rest is infrequently associated with pathology. However, discitis or a spinal tumour in a toddler may present with obvious discomfort when lying supine for nappy changes, with relief of symptoms when picked up under the arms. History of a limp or altered gait raises similar suspicions in older children. Timing of the pain is important. Early morning stiffness and "gelling" (stiffness after a period of inactivity) suggest an inflammatory process, while nocturnal pain is highly indicative of a tumour or infection. Pain later in the day or after an active day is much less worrying. A history of recent infection (for example, urinary tract or staphylococcal infection with the possibility of haematogenous spread), exposure to atypical infections such as tuberculosis (TB), foreign travel and trauma may be relevant. The response to analgesia may give clues to an underlying cause. For example, pain related to osteoid osteoma, a benign bone tumour, characteristically shows marked improvement with non-steroidal anti-inflammatory drugs (NSAIDs), whereas unremitting pain points to infection or malignancy. Constitutional features such as reduced appetite, weight loss, fevers and night sweats also point to malignancy or infection, whereas abnormal bleeding or bruising suggests a haematological or disseminated malignancy. Enquiry into the menstrual history and presence of abnormal symptoms, such as vaginal discharge in girls, may be helpful, because gynaecological disorders such as pelvic inflammatory disease (PID) or ovarian pathology are occasionally associated with back pain.

A history of change in posture or mobility should be sought, and direct enquiry made regarding the presence of neurological symptoms such as weakness or "pins and needles" and any alteration

Table 1 Symptoms and signs that may indicate serious underlying pathology in children with back pain—"red flags"

Red flags: history	Red flags: examination
Pre-pubertal children especially <5 years	Fever, tachycardia
Functional disability	Weight loss, bruising lymphadenopathy or abdominal mass
Duration >4 weeks	Altered spine shape or mobility
Recurrent or worsening pain	Vertebral or intervertebral tenderness
Early morning stiffness and/or gelling	Limp or altered gait
Night pain	Neurologic symptoms
Fever, weight loss, malaise	Bladder or bowel dysfunction
Postural changes: kyphosis or scoliosis	
Limp or altered gait	

in bladder or bowel function. Medication history should identify use of drugs such as corticosteroids which are associated with osteoporotic vertebral crush fractures. Other relevant factors are hobbies (particularly sporting activities) and the type, style of carrying and weight of the school bag. It is also important to ask whether school teachers have noticed a change in the child's behaviour or activity and if the child is missing school. Family members' experience of back pain (and its cause), or illness associated with chronic pain may be relevant either due to an increased risk of a particular condition, or it may support abnormal illness behaviour. Important or disrupting issues related to the family or school environment should also be noted.

EXAMINATION

A general physical examination including pulse and temperature is indicated to detect any evidence of infection or malignancy, such as lymphadenopathy or an abdominal mass. Skin rashes, joint changes, muscle spasm or wasting are important findings. A full spinal examination includes palpation of the entire spine for vertebral or intervertebral point tenderness, which can be an indication of bone or disc pathology. The antero-posterior curvatures of the spine should be assessed, looking for kyphosis, or flattening of the lumbar curve along with a measurement of forward flexion, lateral flexion and extension. Forward flexion is gauged by Schober's measurement. A mark is placed 10 cm above and 5 cm below the level of the sacral dimples and the distance between the two marks measured. The patient then attempts to touch their toes. An increase in the distance between the two marks of less than 7 cm raises concerns about lumbar mobility.

If there is a scoliosis, this should be assessed to determine whether it is *structural* (fixed) or *non-structural* (reversible). In structural scoliosis there is always vertebral rotation or a bony abnormality such as a congenital vertebral anomaly. The curve is fixed and does not disappear with changes in posture, such as bending forward or sitting. Non-structural scoliosis disappears with postural changes or when the underlying cause is removed. Causes include abnormal posture, pelvic tilt due to muscle spasm, hip pathology or leg length discrepancy which should be identified during the examination. The sciatic stretch test is performed by elevating the straight leg to approximately 60° and dorsiflexing the foot; induced pain is an indication of sciatic nerve root irritation. Finally, a detailed neurological examination should be performed including an assessment of the gait.

INVESTIGATIONS FOR BACK PAIN

There is no standard work-up for back pain. Rather, the history and examination findings should highlight those patients requiring investigation and help to determine which investigations are performed. Certain symptoms and signs are "red flags" that should prompt investigation, particularly in pre-pubertal children (table 1). If there are signs of infection, microbiological investigation including blood cultures, radiograph (CXR) and Mantoux test are indicated. Concerns over possible malignancy warrant assessment of the acute phase reactants, full blood count with blood film and lactate dehydrogenase (LDH). Abdominal ultrasound and urinary catecholamines may also be indicated, the latter to exclude neuroblastoma.

THE ROLE OF IMAGING IN BACK PAIN

Diagnostic imaging is indicated when pain fails to settle with conservative measures such as analgesia and physiotherapy; if pain or functional difficulty escalates, or when the symptoms and signs are suspicious of an underlying condition such as those discussed below. However, it is important to remember that underlying pathology is uncommon in children, with some conditions (for example, intervertebral disc herniation or spinal tumours) being extremely rare. For patients in whom imaging is warranted, spinal radiography may be helpful in the first instance to search for a bony tumour, osteomyelitis, discitis, Scheuermann's disease, spondylolisthesis and spondylolysis, but does have serious limitations. In the majority of cases the next appropriate investigation is MRI. However, the choice of CT, MRI or even bone scintigraphy will depend on the suspected underlying pathology and the child's age.

Increasingly, MRI is considered to be the "gold standard" and used as the first-line imaging investigation when a serious underlying pathology is suspected. In fact, a normal MRI scan effectively excludes most pathology but it is an expensive investigation and requires sedation or anaesthesia in younger children, often limiting its availability to specialist children's hospitals or sites where there is suitable anaesthetic support. Where there is particular clinical concern, or any uncertainty regarding the most appropriate use of imaging, individual cases should be discussed with a radiologist with appropriate paediatric musculoskeletal experience. Absence of local radiological expertise should not prevent a child from having the correct

imaging investigation. Such cases may require advice from or referral to the local specialist centre.

JUVENILE IDIOPATHIC ARTHRITIS

The cervical spine is typically involved in juvenile idiopathic arthritis (JIA). The thoracic and lumbar spine are not usually affected—therefore back pain associated with JIA is very rare. Onset of enthesitis-related JIA is usually in late childhood or adolescence, with boys being affected much more than girls. There may be evidence of sacro-iliitis at presentation but spinal involvement is usually much later, and the pain is associated with stiffness. Loss of lumbar lordosis and flattening of the lumbar spine on forward flexion, with reduced range of spinal motion are found on examination.

Plain radiographs have a low sensitivity for detecting early changes of sacro-iliitis and spinal involvement in JIA. Both bone scintigraphy and CT will detect disease earlier but involve considerable radiation exposure. MRI (with gadolinium-based contrast enhancement) is recommended because it can detect synovial changes and inflammatory changes in the marrow, without irradiation.[2]

Patients with JIA should be referred to a specialist paediatric rheumatologist for further management, which may be shared with the referring paediatrician. Physiotherapy has an important role to establish good posture and prevent loss of range of movement in patients with inflammatory arthritis.

