

Intermittent irritability in a toddler

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DESCRIPTION

We present the case of a healthy 15-month-old boy, who presented to our emergency department with intermittent unspecified dorsal pain and periods of irritability for the last 2 weeks. During the last week, parents reported significant worsening of his general condition with episodes of polypnoea, nocturnal awakening and progressive gait refusal. Pain was relieved in supine position and responded well to ibuprofen. No other symptoms were reported except diarrhoea 2 weeks prior to the beginning of symptoms. A fall from height was reported 7 weeks prior to admission, resulting in a minor lip incision with spontaneous healing; no history of trauma involving the back or legs was found. No relevant epidemiological context or family history was identified. On admission, he looked prostrated, uncomfortable while sitting, referring no pain on palpation/percussion of the spine. Spinal flexion and extension were limited by pain with asymptomatic passive mobilisation of the lower limbs. He refused standing or walking due to pain. On admission, he was afebrile (tympanic temperature 36.8°C), with a pulse of 132 bpm and blood pressure 98/54 mm

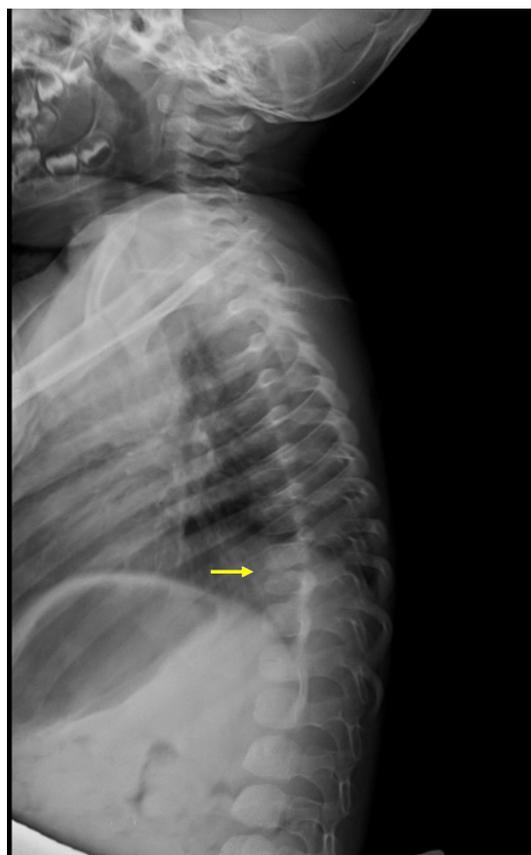


Figure 1 Vertebral X-ray, showing a decrease in disc height involving D9 and D10 vertebrae (arrow).



Figure 2 Vertebral CT, showing irregularity of D9-D10 vertebral bodies (arrow), with possible relation to bone destruction.

Hg. He started fever at the hospital that persisted for 2 days. Chest X-ray was normal but on lateral view, a decrease in disc height was evident on the low thoracic spine (figure 1). The blood panel revealed increased erythrocyte sedimentation rate (55 mm/hour) and alkaline phosphatase (1076 U/L), with normal C reactive protein and blood count. Spinal CT showed vertebral irregularity of D9-D10 with possible relation to bone peridiscal erosions suggesting spondylodiscitis (SD) (figure 2). MRI confirmed the diagnosis (figure 3). No agent was identified in microbiology and molecular studies. Mantoux test was not performed, but an Interferon Gamma Release Assays (IGRA) test for *Mycobacterium tuberculosis* was negative. Blood cultures for identification of *Kingella kingae* were also negative. After 8 days of intravenous ceftriaxone, he was asymptomatic and was discharged with oral amoxicillin and clavulanic acid for 5 weeks. No intercurrents or sequels were documented; at 2-year follow-up he had normal activity and X-ray showed a complete disc height restoration.

SD is a rare entity in paediatrics and diagnosis can be delayed because of low incidence and lack of awareness. In children, the incidence is about 0.3/100 000.¹ SD is characterised by an inflammatory process involving the intervertebral disc and adjacent vertebral bodies, resulting in symptomatic narrowing of the intervertebral space. It typically occurs in children younger than 6 years, and the lumbar discs are most commonly affected, but any disc may be involved.²⁻⁴ In most patients, spinal seeding occurs haematogenously from a previously existing site of infection. Pathogens however, can be exceptionally inoculated from a diagnostic/surgical procedure and following trauma.² In more than



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Figure 3 Vertebral MRI, with evidence of an inflammatory process involving D9 and D10 (arrow) with disruption of bone structure and intervertebral disc destruction.

half of the cases no micro-organism can be identified, but when isolated *Staphylococcus aureus* prevails.¹⁻⁴ Clinical manifestations vary with age. It is not unusual for toddlers to present only with non-specific signs such as irritability, and not the classic low back pain and antalgic gait.³ This can explain why diagnosis of SD can be difficult, as little advantage is derived quite often from the use of laboratory tests and conventional radiography.

Learning points

- ▶ The beginning of symptoms in spondylodiscitis is usually insidious and physical examination is often poor, therefore, it can go unnoticed.
- ▶ The low specificity of symptoms, such as irritability and normal range laboratory data, the scarcity of physical examination findings and the late radiographic changes are responsible for the usual late diagnosis of this disease, which depends largely on the clinician's suspicion.
- ▶ *Staphylococcus aureus* is still the most common isolated agent, although in the majority of reported cases no agent is identified.

A delay in establishing the diagnosis has been found, with studies reporting an average of 27 days going up to between 4 and 6 months.⁴ MRI is highly sensitive for an early diagnosis and tandem to clinical evaluation, an excellent examination for monitoring disease progression. Treatment includes rest, non-steroidal anti-inflammatory drugs, antibiotics, casting in some patients and physiotherapy if appropriate. Prognosis is generally good, especially with early diagnosis and adequate therapy followed by close clinical follow-up.^{2,3}

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