Intraspinal metallosis are rare. The authors present a case after implantation of two titanium threaded interbody cages at the L4L5 level, without posterior instrumentation. To their knowledge this is the first case due to intervertebral cages. The lack of additional instrumentation had probably allowed the cages to make contact. Subsequently, friction generated wear débris, which led to the formation of a granuloma, responsible for compression of the dural sac. Intraspinal metallosis should be kept in mind as an infrequent cause of delayed neurological symptoms after spinal surgery with metallic instrumentation.

**Keywords**: metallosis; metallasma; spinal stenosis; interbody cages.

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**INTRODUCTION**

There is a great deal of clinical experience with permanent and temporary metal implants in orthopaedic surgery. The biocompatibility of these implants with the surrounding tissues has always been a concern. The implant, or wear débris generated from it, may release chemically active metal ions (7). These ions may stay in place, and cause local problems, such as metallosis, bone resorption or mechanical compression of important soft tissues (3). They may also diffuse to the bloodstream and the lymphatics and, hence, to remote organs (9) with unclear consequences. These problems are well known in the field of arthroplasty (5,8,13), but the increasing use of spinal instrumentation has led to similar concerns.

**CASE REPORT**

A 46 year-old male patient was referred because of low back pain with ambulation and neurogenic claudication. Eight years before he had undergone a microdiscectomy L4L5 for a disc herniation, in another hospital. However, after a few months the complaints recurred. MRI demonstrated a decreased cross-sectional area of the spinal canal, secondary to a disc hernia L4L5, and bilateral lateral recess stenosis at the same level. The next step, 4 years before referral, consisted of open discectomy, bilateral flavectomy and posterior lumbar interbody fusion L4L5 with two threaded cylindrical titanium cages. Neither posterior instrumentation nor posterolateral fusion were added.
The patient was referred to the authors’ hospital, 8 years after the first operation. The neurogenic claudication was now extreme. Symptoms were relieved with flexion of the lumbar spine, hips and knees. There was no neurological deficit. The biochemistry was normal. Radiographs showed contact between the cages, and a slight subsidence (Fig. 1). CT and MRI (Fig. 2) showed a mass at the L4L5 level, displacing the dural sac posteriorly. No metallic artifacts were noted.

An L4L5 decompression was performed, the mass was excised, and the cages were removed. The cages were in contact and loose, the brightness of the metal was lost, and wear was noted in the friction area (Fig. 3). The mass consisted of dark gray granulation tissue. All metallic débris were removed, and tissue samples were obtained for microbiological and pathological examination. A circumferential L4L5 fusion was performed with four pedicle screws (Malibu®, SeaSpine, USA) and a 13 mm Olys® PEEK interbody cage (BioradMedisy Pvt. Ltd.). Local bone grafts, augmented with Vitoss® bone graft substitute (Orthovita, Malvern, PA, USA), were added.

No microorganisms were identified in the removed material. Haematoxylin-eosin stained sections (Fig. 4) of the paraffin-embedded material showed dead bone trabeculae adjacent to dense fibrotic tissue, heavily stained with predominantly extracellular black metal débris. There were numerous macrophages heavily laden with the same débris.

The postoperative course was uneventful, and the patient was symptom free three months later.

**DISCUSSION**

**Rarity of spinal mettalomas**

There are few reports about metallosis in the field of spinal instrumentation. Guyer et al (6) and François et al (2) found metallosis after failure of metal-on-metal lumbar and cervical total disc replacement; explantation and spinal fusion were necessary. Takahashi et al (11) and Tezer et al (12)
reported on 3 cases of intraspinal metalloma causing neurological symptoms in patients who underwent posterior stabilization of the lumbar spine with hooks and rods. Granulation tissue had formed in the spinal canal adjacent to a metallic device. Months to years later, neurologic impairment developed.

Intraspinal metallosis, caused by metallic interbody cages, as described here, has not yet been reported, to the authors’ knowledge. They hypothesize that abnormal implant motion with friction, generating metallic débris, and chemical stimuli by metal particles, were both responsible for the formation of the growing metalloma around the implants, causing a progressive lumbar stenosis.

Pathogenesis of spinal metalmomas

Motion at a bone-metal interface generates metal wear. If, moreover, there is motion between two metal pieces, wear and corrosion at the interface are accelerated, which in turn leads to rapid release of metal particles into the interstitial fluid. Phagocytosis of these particles initiates a series of inflammatory reactions \((7,11)\), which may cause further acceleration of the metal degradation, leading to a vicious circle. The inflammatory granulation tissue or metalloma may lead to compression of the cauda equina.

In the case reported, no additional instrumentation had been used, besides the two cages. This might have caused the collision, and subsequently the friction, of the cages. Indeed, the use of titanium threaded cages as stand-alone devices (not supplemented with some form of posterior fixation) has been questioned \((1)\). Fuji et al \((4)\) conducted a study to evaluate the bony union obtained through

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**Fig. 3.** — Titanium threaded cages after removal. Wear in the friction area (arrows). Loss of brightness.

**Fig. 4.** — Histological sections. A: abundant black metal wear débris, predominantly extracellularly, and macrophages heavily laden with the same débris (hematoxylin-eosin, \(\times400\)). B: dead bone trabeculae adjacent to fibrous tissue (above); necrosis and metallic wear débris (below) (hematoxylin-eosin, \(\times100\)).
posterior lumbar interbody fusion using Ray’s threaded fusion cages without other instrumentation, such as pedicle screws. They noted, with a mean follow-up of 44 months, a nonunion rate of 72%. Shetty et al (10) compared interbody fusion with 1 : cages only, 2 : cages supplemented with posterolateral bone grafts, and 3 : cages supplemented with posterolateral pedicle screw fixation. They found that subsidence was more common in the groups without pedicle screw fixation. They suggested that pedicle screw fixation should be added to the cages when operating at levels above L5S1.

**CONCLUSION**

The authors wish to warn against placing interbody metallic devices too close to one another in disk surgery, more specifically if no additional instrumentation is used, so facilitating friction. Ideally, posterior instrumentation should be added. Intraspinal metallosis should be kept in mind as one of the late complications associated with spinal instrumentation.

**REFERENCES**