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Metallosis with spinal implant loosening after spinal instrumentation: A case report

Yiu Hin Kwan, Hong Lee Terry Teo, Shree Kumar Dinesh, Wee Lim Loo

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Abstract

BACKGROUND

Spinal metallosis is a rare complication following spinal instrumentation whereby an inflammatory response to the metal implants results in the development of granulomatous tissue.

CASE SUMMARY

We describe the case of a 78-year-old woman who had recurrence of back pain 5 years after lumbar spine posterior decompression and instrumented fusion. Lumbar spine radiographs showed hardware loosening and magnetic resonance imaging showed adjacent segment disease. Revision surgery revealed evidence of metallosis intraoperatively.

CONCLUSION

Spinal metallosis can present several years after instrumentation. Radiography and computed tomography may demonstrate hardware loosening secondary to metallosis. Blood metal concentrations associated with spinal metallosis have yet to be established. Hence, metallosis is still an intraoperative and histopathological diagnosis. The presence of metallosis after spinal instrumentation likely indicates a more complex underlying problem: Pseudarthrosis, failure to address sagittal balance, infection, and cross-threading of set screws. Hence, identifying metallosis is important, but initiating treatment promptly for symptomatic implant loosening is of greater paramount.

Key Words: Metallosis; Spine; Instrumentation; Implant loosening; Corrosion; Case report

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Core Tip: This paper describes a rare case of metallosis after spinal instrumentation and discusses the methods of diagnosing and risk factors contributing to spinal metallosis. A review of the current literature as presented in this paper demonstrates the scarcity of studies on spinal metallosis after spinal instrumentation, despite the fact that a diagnosis of spinal metallosis should be promptly identified and treated by revision surgery. It is also important to understand that the presence of metallosis after spinal instrumentation likely indicates a more complex underlying problem, such as instability of the spinal implants.

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INTRODUCTION

Metallosis is postulated to occur due to the corrosion of metal implants leading to metal debris build-up in periprosthetic soft tissue and bone. This precipitates a granulation-type reaction involving phagocytosis of metal particles resulting in osteolysis and local reactions, namely, aseptic fibrosis, local tissue necrosis, and implant loosening, as well as systemic toxicity such as cardiomyopathy and abnormal thyroid function[1]. Posterior spinal fusion involves the placement of metallic rods and screws and these fixtures are not routinely removed except for reasons such as localised pain over implant site, prominent hardware, implant failure, or infection[2]. Metallosis is not uncommon following joint arthroplasties but only a few cases of spinal metallosis have been described in the literature. Hence, we report a case of spinal metallosis with implant loosening after posterior spinal instrumentation.

CASE PRESENTATION

Chief complaints

A 78-year-old woman was admitted in 2019 to the Department of Orthopaedic Surgery in Changi General Hospital, a tertiary hospital in Singapore, for a 4-mo duration of worsening lower back pain with claudication and right lower limb radiculopathy, with onset 5 years after a spinal surgery.

History of present illness

The patient's past medical history was significant for well-controlled hypertension and type 2 diabetes mellitus, gout, gastroesophageal reflux disease, and obesity [body mass index (BMI) of 31.2].

She also had a significant past surgical history of L3 to S1 posterior decompressive laminectomy, stabilisation with pedicle screws, and posterolateral fusion with local bone grafting performed at the same hospital 5 years ago. The surgery was performed for a diagnosis of lumbar spondylosis with central canal stenosis, for which she presented with chronic lower back pain radiating down her bilateral lower limbs. Titanium polyaxial screws (DePuy Synthes) were used in that surgery. The patient's symptoms were relieved after the surgery and she recovered. Postoperative lumbar spine radiographs showed the proper positioning of the spinal implants (Figure 1).

History of past illness

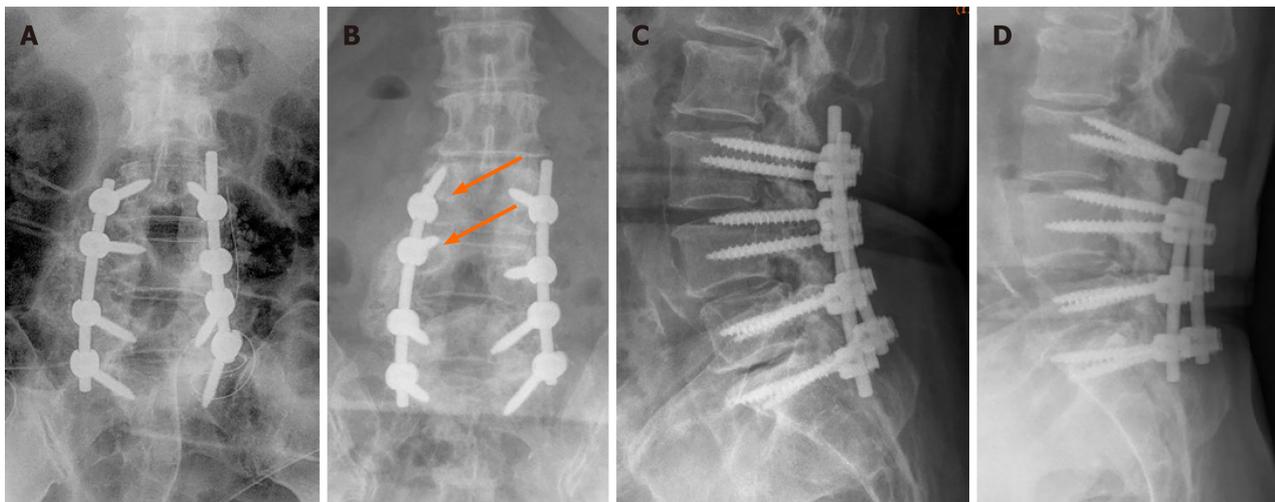
The patient was followed up in the orthopaedic surgery specialist outpatient clinic regularly after her spine surgery. Five years after the surgery, she started to develop progressively worsening lower back pain which radiated to her right lateral thigh and calves. Four months later, her symptoms had slowly deteriorated to the point where she required a motorized wheelchair as she was unable to ambulate long distances due to pain.

