Acetabular Rim Fracture after Adult Spinal Deformity Surgery-Induced Secondary Hip Osteoarthritis: Two Case Reports

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The incidence of adult spinal deformity has increased, resulting in an increase in the number of spinal fusion surgeries. Spinal fusion surgery has several issues. Recently, several studies reported that adjacent joint disease on hip joint is a complication after spinal fusion surgery[1-3]. We encountered two cases of hip osteoarthritis (HOA) with acetabular rim fractures after spinal fusion surgery.

A 76-year-old woman underwent spinopelvic fixation surgery from T9 to the ilium because of degenerative kyphosis due to proximal junctional kyphosis after spinal fusion surgery from L3 to L5 (Fig. 1). After the surgery, lumbar lordosis (LL) and pelvic tilt (PT) changed from −18° to 49° and 28° to 25°, respectively, with pelvic incidence (PI) of 50°. She could walk; however, her right hip joint pain developed during the 4 months following spinopelvic fixation surgery without any history of trauma. Her bone mineral density (BMD) was 860 (mg/cm^2) (T score, −0.2). Four months later, radiography showed that her acetabular rim was fractured. The Kellgren-Lawrence (KL) grade changed from 0 to 3. She underwent total hip arthroplasty (THA) 6 months after spinopelvic fixation surgery. The patient's postoperative course was uneventful for 5 years after THA (Fig. 1).

A 76-year-old woman complained of low back pain, which was thought to be induced by degenerative scoliosis at the proximal junction after spinal fusion surgery from L4 to L6. L6 was fused with the sacrum alar through the transverse process, and the bony union was recognized at L4-L6 after the previous surgery. It was thought that the L4 to pelvis was one unit, so she underwent spinal fusion surgery from T9 to L6. Her BMD was 644 (mg/cm^2) (T score, −2.4). LL, PT, and the Cobb angle (L2-L5) improved from 48° to 50°, 24° to 23°, and 53° to 20°, respectively, with a PI of 60°. After the surgery, her low back pain was improved and she could walk; however, the acetabular rim was fractured and her right hip joint pain occured 5 years later without any history of trauma. She could control her hip pain using medication and walk with a gait aid 7 years after her surgery, but the KL grade changed from 0 to 3 (Fig. 2).

There were no reports on how the stress on the acetabular rim changed after spinal fusion surgery. Hence, we performed finite element analysis to investigate the relationship between spinal fusion and acetabular rim stress using a healthy model from L4 to the femoral model, which was validated by a previous study[4]. In this study, we could not use these cases as patient models because of the influence of implants on computed tomography. We set three models, nonfusion, L4 to L5 fusion, and L4-S2 alar-iliac (AI) fusion and added 400 N at the L4 vertebra and followed a 10-Nm flexion moment. In the L4 to S2 AI fusion model, followed by L4 to L5 fusion model, stress concentration was recognized at the acetabular rim compared with that in the nonfusion model (Fig. 3), which might be associated with the latency between spinal fusion surgery and occurrence of rim fracture in Case 2.

There are several reports on sacral[5], iliac crest and rami[6-8], and hip[6,9] fractures. Past studies reported that they might be due to the stress translation and hypermobility from the fused spine to the next mobile segments[4,10]. We thought that these mechanism was based on adjacent segment disease: the stress shielding within the long spinal fusion construct and induced stress concentration at the distal

