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Case Report

Impingement of the Sciatic Nerve due to a Protruding Acetabular Cage Rim

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ABSTRACT

Sciatic nerve damage is a well-known complication that occurs in 1.5% of patients after primary total hip arthroplasty and in 8% after revision total hip arthroplasty. Yet when considering re-revision arthroplasty and acetabular cage implantation, incidence and management remain unclear. This case report describes a young female patient with sciatic nerve impingement after acetabular cage implantation. Her primary complaint was shooting sciatic left leg pain, worsening on ambulation and when seated. A complete workup was negative for spinal impingement or infection, and axonal nerve damage was confirmed through nerve conduction studies. The intraoperative findings showed that it was the acetabular cage rim that stretched the sciatic nerve. The rim was adjusted using a diamond burr to provide a specific solution without sacrificing the acetabular anchorage. Postoperative findings showed an excellent return to previous mobility and resolution of pain. This case provides a rare example of sciatic nerve impingement, showing that nerve palsies in the revision total hip arthroplasty setting may require patient-specific solutions.

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Introduction

Total hip arthroplasty (THA) is a common and effective procedure to relieve pain in patients with osteoarthritis of the hip [1]. With increasing demand for primary arthroplasty, the need for revision arthroplasty is rising as well [2,3]. In this respect, some projections note a growth of revision hip arthroplasty up to 70% by 2030 [4]. Especially in young patients with higher functional demands, the need for cup revisions due to acetabular osteolysis is expected to gain importance [5]. The incidence of sciatic nerve injury after THA is 1.5% [6,7], which increases to 8% in revision THA [7]. The incidence after multiple revisions or after cage implantation remains unknown. Nerve impingement after such acetabular cage insertion is a rare but severe complication, causing intense shooting pain, immobility, and disability. To our knowledge, no report exists on management of sciatic nerve impingement after cage implantation. We present a case of position-dependent sciatic

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nerve impingement after revision cage and cemented cup implantation, treated by local neurolysis and cage design modification without explanting implants. This report was written in line with the Surgical CAse REport (SCARE) consensus guidelines [8].

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Case history

A 47-year-old woman with juvenile rheumatoid arthritis presented to our outpatient clinic with recurrent shooting sciatic pain in the left buttocks and left leg. Owing to her condition, longer distances had always been by wheelchair since childhood; however, walking for short distances was possible without walking aids. Her surgical history involved multiple arthroplasties including a left shoulder arthroplasty in 2002, a staged bilateral total knee arthroplasty in 1993 and 1994, and a staged bilateral primary THA in 1988. Furthermore, she had undergone total knee arthroplasty revisions in 2005 and 2009, an ankle arthroplasty in 2000, and revisions thereof in 2012 and 2014. Finally, a staged bilateral elbow arthroplasty was performed in 1995 and 1996 and a C0/C3 spinal fusion was performed in 1993.

A revision THA of the left hip was performed 29 years after index surgery because of aseptic component loosening. A posterior

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approach was used here to implant an acetabular cage with cemented acetabular allograft and ceramic bearing surfaces. Six months thereafter, she presented to our outpatient clinic complaining of shooting sciatic pain in this hip on ambulation. The pain started several weeks after the operation and worsened over time. Mobilization without walking aids was not possible, limiting her to wheelchair mobility. When seated in the chair and shifting her weight to the left, intense pain started in the posterolateral thigh and stretched down to the anterolateral leg. On physical examination, a body mass index of 22.2 kg/m² (height: 144 cm and weight: 46 kg) was noted. The left hip range of motion was free, although straightening the leg with the hip flexed at 90 degrees was very painful. The described shooting pain was evoked by hip motion in general without a clear joint position in which pain persisted. Clinical motor and sensory examination was normal without a leg-length discrepancy. There were no clinical signs of infection, with laboratory workup showing a C-reactive protein value of 0.57 mg/L (normal value: <5 mg/L) and a leukocyte count of 5.16/nL.