Personal and family history

The patient had no relevant personal and family history.

Physical examination

The most significant finding on examination was reduced power in the right L2 and L3 myotomes [grade 4 out of 5 on the Medical Research Council (MRC) scale for muscle strength]. The rest of the myotomes from L2 to S1 were normal, with a grade 5 out of 5 power on the MRC scale. She also had a large body habitus. Her gait was slow but steady over a short distance. The rest of the physical examination was unremarkable: There was no spinal tenderness or significant muscle wasting of her back or lower limbs. The range of movement of her cervical and thoracolumbar spine was normal. Sensation was intact over all dermatomes. Bilateral knee and ankle reflexes were normal. The straight leg raise test was negative as well.



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Figure 1 Postoperative lumbar spine radiographs showing the proper positioning of the spinal implants. A: Lumbar spine antero-posterior radiograph after initial surgery for posterior decompression and instrumented fusion; B: Lumbar spine antero-posterior radiograph at 5 years after initial surgery, showing stable periprosthetic radiolucencies (indicated by arrows) surrounding the right L3 and L4 screws suggestive of instrumentation loosening; C: Lumbar spine lateral radiograph after initial surgery; D: Lumbar spine lateral radiograph at 5 years after initial surgery.

Laboratory examinations

Laboratory blood tests showed normal values, including a white blood cell count of $8.2 \times 10^9/L$ and C-reactive protein level of 4.1 mg/L. Other biochemical parameters were within the normal range. Serum metal concentrations were not performed for the patient due to cost issues.

Imaging examinations

Lumbar spine radiographs and magnetic resonance imaging (MRI) were performed prior to the revision surgery. Lumbar spine anteroposterior and lateral radiographs revealed stable periprosthetic radiolucency surrounding the right upper two screws (L3 and L4 pedicle screws) that suggested loosening of the instrumentation (Figure 1). There was no evidence of fracture of the pedicle screws and rod instrumentation. Narrowing of the L4-L5 and L5-S1 intervertebral disc spaces was noted but vertebral body heights were largely maintained. Spondylotic changes and facet arthropathy were seen. There was no instability noted in the flexion and extension views.

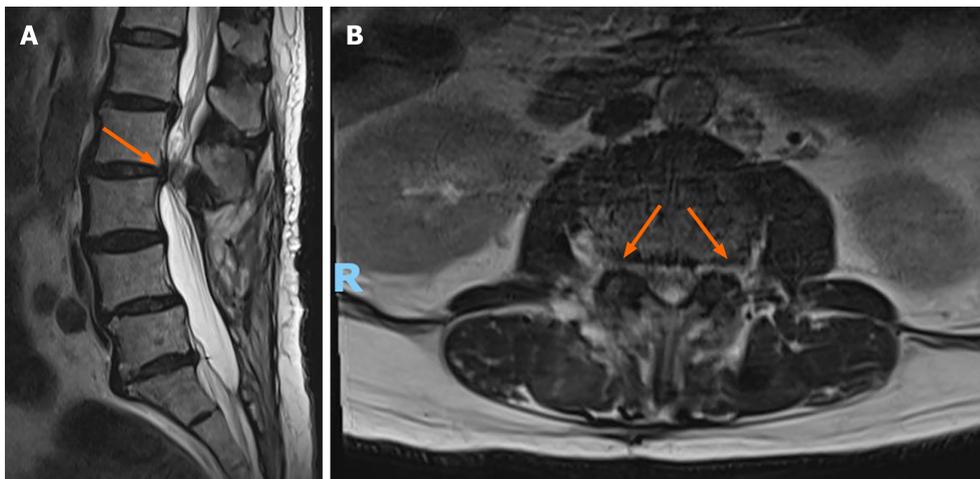
Lumbar spine MRI revealed severe spinal canal stenosis at the L2-L3 level with compression of the cauda equina nerve roots, severe bilateral lateral recess, and neural foraminal stenosis (Figure 2). There was moderate spinal canal stenosis with crowding of the cauda equina nerve roots at L1-L2. Mild peri-screw bony edema was observed around the left L4 screw (Figure 3), otherwise there was no significant evidence of peri-screw edema and screw loosening around the rest of the L3 to S1 screws.

