

Mehmet Tezer · Unal Kuzgun · Azmi Hamzaoglu
Cagatay Ozturk · Fevziye Kabukcuoglu
Mustafa Sirvanci

Intraspinal metalloma resulting in late paraparesis

Received: 16 September 2004 / Published online: 18 June 2005
© Springer-Verlag 2005

Abstract The metal-related complications caused by orthopedic implants have long been a concern, but these problems have been considered mostly in the field of arthroplasty or internal fixation of fractures. The recent prevalence of spinal instrumentation has evoked a similar concern among spine surgeons. Here, we present a case of intraspinal metallosis adjacent to the pedicular hook occurring after treatment of vertebral fracture by posterior spinal instrumentation and fusion, and causing paraparesis at the 3rd postoperative year. Metallic granulomas can appear around the pedicular hooks as in the reported case. Crevice and fretting corrosion are results at the junctions of rod-screw, rod-hook, transverse connector rod and other connector rods in modular spinal implants. Adequate usage of transpedicular screws may inhibit the occurrence of such a complication. For this reason, further studies are necessary to increase metallic corrosive resistance to inhibit crevice and fretting corrosion.

Keywords Metallosis · Pedicular hook instrumentation · Neurological complication · Paraparesis

Introduction

The metal-related complications caused by orthopedic implants have long been a concern, but these problems have been considered mostly in the field of arthroplasty or the internal fixation of fractures [7, 8, 9, 10, 12, 14, 20]. The recent prevalence of spinal instrumentation has evoked a similar concern among spine surgeons [31, 32, 33]. To the authors' knowledge, however, no metal-related complication has been reported yet regarding spinal instrumentation, except the case reported by Takahashi et al. [28], who reported two cases in which neurological symptoms in the form of radiculopathy appeared months to years after surgery from the formation of an epidural metallosis adjacent to a laminar hook without migration or dislodgement of the implants.

A case of intraspinal metallosis adjacent to the pedicular hook occurring 3 years after surgical treatment of vertebral fracture by posterior spinal instrumentation and fusion and causing paraparesis is presented in this study.

Case report

A 57-year-old male patient was referred to our clinic with a fracture of Th8-9 vertebrae as a result of falling from height. The neurological examination was found to be normal. He underwent posterior spinal surgery with a pedicle screw-hook combination system made of stainless steel with a diagnosis of Th8-9 compression fracture 2 days after the trauma. In the early postoperative period, no clinical or radiological abnormality was detected. During the regular follow-up visits, the patient

M. Tezer (✉) · C. Ozturk
Department of Orthopedic Surgery,
Florence Nigthingale Hospital,
Abide-i Hürriyet Caddesi no. 290,
80220 Istanbul, Turkey
E-mail: rezocagatay@hotmail.com
Tel.: +90-212-2244950
Fax: +90-212-2348689

U. Kuzgun
Department of Orthopedic Surgery, Sisli Etfal Hospital,
Istanbul, Turkey

A. Hamzaoglu
Department of Orthopedic Surgery,
Kadir Has University Medical School,
Istanbul, Turkey

F. Kabukcuoglu
Department of Pathology, Sisli Etfal Hospital,
Istanbul, Turkey

M. Sirvanci
Department of Radiology, Kadir Has University Medical School,
Istanbul, Turkey

was seen at the clinics every 6 weeks without any clinical and radiological abnormalities. At the end of the 3rd year, progressive paraparesis developed, beginning in his left lower extremity.

The last follow-up X-rays revealed caudal migration of the blocker of the pedicle hook at the Th6 level. The fractures had healed, and the sagittal and frontal spinal plane alignments were normal. Laboratory test results were insignificant.

In his neurological examination of the right leg, the muscle strengths of the flexors were 2/5 in the proximal, 5/5 in the distal, and normal in the extensors. In his left leg, proximal and distal flexion muscles were 0/5 in strength, and the extensors were normal. The superficial abdominal reflexes could not be obtained. There was sensory deficit at the level of Th5-6. Patellar reflexes were increased, and there were no Achilles reflexes. The bilateral sole reflexes were indifferent. There were no pathological findings in the upper extremities. The electromyographic examination revealed normal sensory transmission values. Denervation potential or motor unit action was not encountered in the lower extremities. In the muscles innervated by Th6-7 nerves, partial denervation findings were identified. With this finding, it was concluded that the lower extremity was affected at the level of the first motor neuron, and there were no other pathologies of the second motor neuron level, except the probable radicular involvement at Th6-7.

