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Avascular Necrosis of the Femoral Head After Surgery for Lumbar Spinal Stenosis

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Study Design. Case report.

Objective. To report a previously undescribed complication of lumbar spinal surgery under prolonged hypotensive anesthesia.

Background Data. Avascular necrosis of bone most commonly affects the femoral head. The etiology of the condition is understood in only 75% of cases. There have been no prior reports of this condition following lumbar spine surgery carried out under hypotensive anesthetic.

Methods. Notes review, clinical examination, plain radiographs, and magnetic resonance imaging diagnosed three patients who developed avascular necrosis of the femoral heads (five joints in total) after surgery for lumbar spinal stenosis. All three were treated with total hip replacement (five joints), and the diagnosis of avascular necrosis was confirmed in two by histopathological examination.

Results. All three patients have recovered full mobility following hip replacement surgery. None had any residual symptoms of lumbar spinal stenosis or hip disease, and none of them had shown any clinical evidence of avascular necrosis in any other bone.

Conclusions. The development of avascular necrosis of the femoral heads following surgery for spinal stenosis may be due to hypotensive anesthesia, prone positioning on a Montreal mattress, or a combination of the two. Careful intraoperative positioning may reduce the risk of this occurring after spinal surgery. However, close postoperative surveillance and a high index of suspicion of worsening hip pathology in patients who appear to mobilize poorly after lumbar spinal surgery may be the only method of early detection and treatment for this condition. Key words: lumbar spinal stenosis, avascular necrosis of bone, femoral head, osteoarthritis, hypotensive anesthesia, intraoperative positioning] **Spine 2003; 28:E364–E367**

Nontraumatic avascular necrosis (AVN) or osteonecrosis of bone is a well-studied clinical entity but the underlying pathophysiology is unclear in many cases. Any bone that is formed in cartilage may be affected by AVN, but the most common and best-described site is the femoral head.[1] Many systemic conditions are associated with AVN, but 25% of all cases are described as idiopathic in which the etiology is obscure and often thought to be due to a combination of factors rather than a single injury or isolated pathologic process.[2–5] Chang *et al* suggest all cases of nontraumatic AVN should be considered idiopathic until it is known what is occurring at a cellular level.[1]

Occlusion of subchondral arterioles by intravascular fat emboli, compression of vessels by progressive accumulation of marrow fat stores, and intraosseous hypertension are some mechanisms implicated as local causes of ischemia and cell death.[1,4–6] At a cellular level, local alteration of arteriolar and capillary blood flow can be postulated as the final common pathway resulting in ischemia and ultimately AVN.[1,5,7]

Gross arterial and venous occlusion produces tissue ischemia, and the latter may be implicated in AVN of the femoral head associated with some forms of arthritis.[7] If the pressure within a hip joint rises as a result of an effusion, the subcapsular vessels may be compressed enough to produce venous hypertension of the femoral head leading to low local oxygen tension, marrow edema with increase in marrow pressure, and thereby further ischemia.[8] Although there is a rich anastomosis of vessels around the hip, it is easy to postulate that critical compression of large vessels at the level of the inguinal ligament might also produce sufficiently altered local hemodynamics that would lead to venous hypertension. If the arterial pressure was low at the same time, the combined effect might be sufficient to produce local ischemia and subsequently AVN in a hip at risk, such as one with mild to moderate osteoarthritis. There have, however, been no reports to our knowledge linking AVN of the femoral head with hypotension and local vascular impairment during anesthetic.

We describe a series of three patients who underwent a multiple level lumbar spinal decompression for spinal stenosis using a well-described technique of hypotensive anesthesia in the prone position. Two subsequently developed bilateral AVN of the femoral heads and 1 unilateral AVN within 4 months of recovery from the spinal surgery. None of them had any recognized risk factors for the development of AVN.

