Posttraumatic Osteonecrosis of the Femoral Head after Nine Years of Posterior Femoral Head Fracture Dislocation

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Posttraumatic osteonecrosis of the femoral head (ONFH) is the most serious complication after fracture dislocation of the femoral head. The rate of this complication was reported to range from 1.7% to 40%. Although the development of posttraumatic osteonecrosis normally occurs within 2 years of injury, there are some reports of the late development ONFH. The authors encountered a case of posttraumatic ONFH that developed after 9 years of a Pipkin type I fracture dislocation. The patient was treated by modified transtrochanteric rotational osteotomy. We report this rare case with a review of the relevant literatures.

Key words: femoral head, posttraumatic osteonecrosis, fracture dislocation, transtrochanteric rotational osteotomy

CASE REPORT

A 31-year-old woman presented with complaint of left hip pain of two months duration. She had injured a left femoral head fracture and posterior dislocation 9 years ago by an accident while driving a car, and had been treated by open reduction and internal fixation with three screws (Herbert's screws; Zimmer Inc., Warsaw, IN, USA; 4.5×30 mm) via a Smith–Petersen anterior approach. Although the fragment was located below the fovea centralis (Pipkin type I), it was intraarticular and relatively a large size, we decided to reduce it anatomically and fix it (Fig. 1). At that time, closed reduction of the dislocation was performed within six hours after injury. But for the treatment of a concomitant lung injury (bilateral hemothorax), fixation of the head fragment were delayed until seven days after injury. After surgery, she was followed-up for four years.

In nine years after injury, the patient complained of a left hip pain for 2 months which was getting worse recently. A physical examination revealed a limp and a mildly restricted range of motion of the left hip joint (flexion 110°, extension 0°, internal rotation 25°, external rotation 40°, abduction 40°, adduction 30°). The Patrick test was positive.

Radiographs of left hip showed bony union with screws in situ without joint space narrowing or other signs of osteoarthritis. There was no evidence of femoral head collapse (Fig. 2A, 2B). Magnetic resonance imaging of the hip showed small area of signal change in
the anterosuperior portion of the femoral head with positive double line sign (Fig. 2C, 2D). The radiologic diagnosis of ONFH was confirmed by two independent radiology specialists.

Extensive investigations for risk factors of ONFH were performed. She had no history of taking steroids for therapy or any other purpose, denied alcohol abuse; her serum ethanol level was low and her carboxy-deficient-transferrin levels were normal, which ruled out chronic alcoholism. The findings of tests for systemic thrombophilia were normal or negative; these included, blood clotting time, prothrombin time, activated partial thromboplastin time, protein–C and protein–S levels, and resistance to activated–protein–C. No blood cell disorders associated with blood vessel occlusion were found, and her serum bilirubin level was normal. The findings of tests for antibodies associated with connective tissue disease were normal or negative, these included antinuclear antibody, ds–DNA, and antineutrophil cytoplasmic antibody.

Because of the absence of any risk factor other than a history of trauma, we diagnosed posttraumatic ONFH which developed by

Figure 1. (A) Anteroposterior radiograph of both hips show a Pipkin type I fracture dislocation. (B) Radiograph taken after a closed reduction. (C) Computed tomography scan of the left hip after reduction shows a large anterior bony fragment. (D) Intraoperative photograph shows a relatively large fragment involving the anteroinferior portion of the femoral head. (E) Intraoperative photograph after fixation of the fragment. (F) Radiograph taken after open reduction and fixation with three-screws.
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Because the patient was young and active, modified transtrochanteric rotational osteotomy was planned to preserve the femoral head (Fig. 3).

At postoperative three years of follow-up, she had no pain or limp and could sit cross legged and squat (Fig. 4). The patient provided informed consent that case data could be submitted for publication.

**DISCUSSION**

The rate of posttraumatic ONFH after hip posterior dislocation has

**Figure 2.** Anteroposterior (A) and femoral head lateral (B) radiographs taken 9 years after injury show good maintenance of the joint space and bony union without subchondral fracture or other sign of osteonecrosis of the femoral head. T1-weighted magnetic resonance imaging axial (C) and coronal (D) view show a small region of the osteonecrosis anterosuperior of the femoral head (white arrows).

**Figure 3.** Immediate postoperative anteroposterior (A) and lateral (B) radiographs of both hips show the osteomy site fixed with two cannulated screws. The last follow-up anteroposterior (C) and lateral radiographs (D) taken 3 years after the rotational osteotomy show good bony union.
been reported to range from 1.7% to 40%. The most significant clinical factor of ONFH in this setting is the length of time a hip remains dislocated. If a hip is reduced within the confines of the acetabulum within 6 hours of dislocation, the rate of ONFH has been reported to decrease to 0% to 10%. The causes of posttraumatic ONFH are thought to be mainly two. The cervical vessels to the head and the contributions from the ligamentum teres are damaged at the time of injury. Secondarily, an ischemic insult to the femoral head while it is dislocated affects outcome.