Computed tomography (CT) and magnetic resonance imaging (MRI) could not be obtained because of diffuse metal artifacts. Myelography and myelo-CT were applied. In the left posterolateral epidural area at the Th6-7 level, a focal image of a mass was seen anterolaterally displacing the dural sac and the spinal cord (Fig. 1A, B).

Fig. 1 Anterior-posterior (**A**) and lateral (**B**) myelography views showing the interruption at the Th7 level



Posterior surgical procedure was applied to the patient for excision of the mass. At the pedicle hook, the rod and the blocker, the brightness of the metal was lost; it was corroded and turned black (Fig. 2). At the other implanted parts, there was no loosening, color change of the metal or metallic area.

The construct was stable and strong. The fusion was complete. The implants were completely extracted, and the mass was completely excised (Fig. 3A, B). There was a defect of 1.5 cm in diameter in the lamina and pedicle. The metallic debris had seriously pushed the dural sac and the spinal cord to the anterior and contralateral side by more than 50%.

All metallic debris was cleaned, and tissue samples for microbiological and pathological examination were taken. The metals were kept for metallurgic analysis (Table 1).

No microorganisms were identified in the removed material. In the histopathological analysis, hematoxylin-eosin stained sections of the paraffin-embedded material showed dense fibrotic tissue heavily stained with black metal debris. There were numerous macrophages. Some foreign body giant cells were encountered around metallic debris. Iron staining of the tissue sections by Perls method showed the widespread presence of iron within macrophages (Fig. 4). Neurological deficit was completely improved at the 3-month follow-up period, and the patient is symptom-free.

Discussion

Potential adverse effects associated with the use of orthopedic metal implants include systemic metabolic, bacteriologic, immunologic, and neoplastic effects as

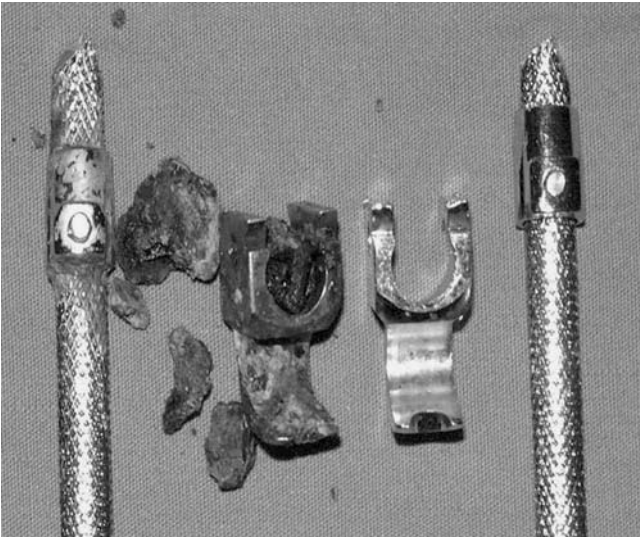


Fig. 2 Foreign body reactions and corrosions on the affected pedicular hook

well as local effects such as metallosis, bone resorption and mechanical compression of important soft tissues [12].

After the metallic implants are applied, tissue fluid accumulates around these metals, and a fibrous sheath forms in time [1, 15, 19, 22, 24, 32]. Metallic corrosion forms at the metal-bone union after micromovements [4,

6, 15]. Loosening at the metal-to-metal junctions and abnormal micro- or macro-movements cause metallic corrosion, and metallic debris start to accumulate in the fibrous tissue, forming a pseudosheath. These micro-particles trigger the foreign body reaction, with migration of the phagocytes [1, 13, 15, 18, 19, 21, 22, 26, 31, 32]. In the study of Case et al. [10] with a post-mortem control group, metallic debris infiltration in the spleen, liver and bone marrow in addition to the local lymph nodes has been shown in cases where stainless steel and cobalt-chrome prosthesis and implants were used. Accumulation of wear debris, which is biologically inert, causes loosening of the implant [17], resorption of bone [16], granuloma formation [29] and necrosis of bone marrow [2]. If this abnormal movement and inflammatory reaction is not interrupted, it continues as a vicious circle. We agree with Takahashi et al. [28] that both the abnormal movement and the chemical reaction caused by the metallic particles may cause metallosis. The abnormal metallic distribution of the metallic debris as detected by metallic analysis also shows that chemical features may also contribute to the formation of the reaction.