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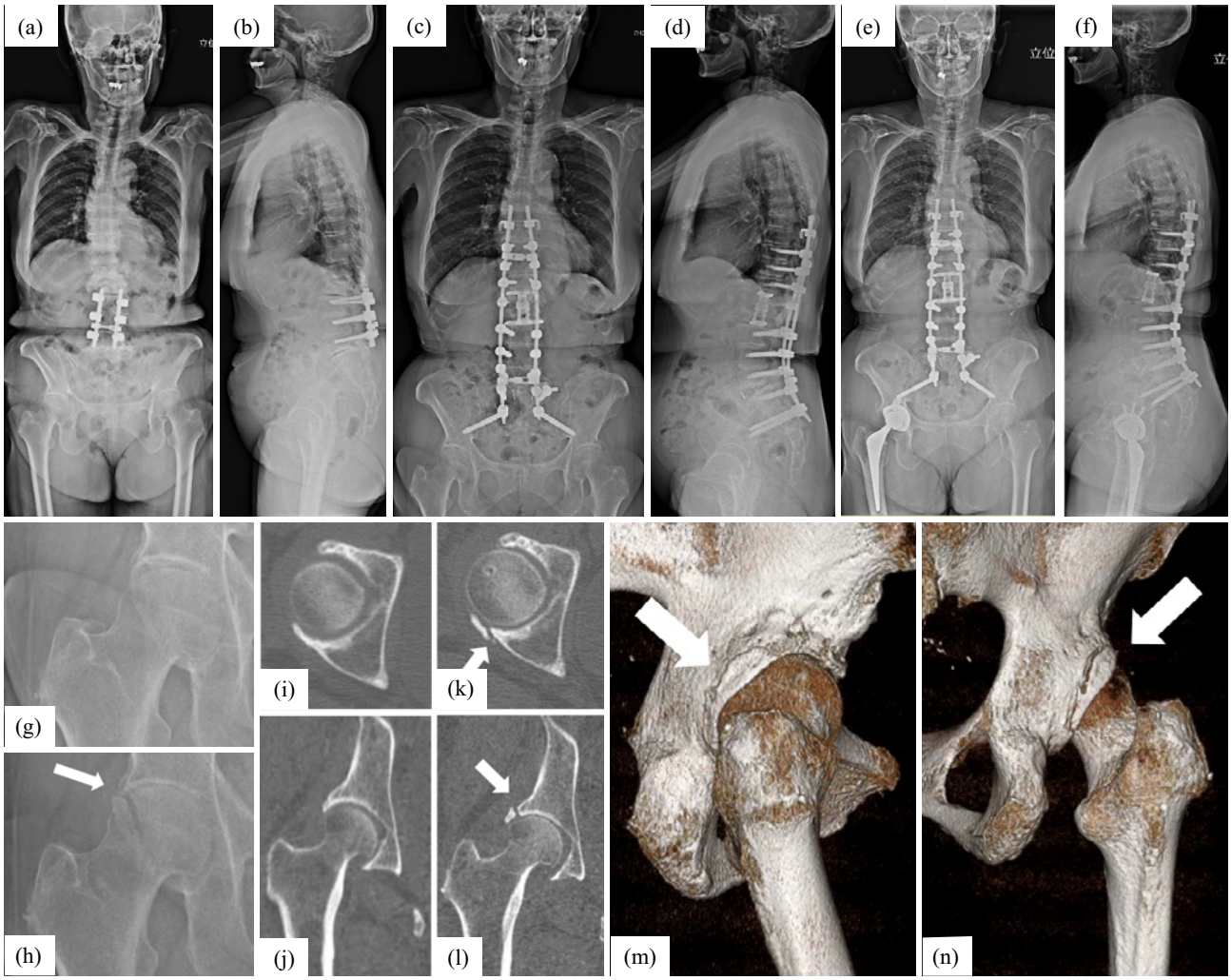

Figure 1. Case 1. Standing radiograph reveals the change in thoracolumbar alignment before (a, b) and after (c, d) adult spinal deformity surgery and total hip arthroplasty (e, f). Standing X-ray imaging shows the hip joint without osteoarthritis 1 month after adult spinal deformity surgery (g), but acetabular rim fracture and progression of hip osteoarthritis occurred 4 months later (h). The acetabular rim was not fractured before spinal long fusion surgery (i, j) but fractured at 4 months after adult spinal deformity surgery on computed tomography (k, l) and three-dimensional images (m, n).

aspect of the fusion mass. The immobilization at the lumbar lesion was compensated by the hypermobility at the hip joint as the next mobile joint.

Here, we report two cases in which HOA progressed after spinal fusion surgery. The prevalence and relationship between HOA and acetabular fractures remain unclear, but it seemed that acetabular fracture was associated with HOA after spinal fusion surgery. The acetabular fracture after spinal deformity surgery thinned the cartilage of the hip joint and might induce HOA. The incidence of these problems seems to increase in an aging society. We should research on hip joint disorders after spinal fusion surgery for pain, range of motion, radiological, and biomechanical considerations.

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Ethical Approval: This study was approved by the Institutional Review Board of Wakayama Medical University (No. 2511).

Informed Consent: Informed consent for participation and publication was obtained from two patients in this study before spinal surgery.

