

# Rapidly Progressive Osteonecrosis of the Humeral Head after Arthroscopic Bankart and Rotator Cuff Repair in a 66-Year Old Woman: A Case Report

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Humeral head chondrolysis has been widely reported as a devastating complication after arthroscopic shoulder surgery; however little is known about post-arthroscopic humeral head osteonecrosis. We experienced a 66-year-old female patient with rapidly progressive osteonecrosis of the humeral head only seven months after arthroscopic Bankart and rotator cuff repair. The patient had no systemic risk factors for osteonecrosis. A satisfactory result was achieved with reverse total shoulder arthroplasty for severe humeral head destruction and an irreparable massive rotator cuff tear. Shoulder surgeons should be aware of such severe complication, perform routine radiographs, and pay close attention to the presence of constant pain or loss of motion after arthroscopic shoulder surgery.

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**Key Words:** Shoulder arthroscopy; Osteonecrosis; Reverse total shoulder arthroplasty

Arthroscopic shoulder surgery has become a popular procedure in the last two decades. It is generally regarded as a safe procedure, with few risks. Recently, however, some disastrous complications, including glenohumeral chondrolysis and osteonecrosis of the humeral head after an arthroscopic procedure, have been reported.<sup>1-4)</sup> Glenohumeral chondrolysis after arthroscopic shoulder surgery has been widely reported, and many causes were suggested, including local anesthesia through a pain pump, radiofrequency device, and so on.<sup>1)</sup> However little is known about post-arthroscopic humeral head necrosis. To the best of the author's knowledge, there are only three reports in English literature on humeral head osteonecrosis after such surgery.<sup>2-4)</sup> Despite its rarity, humeral head osteonecrosis after arthroscopic shoulder surgery requires early diagnosis and proper treatment to prevent development into a serious glenohumeral arthrosis. We report on a case of osteonecrosis of the humeral head after arthroscopic Bankart and rotator cuff repair which showed rapid progression with a review of the literature.

## Case Report

A 66-year-old right-handed female patient was referred to our clinic with complaints of pain and limited motion in her right shoulder after arthroscopic surgery at an outside facility. Meticulous history taking revealed that the patient had fallen from a height of about one meter about seven months earlier, and she was diagnosed with an anterior shoulder dislocation with bony Bankart lesion (Fig. 1A, B), and rotator cuff tear, however the marrow signal of her proximal humerus was nonspecific on pre-operative magnetic resonance imaging (MRI) (Fig. 1C, D). Subsequently she underwent arthroscopic Bankart and double row rotator cuff repair with bioabsorbable suture anchors (3 anchors for bony Bankart lesion, 4 anchors for rotator cuff tear). Post-operative MRI taken one week later showed that both lesions were well repaired (Fig. 2). According to the medical records from the previous hospital, she had started performing range-of-motion exercises from 6 weeks postoperatively and presented 90 degrees of active forward flexion and 40 degrees of active

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Fig. 1. Initial X-ray shows anterior shoulder dislocation (A) and bony Bankart lesion after reduction (B). Axial and coronal T2-weighted magnetic resonance images show bony Bankart lesion (C) and rotator cuff tear (D). Arrows indicate bony Bankart lesion.



Fig. 2. Magnetic resonance imaging one week after the initial operation. T2-weighted axial (A) and coronal images (B) show that bony Bankart lesion and rotator cuff tear were repaired successfully.

external rotation at that time. She had an interval of a couple of months with mild improvement of her shoulder motion after the exercises. At 4 months after the operation she underwent a sonographic examination and the repaired cuff was observed with mild effusion. However, her shoulder motion and pain were worsening and on the seven-month follow-up, she presented with increasingly severe shoulder pain and gross restriction of range of motion. She was transferred with clinical suspicion of osteonecrosis of the humeral head based on plain radiographs. She had no particularly remarkable medical history including diabetes or systemic risk factors of osteonecrosis.

In our hospital, she complained of pain and markedly limited active motion of her right shoulder. The extent of its active forward flexion was 80 degrees; external rotation, 20 degrees; and on internal rotation, she could only reach the buttock level. The shoulder radiographs obtained upon the patient's presentation showed destruction of the humeral head and abundant bone debris and upper migration of the proximal humerus (Fig. 3A). The 3-dimensional computed tomography also showed collapse of the humeral head with multiple intra-articular bone debris (Fig. 3B). The MRI findings showed large amounts of effusion, massive rotator cuff tear, and humeral head destruction (Fig. 3C, D).