Plain radiographs obtained during the initial evaluation showed one of the acetabular screws overriding the arcuate line and cement entering the pelvis (Fig. 1a and b). The overriding screw in proximity of the sacral plexus and sciatic nerve was suspected as the culprit. A pelvic computed tomography scan confirmed the position of the screw (Fig. 2a and b). To exclude spinal causes of sciatic pain, a magnetic resonance imaging scan and neurosurgical consultation were performed. Thereafter, proof of axonal nerve damage was provided through electromyography and nerve conduction studies which showed reduced amplitude of the compound muscle action potentials and nonverifiable F-wave latency in the left peroneal nerve (Table 1). As the patient had undergone extensive surgery of the left ankle as well, the neurologist conducting the nerve conduction studies could not exclude a possible lower impingement site of the peroneal nerve. A transgluteal infiltration with bupivacaine of 5 mg/mL (0.5%) and triamcinolone acetonide of 40 mg/mL (4%) was then performed as the last phase of staged diagnostics. The infiltration was performed under fluoroscopic control around the point of maximum pain and had a positive effect on pain and mobility for a two-week interval, after which the patient's symptoms returned. Consequently, the patient was planned for operative therapy. The indication for surgery was the suspected impingement of the sciatic nerve by the protruding screw.

Therapeutic intervention

A screw shortening and neurolysis of the impinged sciatic nerve was planned through a Kocher-Langenbeck approach. The intraoperative findings were however different than expected. The exposed sciatic nerve was not impinged by any screw but seemed to be stretched by the rim of the acetabular cage (Figs. 2c and 3a). Metal debris present at this site was cleared out. No signs of gross infection were found. Using a diamond burr and an electrical power system, the acetabular cage rim was thereafter tapered until the sciatic nerve was no longer at tension (Aesculap/Braun ELAN 4 electric power system; Hi-Line XS diamond cutting disk GE530SU; Hi-Line 6.0 mm diamond burr, size II BD802R, Tuttlingen, Germany). A smooth elliptical resection of the cage was performed, which prevented hooking of the nerve on the implant (Fig. 3b). The overriding screws were shortened by the same method to create a smoother surface of the quadrilateral plate. The surgical site was



Figure 1. Initial imaging of the THA showing the Müller cage and protruding screw. (a) An anteroposterior radiograph of the left hip. (b) An axial radiograph of the left hip.



Figure 2. CT scan transection slices of left THA with the Müller cage: (a) the transverse slice showing a protruding screw (arrow); (b) the coronal slice showing a protruding screw (arrow); (c) the transverse slice showing a protruding acetabular rim (arrow). CT, computed tomography.

flushed continuously during the resection of the rim and thoroughly thereafter to prevent thermal necrosis. The overriding screw tip was shortened as well because of its possibly dangerous proximity to the sacral plexus and sciatic nerve origin. A soft-tissue layer was placed over the tapered cage rim to cushion the sciatic nerve and to avoid direct contact between the nerve and the acetabular cage. Postoperatively, there were no signs of new sensory or motor sciatic nerve damage and pain due to hip mobilization had disappeared. Full weight-bearing as tolerated was allowed on the ward.

Outcomes and follow-up

On the second week after the operation, the patient noticed that the shooting pains had now been replaced by muscle soreness. At 3 months, all wounds had healed uneventfully, and standing as well as ambulation for short distances was possible without pain. Assistive devices while mobilization were no longer needed. A mild Trendelenburg limp due to muscle atrophy was present after a long phase of wheelchair mobility. At 6 months, the positive results persisted. Ambulation without walking aids and sitting for extended

Table 1

Nerve conduction velocity studies.

Nerve	Distal latency (ms) measured value (normal value)	l Distal amplitude (mV/µV) * measured value (normal value)	Conduction velocity (m/s) measured value (normal value)	F-wave latency (ms) measured value (normal value)
Left peroneal motor	4.08 (<5.5)	3.6 (>4)	58.3 (>45)	No wave obtained
Right peroneal motor	3.54 (<5.5)	5.3 (>4)	48.8 (>45)	42.2 (<56)
Left sural sensory	2.05 (<3.6)	19.7 (>4)	43.9 (>42)	
Left tibial motor *mV for motor	5.42 (<6.1)	14.3 (>3)	44.8 (>45)	42.8 (<56)
nerves, µV for sensory nerves				



Figure 3. Intraoperative findings by the Kocher-Langenbeck approach: (a) tensioned sciatic nerve (thick arrow) over the acetabular cage rim (thin arrow); (b) after tapering of the rim and neurolysis, the sciatic nerve is no longer at tension.

periods of time without pain were possible. Her mobility had returned to the state before the initial operation. The patient was contacted by telephone 15 months after the operation. Here, she stated not to have any pain seated or standing, stated that her mobility had stayed the same, and denied any performed or planned treatment of the hip since her last clinical follow-up in our clinic.