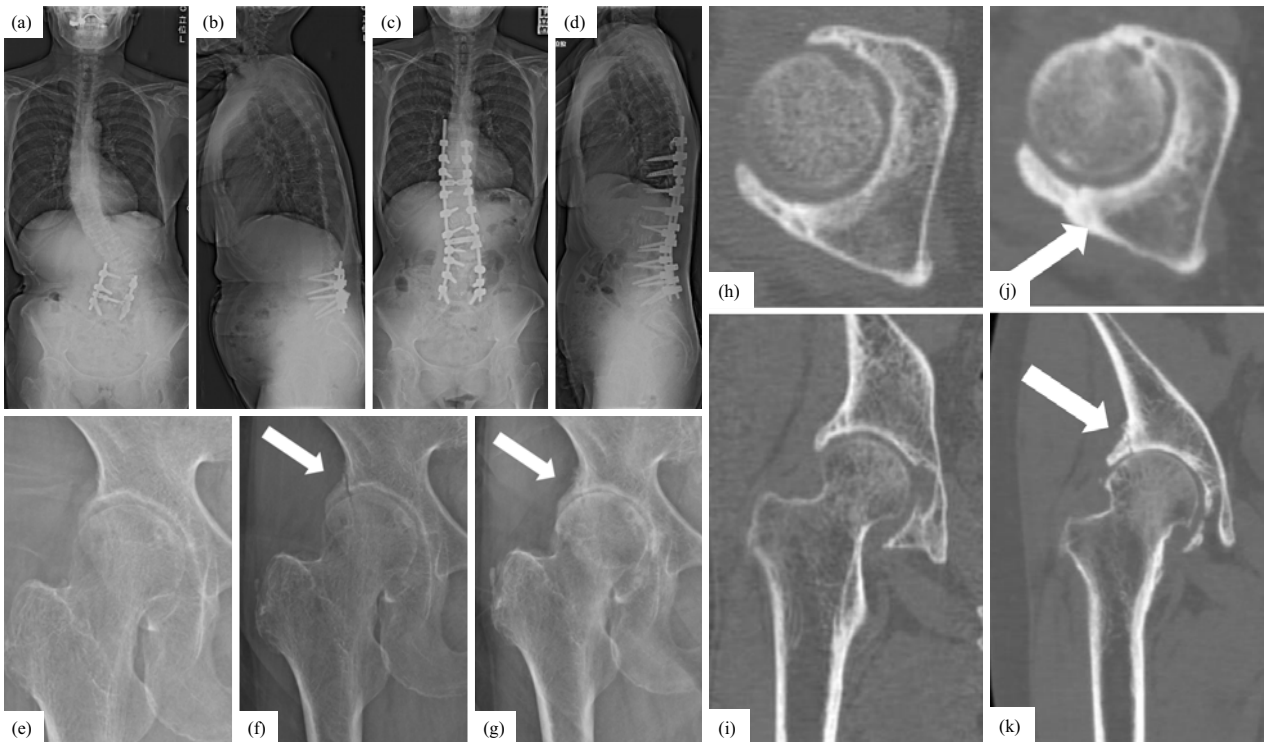

Figure 2. Case 2. Standing X-ray revealed a change in thoracolumbar alignment before (a, b) and after (c, d) adult spinal deformity surgery. She did not feel hip pain 4 years later (e), but her acetabular rim was fractured 5 years after adult spinal deformity surgery (f). The progression of hip osteoarthritis was confirmed radiographically 7 years after adult spinal deformity surgery (g). The acetabular rim was not fractured before adult spinal deformity surgery (h, i) but fractured after surgery. The fracture site tended to bony fusion at 7 years after adult spinal deformity surgery at axial (j) and coronal plane (k) on computed tomography.

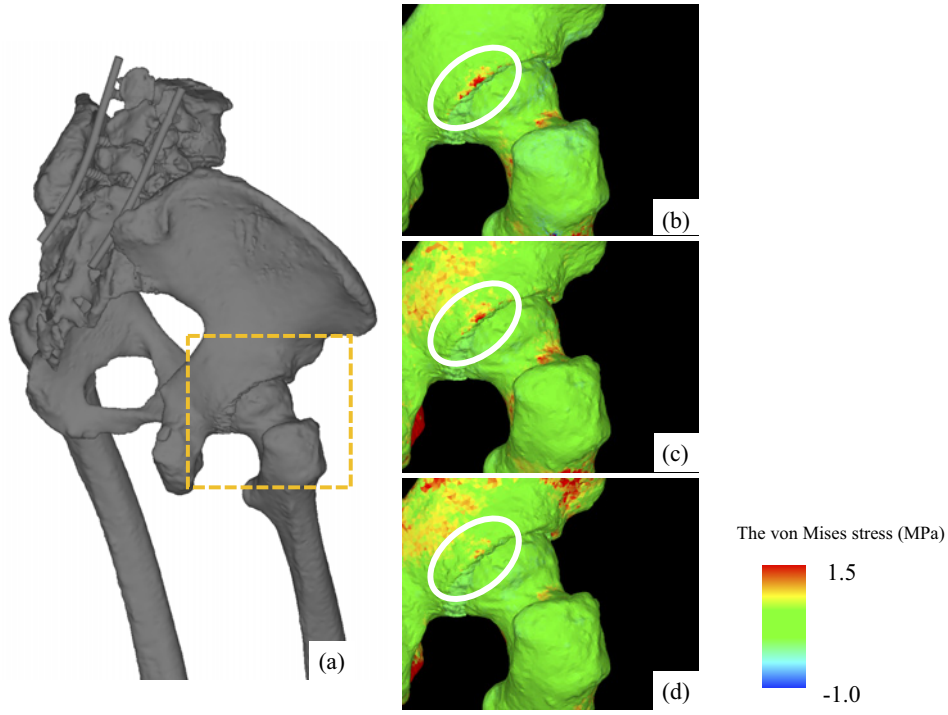

Figure 3. We also underwent finite element analysis using a healthy model from L4 to the femoral model (a). We created the following three models: nonfusion, L4 to L5 fusion, and L4 to S2 alar-iliac fusion. We added 400 N at the L4 vertebra and following a 10-Nm flexion moment. A resultant stress concentration was recognized at the posterior-superior wall of the acetabular rim in the L4-S2 alar-iliac fusion model (b), followed by the L4 to L5 fusion (c) and nonfusion (d) models.

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