The most common corrosion type in metallic implants is crevice corrosion. This type of corrosion is encountered in 50% of extracted modular implants [25]. Crevice and fretting corrosion can be seen in three fourths of all stainless steel implants [11]. Crevice corrosion causes extreme accumulation of corrosion

Fig. 3 Peroperative views of the metallosis before (A) and after (B) extraction of the materials

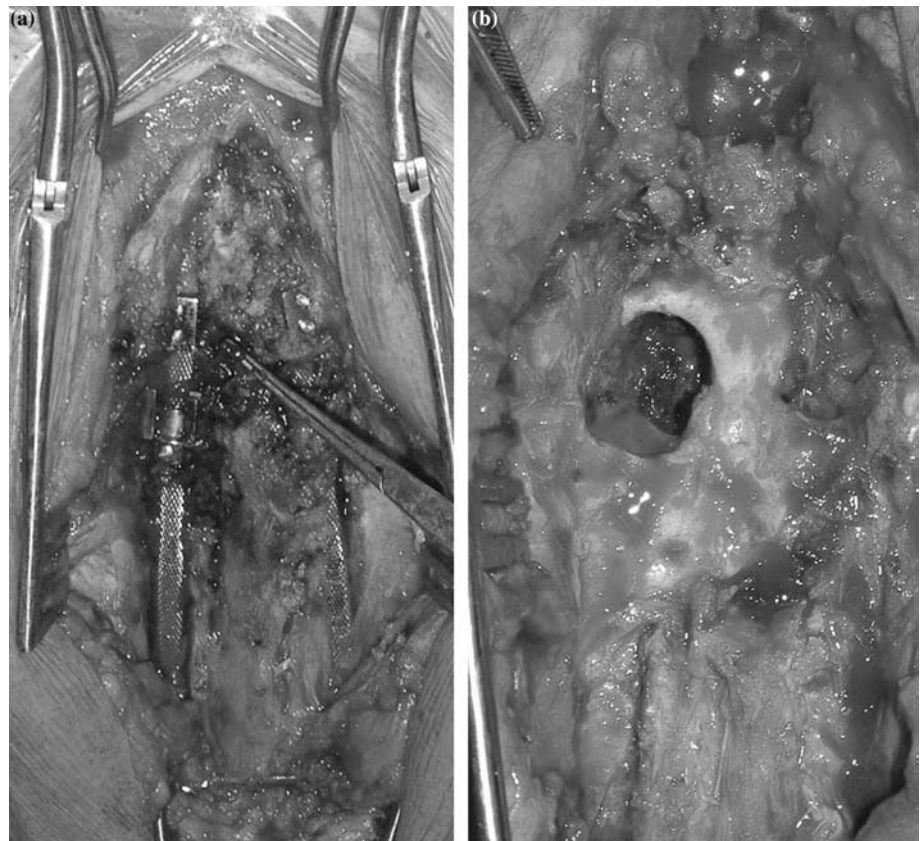


Table 1 The concentration of the metals in the excised tissue

Metal	Rates (%)
Mo	2.05
Cr	16.94
Ni	9.40
C	0.02

products or breakage of the implant by formation of cracks. Titanium can take a more corrosion-resistant form as TiO₂ after passivation, but the mechanical resistance is less than stainless steel and is subject to fretting corrosion [23]. In vitro studies have shown that the debris material of titanium inhibits osteoblastic activity, accelerates osteoclastic differentiation and causes osteolysis [5, 34]. Stainless steel is not a self-passivating material like titanium. When the oxidized film is broken, corrosion starts and progresses [23, 30].

Metal implants used in spinal instrumentation surgery may cause iatrogenic compression injury of the neural tissues, but delayed occurrence of such a complication is rare. The two cases reported by Takahashi et al. [28] were unusual in that the neurological complications occurred via the formation of an intraspinal soft tissue mass adjacent to a metal implant, and in that no direct compression by the implants was responsible for the neurological deficit.