SPONDYLOLYSIS AND SPONDYLOLISTHESIS

Spondylolysis is the term used to describe an anatomic defect or disruption in the pars interarticularis, which is the weakest part of the vertebral body and located between the lamina and superior facet. Spondylolysis is thought to arise from repetitive sub-clinical injury or microtrauma, analogous to a stress fracture. It is more common in patients who take part in sports that involve repetitive hyperextension and rotational spinal loading—for example, gymnastics, cricket (particularly bowling), contact sports, rowing and dancing.[3] The most commonly affected site is the lower lumbar spine, mainly L5, although patients with L4 spondylolysis are more frequently symptomatic.[4] However, the majority of children with spondylolysis are actually asymptomatic. Hereditary factors may predispose some patients to develop spondylolysis and some pars interarticularis defects occur secondary to minor congenital abnormalities of the lumbosacral spine.[5]

Spondylolisthesis is the forward translation of one vertebral segment with respect to the vertebra below, which can lead to compression of the exiting nerve roots. Bilateral pars interarticularis defects allow this translation to happen. In children and adolescents the most frequently affected site is the L5/S1 segment.[5]

Spondylolysis and spondylolisthesis are rare in children under 5 years of age and more likely to be found in patients over 10 years of age.[6] Typical symptoms are low back pain, occasionally radiating to the buttock or posterior thigh. Onset is usually insidious although less commonly it may follow an acute injury. Lumbar flexion and extension are often limited, and lumbar hyperextension may elicit pain.

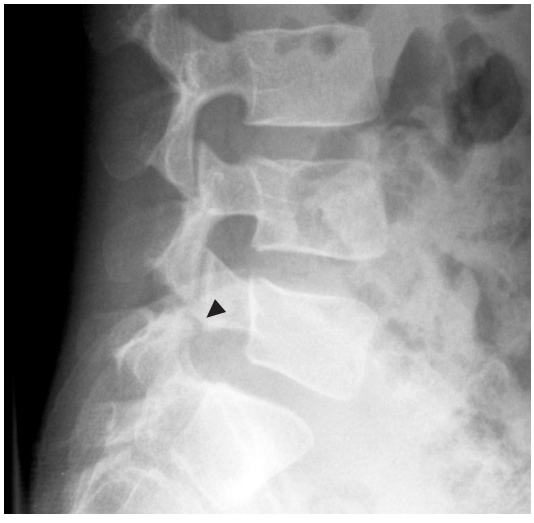

Figure 1 Lateral view of the lower lumbar spine with pars defect (arrowhead) of L5. There is also a low grade spondylolisthesis at L5/S1 indicating a bilateral pars defect. The left and right pars interarticularis are superimposed on the radiograph, there was bilateral abnormality on CT (fig 3).

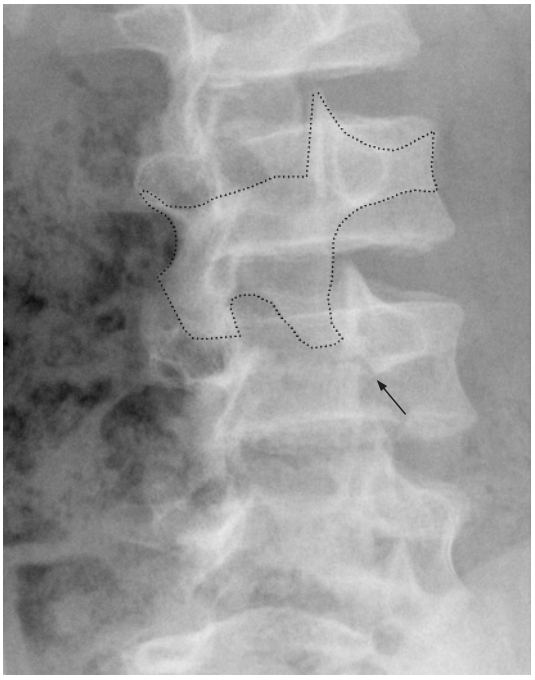

Figure 2 Oblique lumbar spine radiograph to demonstrate the pars interarticularis. A "Scottie dog" appearance is formed by drawing an outline around parts of the vertebral body. In this case, the left superior facet forms the ear, the nose is the left transverse process and the front leg the left inferior facet. A pars defect appears as a lucency giving the appearance of a dog collar (arrow) at the affected level.

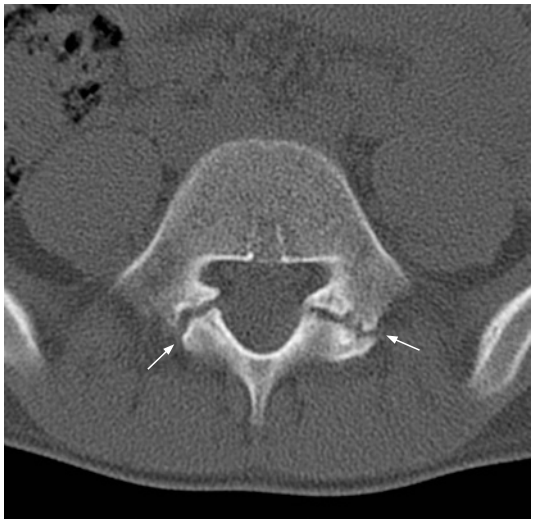

Figure 3 Axial CT showing bilateral pars defects (arrows).

Symptoms of nerve root compression, bladder and bowel disturbance are uncommon in spondylolysis and low-grade spondylolisthesis but may occur in high-grade spondylolisthesis (slip >50% of the antero-posterior vertebral body width), such patients may have a flattened lumbar lordosis and abnormally tight hamstrings on examination. With advanced slip, the sacrum is more vertically oriented and a visible or palpable step-off of the spinous processes at the affected levels may be found.[5]

Pars defects can be identified with plain radiography, bone scintigraphy, CT and MRI. Conventional radiography is the initial imaging modality of choice. Standing posterior-anterior and lateral lumbar spine radiographs including the lower thoracic spine and sacrum should be performed. Spondylolytic defects may be identified on these views (fig 1), particularly if there is accompanying slip, but oblique views of the lumbar spine allow better visualisation of the pars interarticularis (fig 2). However, these views can be difficult to interpret if the image quality is suboptimal due to patient positioning. They are also unreliable with early/acute lesions, including "stress reaction" without disruption of the bony cortex. Therefore, even with normal oblique radiographs, further imaging is indicated if the clinical suspicion remains high.

Localised CT is most accurate at defining the bony abnormalities associated with spondylolysis[7] (fig 3). In view of the limitations of oblique views, it may be appropriate to proceed directly to localised CT of the lumbar spine if the initial posterior-anterior and lateral views are negative, but this will depend on local preference and should be discussed with a radiologist first. A recent study suggests that MRI is an effective and reliable first-line imaging modality in children and adolescents.[8] However, detection of spondylolisthesis is not always possible from routine MRI sequences performed to exclude other pathology in the spine. The authors used specific sequences to visualise the pars interarticularis and also suggest localised CT following MRI for acute or indeterminate defects, with follow-up CT to assess healing. However, MRI is particularly useful when there are neurological symptoms accompanying spondylolisthesis.

