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CASE REPORT

Corrosion of Harrington rod in idiopathic scoliosis: long-term effects

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Abstract
Purpose Metal implants have been used to treat adolescent idiopathic scoliosis since the 1960s. Only recently, however, it has the issue of metal-bone breakdown secondary to metal corrosion in situ come to light, raising concerns of possible long-term complications from the resulting metallosis and inflammation of spinal tissues. We present a case of a patient with neurological deficit, pain, and disability with Harrington rod in place for over 30 years, to bring attention to the issue of bio-corrosion of metal implants and its effect on human tissue. We call attention to the need for protocols to better diagnose and treat these patients.
Methods We provide a complete review of the history and clinical manifestations as well as serum metal, X-ray, and CT/myelogram test results.
Results A 52-year-old female with spinal fusion and Harrington rod presents with pain, lymphedema, disability, and neurological deficits including thoracic outlet syndrome, hyperreflexia, peripheral muscle weakness and atrophy, hypertonicity, Raynaud's phenomenon, and balance and gait abnormalities. Serum chromium levels were elevated (26.73 nmol). X-rays showed no evidence of rod breakdown. Serial X-rays can demonstrate subtle corrosive changes but were not available. Adhesive arachnoiditis was diagnosed via CT/myelogram.
Conclusion We hypothesize that bio-corrosion is present in this case and that it is associated with intraspinal metallosis. Trauma secondary to a motor vehicle accident, as well as arachnoiditis, and their possible effects on this case are outlined. Challenges in proper diagnosis and management are discussed.

Keywords Scoliosis · Spinal implants · Corrosion · Metal ions · Metallosis

Introduction

Metal implants, previously thought to be benign, have been increasing in recent years, and show evidence of having deleterious effects on the human body. Adolescent idiopathic scoliosis (AIS) has been treated with spinal instrumentation as the treatment of choice in moderate-to-severe spinal curvatures since the 1960s. A proportion of patients, with metal implants in place, present with complications secondary to metal breakdown that are at present poorly understood. Recent studies have demonstrated conclusively that metal implants corrode over time and can lead to osteolysis, aseptic loosening, and release of metal wear debris and metal ions into surrounding tissues and distant organs [1–11]. This phenomenon has been correlated with an inflammatory cascade that affects peri-implant tissue [12–15]. There have also been cases of localised neurological damage associated with rod breakdown [11, 16, 17].

A review of the literature on the complications of total hip replacement shows clear evidence of peri-articular adverse reactions, including inflammation, osteolysis, pseudo-tumors, and loosening of prostheses [18]. Studies of thoraco-lumbar fixation prostheses, and total disc replacement showed similar effects with corrosion and metallosis that were evident by direct observation during revision surgery [19–21].

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In spite of this information being available in the literature, the clinical complications of bio-corrosion in spinal implants have not been recognised and diagnosis has been an arduous process. Standardised protocols for testing and diagnosis are not in place. Treatment options are not made available and complications are poorly understood. We bring this case forward in an effort to challenge the current thinking about metallosis-related spinal problems to begin a much needed conversation about how best to manage the effects of bio-corrosion on those with long-term implants.

We present a case of AIS corrected by Harrington rod and posterior spinal fusion, in which several late onset signs and symptoms occurred, following a motor vehicle accident (MVA) 14 years after the initial surgical correction. We will explore the nature of bio-corrosion and its diagnosis. We will also discuss intraspinal metallosis and its possible neurological effects as well as the effects of trauma on this case.

Case report

A 52-year-old female, who underwent posterior spinal fusion with Harrington rod, presents with pain, disability, and neurological signs that interfere with activities of daily living.

Over the initial 14 years after surgery, this patient functioned well. She experienced some minimal back pain that did not necessitate time away from work or interfere with sport or leisure activities. However, after the MVA in 1993, this patient's status changed.

Assessment immediately following the MVA indicated acute spinal pain, headache, paresthesia, and muscle spasm consistent with a whiplash type injury. Neurological testing immediately after the MVA was normal. The patient did not progress as expected, but instead seemed to get worse over time. Axial pain and myofascial restriction persisted over several months. Gradual onset of lymphedema occurred. After 18 months, neurological examination revealed thoracic outlet syndrome, hyperreflexia, Raynaud's phenomenon, peripheral muscle atrophy and weakness, and hypertonicity of flexor muscles which persisted over time. Gradual deterioration of motor function significantly impaired movement, with general muscle weakness varying from grade 2–4. Eventually, balance and gait pattern were affected resulting in frequent falls. Loss of proprioception further disrupted gait and task performance. Oswestry disability index testing performed 2 years ago, resulted in a score of 70.

Concern about rod status with regard to possible corrosion or breakage led to further investigation. X-rays of the spine indicated that all metal remained in situ and that no breakage was evident. Artifact around the metal rod does not allow magnetic resonance imaging (MRI) to be a useful diagnostic tool. Bone density testing indicated osteoporotic changes. Blood tests indicated plasma chromium levels of 26.73 nmol/l (normal range 1.9–5.8 nmol/l). Nickel levels were within normal limits. A CT myelogram indicated spinal arachnoiditis, with clumping of nerve roots, consistent with adhesion formation.

