Late Hardware-induced Sciatic Nerve Lesions After Acetabular Revision

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Abstract We encountered late hardware-induced sciatic nerve lesions after acetabular revision in six patients. There were five female patients and one male patient. The mean age of the patients at the time of index acetabular revision was 59.3 years (range, 42–76 years). The interval from the index acetabular revision to the onset of sciatic nerve symptoms averaged 9.4 months (range, 4–16 months) and that from the onset of symptoms to nerve release was 11.3 months (range, 8–13 months), except in two patients with intermittent symptoms in which it was 9 and 10.5 years, respectively. Sciatic nerve release was successful in two patients, but in four patients, the nerve had been partly or entirely cut by the metallic hardware resulting in a permanent deficit. The minimum followup was 2 years (mean, 4 years; range, 2–7 years). Mechanical irritation should be suspected in the case of any late sign of peroneal neuropathy after acetabular revision with a macropup or antiprotusion device, and plate fixation of the posterior column. We recommend exploration and nerve release before a permanent lesion of the nerve has developed.

Level of Evidence: Level II, prognostic study. See the Guidelines for Authors for a complete description of levels of evidence.

Introduction

Nerve lesions after hip arthroplasty are disabling complications occurring in 0.06% to 2.2% of arthroplasties [4, 7, 10–12, 16]. Most of them are surgery-related and occur during the procedure or are delayed by a few days [9], especially when caused by a hematoma [2, 6]. Late nerve lesions are uncommon and are reported only sporadically [3, 5]. Late hardware-induced sciatic nerve complications are rarely reported [1, 8, 14]. In three such reports, a migrated fragment of a Kirschner wire violated the sciatic nerve 6 years after THA [1], the posterior flange of a protrusion ring cut the sciatic nerve 3 months after THA [8], and a loose screw after pelvic reconstruction with a plate caused sciatic nerve entrapment 6 months after THA [14].

We report six cases in which metallic hardware used for acetabular revision caused severe late problems to the sciatic nerve.

Materials and Methods

From 1991 to 1998, we performed 1570 hip revision arthroplasties in our hospital. Acetabular revision was included in approximately 90% of the patients. According to our practice, the loosened acetabular cup was replaced by a larger porous-coated press-fit cup whenever possible, combined with autogeneic or allogeneic bone transplantation for the cavitary defects. In cases with more severe segmental defects, we used a reinforcement device with

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allogeneic cancellous bone graft. The Burch-Schneider ring (Sulzer Medica AG, Winterthur, Switzerland) was most popular during that period. Plate fixation of the posterior column also was performed in the patients with a posterior column fracture or pelvic discontinuity. We encountered late hardware-induced sciatic nerve lesions in six patients (0.4%). There were five female patients and one male patient. The mean age of the patients at the index acetabular revision was 59.3 years (range, 42–76 years). The interval from the index acetabular revision to the onset of symptoms averaged 9.4 months (range, 4–16 months) and that from the onset of symptoms to nerve release was 11.3 months (range, 8–13 months). In two patients, symptoms were intermittent, and surgery was scheduled 9 and 10.5 years, respectively, after the onset of symptoms. All patients reported pain and numbness in the peroneal area of the ipsilateral lower limb, and peroneal weakness. Some kind of local pain at the hip level also was present. The minimum followup was 2 years (mean, 4 years; range, 2–7 years).

Patient 1

In 1984, a 40-year-old woman had an uneventful THA resulting from congenital hip dysplasia-induced arthritis. She did not have previous hip surgery. She did well for 5 years postoperatively, but in 1991, revision was performed because of loosening of an uncemented threaded cup. A macrocup (68-mm Universal®; Biomet, Inc, Warsaw, IN) was implanted. One year after the revision surgery, the patient reported slight radicular pain in her leg on the surgically treated side, and in 1994, electroneuromyography (ENMG) showed slight neurogenic findings in the muscles innervated by the peroneal division of the sciatic nerve at the level of the hip but no clear injury of the sciatic nerve. She did quite well and could walk 2 to 3 km without difficulties but still reported slight sporadic sciatic pain. In 2001, the patient experienced temporary severe numbness and fatigue in her leg. Electroneuromyography showed slight to moderate neurogenic findings in the muscles innervated by the peroneal division of the sciatic nerve. The injury was localized at the level of the hip. The plain hip radiographs were interpreted as normal. Magnetic resonance imaging of the lumbar spine showed only pre-sacral protrusion. Sciatic nerve exploration was postponed because the symptoms resolved spontaneously. In 2003, 12 years after the cup revision, disabling sciatic nerve symptoms recurred. Radiographs were interpreted as normal (Fig. 1). However, sciatic nerve injury was apparent on ENMG, and surgery was scheduled. The sciatic nerve was tightly adherent to the corner of the macrocup and severely entrapped (Fig. 2). The nerve was released, and a

Fig. 1 A radiograph of Patient 1 taken 12 years after a macrocup revision arthroplasty was interpreted as normal. The arrow shows the anatomic location prone to nerve lesion.