Case Report

Case 1. A 68-year-old woman was referred with symptoms of severe spinal stenosis. She was a healthy nonsmoker with no recognizable risk factors for AVN. Her claudication distance was 25 m. She had no symptoms of hip osteoarthritis. Her spine was virtually immobile. Her right hip was markedly stiff

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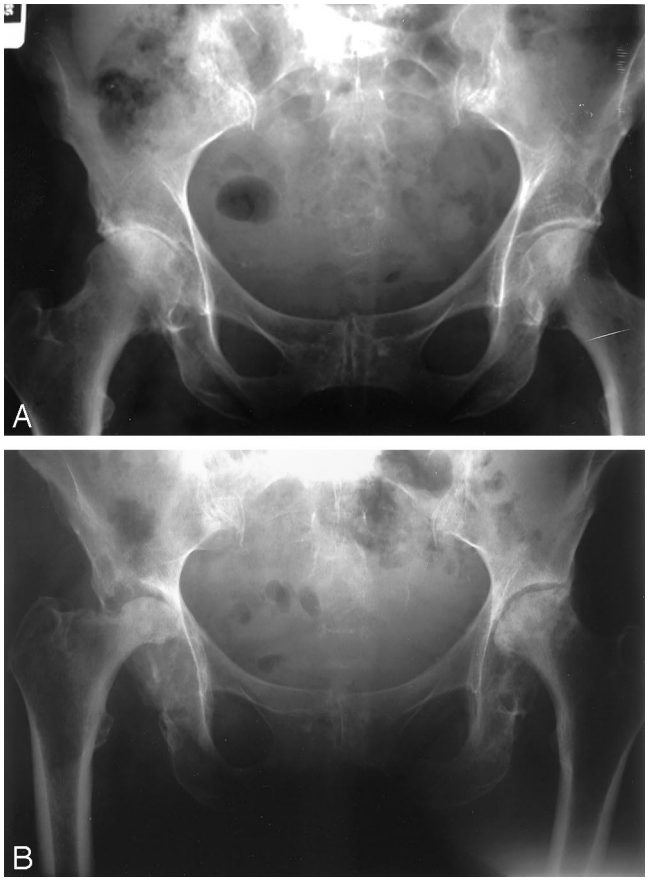

Figure 1. **A,** Plain radiographs of the hips showed osteoarthritis of the right hip and an essentially normal left hip. **B,** Radiographs show collapse of both femoral heads 8 weeks after spinal surgery. Note the degree of shortening, hence the patient's difficulty in maintaining mobility.

in internal rotation and flexion. Her left hip moved normally and neither was painful.

Plain radiographs showed a degenerative spondylolisthesis at L4–L5, degeneration at L5–S1, and gross osteoarthritis of the right hip with minor degeneration in the left hip (Figure 1A). Magnetic resonance imaging (MRI) of the spine showed multiple levels of degeneration with significant spinal stenosis at L4–L5 and L5–S1 in combination with a degenerative (Type III) spondylolisthesis at L4–L5.

A wide decompression and an instrumented fusion from L4–S1 was carried out. The anesthetic time was 160 minutes in total, and there were no immediate postoperative complications. The mean intraoperative blood pressure was 58 mm Hg. She was discharged to home on the fourth postoperative day.

Eight weeks after surgery, her claudication symptoms had improved considerably, but she mentioned aching in her left hip. Her pain worsened over the next 2 months, and radiographs of the hips 4 months after surgery showed a marked deterioration in the hips with collapse of the femoral heads suggestive of bilateral AVN (Figure 1B). Bilateral total hip replacements were successfully performed in stages and histologic review of the femoral heads confirmed AVN bilaterally.

Case 2. A 73-year-old healthy, nonsmoking woman was referred with a 1-year history of bilateral calf and posterior pelvic pain on walking. Her claudication distance at presentation was 200 m. Clinical examination revealed a stiff and painful left hip and a stiff but painless right hip. Her lumbar spine was globally stiff but movements were not painful. The only neurologic loss in the legs was bilaterally absent ankle jerks. Straight leg raising was 75° bilaterally with no sciatic irritation.