Discussion

It has been conclusively demonstrated that corrosion occurs in stainless steel spinal implants [1–6, 8–11]. Three types of corrosion have been discussed in the literature. They are crevice corrosion, fretting corrosion, and galvanic corrosion [1, 8, 22]. Crevice corrosion results as metal is exposed to a tissue fluid environment causing localised corrosion of stainless steel by breaking down the protective oxide layer [1, 5]. Fretting corrosion results from mechanical damage from micro-motion leading to wear debris in the surrounding tissues [1, 8]. In galvanic corrosion, metals are in contact with one another while immersed in electrolytes, so that the corrosion process is accelerated [8]. All three types of bio-corrosion can occur in spinal instrumentation. It is clear in all of these situations that no breakage of the rod is necessary for bio-corrosion to occur [1, 6, 23].

Corrosion has been shown to be a slow and continuous process that leaches metal ions into the body's tissues. Perispinal tissue surrounding the rod, exposed during revision surgery, is discolored and has been shown via electron microscopy to contain microscopic metal particles [2]. Aulisa et al. [2], examining tissue from 20 rod retrieval procedures, found the presence of fully pigmented tissue surrounding the rod and on histological examination found metal particles in this tissue and in adjacent bone. All of these findings occurred in cases in which rods were in place for only 2 years. Several studies describe a cascade reaction to metal that leads to inflammation [3, 15, 18, 24, 25]. Gristina [13] described the process by which inflammation becomes an ongoing chronic state via a self-perpetuating cycle.

The subject of metal debris in peri-articular tissue has been more thoroughly researched in cases of total hip replacement. Drummond et al. [18] correlated pain and dysfunction to adverse reactions to metal debris, locally and distally, in hips with metal-on-metal prostheses. Revision surgery has been performed more regularly in these cases and evidence of necrosis, debris, and local degradation that were not always evident on imaging, which have been clearly observed at time of surgery [27]. Chromium, cobalt, and titanium levels were often elevated [24, 26, 28, 29]. The levels were reduced post-revision, although they remained higher than normal [30, 31].

In other studies of spinal implants, metal ions have been detected in blood [4–6, 32]. Metal debris has also been found in lymph and distant organs, including kidneys, spleen, and liver [26, 33]. The clinical significance of metals in the parenchyma of organs is unknown. Many researchers have questioned its carcinogenic and teratogenic implications [5, 6, 9, 22, 32, 34]. We found no studies which looked at this issue nor did we find studies on spinal implants that were more than a few years post-surgery. Considering that these metal rods are inserted mostly in pubescent children and are left in place for decades, the implications of long-term ongoing metallosis and metal-related systemic changes have the potential to create significant health issues. However, at this time, the correlation between metallosis, its inflammatory cascade, and the patient's symptoms cannot be made. Some studies related to metal-on-metal hip replacement have shown that patients can have metallosis and be asymptomatic [18, 35]. One case report on total disc replacement did report pain relief after revision surgery to remove the metal implant [21]. Another case study showed significant damage in peri-articular tissue that led to dysfunction that persisted after revision surgery and raised the issue of whether signs of metallosis should be used as indicators of possible future problems [31]. It is clear that more study is required to determine if there is correlation between corrosion and symptoms.

Diagnostic testing

Measurement of the bio-corrosion process and its associated metallosis and inflammatory responses has been a medical challenge as no standardised protocols are in place for appropriate diagnosis. To date, for spinal implants, most investigative studies have relied on the measurement of serum and urine metal ions [5, 6, 32, 34, 37, 38].

The major constituent metals found in stainless steel implants are chromium, nickel, iron, and molybdenum [5, 39]. Serum chromium has been found to be increased in patients with spinal implants as compared to controls without implants [5, 32, 34, 36]. In our case study, significantly elevated chromium levels were present, whereas nickel levels were within normal limits. Nickel has been shown to be quickly metabolised and excreted by the kidneys and is an unreliable marker in cases of implant degradation [1, 9]. In our review of the literature on spinal implants, correlation was found between elevated chromium levels and the presence of bio-corrosive metal breakdown [5, 6, 9]. In studies of total hip replacement, elevated chromium, cobalt, and titanium levels were associated with bio-corrosion [18, 28, 31]. A review of studies on total disc replacement revealed elevated chromium and cobalt on serum tests [7, 40]. Thus, in our case, as in other studies of stainless steel implants, chromium was the only reliable indicator of metal ions as cobalt and titanium were not constituent metals of the Harrington rod. Therefore, due to high chromium levels found on serum testing in this case, we came to the conclusion that bio-corrosion is likely present.