In 1962, Brav reported on 189 patients who developed ONFH after reduction for a traumatic hip dislocation. Of these, 98% became symptomatic within 1 year and the remaining 2% became symptomatic between 1 and 5 years. It was also reported that the radiographic changes of ONFH develop within 2 years of injury. Accordingly, it has been recommended that medico-legal reporting should be undertaken 18–24 months from the time of injury.

However, Cash and Nolan reported a case of ONFH of the femoral head that occurred at 8 years after posterior hip dislocation. The hip was reduced within 6 hours of injury and there was no evidence of necrosis during 3 years of follow-up. However, 5 years later, the patient presented with hip pain and was treated by total hip arthroplasty. Not surprisingly, the authors recommended that additional consideration be given to length of follow-up and medico-legal reporting after hip dislocation.

Ksielinski et al. reported a case of ONFH of femoral head 15 years after a transcervical femoral neck fracture in a woman with a 20-year history of daily inhaled glucocorticoid therapy for chronic bronchitis, and recommended that caution be exercised in patients on low-dose inhaled glucocorticoids, particularly those with internal fixation devices implanted to treat femoral neck fracture. It was concluded that the low-dose inhaled glucocorticoid therapy and femoral neck fracture combination may cause osteonecrosis in the long-term. In our case, the absence of steroid use and alcohol abuse and laboratory findings indicated no blood cell disorder, connective tissue disease, or any other risk factor of ONFH, other than a trauma history. The initial posterior dislocation in this patient was reduced within 6 hours and femoral head fracture fixation was done using the Smith–Petersen approach. Stannard et al. undertook an odds ratio analysis on different surgical approaches to femoral head fracture dislocation, and found that the Kocher–Langenbeck posterior approach was associated with a 3.2 fold higher rate of ONFH development than the Smith–Petersen approach. Some surgeons may question the prudence of internally fixing a head fragment which is infra foveal. We believe that the fixation was justified and essential because the fragment was large and the hip was unstable without its fixation.

Several methods can be used to treat early stage, precollapse lesions of the femoral head, e.g., core decompression, nonvascularized bone graft, vascularized bone graft, or osteotomy. Fortunately, unlike ONFH secondary to systemic illness or medication, or idiopathic ONFH, posttraumatic ONFH may be highly localized, and thus, it is more amenable to osteotomy than global ONFH. Accordingly, in our patient, after considering her age, the location and the size of the ONFH, osteotomy was considered as the best treatment option.

Rotational osteotomy was first performed by Sugio in Japan in 1972 and first reported in the English literature in 1978. It involves rotating the femoral head around the longitudinal axis of the neck to
remove the area of necrosis from weight-bearing and transferring shear forces to the healthier posterior cartilage of the femoral head. In the described patient, the rotation involved a movement of almost 90 degrees anteriorly, and thus, the anterosuperior osteonecrosis was relocated anteroinferiorly. The osteotomy site united at 2 months postoperatively.

Summarizing, ONFH can develop 9 years after femoral head fracture and posterior dislocation, and thus, careful serial follow-up is required. Furthermore, patients should be counseled about this complication and advised to report any hip pain, so that diagnosis and treatment can be initiated at the earliest opportunity—before collapse or arthrosis develops. In the present case, the patient underwent modified transtrochanteric osteotomy and good results were achieved. Accordingly, we recommend that the described modified transtrochanteric osteotomy be considered as a good treatment option in such cases.

REFERENCES

후방 대퇴골두 골절 탈구 9년 후에 발생한 외상후성 대퇴골두 무혈성 괴사

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외상후성 대퇴골두 무혈성 괴사는 대퇴골두 골절 탈구 후에 발생하는 가장 심각한 합병증으로 약 1.7%~40%까지 발생하는 것으로 알려져 있다. 대부분의 외상후성 무혈성 괴사는 수상 후 2년 내에 발생하나 몇몇의 증례에서 그보다 더 늦게 나타난 무혈성 괴사를 보고하고 있다. 저자들은 Pipkin I형의 대퇴골두 골절 탈구 9년 후 발생한 대퇴골두 무혈성 괴사 1예에서 변형된 전자간 회전 절골술을 이용하여 성공적으로 치료하여 참고문헌 고찰과 함께 보고하고자 한다.

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