Plain radiographs showed moderate osteoarthritis of both hips. There were established degenerative changes of the lumbar spine especially at L3–L4 and L4–L5.

Initially, she was treated conservatively with physiotherapy and nonsteroidal anti-inflammatory medication.

Over the next 3 months, her hip pain settled, but her claudication distance reduced to 100 m. She developed worsening left L3 and L4 motor and sensory deficits. Magnetic resonance imaging of the lumbar spine showed widespread degenerative changes with marked central and foraminal stenosis at L3–L4 and L4–L5. She consented to operation and underwent a multiple-level decompression 6 weeks later.

The procedure was uncomplicated. The anesthetic lasted 180 minutes, and the mean arterial pressure was maintained at 60 mm Hg. The central canal, lateral recesses and nerve root canals at L3–L4 and L4–L5 were fully decompressed. She made an uneventful postoperative recovery and was discharged to home 4 days after surgery. Six weeks after surgery, her walking distance had improved to 300 m using elbow crutches and she was pain free.

Within the next 2 weeks, she developed increasing groin and lateral thigh pain, which was thought to be reactive pain following her spinal surgery and the physiotherapy she had received after operation. She was next reviewed at 5 months after surgery when her mobility was continuing to deteriorate. Radiographs of the hips at this point showed severe collapse of both femoral heads consistent with AVN. She had bilateral total hip replacements 4 weeks later, from which she made a good recovery.

Case 3. A 73-year-old tree surgeon was referred complaining of left groin pain radiating to the knee and down the front of the shin. His symptoms came on after an hour of walking and made him restless at night. Up until then, he was managing on anti-inflammatories but had been steadily deteriorating.

Examination showed a globally stiff spine. He had a diminished left knee reflex, but no other neurologic loss in the legs. Hip examination was consistent with mild osteoarthritis. Spine and hip radiographs and an MRI of the spine revealed a fixed, degenerate scoliosis of the lumbar spine with spinal stenosis between L2–L3 and L4–L5 and mild osteoarthritis of the left hip (Figure 2A). He was admitted for a spinal decompression of L2–L3, L3–L4, and L4–L5 8 weeks later.

The procedure was uncomplicated, and the anesthetic lasted 190 minutes from induction to extubation with the mean arterial blood pressure maintained at 65 mm Hg. Recovery after operation was uneventful, and at the routine 6-week follow-up he was doing well with a walking distance of 500 m. He was, however, being increasingly bothered by left groin pain. Over the next 4 months this became disabling, and at the 6-month postoperative review, he was found to have severe left hip pain. Radiographs suggested AVN of the left femoral head (Figure 2B). Magnetic resonance imaging confirmed this (Figure 2C).

He underwent left total hip replacement 10 weeks later. Once again, the histopathological evaluation of the femoral head confirmed the diagnosis of AVN in the presence of severe osteoarthritis. He has subsequently regained hip and spine function and has been climbing trees again.