Patients with stress reaction in the pars interarticularis without a defect have the potential to heal and immobilisation or brace treatment is indicated, followed by physiotherapy and gradual re-introduction of activity once pain subsides.[5] Management of symptomatic patients with spondylolysis is aimed at alleviation of pain and improvement of spinal mobility, rather than bony healing. Restricted activity and physiotherapy to strengthen the abdominal and paraspinal muscles, and stretch the tight hamstrings are beneficial.[9]

Patients who do not respond to rest and physiotherapy may require brace treatment. Surgery is generally reserved for patients who do not respond to a minimum of six months of conservative treatment. Various pars repair techniques are described including screw fixation and wiring of the defects to allow them to heal.[10] A similar conservative approach to the child with symptomatic spondylolisthesis with restriction of activity, physiotherapy and brace treatment is indicated in the first instance. Those with high-grade slip are less likely to respond to non-surgical management. Progressive slip and neurological deficit are also indications for surgery, involving fusion with or without reduction of the spondylolisthesis and nerve root decompression.[10]

SCHEUERMANN'S DISEASE

The exact aetiology of Scheuermann's disease is not known, although it has been attributed to a growth variation or repeated trauma. There is a familial association in some patients suggesting a genetic cause. The incidence is probably between 1–8% of the general population although this may be an underestimation with some undiagnosed cases being attributed to poor posture.[11] Onset is usually around puberty and patients present with kyphotic deformity of the thoracic or thoracolumbar spine. Associated pain is usually mild and typically occurs after prolonged periods of sitting or exercise. There may also be focal tenderness at the apex of the thoracic curve. On examination, affected patients have a greater than 40° fixed kyphosis of the thoracic spine that remains visible on hyperextension and forward bending. There is also often exaggeration of the normal cervical and lumbar lordosis although these parts of the spine retain flexibility. In contrast, postural kyphosis results in a uniform rounding of the entire spine with forward bending.

Spinal radiographs are usually sufficient to confirm the clinical diagnosis of Scheuermann's disease. The radiographic criterion for Scheuermann's disease is >5° anterior wedging of at least three adjacent vertebral bodies. There may also be irregularity and flattening of the vertebral end plates, narrowed intervertebral disc spaces and Schmorl's nodes (extrusion of intervertebral disc

substance through the vertebral end plate). Up to a third of patients have scoliosis in addition to kyphosis.[12] MRI is reserved for patients in whom the diagnosis is unclear or when there are symptoms suggestive of other pathology such as intervertebral disc prolapse.

For the majority of patients the course of the disease is benign and symptoms subside with skeletal maturity. Modification of activity and NSAID treatment is sufficient in terms of management for most patients but those with severe deformity may require spinal bracing or surgery.

INTERVERTEBRAL DISC HERNIATION/PROLAPSE AND DISC DEGENERATION

Disc degeneration is reported to occur in 6–16% of 10–19-year-olds.[13 14] However the relation between disc degeneration and back pain in children is controversial. Pain is not always present in association with disc changes detected on MRI, with some studies reporting no significant difference between the incidence of degenerative disc changes detected in symptomatic and asymptomatic adolescents. However, when associated with structural changes—for example, Scheuermann's disease or disc prolapse—disc degeneration is much more likely to be symptomatic.[15] Other authors have associated disc degeneration with increased incidence of recurrent back pain beyond the adolescent growth spurt into early adulthood.[16 17] There is a definite population of children who present with chronic low back pain associated with degenerative disc disease identified on MRI. It is likely that lumbosacral degenerative disc disease—commonly thought to exist only in an older population—actually begins earlier in selected patients.[18] Most children and adolescents with symptomatic disc degeneration respond well to medical management with pain relief, and physiotherapy including advice regarding posture, activities and exercise.

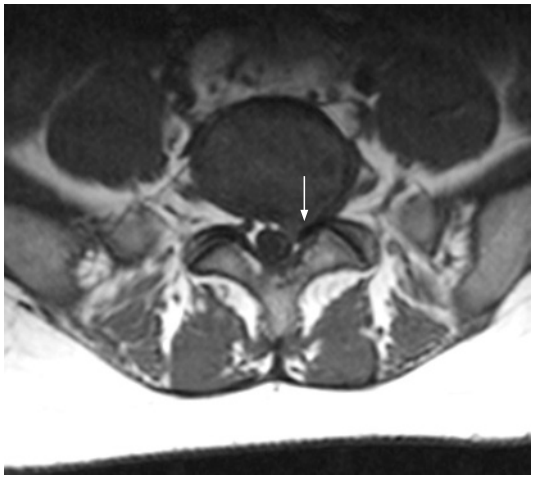

Figure 4 Axial T1-weighted MRI showing a left-sided disc prolapse (arrow) at L5/S1 level in a teenager with back pain and sciatica. The prolapsed disc is encroaching on the neural exit foramen obliterating the fat within it, and the left L1 nerve root is not visible compared with the normal right side.

Herniation or prolapse of intervertebral discs is a common cause of back pain in adults, frequently associated with age-related disc degeneration. It is rare in children and adolescents, only accounting for 1–4% of all cases of disc herniation.[19] It is far more common in adolescents than in pre-pubescent children, although disc herniation in children under the age of 10 years is described,[20] usually following severe trauma such as road traffic accident (RTA). Diagnosis may be delayed if the condition is not suspected or with late presentation. Older children are usually more cooperative with clinical examination including elicitation of signs such as pain on straight-leg raising. They are also able to give a more reliable history of symptoms suggestive of disc herniation such as sciatica.

There are varied reports of the incidence of associated symptoms in published case series. Generally, up to two thirds of patients with disc herniation will have localised pain, and one third or more have symptoms of nerve root irritation or compression such as sciatica.[21 22] Onset of symptoms may occur after strenuous activity such as sport or following an episode of trauma.[19] Disc herniation may be seen in patients with Scheuermann's disease.[23]

MRI is the imaging modality of choice in patients of any age with suspected disc herniation. It accurately defines the affected level, size and site of the herniated disc. For example, whether it is central (posterior disc protrusion) or lateral (fig 4). In children, the L4/5 and L5/S1 discs are most commonly affected[24] and the herniated disc material is usually larger than in adults. Any compressive effect of the herniated disc on nerve roots or at higher levels on the spinal cord can also be assessed, as well as accompanying narrowing or stenosis of the spinal canal or neural exit foramina. Treatment is conservative except in the small proportion of patients with severe or persistent nerve root pain, or progressive neurology who require surgery. Injection of chymopapain (an enzyme obtained from the papaya plant) into the herniated disc has been successful in adolescents with disc herniation.[25]

DISCITIS AND VERTEBRAL OSTEOMYELITIS IN CHILDREN

Discitis and vertebral osteomyelitis are uncommon diagnoses in children but are quite different, both in terms of severity and prognosis compared with adult-onset forms of the disease. Childhood discitis is often a benign, self-limiting condition that responds to conservative management. Although discitis can occur at any age in childhood there is a higher incidence in the toddler age group and a second subtler peak in late childhood/early adolescence.[26]

The blood supply to the intervertebral disc and cartilaginous vertebral end plate in children differs from adults, which could predispose to infectious

agents settling in and around the disc. In the fetus, infant and young child up to around 7 years of age, numerous anastamotic channels in the vertebral end plate communicate with the disc. They may be an important source of nutrition for the disc but also allow haematogenous delivery of bacteria to the disc. The anastamotic channels gradually involute leaving end arteries in the vertebral end plate by adolescence. The nucleus pulposus of the disc contains no blood or lymphatic vessels at any age but blood vessels are present in the annulus fibrosus up to 20 years of age.[27 28]

Childhood discitis frequently presents in a non-specific way, often with mild symptoms, making it difficult to diagnose.[29] A common presenting symptom in toddlers is refusal to walk or sit. Limping, gait disturbance, hip or leg pain or a need to hold onto objects for support are also typical. Back pain is common and may be the presenting symptom at all ages, whereas fever or abdominal pain can be presenting symptoms, more commonly in adolescents.[30] Restricted spinal mobility and loss of lumbar lordosis may be found on clinical examination. Biochemical markers of inflammation are usually only slightly raised and blood cultures usually negative.