FINAL DIAGNOSIS

Histopathology of the stained tissues that were excised revealed fibroadipose tissue and occasional striated muscle bundles exhibiting degenerative changes. There were aggregates of non-refractile, non-polarisable black granular foreign material mostly in a perivascular location, that were consistent with metallosis. There was no evidence of malignancy. Intra-operative tissue cultures were negative for bacterial growth.

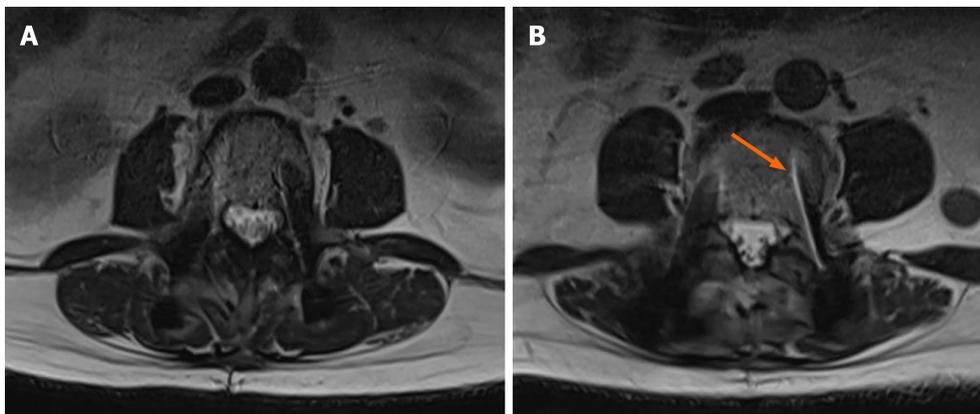
TREATMENT

L2 to S1 posterior decompression and instrumented fusion with O-Arm computer-guided navigation (Medtronic, StealthStation® S7®) were performed. Posterior elements were exposed from L2 to S1, revealing loosened L3 to L5 screws bilaterally, at both the set screw-rod interface and the bone-implant interface. The tissues surrounding the bilateral L3 to L5 polyaxial screw heads and tulips were observed to be stained dark grey (Figure 4). All previous DePuy Synthes screws were removed uneventfully. The loosened screws showed evidence of fretting at the contact surfaces. New titanium pedicle screws (Medtronic, CD Horizon® Solera®) were inserted with new trajectories under O-Arm computer-guided navigation. L2 and L3 laminectomy was carried out. The thecal sac was well decompressed at L2-L3 where there was severe stenosis. A drain was inserted, and the surgical site was closed in layers.



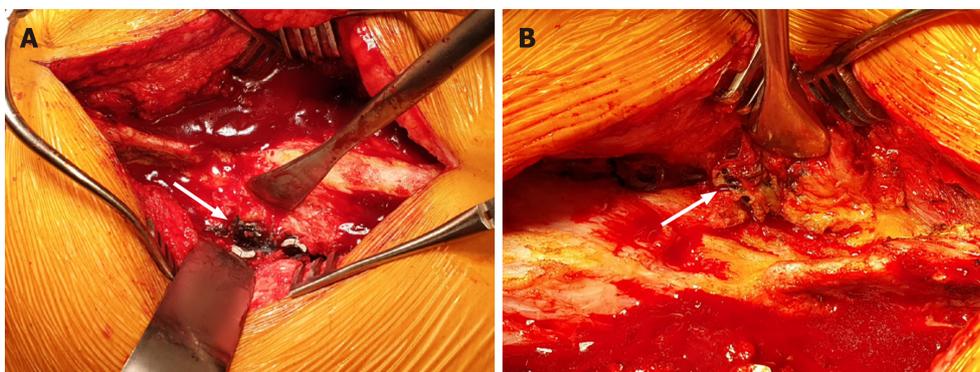
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Figure 2 Lumbar spine magnetic resonance imaging (T2 weighted) 5 years after initial surgery. A: Sagittal view. The arrow indicates herniated disc at L2-L3 level with severe spinal canal stenosis and compression of the cauda equina nerve roots; B: Coronal view demonstrating severe spinal cord compression at L2-L3 level. Arrows indicate protruded disc with bilateral lateral recess and neural foramina stenosis.



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Figure 3 Lumbar spine magnetic resonance imaging (T2 weighted) 5 years after initial surgery. A: Coronal view showing L3 level with no significant peri-screw bony edema; B: Coronal view showing L4 level with the arrow indicating mild left L4 peri-screw bony edema.



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Figure 4 Intraoperative photographs during revision surgery showing metallic grey-stained tissue surrounding bilateral L3 to L5 polyaxial screw heads. The most prominent staining occurred at A: Left L4 screw (indicated by the arrow); B: Right L4 screw (indicated by the arrow).

OUTCOME AND FOLLOW-UP

The patient's postoperative recovery was uneventful. At the 2-mo follow up, her back pain and lower limb weakness had

almost completely resolved. Her postoperative lumbar spine radiographs showed that the new implants were intact (Figure 5).