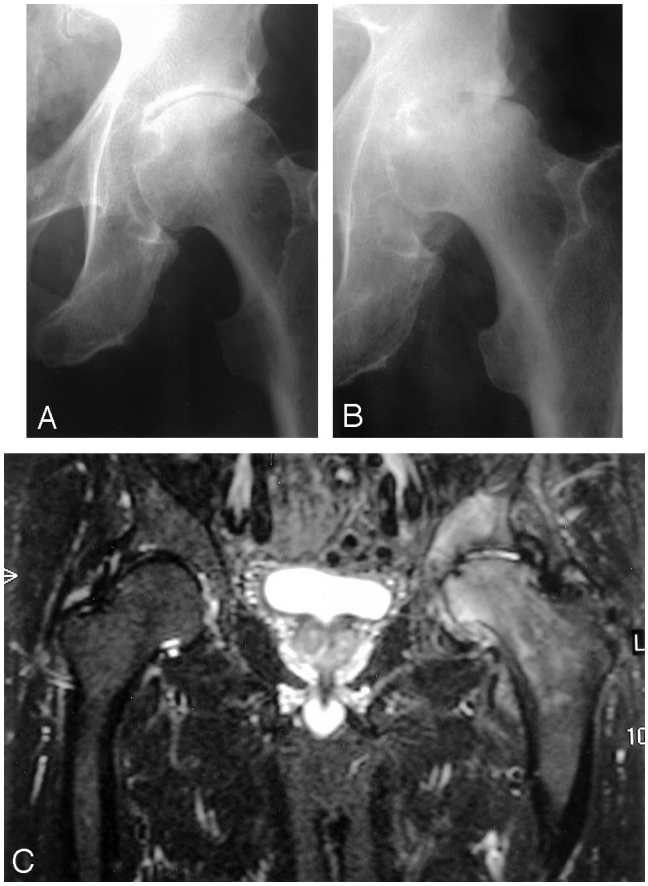

Figure 2. **A,** The osteoarthritis of the left hip was mild in the preoperative review of hips and spine. **B,** Radiograph showed collapse of the left femoral head, suggesting avascular necrosis. **C,** T2 STIR-weighted MRI scan of the hips shows evidence of collapse of almost the entire cortical surface of the left femoral head with edema on both sides of the joint and a small effusion. The appearances are entirely consistent with end-stage avascular necrosis. The right hip appears normal.

The anesthetic technique was similar in all three cases and provided by the same consultant anesthetist. General anesthesia was induced with midazolam 0.05 mg/kg, followed by propofol 1–1.5 mg/kg, mivacurium 0.25 mg/kg, and remifentanil 1.0 μg/kg. The lungs were ventilated with oxygen (FiO_2 0.5 L), nitrous oxide, and isoflurane (E_T 0.8%) *via* a circle system. An infusion of remifentanil (0.1–0.15 μg/kg/min) was established after the patient was turned prone for surgery and titrated according to the mean arterial pressure, with a target level of 50 to 60 mm Hg during surgery. Thirty minutes before the end of surgery, the remifentanil infusion was reduced to 0.05 μg/kg/min to allow the resumption of normal spontaneous respiration after surgery. Morphine (0.15 mg/kg) was administered intravenously 20 minutes before the end of surgery to provide postoperative pain relief.

■ Discussion

Hypotensive anesthesia is a well-described technique, widely accepted as an effective method of attaining a bloodless field and ensuring minimal blood loss in a variety of surgical procedures.[9] By artificially lowering the intraoperative blood pressure, the anesthetist is able to ensure minimal loss of blood without compromising essential perfusion of vital organs. Hepatic function,[4,9,10] renal function,[10,11] cardiac, or cerebral function[10] are not altered, although there may be short periods in which the function of these organs is impaired before returning to their usual state.[12] Proper positioning of the patient and controlled ventilation aid the technique of hypotensive anesthesia, but the risk of underperfusion of vital organs, *e.g.*, the brain, spinal cord, and myocardium, still exists.[7] During deliberate hypotension induced by a number of agents, including isoflurane, $Pa0_2$ decreases to a considerable degree due to suppression of hypoxic pulmonary vasoconstriction.[13] Prevention of underperfusion and the resultant hypoxia is achieved by monitoring the blood pressure at regular intervals throughout the procedure and maintaining mean arterial blood pressure at between 50 and 60 mm Hg.

Ischemia in the legs due to occlusion of the femoral artery during spinal surgery in the prone position is described.[14,15] Limb ischemia, however, has not been related to hypotensive anesthesia nor has femoral artery occlusion to AVN of the hips.