Discitis can affect any spinal level but most frequently affects the thoracic and lumbar spine. Usually only one disc space is affected. If more than one level is affected, tuberculosis (TB) infection should be excluded. Radiographic signs of discitis may not be seen until 3–8 weeks after the onset of symptoms when loss of disc height and end-plate irregularity is seen (fig 5). The imaging modality of choice is MRI of the spine. Characteristic findings of reduction in disc height and abnormal disc signal with accompanying vertebral end plate irregularity are seen (fig 6). Abnormal enhancement of the disc and adjacent vertebral bodies, particularly the end plates, is frequently demonstrated. Enhanced scans may also demonstrate paravertebral inflammatory masses and abscesses.

Most authors propose an infective cause for childhood discitis,[26] however most cultures, including vertebral and disc biopsies, are sterile and some patients recover without antimicrobial treatment. Trauma and an inflammatory process of unknown cause have also been implicated in the aetiology of discitis. This uncertainty makes decisions about treatment more difficult, although the usual choice is anti-staphylococcal antibiotics. Depending on local resources and experience, biopsy may be performed, although it is often reserved for patients not responding to antibiotic treatment, immunocompromised patients and those in whom TB or fungal infections are suspected. Some centres do not routinely prescribe antibiotics unless there are signs of systemic toxicity. Generally there is a good prognosis for recovery and lack of long-term complications following discitis in childhood.[31]

Pyogenic vertebral osteomyelitis and TB spondylitis are thought to occur due to organisms lodging in low flow end vessels close to the cartilaginous vertebral end plate following haematogenous spread.[26] Again, differences in the vascular anatomy

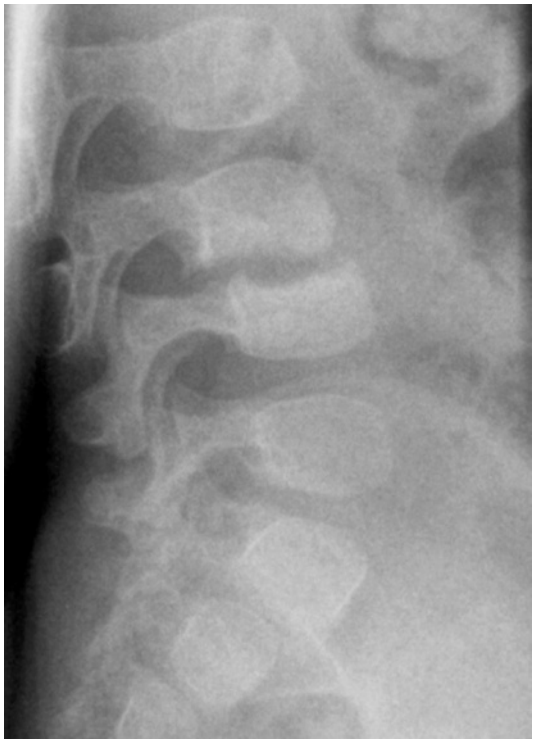

Figure 5 Lateral lumbar spine radiograph of a 13-month-old who refused to sit. There is disc space narrowing at L3/4 with associated superior and inferior end plate irregularity. There is also slight posterior slip of L3 on L4. MRI confirmed discitis with end plate bony changes. Biopsy was performed but there was no bacterial growth on prolonged culture.

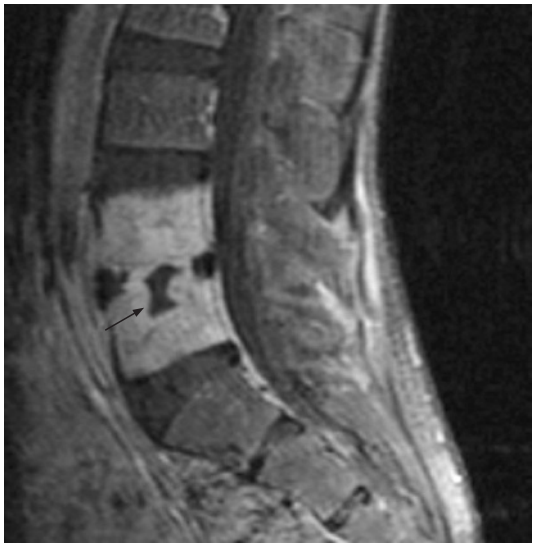

Figure 6 Enhanced T1-weighted sagittal MRI with fat suppression. There is L4/5 discitis with an associated abscess that does not enhance (arrow). There is also abnormal enhancement of the adjacent vertebral bodies indicating osteomyelitis. Tuberculosis is a possible cause. The patient is an 11-year-old boy with new onset back pain; results of microbiological investigation are awaited.

of the vertebral bodies in children and adults result in differences in spinal infections in these two groups. Infants and small children have widespread anastamoses between intraosseous arteries of the vertebral bodies which decrease the chance of infarction, and also enhance the likelihood of clearance of bacteria after septic embolisation to the vertebral body. These anastamotic channels decrease in number leading up to adolescence and have disappeared by adulthood.[32] The peak incidence of vertebral osteomyelitis occurs during adolescence and in those aged over 50 years.

