CASE REPORT

Acute haematogeneous upper cervical osteomyelitis in neonates
A REPORT OF TWO CASES

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Haematogenous osteomyelitis in newborns and infants usually occurs in the long bones and is rare in the short or flat bones. We present two neonates with osteomyelitis of the upper cervical spine affecting the second to fourth cervical vertebrae and the first and second cervical vertebrae, respectively. Despite some delay in diagnosis, both responded successfully to conservative treatment with antibiotics, a cervical collar and needle puncture. The latest follow-up at six and seven years, respectively, showed no persistent neurological deficit and a normal diameter of the cervical spinal canal on MRI.

Haematogenous osteomyelitis in newborns and infants occurs typically in long bones, the inflammatory process affecting mainly the metaphyses of the femur, humerus and tibia. Short or flat bones such as the scapula, ribs, calcaneal and ankle bones, metatarsals, metacarpals, pelvic and skull bones are less frequently involved.1-3 Characteristic symptoms are swelling, erythema, metaphyseal and articular tenderness and limitation of movement.3

When the inflammatory process develops in the axial skeleton, early diagnosis can be difficult owing to the limited range of joint movement and accessibility to physical examination. Local symptoms tend to be discrete, and may go unnoticed.1 Purulent vertebral osteomyelitis is rare in both children and adults, and there is nothing in the literature on the subject. We present two cases of cervical spine infection in a neonate.

Case 1
A baby boy was born in the 40th week of gestation weighing 4110 g. The pregnancy and delivery were normal. He was discharged from hospital on the fourth day. While at home he became restless and jaundiced, with episodes of apnoea and skin cyanosis, but was afebrile. Since the episodes of apnoea were becoming more severe, he was referred to hospital on day 19. His condition was poor on admission and investigations revealed elevated inflammatory markers: ESR 43 mm/h, serum CRP 96 mg/l and WBC 30.8 × 10³/mm³. Lumbar puncture and blood cultures proved positive for Staphylococcus aureus. A diagnosis of purulent meningitis and bacteriaemia was made and cefuroxime and netromycine therapy was started immediately. His condition improved, but seven days after admission he deteriorated, with respiratory insufficiency and episodes of apnoea. He was intubated and ventilated. Radiographs confirmed the diagnosis of pneumonia. After three days his condition stabilised and he was extubated. Inflammatory markers returned to normal. The following day a tremor was observed in the right upper limb. MRI did not reveal any abnormality in the brain, but considerable pathology was found within the cervical spine with heterogenous echogenicity in the second and third cervical vertebrae and marked enhancement following administration of contrast. The changes were reported to be inflammatory. Narrowing of the spinal canal and cord compression were also seen at this level. The lesions extended into the pre-spinal space, deforming the posterior pharyngeal wall on plain radiography (Fig. 1). There was also osteolysis of the third cervical vertebrae and changes in neighbouring vertebrae (Fig. 2).

Fine-needle puncture showed a non-specific inflammatory process, with cultures positive for Staph. aureus and Klebsiella pneumoniae. Intravenous antibiotic treatment with clindamycin and then meropenem (30 mg/kg/day) was commenced, and continued for eight weeks. The cervical spine was immobilised in the posterior pharyngeal wall on plain radiography (Fig. 1). There was also osteolysis of the third cervical vertebrae and changes in neighbouring vertebrae (Fig. 2).

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849
fourth cervical vertebrae, with persistent anterior and posterior bulging, but no compression of the spinal cord. Administration of contrast material yielded no signal enhancement. The retrospinal fluid space did not show any abnormality. The cervical 2/3 and 3/4 intervertebral discs were very thin. MRI of the cervical spine six years later showed persistent changes in the second and third cervical vertebrae but no reduction in the volume of the spinal canal (Fig. 3). There was no neurological deficit or limitation of movement of the cervical spine at this time.

Case 2
A baby girl with a weight at birth of 3100 g was born following a normal pregnancy and delivery. She was severely jaundiced at birth with a serum bilirubin level of 18.9 mg%. She was discharged from hospital on the seventh day after delivery. Seven days later she developed a pyrexia of 39°C, and paresis of the right upper limb.

Treatment with cefuroxime was started in the outpatient department. A neurological diagnosis of a right brachial plexus palsy was made and physiotherapy started. She became restless and at the age of five weeks developed paresis of the left upper limb and physiotherapy continued for the next seven days. At this time she was referred to hospital and on admission had a bilateral flaccid paresis with areflexia, including an absent Moro reflex. There was muscular atrophy of the shoulder girdle and upper limbs. Laboratory investigations revealed an elevated ESR 43 mm/h, CRP 78 mg/l and WBC $16.7 \times 10^3$ mm$^{-3}$. CT and MRI of the cervical spine revealed heterogenous changes in the posterior pharyngeal wall, which enhanced following administration of contrast (Fig. 4). The changes extended into the foramen magnum and the spinal canal, causing narrowing at the craniocervical junction. Bony degeneration was seen in the first and second cervical vertebrae. Intravenous antibiotics were administered for six weeks (meropenem 30 mg/kg/day, clindamycin 20 mg/kg/day) and a cervical collar was used for eight weeks. Her condition improved, her upper limb paresis recovered and her weight increased. Bacteriological investigations failed to detect the responsible pathogen, probably as a result of early outpatient antibiotic treatment. The serum CRP level at six weeks after treatment was 1 mg/l. MRI at the age of seven years showed remodelling of the first and second cervical vertebrae (Fig. 5), and at this time she was asymptomatic and there was no neurological deficit.