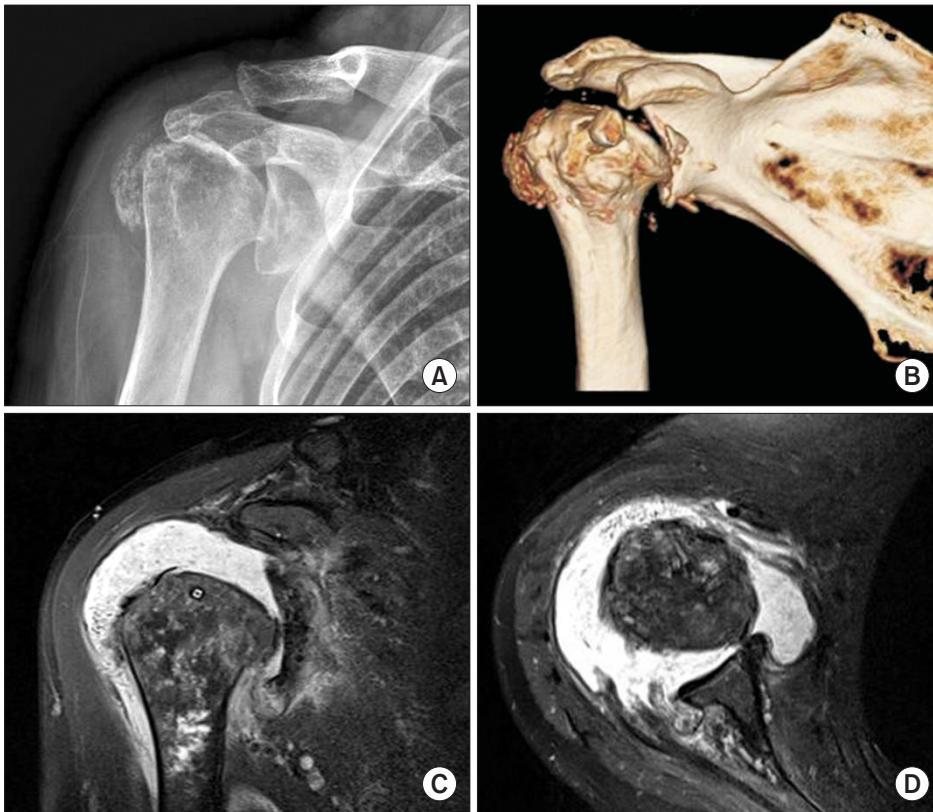


Fig. 3. Radiologic images seven months after the initial operation. X-ray (A) and 3-dimensional computed tomography (B) show marked destruction of the humeral head, bone debris, and upper migration of the humeral head. Axial (C) and coronal T2-weighted magnetic resonance images (D) show marked effusion, humeral head destruction and a massive rotator cuff tear.



Fig. 4. (A) Intraoperative photograph showing collapse of the humeral head and previous suture materials in greater tuberosity. (B, C) Radiographs at 1 year postoperative showing a successful reverse total shoulder arthroplasty.

A variety workup was performed to determine the cause of the rapid progressive humeral head destruction. The white blood cell count, C-reactive protein, and erythrocyte sedimentation rates were within the normal range. Approximately 25 ml of glenohumeral synovial fluid was aspirated, however no bacterial growth was detected after three weeks and no uric acid or calcium pyrophosphate dehydrate crystal was observed under a polarizing microscope. Alizarin red staining could not be performed to rule out Milwaukee disease; however, the biopsy

specimens obtained during the operation showed osteonecrosis.

Due to pain, poor function, irreparable rotator cuff, and severe glenohumeral joint arthrosis, the patient underwent reverse total shoulder arthroplasty seven months after her previous operation. The intraoperative findings showed destruction of the humeral head with chondral delamination compatible with humeral head osteonecrosis (Fig. 4A). One year after the second operation, she registered 170 degrees of forward flexion, 35 degrees of external rotation, and three lumbar vertebral levels

in the internal rotation and the prosthesis was well maintained on final radiographs (Fig. 4B). The patient is not impaired in her daily activities and is satisfied with the outcome.

## Discussion

The incidence of osteonecrosis of the humeral head after arthroscopic shoulder surgery has not yet been investigated and reported cases are rare. In addition, its etiology has not yet been clearly established. In 2010, Beauthier et al.<sup>2</sup> reported the first case of osteonecrosis after an arthroscopic procedure, and they believed that the blood supply was disrupted after placement of multiple metallic suture anchors for rotator cuff repair. Dilisio et al.<sup>3</sup> reported three cases of osteonecrosis after arthroscopic surgery, two of which had no hardware placed, so that they could not conclude that osteonecrosis was due to the aberrant anchor placement. They assumed that a kind of peri-operative insult led to the disruption of the humeral head blood supply. Goto et al.,<sup>4</sup> who reported another case of osteonecrosis, also reported that the metal anchor injury to the anterolateral branch of the anterior humeral circumflex artery, which runs parallel to the lateral aspect of the tendon of the long head of the biceps, penetrated the humeral head.