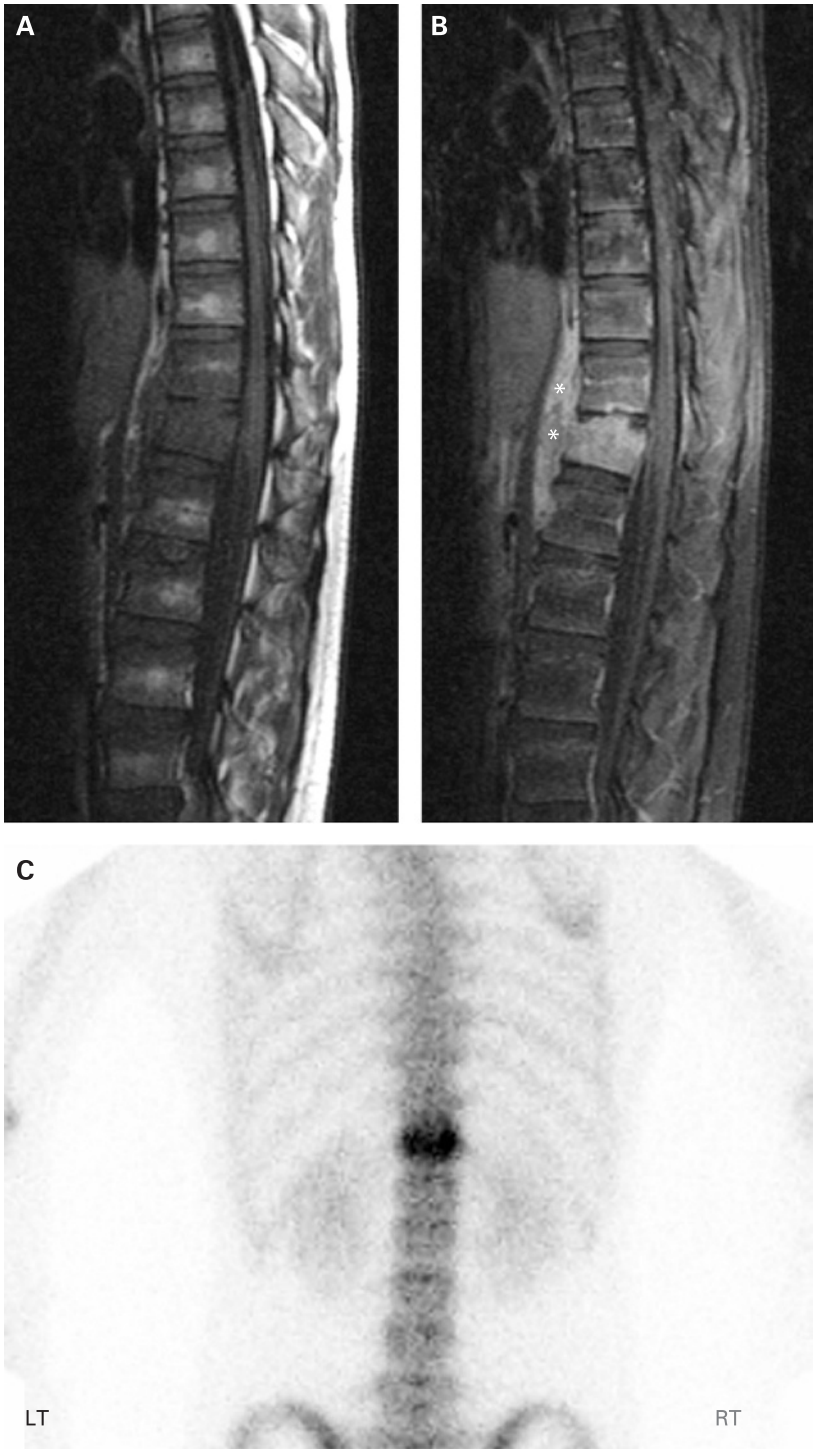

Figure 7 Pre- (A) and post-contrast (B) T1-weighted sagittal MRI scans showing T11/12 discitis and T12 vertebral osteomyelitis in an adolescent with back pain. The affected areas enhance and there is pre-vertebral enhancing soft tissue in addition (arrow). No organism was identified and he recovered with antibiotic treatment. (C) This patient was initially investigated with bone scan because he is severely autistic and required general anaesthetic for MRI. There is increased uptake of isotope at the affected level.

Back pain is common in vertebral infection. Patients with TB spondylitis frequently present with abnormal neurology, bladder or bowel symptoms, although the onset of the illness is usually insidious which helps to differentiate it from pyogenic vertebral osteomyelitis.[33] In suspected pyogenic or TB vertebral infection, the primary imaging modality of choice is MRI. This demonstrates the extent of vertebral and intervertebral disc involvement, together with complications such as paraspinal, epidural or vertebral abscess formation or soft tissue inflammatory masses (fig 7A–C). Identification of the infecting organism may require imaging-guided or open biopsy with culture and histological examination of the material obtained, in addition to blood cultures and culture of other potential sources of infection such as urine. Treatment is with anti-TB chemotherapy or appropriate intravenous antibiotics, usually for 2–6 weeks' duration, followed by prolonged treatment with oral antibiotics. Response to treatment can be monitored using the ESR and CRP. Immobilisation may be required for pain relief and surgical drainage of abscesses is indicated. Acute surgical intervention may be required if there is neurological deficit or to stabilise the spine.[34]

CHRONIC RECURRENT MULTIFOCAL OSTEOMYELITIS

Chronic recurrent multifocal osteomyelitis (CRMO) is an unusual form of chronic non-suppurative inflammatory bone disease involving multiple sites, characterised by multiple exacerbations and spontaneous remissions. Some children do not have multiple lesions or a recurrent course and these children have a better prognosis. Some authors have used the term chronic non-bacterial osteomyelitis (CNO) to describe this form of the disease.[35] It is likely that the accepted terminology will change and CNO will replace CRMO in the future.

The aetiology of this disorder is uncertain—so far, no infective cause has been identified—but the disorder may be immunologically mediated and there is evidence of genetic susceptibility in family studies.[36] Children and adolescents are most frequently affected with lesions found most commonly in long and short limb bones (tubular bones), ribs and clavicle. The spine and pelvic bones are less frequent sites but, if affected, back pain may be the presenting feature. Primary spinal involvement is rare.[37]

Radiographically, CRMO resembles subacute or chronic osteomyelitis but can also mimic tumour such as Ewing's sarcoma. Vertebra plana deformity (anterior wedging and reduced height of the vertebral body) may occur and the adjacent intervertebral discs may be involved. Spinal lesions

in CRMO are frequently multiple but not often in adjacent vertebral bodies. Furthermore CRMO is not associated with formation of abscesses or soft tissue inflammatory masses which are seen in TB or pyogenic osteomyelitis.[37] Histopathology and laboratory findings are non-specific and bacterial culture is usually negative. These conditions are diagnosed in patients with a characteristic clinical course after exclusion of bacterial infection and tumour, which may require biopsy. Bone scintigraphy and MRI have an important role in diagnosis,[38] because they can identify clinically occult lesions. Both conditions are treated with NSAIDs, but oral corticosteroids may be necessary in severe recurrent cases.

TUMOURS

Spinal tumours may be primary or secondary affecting the bony spine (vertebral tumours), leptomeninges or the spinal cord. A painful scoliosis, persistent pain with night pain and steadily increasing pain or disability should prompt careful evaluation for a spinal tumour. However, many patients present with vague symptoms leading to delayed diagnosis. Neurological complaints are uncommon. Clinically, the symptoms may be similar to infection—therefore appropriate laboratory studies are also indicated.

The most common benign primary vertebral tumours are osteoid osteoma, osteoblastoma and aneurysmal bone cyst. Malignant tumours of the spine in childhood and adolescence include Ewing sarcoma, lymphoma, leukaemia, and metastatic disease. As a general rule, benign lesions are more frequently found in the posterior elements of the vertebral body. Common metastatic lesions in the paediatric age group include neuroblastoma (which is rare over 7 years of age), rhabdomyosarcoma, Ewing's sarcoma, malignant germ cell tumours and lymphoma. They are usually lytic destructive bone lesions with a large soft tissue component that may invade the spinal canal and cause neurological symptoms. Metastases may also disseminate in the cerebrospinal fluid (CSF).