Diagnosis of this condition was not simple. Magnetic resonance imaging (MRI) and CT imaging are not useful because of the artifacts around the metal implants, and electromyography generally does not provide specific localizing information. Myelography appears to be the sole informative diagnostic method. Myelography has also contributed to the diagnosis in our case of a space-occupying lesion starting at the hook of the pedicle and progressing into the canal. Therefore, it should

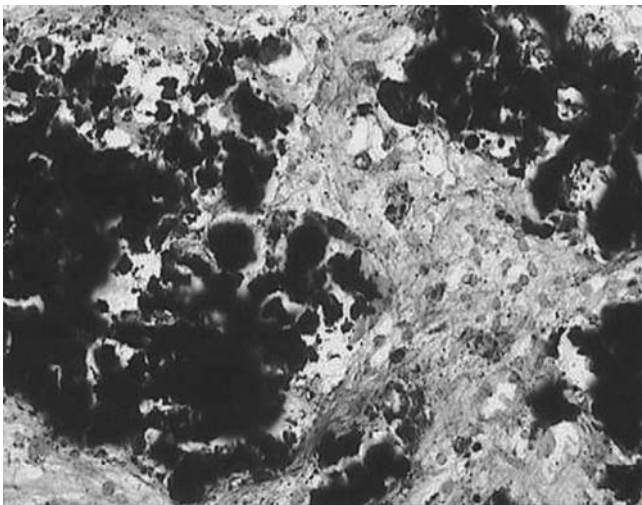


Fig. 4 Iron staining by the Perls method showing widespread presence of iron (Perls $\times 200$)

be performed without delay when intraspinal metallosis is suspected. Intraspinal metallosis should be kept in mind as one of the late complications associated with spinal instrumentation surgery.

Corrosion after spinal instrumentation may be due to metallurgical composition as well as the specific construction of the instrument [30]. In cases of metallosis, micromovements, especially at the rod-crosslink junction causing sterile flow without neurological deficit, have been reported after spinal instrumentation [3]. It has been reported that the caudal end of the spinal instrumentation is the most stressful part and is open to micromovements and pseudoarthrosis [27]. Also at the distal area of a fusion applied scoliotic curve, degenerative changes may occur. However, the granuloma formation was at the center of the metallic construct in our case. At the cranial part, there was a pedicle hook and a transverse process hook in the claw position, so it was an area where stress was not concentrated. The non-uniform distribution of the chemical properties and the long-term degeneration of the micromovements would be reasons for the granuloma formation in this area.

Metallic granulomas can appear around the pedicular hooks as in the reported case, similar to the other two cases reported in spinal implantology. Adequate usage of transpedicular screws may inhibit the occurrence of such a complication. Crevice and fretting corrosion are results at the junctions of the rod-screw, rod-hook, transverse connector-rod, and other connector-rods in modular spinal implants. For this reason, further studies are necessary to increase metallic corrosive resistance to inhibit crevice and fretting corrosion.

References

1. Agins HJ, Alcock NW, Bansal M, et al (1988) Metallic wear in failed titanium-alloy total hip replacements. A histological and quantitative analysis. *J Bone Joint Surg Br* 70:347–356
2. Amstutz HC, Campbell P, Kossovsky N, et al (1992) Mechanism and clinical significance of wear debris-induced osteolysis. *Clin Orthop* 276:7–18
3. Aydinli U, Karaeminogullari O, Tiskaya K (1999) Postoperative deep wound infection in instrumented spinal surgery. *Acta Orthop Belg* 65:182–187
4. Been HD, Kalkman CJ, Traast HS, et al (1994) Neurologic injury after insertion of laminar hooks during Cotrel-Dubousset instrumentation. *Spine* 19:1402–1405
5. Bi Y, Van de Motter RR, Ragap AA, et al (2001) Titanium particles stimulate bone resorption by inducing differentiation of murine osteoclasts. *J Bone Joint Surg Am* 83:501–508
6. Bischoff UW, Freeman MAR, Smith D, et al (1994) Wear induced by motion between bone and titanium or cobalt-chrome alloys. *J Bone Joint Surg Br* 76:713–716
7. Black J (1988) Editorial. Does corrosion matter? *J Bone Joint Surg Br* 70:517–520
8. Black J, Sherk H, Bonini J, et al (1990) Metallosis associated with a stable titanium-alloy femoral component in total hip replacement: a case report. *J Bone Joint Surg Am* 72:126–130
9. Bullough PG (1994) Metallosis (editorial). *J Bone Joint Surg Br* 76:687–688
10. Case CP, Langkamer VG, James C, et al (1994) Widespread dissemination of metal debris from implants. *J Bone Joint Surg Br* 76:701–712