DISCUSSION

Literature review

Metallosis was first identified as a complication of metal-on-metal total hip arthroplasty[1]. The incidence of metallosis following total hip arthroplasty has been described to be 5.3% and 0.3% after lumbar arthroplasty[3], but the incidence following spinal instrumentation such as posterior spinal fusion is not well estimated currently due to the scarce literature. A literature search detailing other cases of metallosis after spinal instrumentation revealed that it is a rare occurrence. A detailed look of the reported cases of metallosis after spinal instrumentation is summarised in Table 1.

The earliest cases of metallosis following spinal instrumentation were reported by Takahashi *et al*[4]. The authors described two cases of delayed neurological deficits secondary to intraspinal metalloma adjacent to loosened infralaminar hooks. One of the patients had undergone posterior correction and stabilisation and the other had undergone posterior correction and arthrodesis for degenerative scoliosis. Radicular symptoms resolved entirely after revision surgery. The authors speculated that metallosis was caused by abnormal implant movements and chemical reactions from the metal particles. Tezer *et al*[5] then described a case whereby paraparesis secondary to intraspinal metallosis adjacent to the pedicular hook occurred 3 years after posterior spinal instrumentation and fusion for a vertebral compression fracture. The patient's neurological symptoms resolved completely following the excision of the metalloma and removal of the affected instrumentation. The authors concurred with the pathophysiology of metallosis described by Takahashi *et al*[4], and recommended using transpedicular screws sufficiently while carrying out further research to improve the corrosive resistance of spinal instrumentation. Goldenberg *et al*[6] also reported a case of spinal metalloma 18 mo after lumbar laminectomy, posterior spinal instrumentation, and fusion using titanium instead of stainless-steel alloy components. The authors concluded that the metallosis in their case occurred due to the interaction between titanium and the surrounding tissue structures rather than as a result of implant failure, corrosion, or infection as described in previous cases. Li *et al*[7] described another case of metalloma attributed to the wear and loosening of implant. A 2-cm large metalloma could be visualised on MRI. Prior to this study, MRI had not demonstrated much utility in the investigation of metallosis. Subsequently, Ayers *et al*[8] described three more cases of spinal metallosis, two of which had undergone multiple previous spine surgeries complicated by pseudarthrosis and infection and the last had undergone single-level lumbar stabilisation. Neurological symptoms improved in all three cases following revision surgery. The authors hypothesised that biologic mechanisms such as bacterial growth could influence fretting and corrosion of spinal instrumentation leading to metallosis. Richman *et al*[9] then described a young patient with acute onset pain and neurological deficits that progressed quickly. Previous instrument made of stainless steel was removed. They also noted high serum chromium levels. Most recently, Mazur-Hart *et al*[10] reported a case of unilateral metalloma from mixed-metal (titanium and cobalt chrome) instrumentation that resulted in progressive neurological deficit, but not hardware failure. Another unusual finding was the absence of metallosis on the side where the patient had also undergone a hip arthroplasty comprising of the same materials.

Clinical presentation and diagnosis of spinal metallosis

Metallosis is often diagnosed incidentally through intraoperative findings of grey-stained local tissue[8], and definitively through histopathological evidence of macrophages containing metal debris[11]. This is because of the non-specificity of clinical presentations, such as pain, symptoms of infection, and neurological deficits[9]. Based on case studies in the literature (summarised in Table 1), patients with spinal metallosis most commonly presented with lower back or radicular pain. Other symptoms included neurogenic claudication and progressive paraparesis of the lower limbs. It is also challenging to visualise metallosis through standard radiographic evaluation[8]. MRI and computed tomography (CT) are not able to definitively diagnose spinal metallosis due to the presence of artifacts around the metal implants[4,5]. Ayer *et al*'s study showed that all three cases did not have evidence of metallosis on CT prior to surgery[8]. This differs from the usefulness of CT in the diagnosis of metallosis in total hip arthroplasties, in which metallic debris or a high-density material outlining the joint capsule or bursa can be visualised[12]. On the other hand, CT myelography has been the diagnostic imaging modality of choice in a number of studies on spinal metallosis, showing stenotic lesions adjacent to previous instrumentation[4,5,6,9]. In our case, lumbar spine radiographs showed evidence of loosening of pedicle screws. Screw loosening can be secondary to a variety of factors including metal wear debris, microfracture, infection, tumour, and metabolic diseases, with a greater incidence in patients with osteoporosis[13]. Our patient had type 2 diabetes mellitus but was not known to have osteoporosis. She did not have constitutional symptoms and her preoperative routine blood tests were unremarkable. Therefore, osteolysis secondary to fretting of instrumentation was a more probable mechanism for the loosening of screws in this case. MRI was not useful in identifying hardware loosening or spinal metallosis in our case. In a recent clinical trial by Spirig *et al*[14], CT was more sensitive and specific in detecting screw loosening despite applying metal artifact reduction techniques with MRI. However, MRI may be of utility in cases where the metalloma is large enough with compression or extension into surrounding structures, such as in the cases reported by Li *et al*[7] and Mazur-Hart *et al*[10]. Comparing plain radiographs and CT findings of previous studies (Table 1), we suggest adopting a high index of suspicion of metallosis when screw loosening is evident on radiographical imaging in a patient with persistent postoperative back pain or radiculopathy but otherwise medically well. If plain radiographs do not show hardware abnormalities, it may be prudent to proceed with a CT scan or myelogram instead.