Spinal cord tumours include astrocytoma and ependymoma and are rare in childhood. They are most frequently located in the cervical spinal cord. Pain is the most common presenting symptom, usually preceding other symptoms such as weakness, gait deterioration, torticollis, sensory disturbance and sphincter disturbance.[39] Tumours arising from the nerve sheath include neurofibromas and schwannomas. Neurofibromas may be solitary or multiple and are associated with NF1. A detailed description of the clinical and imaging features of spinal tumours can be found in several recent review articles.[39–41]

Radiographs of the spine may detect tumours but will then require further evaluation with MRI or CT. In the case of vertebral tumours the two techniques are often complimentary (fig 8A and B). MRI is mandatory for meningeal, cord or nerve sheath tumours (fig 9). If the initial radiograph is negative and MRI is not available, bone

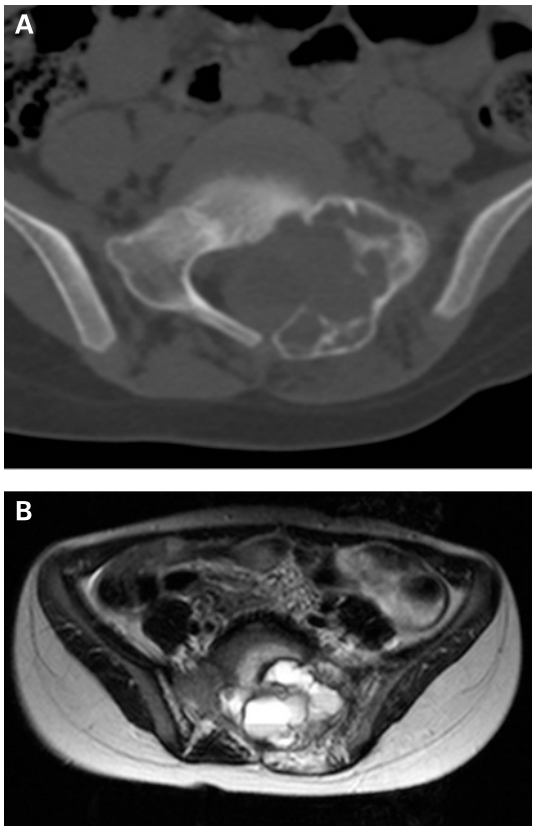

Figure 8 (A) Axial CT of the upper sacrum in a 4-year-old child with back pain and altered gait. There is a left-sided expansile destructive bone lesion, predominantly affecting the posterior elements, with extension into the sacral canal. (B) Axial T2-weighted MRI scan demonstrates a multiloculated mass with fluid-fluid levels consistent with an aneurysmal bone cyst.

scintigraphy may be used to evaluate the bony spine; however, such studies have a low sensitivity for detecting vertebral tumours.[42] Furthermore, CT or MRI would be required to evaluate any abnormality detected scintigraphically. However, bone scintigraphy is necessary in the staging of malignant bone tumours following diagnosis. Children with vertebral tumours should be managed in a specialist bone tumour unit. These tumours are often difficult to treat and management issues include maintaining spinal stability, preservation of neurological function and the effects of treatment on the immature skeleton.[43] Similarly leptomeningeal and spinal cord tumours require appropriate management from specialist paediatric oncology and neurosurgical teams.

METABOLIC AND SYSTEMIC CAUSES OF BACK PAIN

Osteoporosis is characterised by a generalised decrease in bone mass with a normal mineral to matrix ratio. Osteopenia or increased radiolucency of bone has numerous causes including osteoporosis and occurs when bone resorption exceeds bone formation. Primary osteoporosis is rare in children and usually caused by osteogenesis imperfecta (OI) (fig 10). Idiopathic juvenile osteoporosis is an

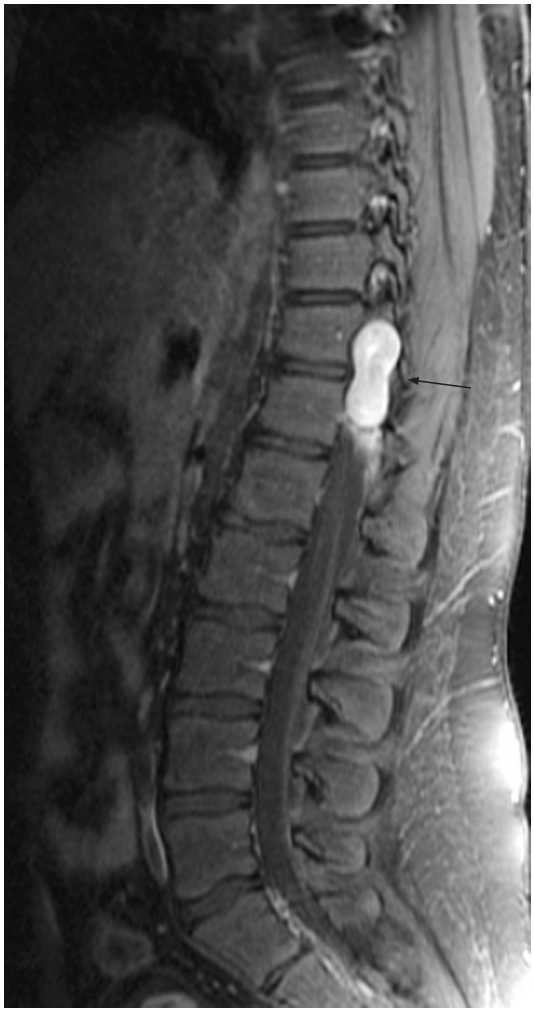

Figure 9 Enhanced T1-weighted sagittal MRI scan (with fat suppression) of an adolescent with back pain and upper motor neurone signs in both lower limbs. There is a well-defined enhancing tumour in the spinal canal at T11–12 level (arrow). Histology following excision confirmed this was a benign schwannoma.

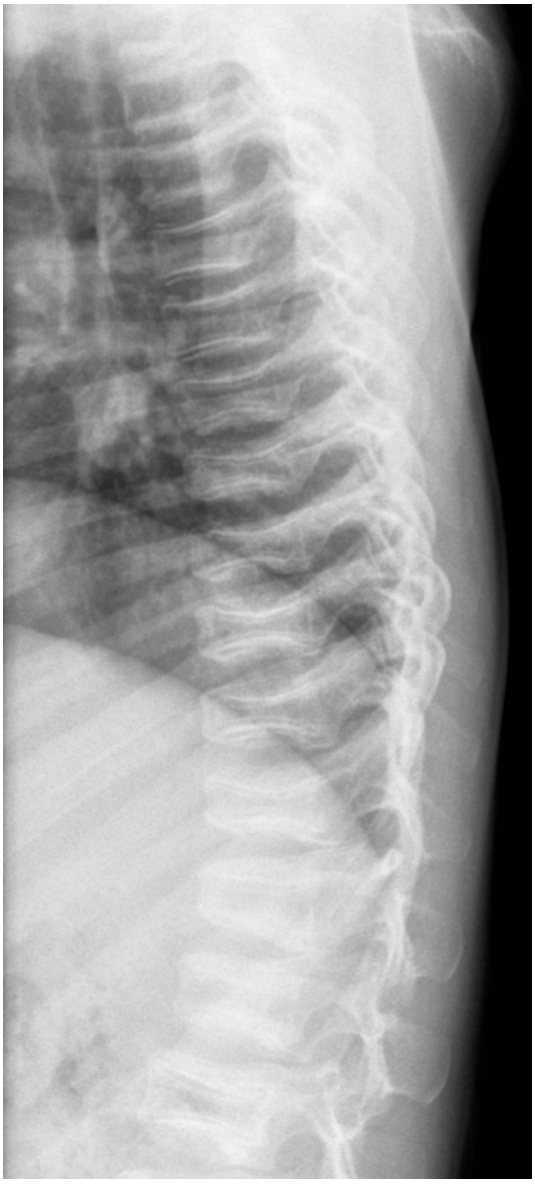

Figure 10 Generalised vertebra plana deformity due to insufficiency fractures of the vertebral bodies in a 10-year-old with osteogenesis imperfecta.

uncommon self-limiting disease of childhood. Presentation is usually about two years before puberty with spinal and extraspinal symptoms that may simulate an inflammatory arthritis. The mechanism may be due to defective osteoblastic function or increased osteoclastic activity leading to excessive resorption of bone, however the exact cause is not known. Most laboratory parameters are normal including serum calcium, phosphorus and alkaline phosphatase levels.[44]

Osteoporosis can also occur secondary to chronic diseases and their treatment, including JIA (particularly the systemic onset subgroup), hepatobiliary and inflammatory bowel disease, nutritional disturbance (for example, malabsorption, anorexia nervosa), renal disease, malignancy, endocrine disease and drugs, particularly corticosteroids. Osteoporosis in itself is not painful but osteoporosis affecting the thoracic and lumbar spine may be combined with insufficiency fractures (vertebral crush/compression fractures). The pain is typically acute in onset following trauma or activity which may not necessarily be strenuous or prolonged. Spinal radiographs usually confirm the diagnosis of vertebral compression fractures (fig 11), although subtle fractures may only be visible on MRI.

