

Intraspinal canal rod migration causing late-onset paraparesis 8 years after scoliosis surgery

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Abstract



Introduction Complete intraspinal canal rod migration with posterior bone reconstitution has never been described in the adolescent idiopathic scoliosis (AIS) population. We present an unusual but significant delayed neurological complication after spinal instrumentation surgery.

Case report A 24-year-old woman presented with lower limb weakness (ASIA D) 8 years after posterior instrumentation from T2 to L4 for AIS. CT scan and MRI

demonstrated intra-canal rod migration with complete laminar reconstitution. The C-reactive protein was slightly elevated (fluctuated between 10 and 20 mg/l). Radiographs showed the convex rod had entered the spinal canal. The patient was taken into the operating room for thoracic spinal decompression and removal of the convex rod. This Cotrel–Dubousset rod, which had been placed on the convexity of the thoracic curve had completely entered the canal from T5 to T10 and was totally covered by bone with the eroded laminae entirely healed and closed. There was no pseudarthrosis. Intra-operatively, the fusion mass was opened along the whole length of this rod and the rod carefully removed and the spinal cord decompressed. The bacteriological cultures returned positive for *Propionibacterium acnes*. The patient recovered fully within 2 months post-operatively.

Conclusion We opine that the progressive laminar erosion with intra-canal rod migration resulted from mechanical and infectious-related factors. The very low virulence of the strain of *Propionibacterium acnes* is probably involved in this particular presentation where the rod was trapped in the canal, owing to the quite extensive laminar reconstitution.

Keywords Adolescent idiopathic scoliosis · Rod migration · *Propionibacterium acnes*

Case presentation

A 14-year-old female (with a Risser 3) underwent a T2–L2 posterior arthrodesis in 1998 for AIS. As a result of pull out of the superior hooks 8 months later, she underwent a re-operation necessitating removal of proximal hooks and shortening of rods. Subsequently, due to progressive

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kyphosis, an extension of the arthrodesis T2–L4 was performed in 1999. This patient continued with diffuse mid/low back pain, which was present at rest and worsened after a physical activity with a moderate effect on her daily activities. The subsequent X-rays were inconclusive (Fig. 1) and the C-reactive protein oscillated between 10 and 20 mg/l.

In 2002, a spinal CT scan from T11 to L5 and bone scintigraphy did not show any abnormalities. It was postulated that the pain was due to irritation of the 11th intercostal nerve, and therefore local anaesthetic infiltration was performed which vastly improved her symptoms for 3 years. A second infiltration was performed in 2007, but did not bring any improvement.

The patient was admitted to the emergency department in January 2008 complaining of difficulty with ambulation and lower limb paraparesis for 2 days (ASIA D), especially on the right side. On examination, she had spastic paraparesis with positive Babinski's sign on both lower limbs. A repeat CT scan showed intra-canthal migration of the convex rod with an overlying bony growth from T5 to T10 (Fig. 2) and interestingly, there had been no progression of the thoracic curve. The MRI scan revealed the thoracic spinal cord was being compressed by the rod, with a hyper-intensity signal spanning from T7 to T9 (approximately 5 cm in length) on the T2-weighted imaging (Fig. 3). The blood parameters showed an elevated C-reactive protein (CRP) at 37 mg/l.

Diagnostic imaging section

See Figs. 1, 2 and 3.

Historical review, epidemiology, diagnosis, pathology and differential diagnosis

Severe neurological complications related to spinal instrumentation for AIS usually occur during or shortly after the surgery. These can be due to either a malposition of the implants or a postoperative mechanical failure. Delayed neurological complications are caused by adjacent level disease in most cases. Late-onset spinal cord compressions by epidural abscess or epidural metallosis have been reported [1–4], but to the authors' knowledge, no rod-related compression has yet been described.

Delayed neurological complications after spinal instrumentation for AIS due to junctional problems are usually obvious on X-rays, showing adjacent disc degeneration and instability. Some authors [1–3] have found metallosis to be responsible for paraparesis due to compressive intra-canthal



Fig. 1 Lateral and antero-posterior X-rays, 1 year after T2–L4 posterior stabilisation

granuloma. In the two cases reported by Takahashi et al. [1], metallosis was related to loosening of a rod and hook at the lower end of a long fusion. However, the possible presence of low virulence organisms was not screened. Beguiristain et al. [3] pointed out the potential relationship between corrosion and biological agents. They described a case where metallosis was associated with a *Propionibacterium acnes* infection 14 years after the surgery. According to these authors, intra-operative contamination was unlikely given the delay between the surgery and the diagnosis of infection. The bacteria were probably attracted to the area of metallosis by haematogenous seeding and then interacted with the metal surface and corrosion products, leading to increasing corrosion of the metal.