Table 1 Summary of the literature

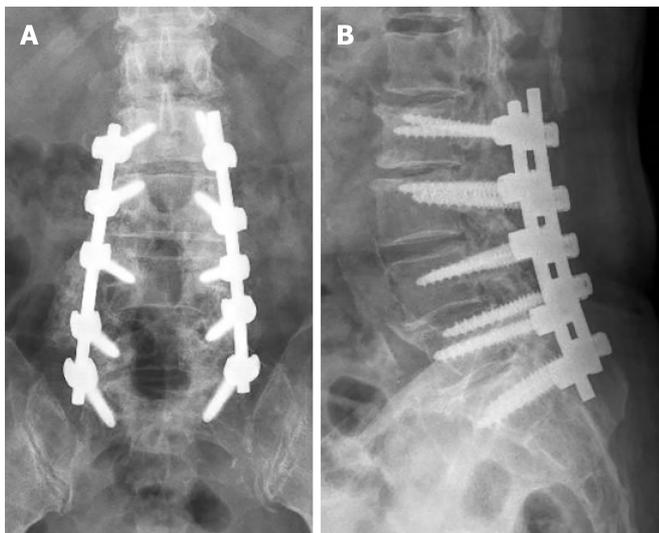
Ref.	Patient biodata	Type of surgery undergone	Instrumentation	Symptoms leading to revision surgery	Radiological findings	Revision surgery	Intraoperative findings	Histopathological findings	Patient outcome
Takahashi <i>et al</i> [4], 2001 (case series)	1 Female, aged 58	Posterior correction and stabilisation T10-L3 (no decompression) for degenerative thoracolumbar scoliosis	Stainless steel Cotrel-Dubousset	Left L4-L5 radicular pain several months post-op	Plain radiographs: No implant dislodgement or spinal instability; myelography: Shadow defect adjacent to the tip of the L3 infralaminar hook and dura mater was compressed from the posterior	11 mo post-op; removal of L3 pedicular screws and left L3 infralaminar hook, L3 laminectomy, excision of metallic mass, instrumentation elongated to L4 with connecting pieces, posterolateral fusion	L3 hook loose from rod, macroscopic metallosis (8 mm mass of dark grey granulation tissue) at hook-rod junction extending to surrounding fibrous tissues, L2-L3 pseudarthrosis	Not described	Immediate resolution of radicular pain, but continued to have slight low back pain during active trunk motion
	2 Female, aged 54	Posterior correction and arthrodesis T12-L4 for symptomatic degenerative lumbar scoliosis	Stainless steel Cotrel-Dubousset	Right L5 sciatic pain 4 yr post-op	Plain radiographs: No implant dislodgement or spinal instability; myelography: Stenotic lesion at lowest level of instrumented lumbar spine but undisplaced implants; myelotomography: No migration of the hooks in the spinal canal, stenotic lesion adjacent to tip of L4 supralaminar hook	5 yr post-op; L4 and L5 laminectomy, excision of metallic mass, instrumentation elongated down to sacrum	1 cm × 1 cm × 2 cm dark grey granulation tissue under L4 lamina continuous with fibrous membrane of the same colour surrounding right L4 supralaminar hook and compressing right L5 root, loosening implant connection and wear of rod at hook-rod junction, no pseudarthrosis	Granulation tissue consisting of metallic debris – iron staining showed widespread intracellular iron, spectrometry analysis of metal concentrations showed presence of iron, nickel and chromium	Radicular symptoms resolved
Tezer <i>et al</i> [5], 2005 (Case report)	Male, aged 57	Posterior spinal instrumentation for T8-9 compression fracture	Stainless steel pedicle screw-hook combination system	Progressive paraparesis 3 yr post-op	Myelography and myel-CT: Focal image of a mass at T6-7 antero- laterally displacing the dural sac and spinal cord; CT and MRI could not be done due to diffuse metal artefacts	Posterior surgical procedure, complete removal of implants, excision of mass, all metallic debris cleaned	Corroded, black-coloured pedicle hook, no loosening or colour change of other implanted parts, construct stable and strong, fusion complete, granuloma formation in centre of metallic construct, metallic debris had pushed dural sac and spinal cord to anterior and contralateral side resulting in defect of 1.5 cm in diameter in lamina and pedicle	Hematoxylineosin stained sections of paraffin-embedded material showed dense fibrotic tissue heavily stained with black metal debris, foreign body giant cells seen around metallic debris, iron staining by Perls method showed widespread iron within macrophages	Symptom-free 3 mo post-op
Goldenberg <i>et al</i> [6], 2016 (Systematic review)	Male, aged 75	Single-level lumbar laminectomy, posterior instrumentation and fusion	Bilateral L4 and L5 titanium alloy pedicle screws, dual interconnecting vertical rods, single interconnecting horizontal rod using the	Persistent and progressive severe lower back pain since the surgery, associated with severe left-sided sciatica	CT myelography: Posterior epidural mass causing canal stenosis, no features suggestive of corrosion or loosening of metalwork; SPECT:	Explorative lumbar canal decompression and nerve root neurolysis, dissected down to area of previous surgery, removal of scar tissue and rostral part of L5	Scar tissue in area of previous surgery, intermixed dark brown and pale pink roughened firm tissue compressing thecal sac,	Dense fibrohistiocytic reaction and cystic change associated with granulomas and calcification, multinucleated giant cells both encasing and adjacent to	Satisfactory clinical improvement in back pain and sciatica