Discussion
The anatomical structure and vascular supply to the long bones may account for the primary location of haemato-
genous osteomyelitis in the metaphyses. However, the pathogenesis of haematogenous osteomyelitis in the flat and short
bones is not fully explained. So far, it has not been established where inflammation starts in the spine, whether within a vertebral body or an intervertebral disc. Song et al compared the structure of a developing vertebral body to that of a long bone with proximal and distal epiphyses and growth cartilage, but such a structure is more characteristic of other mammals, where distinct ossification areas are seen at the vertebral borders. Nevertheless, they believed that factors promoting inflammation within the metaphyses of long bones could also be active in vertebral bodies, especially their anterior portion, as the blood supply to the developing posterior area (arch, spinous process) is less abundant. The blood flow through the intervertebral discs is also subject to change; it is quite abundant in infants, decreases in children, and is minimal in adults. The abundant blood flow in neonates and infants could cause rapid spread of infection into the adjacent vertebrae, as was seen in our patients. Acute haematogenous osteomyelitis at this age can develop very rapidly, as in long bones leading to progressive osteolysis. Diagnosis is often delayed because physical examination can be difficult. A neonate may present with non-specific symptoms such as drowsiness, fever, reluctance to feed, vomiting, irritability, dysphagia, weight loss, episodes of apnoea, bradycardia and circulatory disturbances. Laboratory tests will frequently show elevation of inflammatory markers. Later on, symptoms may appear in the upper limbs, such as tremor, paresis and muscle wasting. Careful physical examination is essential. In the differential diagnosis the following should be considered: perinatal injury to the cervical spine or brachial plexus, a neoplastic lesion, especially neuroblastoma, and a specific inflammatory process such as tuberculosis.

Dich, Nelson and Haltalin, in a review of 163 cases, reported that the inflammatory process was located mainly in the vertebrae in only 1.2% of children treated for osteomyelitis. The percentage was even lower in infants and neonates. Vertebral infection is more common in adults, especially in post-operative patients with bacteriemia. Fernandez, Carrol and Baker reported that the mean age of a child with vertebral osteomyelitis is between six and nine years. The most frequent pathogen is *Staph. aureus*, followed by *Salmonella, Streptococcus pneumoniae* and *Pseudomonas aeruginosa*. However, in around a third of all patients identification of the pathogen is not possible. Aspiration or biopsy should be performed and the material sent for culture and histological analysis. Birmingham et al have devised a real-time quantitative reverse transcription polymerase chain reaction test. This method of detecting live bacteria and antibiotic sensitivity has the potential to dramatically improve the diagnosis and treatment of osteomyelitis. In neonates the diagnosis of osteomyelitis can be delayed owing to the absence of characteristic symptoms. However, because of the accompanying sepsis, antibiotic treatment may be started early. Later the development of neurological signs such as tremor, paresis, or episodes of apnoea and dysphagia suggest that the search for the site of infection should be continued, and diagnostic imaging ordered. The optimal antibiotic regimen and duration of therapy not have been clearly defined, and should be individualised for each patient. Initial antibiotic therapy should cover the most common causative pathogens and can be changed later, based on the results of microbiological tests. Antibiotic treatment may be required for six weeks. However, Jagodzinski et al have recently recommended a shortened regimen for the treatment of acute osteomyelitis, and septic arthritis in children. Children with methicillin-resistant *Staph. aureus* infection tend to have higher inflammatory parameters, longer hospitalisation and antibiotic needs, and a greater number...
of complications than those with a methicillin-sensitive *Staph. aureus* infection. In our two patients inflammation of the cervical spine was diagnosed relatively late, when bone destruction, seen on radiographs and MRI, was already quite severe (Figs 1, 2 and 4). Such damage to the vertebral bodies occurs with inflammatory conditions of two to three weeks’ duration. Radioisotopic bone scanning is more sensitive than plain radiography in detecting early bone involvement. CT and technetium bone scan, can be very useful in the further evaluation of these patients. Radioisotopic bone scanning is more sensitive than plain radiography in detecting early bone involvement. CT and MRI seem to be equally successful in revealing osteomyelitis. MRI examination has been shown to be more sensitive and specific, delineating the extent of bone and soft-tissue involvement. In our two patients, despite the delayed diagnosis the results of treatment were satisfactory. The inflammatory process subsided, bone lesions remodelled, and the neurological sequelae recovered. However, we believe they require long-term follow-up into adolescence and adult life. This will allow continuing assessment of the rate and degree of remodelling of the diseased vertebrae and intervertebral discs. Re-ossification can start several years after the infection. Although the infective process may involve the ossification centre of the epiphysis, it can spare the cartilaginous portion, which contains resting stem cells with the potential to regenerate the damaged portion of the epiphysis.

Like other authors, we can confirm the diagnostic value of MRI when inflammatory changes to bone are suspected, especially in the axial skeleton. MRI was also useful for the assessment of the results of treatment.

There were no indications for operative management in our two patients such as a large abscess, spinal cord compression or severe vertebral degeneration with a sequestrum.

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References