In the case of our patient, we found no sign of corrosion or metallosis and the local reaction to the presence of *Propionibacterium acnes* was extremely low, since no collection or false membrane was found. However, the bacteria may have played a role in the laminar erosion and intra-canthal migration of the rod by inducing an acceleration of the bone remodelling phase. At the apex of the scoliosis, the convex rod generates compressive forces on the posterior arch. The micro-motion between the implants may

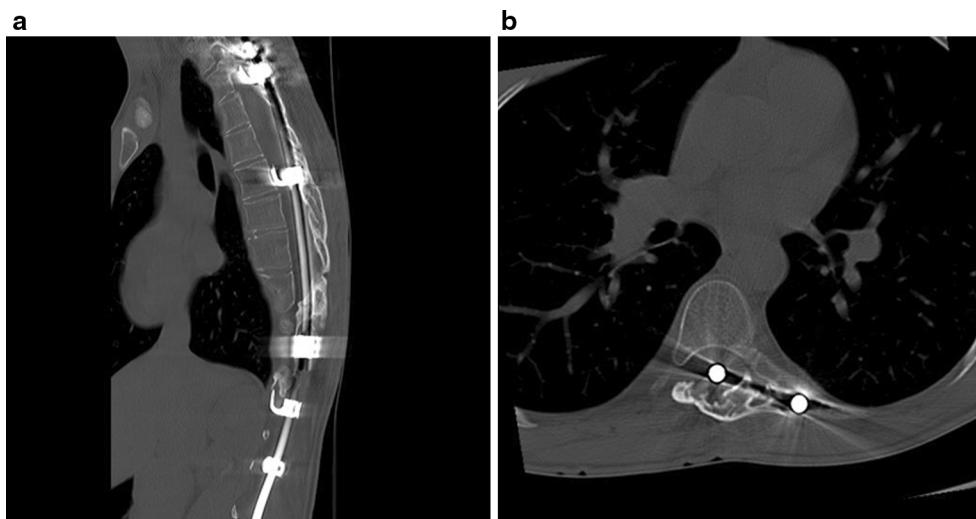


Fig. 2 CT: **a** sagittal reconstruction and **b** axial section showing the intraspinal migration of the convex rod

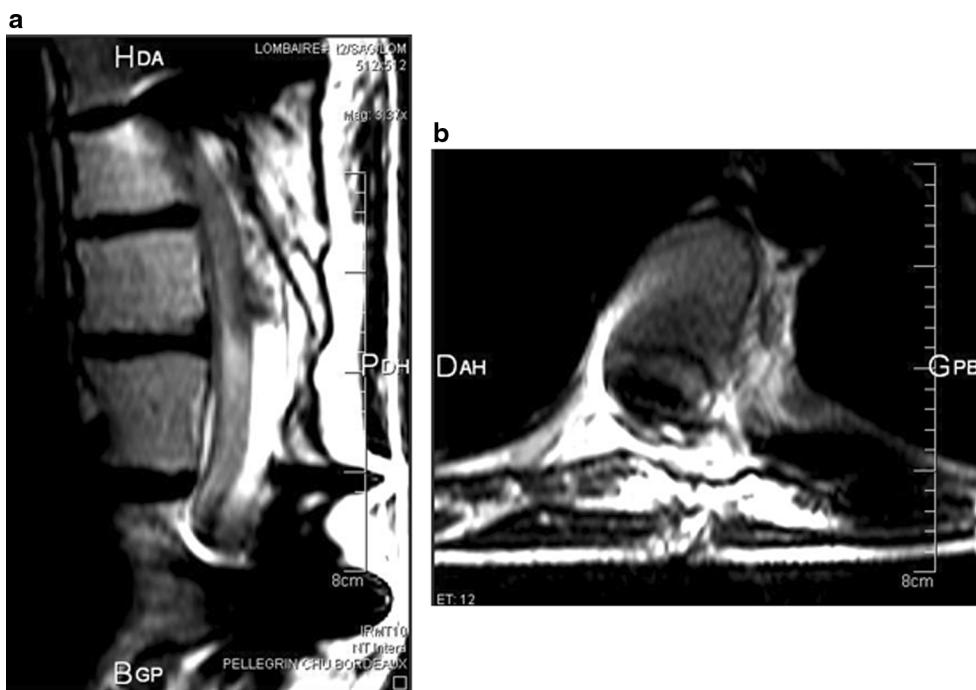


Fig. 3 T2-weighted MRI: **a** sagittal and **b** axial section showing the rod compressing the spinal cord