			DENALI K2M system, interbody device containing bone graft admixed with bone morphogenetic protein, high speed burr used but no contact between metalwork and drill		Increased uptake in keeping with discovertebral disease; MRI not done as incompatible cardiac pacemaker	lamina and spinous process, debulking of mass	no implant loosening or corrosion	foreign black pigmented particles, presence of degenerate bone, cartilaginous material and skeletal muscle, no micro-organisms identified	
Li <i>et al</i> [7], 2016 (Case report)	Male, aged 58	Posterior decompression and instrumented fusion	Titanium implant (surgery was done at another institution)	Recurrent lower back pain radiating to left lower limb, dysesthesia, neurogenic claudication	MRI: Severe adjacent stenosis at L3-4, intraspinal extradural tumor-like mass with compression of the neurological elements	Spinal decompression, excision of mass, and extension of instrumented fusion	Metallic soft tissue and a well-capsulated tumor-like mass	Hematoxylin and eosin staining of mass showed many spindle-shaped; fibroblasts. Many macrophages containing dark metallic wear particulates with phagocytosis	Follow-up not reported
Ayers <i>et al</i> [8], 2017 (Case series)	Male, aged 74	Multiple previous spinal surgeries including limited lumbar fusion complicated by pseudarthrosis, revision with extension of fusions and infection at subsequent operations	Mix of alloy rods (CoCrMoC, ASTM F-1537 specification) and titanium alloy (Ti6Al4V ASTM F-136 specification) screws	Continued mechanical back and radicular pain	CT: Hardware failure with bilateral fractured L5 screws and sagittal plane deformity	Staged revision surgery; (1) Initial surgery - removal and cleaning of T10-S1 hardware, evacuation of fluid collection, wound debridement, intra-op cultures, and exploration of the fusion, subfascial drains inserted; (2) then 2 further irrigation and debridement procedures until cultures negative and tissues appeared viable; and (3) after 6 wk, final stage - evacuation of smaller fluid collection, revision posterior instrumentation with L3 pedicle subtraction osteotomy	(1) Initial surgery: Large fluid pocket containing approximately 500 mL of grey-black fluid, black discoloration of posterior soft tissues, all rods showed significant evidence of fretting, galling, pitting and crevice corrosion; and (2) final stage: Smaller fluid collection of 300 mL in posterior soft tissues, gram stain negative	Excised tissue consisted of necrotic fibrous tissue with areas of viable fibrous tissue and particle laden histiocytes. Soft tissue, pseudomembrane from L3-S1 consisted of fibrous tissue with refractile material and calcification. Cell culture of infected tissue/fluid showed presence of propionibacterium acnes and staphylococcus aureus	Significant reduction in pain and symptoms 1 yr post-op
	Male, aged 47	Multiple previous lumbar spine procedures complicated by pseudarthrosis and infection	Titanium alloy (Ti6Al4V) components	Recurrent pulmonary infections and continued back and radicular leg symptoms	CT: Likely pseudarthrosis at multiple lumbar spine levels	2 yr post-op; Staged surgery; (1) Initial surgery - wound exploration, removal of hardware, formal irrigation-and-debridement, deep drains placed; (2) another irrigation-and-debridement with post-op antibiotics × 6 wk; (3) after 6 wk, instrumented fusion from T10-Ilium with revision TLIF at L2-3 and Smith-Petersen Osteotomy; (4) irrigation-and-debridement; and (5) removal of right S1 screw as	(1) Initial surgery: Significant fluid collection, soft tissues stained black, all rods showed significant evidence of fretting, galling, pitting and crevice corrosion	(1) Initial surgery: Excised tissue comprised of necrotic adipose and fibrotic connective tissue; and (2) instrumented fusion stage: Cultures grew Mycobacterium phlei	No back or leg pain at follow up (recent to when paper was written)