Alteration of the blood supply to vital organs during hypotensive anesthesia is well established.[4,9–11] Dastych *et al* describe altered creatinine clearance in the kidney for up to 42 hours following hypotensive anesthesia.[12] This would suggest that although vital organ function is not affected in the long-term, other perhaps more vulnerable systems might, in certain circumstances, receive enough of an injury to tip the balance, particularly when autoregulatory systems are not in place.

Due to the compartmental nature of the bone unit, intraosseous hypertension due to venous congestion has been proposed as a mechanism for damage to the joint.[3,5] Vossler *et al* describe altered blood flow through the femoral arteries during spinal surgery in the prone position resulting in limb ischemia,[14] whereas Ziser *et al* describe raised creatinine kinase and myoglobinuria due to muscle damage.[15] Akagi *et al* suggest that prolonged direct pressure on the inguinal area, as is potentially produced during prone positioning, can result in arterial thrombosis. This area is potentially at greater risk with the obese patient, long operating times, and thrombotic disorders.[16] During the course of the operative procedure in the prone position, enough pressure is placed on the arteries to limit their already altered efficiency due to the hypotensive anesthetic technique.[14,16] In addition, venous obstruction at this level is likely to result in intraosseous venous congestion and an increase in intraosseous back pressure without adequate perfusion at the arteriolar and capillary level of bone. With mean arterial pressure usually in the range of 50 to 60 mm Hg, the flow to the femoral head is potentially sufficiently compromised so as to act as the "final hit" in an accumulative stress theory, as suggested by Kenzora and Glimcher.[4] It is questionable as to whether this alone would be enough to explain the development of AVN, as the stress the joint is placed under would only be short

lived, and some authors state that a single stress would be insufficient. We know that reduction time in dislocation of the hip plays a role in the risk of development of later AVN,[2] but it is hard to assess whether the brief period of surgery would be enough to result in bone death. It is easier to explain if the bone is already significantly compromised and the temporary ischemia is the final factor. Another contributory factor may be the increased mobility in patients following decompressive spinal surgery with relief of leg claudication. This would lead to increased strain on already weak bone and therefore encourage damage to the bone trabeculae.[10]

The three patients we describe had their spinal and hip surgery performed by an experienced orthopedic surgeon with a special interest in lumbar spine surgery. During the period of this study, 118 other patients underwent similar procedures. A single consultant anesthetist administered the anesthetics. The technique of hypotensive anesthesia employed was standard as described above.

The lengths of case were not unusual, nor were any of the pre- and postoperative factors. What links these cases, however, is the preoperative clinical and radiologic osteoarthritis of the hips. All five affected joints showed some degree of osteoarthritis before their spine surgery. It seems likely that the impact of spinal surgery, with the combination of arterial hypotension and venous congestion, can produce critical ischemia in a femoral head at risk given the known association of osteoarthritis and AVN.[7]

■ Conclusion

No single factor can be clearly defined as the cause of AVN in these patients, although hypotensive anesthesia and patient positioning must certainly be considered to be possible contributors. Because the early detection of this condition can have an influence on the eventual prognosis, recognition of this complication must be considered in patients with altered symptoms during their recovery from spinal surgery performed under hypotensive anesthesia, as symptoms may precede the plain radiologic appearances suggesting AVN by 2 months.[17] It is possible, however, to detect AVN changes on MRI much earlier. Patients ought to be aware of the existence of this potential complication when consenting to spinal decompression, with or without fusion. Surgeons, anesthesiologists, and theater personnel also need to be aware of this potential problem and take particular care in managing the patient intraoperatively so as to protect against this potentially avoidable complication.

■ Key Points

- Multiple theories regarding the pathogenesis of osteonecrosis of the femoral head are evaluated.
- Osteonecrosis of the femoral head following spinal surgery under hypotensive anesthesia has been observed in three patients involving five femoral heads.
- This condition has not been previously described in this group of patients or following this method of anesthetic.
- We suggest possible explanations to the pathogenesis.

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