Spinal implants that have been retrieved in revision surgery have macroscopically shown bio-corrosion that was not evident on pre-operative X-rays [5, 6, 8, 19]. It is clear that single X-rays cannot be relied upon to provide useful information as bio-corrosion has been shown to occur without gross changes to metal on X-ray [21, 34]. However, sequential X-rays compared over time demonstrate a progressive decrease in metal density, indicating corrosive changes [6]. Thus, it is not surprising that this patient's single X-ray was of little use in diagnosis.

In light of this information about the travails of metal and X-ray testing, it is clear that there is a need for standardised testing with regard to differential diagnosis in cases of bio-corrosion. At present, for spinal implants, there are no standardised testing protocols and no consistent measurement parameters from lab to lab. In the case of total hip replacement, guidelines for management after surgery have been outlined. Protocols for post-surgery monitoring are in place that include, blood metal testing, imaging, and follow up medical examinations [18]. In our case, there was no standard testing made available to the patient and diagnosis was a long and arduous process. Our experience has been that many health practitioners were unaware of this problem and, as a result, had no suggestions for testing or treatment.

In summary, diagnosis of bio-corrosion was made based on the elevated chromium levels, since sequential X-rays were not available and other testing was of little value.

Neurological complications

In this case, lower motor and sensory nerve dysfunction has been a significant finding that has affected muscle and gait function. We propose that late onset intraspinal metallosis is a potential complication of spinal instrumentation surgery and should be considered as readily as extraspinal metallosis. Inflammation around the dural sac could be responsible for slow and gradual onset of lower motor neuron and sensory neuron lesions. The bio-chemical effects of meningeal exposure to metallosis are unknown. However, the possibility for diffuse neural-based signs and symptoms to develop cannot be ruled out. To our knowledge, this is the first case of neurological complications in a spinal implant for AIS that is not attributable to compression from a granulomatous mass or metal dislodged from the implant into the spinal canal.

Takahashi et al. [16] studied two cases of intraspinal metallosis in which delayed neurological symptoms arose. In each case, accumulation of metal debris occurred in the spinal canal, and was followed by radicular symptoms secondary to granuloma compression and adherence to the dural sac. Although, in our case, no localised granuloma was found on X-ray or myelogram, adhesive arachnoiditis was diagnosed. Arachnoiditis is a form of diffuse and extensive intraspinal inflammation that can lead to adhesive clumping of nerve roots and the development of neurological signs and symptoms. Considering the diffuse nature of the neurological complications in this case, they could be secondary to arachnoiditis. However, the question remains: is it possible that arachnoiditis is a complication of intraspinal metallosis?

Trauma

In our review of the literature, we found no reference to either the effects of trauma on spinal implants or the effect of trauma on bio-corrosion. Since bio-corrosion occurs in all metal implants, it was likely present in our case before the MVA but was not creating any symptoms. Symptoms arose after trauma and were systemic and neurological. These symptoms are not consistent with the usual post-traumatic biomechanical effects from a typical whiplash injury. Trauma-induced neurological damage is immediate in presentation, whereas in our case, these symptoms began slowly at 18 month post-MVA and became progressively worse over a prolonged period. The escalation of inflammatory and neurological symptoms could be a result of trauma only if another underlying condition was already in place. Despite receiving regular intensive conservative treatment post-MVA, her symptoms gradually worsened eventually including neurological signs.

What is the relationship between trauma, arachnoiditis, and bio-corrosion? Did the trauma have an effect on the inflammatory cascade, causing an increase in its presentation in the body? Did the trauma exacerbate the arachnoiditis? Or did trauma leave the scenario unchanged and have no effect? Would the signs and symptoms we see in this case have arisen anyway? The fact that this patient had the implant in place for several years with no ill effect, and that symptoms arose only after trauma, makes one question whether trauma acted as an accelerating factor. Takahashi et al. [16] described a vicious cycle of metal wear and inflammation caused by motion at the bone metal interface. As inflammation progressed, wear and mobility at the interface increased causing more inflammation. We question whether trauma could be a catalyst to this ongoing circular process.

Conclusion

We chose to bring this case forward to elucidate the issues facing an entire patient population that has previously been unrecognised. The clinical complications of long-term spinal implants have not been studied to date, yet most researchers are questioning the effects of metal breakdown on the body. All the studies we reviewed confirmed that bio-corrosion is a definitive result of spinal implants. The implications of this reality, however, are unstudied at this point. Clinically, those patients with implants in place for many years are presenting with signs and symptoms of dysfunction and pain. Other than one study [32], research to date has been done on patients with implants in place for 10 years or less. It is important to note that 30 years or more on, it is not possible to remove these implants without a significant risk. Better diagnostic and treatment protocols are imperative for this population especially when consideration is given to evidence that long rods are more susceptible to wear and corrosion [19, 34]. Analysis of pre- and post-operative testing through serial imaging and blood ion testing would allow for improved monitoring of these patients. Earlier removal of spinal implants after arthrodesis should be considered. Prevention of the problem by finding alternative treatments for scoliosis at the outset is an important goal.

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Compliance with ethical standards

Conflict of interest None of the authors has any potential conflict of interest.

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