	Female, aged 61	Single level lumbar stabilisation procedure including instrumentation with pedicle screws and PEEK rod	Titanium alloy (Ti6Al4V) components	Significant sagittal plane deformity and significant back/radicular leg symptoms	CT: Significant sagittal plane deformity	Instrumentation from T4-pelvis with hardware removal and pedicle subtraction osteotomy, including removal of L2-3 disc to allow greater correction	Significant black staining of the posterior soft tissues, all rods showed significant evidence of fretting, galling, pitting and crevice corrosion	Tissues not submitted to pathology	Complete symptomatic relief at 6 mo post-op
Richman <i>et al</i> [9], 2017 (Case report)	Male, aged 19	Posterior spinal fusion	Stainless steel implants	Low back pain, urinary hesitancy, and parasthesias on bilateral anterior thighs, that quickly progressed to flaccid paraparesis, hypoaesthesia, and urinary retention	CT: Cavitation around right L1 pedicle screw CT myelogram: Irregular and inadequate opacification of the thecal sac at L1	(1) Initial surgery: Removal of screw; and (2) posterior laminectomy and decompression from T12 to L2 with removal of all instrumentation	(1) Initial surgery: Black and yellowish corrosive film and tissue around right L1 screw; and (2) subsequent surgery: Gritty yellow-black material tracking through the L1 foramen around left L1 pedicle screw, causing thecal sac compression at T12-L2	Pathologic diagnosis was consistent with metallosis	Pain and urinary retention resolved, complete motor and sensory recovery, but presence of bilateral clonus 3 yr post-discharge
Mazur-Hart <i>et al</i> [10], 2022 (Case report)	Male, aged 79	2 previous lumbar decompression, posterior instrumentation and fusion 2 yr apart. Right hip arthroplasty 1 yr later	First surgery: Cobalt chrome rods and titanium screws. Second surgery: PEEK spacer and titanium screws and plates	Worsening falls, ataxia and pseudo-claudication	CT and MRI: T1 and T2 hypointense non-enhancing mass around right-sided paraspinous rod extending into spinal canal and surrounding bones and muscle on the same side	L4-S1 biopsy and subtotal resection of paraspinous mass with removal of hardware at L2-S1	Dense fibrotic tissue, black granular material on screws and rods, black staining of adjacent soft tissues and lumbar bone	Extensive necrosis with surrounding inflammation and fibrosis with focal deposition of black pigment of exogenous origin (metallic <i>vs</i> carbonaceous), lymphohistiocytic reaction with giant cell formation in rare areas. Gram stain and culture negative	Weaned off walker, reduced dysesthesia but leg weakness still present 3 mo post-op. Leg strength and ambulation continued to improve 7 mo post-op

CT: Computed tomography; MRI: Magnetic resonance imaging; PEEK: Polyetheretherketone.

In regards to the relationship between serum metal concentrations and the development of metallosis, Richman *et al*[9] noted that in asymptomatic patients, serum chromium levels of more than 0.6 ng/mL and more than 3.75 µg/L were indications of implant malfunction and corrosion, respectively. Fernández Bances *et al*[15] found a significant rise ($P = 0.00049$) in serum titanium concentrations from the levels prior to posterior spinal fusion using titanium instrumentation, similarly to previous studies; however, the correlation of serum titanium concentrations and metallosis was not explored in their study. Cundy *et al*[16] found that serum titanium and niobium levels in children 2 years after instrumented spinal fusion were significantly increased but their clinical significance was not explored. Ayers *et al*[8] reported that muscle concentrations of various metals, namely, aluminium, cobalt, vanadium, and molybdenum, were higher than normal levels in their cases with spinal metallosis due to observed wear and corrosion of the metal instrumentation. However, none of the patients had elevated concentrations of metal in blood. So far, serum metal levels indicating metallosis have yet to be well-defined. We did not check serum metal concentrations in our patient as she did not have symptoms of metal poisoning, there was no baseline data prior to her previous spinal surgery, and investigating metal concentrations



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Figure 5 Lumbar spine antero-posterior and lateral radiographs showing proper positioning of the implants after revision L2-S1 surgery.

this time would be costly and purely academic. However, in patients exhibiting symptoms of metal poisoning, serum metal concentrations will be helpful in confirming a diagnosis of metallosis, potentially leading to timely intervention.

Effect of implant material and factors contributing to metallosis

The mechanism for the development of metallosis following spinal instrumentation is yet to be well-ascertained. It has been postulated that the rigidity that results from instrumentation leads to accelerated degeneration at adjacent spinal levels[7]. The metallic debris from the degeneration of the spinal implants in turn results in a chronic inflammatory process involving a foreign-body granulation-type reaction[1,7]. Spinal metallosis has been described in studies that involved titanium or stainless steel implants[3]. Spinal instrumentation of different metals is commonly used in combination as each metal has a particular mechanical and physical property; for example, cobalt chromium rods have been described to provide stronger correctional forces for scoliotic curves as compared to rods made of other materials [17]. Titanium may be more resistant to crevice corrosion than stainless steel but it has less mechanical resistance and may even stimulate osteolysis[4]. Singh *et al*[18] demonstrated that posterior spinal fusion constructs made of stainless steel were more prone to fretting corrosion as compared to those made with a combination of cobalt chrome with titanium alloy or pure titanium with titanium alloy in a simulated *in vitro* experiment using normal saline. However, the study was unable to account for the actual inflammatory environments present in the human body. Panagiotopoulou *et al*'s study on retrieved spinal implants demonstrated that the risk of corrosion was not increased when two dissimilar metals, namely, cobalt chromium alloy rods and titanium screws, were used in combination[17]. The authors suggested that metallosis may be more dependent on patient factors rather than the corrosiveness of the metals. However, the main limitation of that study was its small sample size, whereby a combination of metals was employed only in two out of seven patients [17].