lead to an increased mechanical stress between the rod and the laminae. The infection may have contributed to the progressive laminar erosion around the rod and the virulence of the bacterial strain was sufficiently low to enable the bone formation at the same time. The rod was therefore trapped in the canal.

Tribus and Garvey [5] reported a case very similar to ours but the infection was at the forefront of the clinical presentation. Their patient underwent irrigation, debridement and hardware removal but the authors discovered a laminar defect and intra-canal rod migration. The

mechanism was probably the same as in our patient, combining mechanical with infectious factors, but the infection was more severe, leading to laminar erosion without reconstitution.

Rationale for treatment

Another problem is that of the difficulty in diagnosing late infections which occur in 2.6–6.9 % of cases [6, 7], but the signs are sometimes very mild. However, as illustrated by



Fig. 4 Intra-operative photograph showing the fusion mass covering the convex rod which had entered the spinal canal

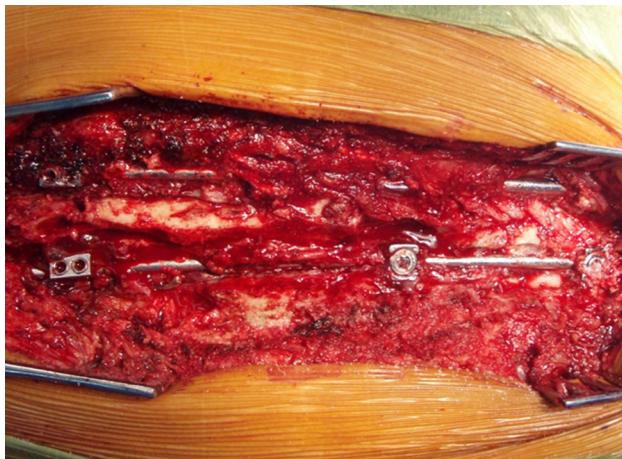


Fig. 5 Intra-operative photograph after opening the spinal canal along the entire length of the convex rod

our patient, the consequences can be very severe. Our patient presented with pain and paraparesis (ASIA D). The bone scintigraphy was not informative and the CRP slightly elevated (37 mg/l). Our radiological investigations (CT/MRI scans) had confirmed the diagnosis of intra-canal migration of the convex rod and resulting thoracic spinal cord compression.

Operative procedure

The patient was taken to the operating room for emergency spinal cord decompression and instrumentation removal. We discovered a Cotrel–Dubousset rod placed on the convexity of the thoracic curve, which had completely entered the spinal canal from T5 to T10. This

rod segment was totally covered by the fusion mass (Fig. 4). All the levels from T4 to L4 were completely fused without any evidence of bony erosion or pseudoarthrosis. The tissues in contact with the instrumentation were suspected of chronic infection, but we did not find any false membranes or liquid collection. The fusion mass was opened along the entire length of the rod (Fig. 5) to carefully remove all the instrumentation and decompress the spinal cord. The tissues surrounding the instrumentation were debrided and irrigation was performed.

15 days after the surgery, the bacteriological cultures returned positive for *Propionibacterium acnes* for four out of five samples. The histopathological results showed non-specific inflammatory lesions, but no metallosis. According to the recommendations of the infectious disease specialists, the patient was treated with a 2-week course of intravenous antibiotics followed by a 10-week oral intake.

Procedure imaging section

See Figs. 4 and 5.

Clinical outcome

The clinical recovery was complete (ASIA E) at the 2-month follow-up, and all the back pain and ribcage symptoms had entirely healed. We report here an exceptional cause of delayed spinal compression 8 years after spinal instrumentation for AIS. The progressive laminar erosion with intra-canal rod migration resulted in our opinion, from mechanical and infectious related factors. The very low virulence of the strain of *Propionibacterium acnes* is probably involved in this particular presentation where the rod was trapped in the canal, owing to progressive laminar reconstitution.

Conflict of interest None.

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