Osteoporosis secondary to an inflammatory condition such as juvenile idiopathic arthritis requires control of the underlying inflammation. In steroid-induced osteoporosis the dose of steroids should be reduced where possible. Calcium and vitamin D supplementation should be considered if diet is inadequate, and weight-bearing exercise encouraged. Bisphosphonates have an established role in the treatment of severe forms of osteogenesis imperfecta and juvenile idiopathic osteoporosis, and may be indicated for osteoporosis accompanying other disorders to reduce the

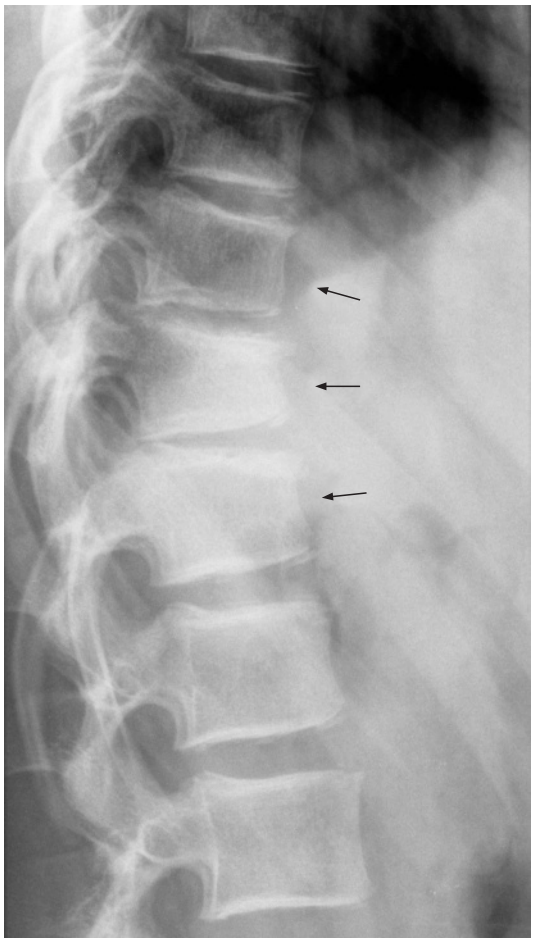

Figure 11 Crush fractures of T10–T12 (arrows) in an adolescent following a road traffic accident. This child did not have osteoporosis.

incidence of fractures. However, bisphosphonates are not licensed in children and treatment should be initiated and monitored in a specialist centre.

In children with sickle cell disease the spine is a common site for acute bony vaso-occlusive crises.[45] Patients may have acute bony tenderness and reduced range of movement during the episode. In the acute setting, adequate pain relief and hydration are required, with some patients requiring blood transfusion. Over time, structural changes occur in the vertebral bodies due to marrow hyperplasia and avascular necrosis, leading to collapse of the vertebral bodies in a significant proportion of patients. Infective spondylitis is a serious complication of sickle cell disease, with some patients needing surgery to decompress the spine, or bone grafting.[46]

OTHER CAUSES OF BACK PAIN

Muscular pain can be caused by asymmetrical load-bearing, such as poor posture, carrying a heavy school bag over one shoulder or a pelvic tilt secondary to a leg length discrepancy. In these cases simple analgesia such as paracetamol or ibuprofen may be useful. Attention should be paid to ensuring good posture as well as teaching techniques for bending and lifting. Advice may be required regarding the appropriate style of school bag and manner in which it is worn (for example rucksack worn over both shoulders rather than one). Specific exercises to strengthen the trunk and lumbar muscles may be indicated. Any leg length discrepancy must be corrected by the use of a shoe raise. Similarly, patients with hypermobility have an altered posture due to increased lumbar lordosis, genu recurvatum (hyperextension of the knees when standing) and pes planus (flat feet). These patients respond well to correction of the pes planus with moulded insoles to improve the medial arch of the foot.

The final group of patients have no demonstrable physical cause for their back pain. This is a diagnosis of exclusion, after appropriate work-up reveals no underlying cause. These conditions have been variously termed fibromyalgia, generalised idiopathic pain syndrome, complex regional pain syndrome and localised idiopathic pain syndrome. Their symptoms may have been initiated by minor trauma but no underlying pathological lesion is found. The symptoms and pain response on examination are out of proportion to the physical findings in such patients. However, their symptoms are real and are due to a vicious circle of initial pain with resultant muscle spasm and poor posture which compound and perpetuate the pain. The role of stresses in the family or school environment is often contributory and appropriate counselling may be indicated in such cases. For these patients, a multidisciplinary approach is required and may include techniques such as graded desensitisation, physical rehabilitation, distraction and relaxation. Simple methods for pain relief such as warm baths or ice packs are often helpful.[47]

CONCLUSION

Back pain in children requires careful, thorough assessment of symptoms and signs. Pre-pubertal children are more likely to have serious underlying pathology, whereas adolescents are more likely to have non-specific back pain with no demonstrable pathological cause. Laboratory and imaging investigations should be targeted towards those with "red flag" symptoms and signs. Imaging has an important role in diagnosis of most underlying conditions, but is also helpful to exclude pathology in selected cases. The choice of diagnostic imaging should be discussed with a radiologist with appropriate paediatric musculoskeletal experience. For the majority of conditions, (with the exception of tumours) surgery is considered only after a thorough trial of conservative management.

Competing interests: None declared.

REFERENCES

1. **Balague F,** Dudler J, Nordin M. Low-back pain in children. *Lancet* 2003;**361**:1403–4.

