Fractures of the acetabulum pose a difficult problem for the patient and the surgeon because of possible complications. Thus any surgeon involved in surgery for fractures of the acetabulum should be aware of the possibility of this potential complication. Here is a 61-year male, who sustained a complex fracture of the acetabulum without hip dislocation, subsequently was treated surgically with internal fixation using an anterior approach, 10 months after surgery patient developed osteonecrosis of the femoral head.

**Key Words:** Acetabular fracture, Osteonecrosis of the femoral head, Acetabulum fracture complication
arterial pressure, leading to decreased blood flow to the femoral head; nature and complexity of the injury and predisposition of the patient.

CASE REPORT

A 61-year male presented to the emergency department with a history of road traffic accident. He arrived hemodynamically stable with a blood pressure of 126/76 mmHg and a heart rate of 78 beats per minute. On plain radiograph (Fig. 1A) anteroposterior and two Judet 45° oblique view and computed tomography (CT) scan of pelvis (Fig. 1B), the findings revealed both column fracture of acetabulum without hip dislocation, but no presence of femoral head fracture or ONFH.

After patient’s vitals were stabilized, we performed the surgical procedure. Buttress plating through ilioinguinal approach was performed using a reconstruction plate, which was supplemented by a compact hand plate. The surgical procedure lasted for 3 hours 44 minutes. The total blood loss intra-operatively was 3,000 mL. The preoperative hemoglobin (Hb) was 9.7 gm%. The patient was transfused 8 units of whole blood, 3 units of fresh frozen plasma and 8 units of packed red blood cells. Intra-operative Hb was 9.7 gm%; the average mean arterial pressure was 91.82 mmHg during the operative procedure. Post-operative blood loss via suction drain was 380 mL. Post-operatively the patient was transfused 2 units of whole blood and 1 unit of fresh frozen plasma. The Hb postoperatively was 10.3 gm%, the patient was shifted to intensive care unit for a day, later was transferred to the ward.

The post-operative X-ray (Fig. 2A) and CT scan revealed an acceptable reduction of the fracture fragments and a concentric hip. Sitting up was performed on the first postoperative day; the patient subsequently began formal physical therapy and active range of motion exercises. Partial toe touch weight bearing (20 to 30 lb; 9 to 13.6 kg) with a walker was maintained for 6-8 weeks. Progression to full weight bearing was started on the basis of the follow-up radiographs. Physical therapy was continued until muscle strength and range of motion were regained. The patient was then followed up on a regular basis, every monthly. His progress was monitored. On subsequent follow-up, the patient had complains of pain in the hip joint. The patient’s 4-month postoperative X-ray revealed a radiolucent lesion in the superolateral part of femoral head, crescent sign, and sclerosis. The 10-month follow up X-ray (Fig. 2B) showed collapse and sclerosis, findings consistent with ONFH. A CT scan (Fig. 3A) confirmed this. ON was diagnosed only when the radiographic findings provided a clear differentiation from wear of the femoral head. The joint pain increased due to the ONFH, we performed a total hip replacement (Fig. 3B) 12 months after the index surgery. Histopathology report of the femoral head confirmed ON.

![Fig. 1. Preoperative images. (A) X-ray image. (B) Computed tomography scan.](image-url)
Late complications of acetabulum fractures include heterotopic ossification and ONFH, which are present in less than 10% of the population\(^3\). The exact cause of these complications are not entirely understood. The incidence of ON described in literature varies from 3\(^\%\) to 53\(^\%\)\(^3\)\(^-\)\(^5\). The incidence of ONFH is known to be high in transverse and posterior wall fractures associated with posterior dislocation\(^6\). ON also occurs in conjunction with approximately 3\(^\%\) of anterior hip dislocations and in more than 13\(^\%\) of posterior hip dislocations. In a recent meta analysis of 3,670 surgically treated displaced acetabular fractures the incidence of ONFH showed an overall incidence of 5.6\(^\%\), suggesting that it is grossly overestimated and that most of the observed changes in the head of the femur are probably due to osteoarthritis\(^5\).

**DISCUSSION**

![Postoperative images](image1)

*Fig. 2. Postoperative images. (A) X-ray image of immediately after surgery. (B) X-ray image after 10 months.*

![Computed tomography scan](image2)

*Fig. 3. (A) Computed tomography scan showing osteonecrosis of the femoral head. (B) Postoperative x-ray image after total hip replacement.*
ONFH is caused by inadequate blood supply to the affected segment of the subchondral bone. When posterior surgical approaches have been used, ON rates as high as 42% within the first year after surgery have been reported\(^7\). The anterior surgical approach to the acetabulum theoretically leads to the least devascularization\(^8\). Many systemic conditions are associated with ON, but 25% of all cases are described as idiopathic and can contribute as a cause\(^9\). Trauma is one of the most common causes of ON, interruption of the blood supply to the affected segment of the bone being the cause of ischemia.

In this case the exact cause of ONFH eludes us, especially in the absence of any patient related predisposing risk factors, except presence of fracture without hip dislocation and subsequent intervention by an ilio-inguinal approach. A probable theory of etiology could be the intra-operative hypovolaemia, low mean arterial pressure, causing compromised flow to the femoral head being so as to act as the final blow. Alteration of the blood supply to vital organs during hypovolaemia is well established. With mean arterial pressure usually in the range of 50 to 60 mmHg, the flow to the femoral head is potentially compromised\(^10\) so as to act in an accumulative stress theory, as suggested by Kenzora and Glimcher\(^9\). It is questionable as to whether this alone would be enough to explain the development of ON. Patient also could have had other unknown risk factors for non-traumatic ON.

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