Discussion

Modern acetabular cages have specific risks that are not fully known yet. Their use is expected to rise with increasing demand for THA and revision total hip arthroplasty (rTHA). A reliable and durable anchorage of the components and the restoration of the correct center of rotation are of utmost importance, especially in an increasingly young population. In our fragile patient, we found the protruding rim of the acetabular cage responsible for intense complaints. As its anchorage was still excellent, we explicitly searched for a way to keep the acetabular cage in place. Revision of the cage construct would have increased the risk of further revisions because of infection and aseptic cup loosening because of the decreased acetabular bone stock [9].

Good data are available on motor palsy after primary THA, where the incidence varies between 0.3 and 3.7%. It is associated with spinal stenosis, smoking, and age younger than 50 years [10]. Revision THA has shown a higher prevalence of motor palsy, with some reports mentioning a rate ranging up to 7% [11,12]. Usually, this is interpreted as a consequence of the technical difficulties during surgery and a generally higher incidence of complications in rTHA [13]. The Kocher-Langenbeck approach is also known to have an increased risk of immediate and delayed sciatic nerve injury [14], most probably due to direct exposure of the sciatic nerve to parts of the acetabular implant. This may develop in time as 10% of posterior hip capsule repairs and 43% of ligated short external rotators may be deficient after 3 months [15]. Managing these types of delayed nerve injury in rTHA can be challenging. In contrast to the wealth of data on THA and rTHA, data on complications after the use of acetabular cages are sparse. Xu et al reviewed 15 cases of delayed neuropathy after THA from 1950 to 2015 and found it to be a result of hardware irritation, component failure, and wear-related pseudotumor formation [16]. When repeating the search for 2015-2020, a single case series of 12 patients could be found [17]. When combining all cases, a very heterogeneous population is found from which no general conclusions can be drawn. More research will be needed to develop this field further.

Motor palsy is not regularly defined as a primary outcome measure as the emphasis of these reports mostly lie on performance of the achieved anchorage. In a review of Aprato et al, on 1327 acetabular cages, motoric deficit due to nerve impingement was not mentioned as a possible complication [18]. The authors suspect the overall incidence of motor palsy after acetabular cage implantation being at least equally high as in rTHA and its absence most likely a sign of under-reporting. Nevertheless, a protruding cage rim as cause of the impingement is decidedly rare.

Management of nerve palsies is mostly conservative to avoid associated intraoperative risks of nerve exploration. Regrettably, this approach has a limited certainty of successful nerve recovery [19]. Our case represents a rare operative indication because of the intermittent and position-depending occurrence of the sciatic pain, which suggests a mechanical cause of the symptoms. Surprisingly, however, our case also shows that even after performing careful staged diagnostics and preoperative planning, one can still be surprised by an unexpected intraoperative finding, as to what the exact cause of impingement is.

Avoiding sciatic nerve impingement can only be performed by careful preoperative planning, proper choice of implant, adjustment of the implant to accommodate the patient's anatomy, and avoiding direct contact between components and the sciatic nerve. Respecting the complexity of acetabular defects and the use of an accurate classification of acetabular defects can help guide preoperative planning and intraoperative management. However, one must be weary of "one-size-fits-all" techniques, as they usually lack respect for the local anatomical restrictions. Neglecting this could increase the risk of loosening, late cage fractures, dislocations, and sciatic nerve injuries [20,21].

To choose the correct implant, the design must be taken into account. The 3 most common antiprotrusion systems are the Müller ring [22], the Ganz ring [23], and the Burch-Schneider cage [24,25]. Of these 3, the Burch-Schneider cage protrudes the most toward the greater sciatic notch from where the sciatic nerve runs down. It therefore was surprising to find the acetabular rim of the Müller ring responsible for the impingement. In our case, the cage was adjusted using a diamond burr to accommodate the anatomy and release the sciatic nerve. In similar cases, it is therefore advised to look intraoperatively to the relation between the nerve and the cage in different positions of the hip joint, especially in small and fragile patients where oversizing can be a problem. In case of dangerous proximity to the sciatic nerve, adjustment of the cage itself may be warranted during implantation.

Summary

A patient with an impingement of the sciatic nerve due to a protruding rim of a revision acetabular cage has been presented. Treatment modalities for acetabular defects must be steered by validated protocols; however, the optimal treatment still must be adjusted to every specific patient profile. "One-size-fits-all" implants must be adjusted to the anatomy of the patients instead of adjusting the anatomy of the patient to the implant. Surgeons must decide on the techniques, implants, and materials carefully to minimize the risk of motor palsy in acetabular cage systems. In case of similar symptoms, nerve impingement as a possible origin of complaints must be considered.

Conflict of interests

The authors declare there are no conflicts of interest.

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