2. **Johnson K.** Imaging of juvenile idiopathic arthritis. *Pediatr Radiol* 2006;**36**:743–58.
3. **Herman MJ,** Pizzutillo PD, Cavalier R. Spondylolysis and spondylolisthesis in the child and adolescent athlete. *Orthop Clin North Am* 2003;**34**:461–7, vii.
4. **Saraste H.** Long-term clinical and radiological follow-up of spondylolysis and spondylolisthesis. *J Pediatr Orthop* 1987;**7**:631–8.
5. **Cavalier R,** Herman MJ, Cheung EV, *et al*. Spondylolysis and spondylolisthesis in children and adolescents: I. Diagnosis, natural history, and nonsurgical management. *J Am Acad Orthop Surg* 2006;**14**:417–24.
6. **King HA.** Back pain in children. *Orthop Clin North Am* 1999;**30**:467–74, ix.
7. **Harvey CJ,** Richenberg JL, Saifuddin A, *et al*. The radiological investigation of lumbar spondylolysis. *Clin Radiol* 1998;**53**:723–8.
8. **Campbell RS,** Grainger AJ, Hide IG, *et al*. Juvenile spondylolysis: a comparative analysis of CT, SPECT and MRI. *Skeletal Radiol* 2005;**34**:63–73.
9. **O'Sullivan PB,** Phyty GD, Twomey LT, *et al*. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine* 1997;**22**:2959–67.
10. **Cheung EV,** Herman MJ, Cavalier R, *et al*. Spondylolysis and spondylolisthesis in children and adolescents: II. Surgical management. *J Am Acad Orthop Surg* 2006;**14**:488–98.
11. **Wenger DR,** Frick SL. Scheuermann kyphosis. *Spine* 1999;**24**:2630–9.
12. **Lowe TG.** Scheuermann's disease. *Orthop Clin North Am* 1999;**30**:475–87, ix.
13. **Powell MC,** Wilson M, Szypryt P, *et al*. Prevalence of lumbar disc degeneration observed by magnetic resonance in symptomless women. *Lancet* 1986;**2**:1366–7.
14. **Miller JA,** Schmatz C, Schultz AB. Lumbar disc degeneration: correlation with age, sex, and spine level in 600 autopsy specimens. *Spine* 1988;**13**:173–8.
15. **Tertti MO,** Salminen JJ, Paajanen HE, *et al*. Low-back pain and disk degeneration in children: a case-control MR imaging study. *Radiology* 1991;**180**:503–7.
16. **Salminen JJ,** Erkintalo M, Laine M, *et al*. Low back pain in the young. A prospective three-year follow-up study of subjects with and without low back pain. *Spine* 1995;**20**:2101–7; discussion 2108.
17. **Salminen JJ,** Erkintalo MO, Pentti J, *et al*. Recurrent low back pain and early disc degeneration in the young. *Spine* 1999;**24**:1316–21.
18. **Dimar JR 2nd,** Glassman SD, Carreon LY. Juvenile degenerative disc disease: a report of 76 cases identified by magnetic resonance imaging. *Spine J* 2007;**7**:332–7.
19. **Epstein JA,** Epstein NE, Marc J, *et al*. Lumbar intervertebral disk herniation in teenage children: recognition and management of associated anomalies. *Spine* 1984;**9**:427–32.
20. **Martinez-Lage JF,** Fernandez Cornejo V, Lopez F, *et al*. Lumbar disc herniation in early childhood: case report and literature review. *Childs Nerv Syst* 2003;**19**:258–60.
21. **Bunnell WP.** Back pain in children. *Orthop Clin North Am* 1982;**13**:587–604.
22. **Mason DE.** Back pain in children. *Pediatr Ann* 1999;**28**:727–38.
23. **Kapetanos GA,** Hantzidis PT, Anagnostidis KS, *et al*. Thoracic cord compression caused by disk herniation in Scheuermann's disease: a case report and review of the literature. *Eur Spine J* 2006;**15**(Suppl 5):553–8.
24. **DeLuca PF,** Mason DE, Weiand R, *et al*. Excision of herniated nucleus pulposus in children and adolescents. *J Pediatr Orthop* 1994;**14**:318–22.
25. **Bradbury N,** Wilson LF, Mulholland RC. Adolescent disc protrusions. A long-term follow-up of surgery compared to chymopapain. *Spine* 1996;**21**:372–7.
26. **Fernandez M,** Carrol CL, Baker CJ. Discitis and vertebral osteomyelitis in children: an 18-year review. *Pediatrics* 2000;**105**:1299–304.
27. **Rudert M,** Tillmann B. Lymph and blood supply of the human intervertebral disc. Cadaver study of correlations to discitis. *Acta Orthop Scand* 1993;**64**:37–40.
28. **Whalen JL,** Parke WW, Mazur JM, *et al*. The intrinsic vasculature of developing vertebral end plates and its nutritive significance to the intervertebral discs. *J Pediatr Orthop* 1985;**5**:403–10.
29. **Karabouta Z,** Bisbinas I, Davidson A, *et al*. Discitis in toddlers: a case series and review. *Acta Paediatr* 2005;**94**:1516–8.
30. **Crawford AH,** Kucharzyk DW, Ruda R, *et al*. Diskitis in children. *Clin Orthop Relat Res* 1991;**266**:70–9.
31. **Kayser R,** Mahlfeld K, Greulich M, *et al*. Spondylodiscitis in childhood: results of a long-term study. *Spine* 2005;**30**:318–23.
32. **Early SD,** Kay RM, Tolo VT. Childhood diskitis. *J Am Acad Orthop Surg* 2003;**11**:413–20.
33. **Andronikou S,** Jadwat S, Douis H. Patterns of disease on MRI in 53 children with tuberculous spondylitis and the role of gadolinium. *Pediatr Radiol* 2002;**32**:798–805.
34. **Tay BK,** Deckey J, Hu SS. Spinal infections. *J Am Acad Orthop Surg* 2002;**10**:188–97.
35. **Girschick HJ,** Raab P, Surbaum S, *et al*. Chronic non-bacterial osteomyelitis in children. *Ann Rheum Dis* 2005;**64**:279–85.
36. **Golla A,** Jansson A, Ramser J, *et al*. Chronic recurrent multifocal osteomyelitis (CRMO): evidence for a susceptibility gene located on chromosome 18q21.3-18q22. *Eur J Hum Genet* 2002;**10**:217–21.
37. **Anderson SE,** Heini P, Sauvain MJ, *et al*. Imaging of chronic recurrent multifocal osteomyelitis of childhood first presenting with isolated primary spinal involvement. *Skeletal Radiol* 2003;**32**:328–36.
38. **Jurik AG.** Chronic recurrent multifocal osteomyelitis. *Semin Musculoskelet Radiol* 2004;**8**:243–53.
39. **Houten JK,** Weiner HL. Pediatric intramedullary spinal cord tumors: special considerations. *J Neurooncol* 2000;**47**:225–30.
40. **Garg S,** Dormans JP. Tumors and tumor-like conditions of the spine in children. *J Am Acad Orthop Surg* 2005;**13**:372–81.
41. **Faingold R,** Saigal G, Azouz EM, *et al*. Imaging of low back pain in children and adolescents. *Semin Ultrasound CT MR* 2004;**25**:490–505.
42. **Sanpera I Jr,** Beguiristain-Gurpide JL. Bone scan as a screening tool in children and adolescents with back pain. *J Pediatr Orthop* 2006;**26**:221–5.
43. **Fenoy AJ,** Greenlee JD, Menezes AH, *et al*. Primary bone tumors of the spine in children. *J Neurosurg* 2006;**105**(Suppl 4):252–60.
44. **Resnick D.** Osteoporosis. In: Resnick D, ed. *Diagnosis of bone and joint disorders*. 4th edn. Philadelphia: WB Saunders Company, 2002:1783–1860.
45. **Roger E,** Letts M. Sickle cell disease of the spine in children. *Can J Surg* 1999;**42**:289–92.
46. **Sadat-Ali M,** Ammar A, Corea JR, *et al*. The spine in sickle cell disease. *Int Orthop* 1994;**18**:154–6.
47. **Isenberg D,** Miller J. *Adolescent rheumatology*. London: Martin Dunitz, 1999.

The investigation and management of back pain in children

P J C Davis and H J Williams

Arch Dis Child Educ Pract Ed 2008 93: 73-83
doi: 10.1136/adc.2006.115535