Fig. 2 A photograph shows an impingement of the sciatic nerve (CHIA) caused by the edge of the acetabular macrocup and scar tissue in Patient 1.

connective tissue flap was developed between the ring and the nerve (Fig. 3). No resection of the corner of the macrocup was performed to avoid excessive metallic debris formation during the procedure. Sciatic pain resolved, and 2 months after surgery, the patient was free of symptoms.

Patient 2

A 59-year-old woman with rheumatoid disease since 1974 had acetabular revision performed in 1996 with a Burch-Schneider ring. Primary THA was performed in 1989. During the next 2 years after the revision, a slight peroneal paresis developed. In December 2005, she experienced severe radicular pain in her ipsilateral lower limb. Lumbar MRI was normal. On plain radiographs, the distal part of

Fig. 3 A connective tissue flap (arrows) was developed between the acetabular shell and the nerve shown in Figure 2.

Fig. 4 A radiograph of Patient 2 taken 9 years after acetabular revision with a Burch-Schneider ring shows some loosening of the distal plate. The arrow shows the level of the nerve injury.

the plate seemed loose (Fig. 4). Electroneuromyography suggested a sciatic nerve lesion at the level of the hip; the peroneal division was severely injured and the tibial division moderately injured. In surgery, the distal half of the plate was loose, the sciatic nerve was adherent to the plate at the joint level, and there were metallosis and severe damage of the nerve induced by the corner of the Burch-Schneider ring. The nerve was released without any attempt at reconstruction (Fig. 5). The Burch-Schneider ring was refixed, and the posterior fracture was reconstructed with a titanium plate (LCP® Reconstruction Plate, Synthes AG, Solothurn, Switzerland) and a titanium mesh,

Fig. 5 The sciatic nerve was severely damaged at the joint level by the Burch-Schneider ring (arrow) in Patient 2. The picture was taken after reconstruction of the posterior column fracture and before creation of a protective connective tissue flap.

also using bone transplants. After that, the nerve was protected with a substantial amount of subcutaneous fat tissue between the reconstruction plate and the nerve. The pain resolved, but the peroneal paresis did not recover.

Patient 3

In 1996, a 65-year-old man underwent acetabular revision with a Burch-Schneider ring. Total hip arthroplasty resulting from primary arthrosis had been performed 10 years earlier. The patient was free of symptoms for 1 year, but the plate was broken. After 2 years, he was still free of symptoms. No cup migration was detected, and bone healing was good. In 2000, the patient reported some pain and numbness in the peroneal area of the ipsilateral lower limb. Peroneal weakness was detected. Electroneuromyography showed severe nerve injury in the muscles and nerves innervated by the peroneal division of the sciatic nerve. The nerve lesion was localized at the level of the hip. At surgery in 2000, the broken end of the plate had violated the peroneal division of the sciatic nerve; approximately $\frac{1}{3}$ of the nerve was lacerated. The broken plate was removed, and the nerve was released but not reconstructed because of the chronic lesion, the age of the patient, and the long distance to the muscles. The pain remitted, but the patient had marked peroneal palsy at the 1-year clinical followup, which remained when the patient was interviewed by telephone 7 years later.

Patient 4

In 1991, a 67-year-old woman had acetabular revision using a 62-mm Universal® cup. The primary THA was performed in 1984 with a Lord prosthesis (Benoist Girard, Bagneux, France) as a result of osteoarthritis. Nine months after the acetabular revision, hip pain developed, and during the next 3 months, peroneal palsy appeared. Electroneuromyography showed a severe lesion of the peroneal division of the sciatic nerve at the level of the hip and moderate changes in the tibial division. Surgery was performed within a few months in 1993. The sharp corner of the metallic acetabular shell had cut the sciatic nerve almost entirely. The nerve was only released. After 4 years, her neuromotor symptoms and signs were unchanged.

Patient 5

In 1996, a 76-year-old woman underwent acetabular revision with a Burch-Schneider ring 14 years after her primary THA. Four months after the revision surgery, sciatic pain developed. There was no muscular weakness. In May 1997, ENMG showed moderate neurogenic findings, but it was impossible to differentiate the findings between L5 nerve root lesion and injury of the peroneal division of the sciatic nerve. Spinal causes of the nerve lesion were excluded by computed tomography, and the pain resolved to some extent. However, during the next few months, peroneal paresis developed, and 1 year after the index surgery, ENMG showed progression of the nerve injury in the peroneal division of the sciatic nerve. There were also slight findings in the muscles innervated by the tibial division. The nerve lesion could now be localized at the hip level. At surgery 1 year after acetabular revision, the sharp corner of the Burch-Schneider plate had cut the peroneal division of the sciatic nerve. The nerve was released, and neurography of the peroneal division was performed by a younger colleague. The pain resolved, but no recovery was detected during the next 4 years before her death resulting from unrelated causes.