Vieweg *et al*[19] in an early study on the corrosion of the internal spinal fixator system described that corrosion occurred due to not only the metallurgical composition but the specific construction of the instrument as well. Cundy *et al* [16] described that crevice corrosion was more likely to occur at rod junctions with increased metal-on-metal sites, contributed by micromovements prior to spinal fusion. Takahashi *et al*[4] noted that the lower end of an instrumented fusion was subjected to greater stress hence predisposing the release of metal debris from the hook-rod junction during flexion-extension movement of the lumbar spine. Comparatively, Tezer *et al*[5] felt that metallosis occurred at the middle levels of the spinal construct in their case because of the unequal distribution of chemical properties and degeneration of micromovements in the long term. Interestingly, metallosis occurred at the upper levels of the spinal construct in our patient, where the burden of flexion-extension is not particularly high in day-to-day activities.

Patient factors such as a high BMI as evident in our patient could have accelerated the wear of the titanium screws. As described in the literature review, previous studies have postulated that metallosis results from abnormal micromovements of hardware and a continuous inflammatory reaction[4-6]. Obesity has been associated with the development of spinal disease through both a chronic low-grade inflammatory response as well as biomechanical alterations in the lumbar spine that lead to increased shear forces and torque on the discs and joints[20], hence potentially predisposing to metallosis. However, it is currently difficult to demonstrate an association between patient demographics and spinal metallosis due to the limited number of case reports and case series in the literature so far.

Metallosis is also likely to be a by-product of unstable spinal instrumentation. The increased cyclical loading as a result of implant loosening causes increased fretting at the contact surfaces. This not only produces the characteristic metal debris in metallosis, but ultimately can lead to implant failure. Pseudarthrosis after lumbar spine fusion is a common cause of spinal implant loosening requiring revision surgery[21]. Chronic low-grade spinal surgical site infection is another potential cause for instrumentation loosening, hence stressing the importance of sterile instrumentation[22,23].

Several procedure-related risk factors for implant loosening have also been described. First, inadequate correction of sagittal imbalance has been associated with a negative prognosis for implant anchorage in bone[24], increasing the risks of screw loosening in posterior spine fusion[25]. Inadequate set screw tightening or cross-threading of screw tulips due to improper insertion of set screws into the screw tulip can also predispose to coupling failure, which can occur at any level of a spinal construct[26]. Hence, spinal surgery should be performed only by well-trained spinal surgeons with vast experience and undertaking these operations regularly to minimise these mechanical risk factors. Other possible risk factors for instrumentation loosening are osteoporosis and cobalt chromium rods[23]. Our patient was not known to have osteoporosis, but it may be prudent to commence osteoporosis treatment prior to surgery in patients who have been diagnosed with osteoporosis so as to improve bone density and potentially increase the strength of screw fixation. Utilising a material that is less rigid than cobalt chromium rods may reduce the risk of implant loosening; however, current options are limited, and cases of metallosis including our patient have mainly involved titanium or stainless steel instrumentation. Our patient's symptoms of progressive lower back and radicular pain were due to severe spinal stenosis and compression of the cauda equina nerve roots. Although implant loosening may present similarly even in the absence of metallosis, there was both intraoperative (corrosion of screws and grey-stained surrounding tissues) as well as histopathological (metallic debris in macrophages) evidence of metallosis at the same spinal levels. Ultimately, it is important to recognise symptoms of spinal implant instability and initiate treatment for patients timely.

CONCLUSION

Identifying metallosis prior to surgical exploration is challenging. Clinical presentation tends to be non-specific, most commonly being lower back or radicular pain. CT appears to be the modality of choice to observe for aseptic hardware loosening and pseudarthrosis, while myelography and MRI are able to suggest the presence of a metalloma. A definitive diagnosis of metallosis can only be made from histopathological results, where metallic debris is seen in macrophages.

To date, the relationship between serum metal levels and the presence of metallosis has yet to be established. Currently, implants of various metallic compositions are used, but the individual metallic properties confer theoretical benefits and disadvantages and no particular material has been identified to be least likely to cause metallosis thus far. Furthermore, patient factors may contribute to metallosis but further studies are required to establish an association.

Finally, we would like to highlight that the presence of metallosis after spinal instrumentation likely indicates a more complex underlying problem. Metallosis can occur due to instability of the spinal implants, which may be secondary to pseudarthrosis, failure to address sagittal balance, infection, and cross-threading of set screws. Spinal implant instability manifests commonly as pain and weakness, which were present in most cases involving instrumentation loosening described within this report to varying degrees. However, regardless of the cause for metallosis, the only definitive treatment to date for symptomatic implant loosening is the removal and replacement of the implants. The rate at which metallosis progress and the onset of symptoms is not known. However, it undoubtedly can lead to significant pain and mobility issues. Hence, it is prudent to identify the underlying cause of implant loosening early and commence treatment promptly.

FOOTNOTES

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