11. Cook SD, Renz EA, Barrack RL, et al (1985) Clinical and metallurgical analysis of retrieved internal fixation devices. *Clin Orthop* 194:236–247
12. Friedman RJ, Black J, Galante JO, et al (1993) Current concepts in orthopaedic biomaterials and implant fixation. *J Bone Joint Surg Am* 75:1086–1109
13. Haynes DR, Rogers SD, Hay S, et al (1993) The differences in toxicity and release of bone-resorbing mediators induced by titanium and cobalt-chromium-alloy wear particles. *J Bone Joint Surg Am* 75:825–834
14. Iida H, Kaneda E, Takada H, et al (1999) Metallosis due to impingement between the socket and the femoral neck in a metal-on-metal bearing total hip prosthesis: a case report. *J Bone Joint Surg Am* 81:400–403
15. Jacobs JJ, Gilbert JL, Urban RM (1998) Current concepts review. Corrosion of metal orthopaedic implants. *J Bone Joint Surg Am* 80:268–282
16. Jasty M, Jiranek W, Harris WH (1992) Acrylic fragmentation in total hip replacements and its biological consequences. *Clin Orthop* 285:116–128
17. Kim KJ, Rubash HE, Wilson SC, et al (1993) A histological and biochemical comparison of the interface tissues in cementless and cemented hip prosthesis. *Clin Orthop* 287:142–152
18. Kummer FJ, Rose RM (1983) Corrosion of titanium/cobalt-chromium alloy couples. *J Bone Joint Surg Am* 65:1125–1126
19. Maloney WJ, Smith RL, Schurman DJ (1993) Fibroblast response to metallic debris in vitro. Enzym induction, cell proliferation, and toxicity. *J Bone Joint Surg Am* 75:835–844
20. Matsuda Y, Yamamuro T, Kasai R, et al (1992) Severe metallosis due to abnormal abrasion of the femoral head in a dual-bearing hip prosthesis: a case report. *J Arthroplasty [Suppl]* 7:439–445
21. Milosev L, Antolic V, Minovic A, et al (2000) Extensive metallosis and necrosis in failed prostheses with cemented titanium-alloy stems and ceramic heads. *J Bone Joint Surg Br* 82:352–357
22. Mody DR, Esses SI, Heggenes MH (1994) A histologic study of soft-tissue reactions to spinal implants. *Spine* 19:1153–1156
23. Pienkowski D, Stephens GC, Doers TM, et al (1998) Multicycle mechanical performance of titanium and stainless steel transpedicular spine implants. *Spine* 23:782–788
24. Pierini G, Fini M, Giavaresi G, et al (1999) Atomic absorption spectrophotometry (AAS) for the evaluation of metallosis in prostheses and artificial organs: a new approach. *Int J Artif Organs* 22:522–527
25. Shahgaldi BF, Heatly FW, Dewar A, et al (1995) In vivo corrosion of cobalt-chromium and titanium wear particles. *J Bone Joint Surg Br* 77:962–966
26. Steinemann SG (1996) Metal implants and surface reactions. *Injury* 27 [Suppl 3]:SC16–22
27. Takahashi S, Delecrin J, Passuti N (1997) Changes in the unfused lumbar spine in the patient with idiopathic scoliosis: A 5- to 9-year assessment following Cotrel-Dubousset instrumentation. *Spine* 22:517–524
28. Takahashi S, Delecrin J, Passuti N (2001) Intraspinal metallosis causing delayed neurological symptoms after spinal instrumentation surgery. *Spine* 26:1495–1499
29. Tallroth K, Eskola A, Santavirta S, et al (1989) Aggressive granulomatous lesions in hip arthroplasty. *J Bone Joint Surg Br* 71:571–575
30. Vieweg U, van Roost D, Wolf HK, et al (1999) Corrosion on an internal spinal fixator system. *Spine* 24:946–951
31. Wang JC, Yu WD, Sandhu HS, et al (1999) Metal debris from titanium spinal implants. *Spine* 24:899–903
32. Xu R, Ebraheim NA, Nadaud MC, et al (1996) Local tissue of the lumbar spine response to titanium plate-screw system. *Spine* 21:81–84
33. Yanase M, Sakou T, Taketomi E, et al (1995) Transpedicular fixation of the lumbar and lumbosacral spine with screws: application of the Diapason system. *Paraplegia* 33:216–218
34. Yau J, Cs-zabo G, Jacobs JJ, et al (1997) Suppression of osteoblast function by titanium particles. *J Bone Joint Surg Am* 79:107–112