Patient 6

In 1998, a 42-year-old woman had acetabular revision with a Burch-Schneider ring. Her primary THA was performed in 1982 as a result of Perthes disease, and successive acetabular revisions then were performed in 1989, 1994, and 1995 as a result of recurrent dislocations. She also had iatrogenic injury of the superior gluteal nerve diminishing the abduction strength of the hip. The patient began to report some pain from 2002 to 2004 in

her ipsilateral lower extremity, and acetabular revision was again scheduled in 2004 as a result of loosening of the cup. During surgery, the broken distal plate of the Burch-Schneider ring was removed, and the sciatic nerve was released. The sciatic nerve was severely tethered to the broken plate and compressed by the plate and scar tissue. Postoperatively, Grade 3/5 peroneal palsy was detected, but it recovered substantially during the next 3 years. In all likelihood, the patient was treated before severe damage of the sciatic nerve had developed from the hardware.

Discussion

A clinical neurologic deficit is not uncommon after THA, occurring in 0.06% to 2.2% of arthroplasties [1, 4, 7, 10–12, 16]; many are related to hematomas or technical errors. In the series of 4339 THAs performed at our institute, we have observed only 27 sciatic nerve lesions (0.6 %) [11]. In another series of 3126 THAs, 42 sciatic nerve lesions occurred (1.3 %) [12]. Few articles describe late neuropathies, especially those induced by hardware. Masses from particulate debris related to acetabular loosening [5] and migration of a trochanteric wire after THA [1] can cause late sciatic neuropathy. An acetabular reinforcement ring has been reported to cause impingement of the sciatic nerve between the dorsal aspect of the acetabular reinforcement ring and scar tissue [13]. We detected similar findings in two patients (Patients 1 and 4). In one reported case [8], an antiprotusion ring caused a sciatic nerve lesion 3 months after THA when the patient fell on his involved hip. The sciatic nerve was cut through by approximately 1/3 apparently by the prominent edge of the nonanatomic antiprotusion ring [8]. The nerve was released approximately 10 cm proximally and distally from the hip and protected using a posterior third of the gluteus medius [8]. Our Patients 2, 3, and 5 showed similar lesions of the sciatic nerve. In another case [14], a loose screw of the reconstruction plate after THA caused sciatic nerve problems 6 months after surgery. Screw removal and nerve release 6 months after the onset of symptoms enabled substantial improvement in clinical symptoms [14]. We observed similar findings in one of our patients (Patient 6).

It is important to recognize the cause of symptoms even years after THA and especially after acetabular revision. Our first patient was fortunate enough to be treated before a permanent lesion had developed. Also, Patient 6 had surgery early enough as a result of the need for revision arthroplasty. At an early stage, it may be difficult to distinguish between spine- and hip-related causes of symptoms, and therefore lumbar imaging modalities might be necessary. We did not find in our material any alerting

radiographic signs, except breakage or loosening of a reconstruction plate.

Mechanical irritation should be suspected in cases of any late sign of peroneal neuropathy after acetabular revision with a macrocup or antiprolusion device and after plate fixation of the posterior column. In posterior column fractures, we have used a locking compression titanium reconstruction plate with combined holes. A straight plate is bent to meet the anatomy of the posterior wall of the acetabulum. A reconstruction plate seems to cause a bigger risk to bring about nerve problems than a macrocup. Ten to 15 years ago, we used antiprolusion ring, macrocup, or plate fixation only in approximately 10% of all acetabular revision cases. If the posterior acetabular wall is so defective that a macrocup extends over the bone rim, the risk seems greater. The most susceptible place for nerve injury is the posterior lateral corner of the macrocup or reconstruction plate in the proximal part of the posterior acetabular wall.

The sciatic nerve should always be identified during THA, preferably by palpation [15]. We now routinely expose the sciatic nerve during revision surgery and develop a protecting connective tissue flap between the nerve and hardware if needed. Ten years ago, that was not routine. We have not observed any adverse effects with nerve exposure, although harmful irritation by hardware would be possible, especially without connective tissue flap protection.

One should always keep in mind the potential risk for nerve entrapment or lesion when using antiprolusion devices or when a posterior column has been reconstructed, and moreover, if a macrocup has been used in acetabular reconstruction. In the case of sciatic nerve symptoms, careful repetitive ENMG examinations are mostly decisive. Exploration and nerve release should be performed before a permanent lesion of the nerve has developed. We recommend using a connective tissue flap between the sciatic nerve and metallic hardware to minimize the risk of late nerve lesions.

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