Previous reports suggest that anterolateral branch injury of the anterior humeral circumflex artery due to the insertion of the anchor on the wrong side is the main cause of proximal osteonecrosis. The anterolateral branch of the anterior humeral circumflex artery is located just lateral to the long head of the biceps, coming close to the origin, thus it can be easily damaged during shoulder arthroscopy according to the anchor insertion site. However, osteonecrosis after shoulder arthroscopy is extremely rare, and, considering that 80% of cases of damage to the anterior humeral circumflex artery are associated with proximal humerus fractures,<sup>5</sup> whereas osteonecrosis is rare, anterior humeral circumflex artery damage cannot be concluded as the solitary cause of osteonecrosis. According to the MRI study of Hettrich et al.,<sup>6</sup> only 34% of the blood supply to the proximal humerus is from the anterior humeral circumflex artery, whereas 64% is from the posterior humeral circumflex artery. Therefore, anterior humeral circumflex artery damage can be compensated for by the intact posterior humeral circumflex artery, and this compensation can explain the low incidence of osteonecrosis.

The recurrence rate for shoulder dislocation is lower for elderly patients than younger patients, and an already thin or already frayed rotator cuff is more often disrupted, but rarely affects the glenoid labrum. Many labral lesions observed in elderly patients were independent of the dislocation. However in this case, the patient had a bony Bankart lesion and repair with rotator cuff concomitant might be reasonable. In consideration of the causes in this case, the patient initially had shoulder dislocation, which could have caused damage to both the anterior and

posterior humeral circumflex arteries. Second, the anchors could have caused damage to the anterolateral branch of the anterior humeral circumflex artery, as in the previous reports. This vessel runs parallel to the lateral aspect of the tendon of the long head of the biceps and enters the humeral head. On postoperative MRI 2 screws were positioned just lateral to the biceps groove, which could cause injury to the vessel. All of the radiographic images in the previous cases presented with medial quadrant destruction of the humeral head. In Hettrich's MRI study, unlike the other quadrants, the medial quadrant received greater blood supply from the anterior humeral circumflex artery. That is, the medial quadrant may be most vulnerable to anterior humeral circumflex artery damage. Also, after the rotator cuff repair, the increase in pressure between the glenohumeral joints might have affected the aggravation of results. Third, although the patient did not have systemic risk factors for osteonecrosis such as steroid induction, alcoholism, Cassion's disease, sickle cell anemia, or connective tissue autoimmune disease, many cases of osteonecrosis are still classified as idiopathic or multifactorial, therefore this case may have an unknown cause that is vulnerable to osteonecrosis, or many factors could have acted as a complex, however further evaluation is needed.

Milwaukee syndrome also presents with rapid collapse of the proximal humerus.<sup>7</sup> Its pathology is believed to be the intra-articular calcium hydroxyapatite deposition, which induces the release of lysosomal enzymes, which attack the peri-articular tissues, including the rotator cuff.<sup>8</sup> Unlike monosodium urate and calcium pyrophosphate dehydrate crystals, calcium hydroxyapatite crystals cannot be observed under a plain and polarized microscope. Use of alizarin red stain enables simple and rapid identification of clumps of calcium hydroxyapatite crystals,<sup>9</sup> but we cannot practice this stain. However the biopsy result of the proximal humerus bone and the synovial membrane of the shoulder joint during the reverse shoulder arthroplasty presented only osteonecrosis and foreign body reaction due to a bio-absorbable screw. In addition, several studies suggested that irrigation of the shoulder joint removes the crystal and has a potential benefit in Milwaukee syndrome.<sup>10</sup> Thus, we can exclude Milwaukee syndrome because our patient's previous surgery was an arthroscopic procedure accompanied by massive saline irrigation.

Radiographs of the shoulder joint after arthroscopic surgery are generally not considered essential examinations, and thus are easily ignored or forgotten. In our case, the radiographs were obtained only once, on the first day after surgery. Seven months after surgery, the patient underwent sonographic examinations three times. However, osteodestruction was not precisely detected on the sonographic study.

Shoulder surgeons should be aware of this potential complication, and carefully decide on the position of the anchor during the operation. After the operation the shoulder surgeon should

check radiographs routinely, and pay careful attention to patients who present with progressive pain and loss of the range of motion of the shoulder after an arthroscopic operation.

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