# Osteonecrosis of the Humeral Head: A Literature Review and Two Case Studies

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*Abstract:* Osteonecrosis of the humeral head may be idiopathic, but it is also associated with a number of known medical conditions. In these patient groups, it is a differential diagnostic possibility that the physical therapist needs to consider. This article discusses histopathology, classification, etiology, history and examination findings, and treatment of humeral head osteonecrosis. It also presents two case studies of patients with undiagnosed osteonecrosis who were referred to physical therapy to illustrate the difficulties and possibilities for correct identification of such patients.

*Key Words:* Osteonecrosis, Humeral head, Bone

Osteonecrosis is defined as the in situ death of cells within the bone due to a lack of circulation and not as a direct result of disease[1,2]. The cells involved may include osteocytes, both in cortical and cancellous bone, and hematopoietic and fat cells in the marrow cavity[1]. Osteonecrosis is also often referred to as avascular or aseptic necrosis[1]. However, osteonecrotic bone is not avascular: the blood vessels are still present, but circulation within them is compromised[1]. The term osteonecrosis is preferred to avascular or aseptic necrosis, as it gives the most appropriate description of the histopathologic process occurring but without suggesting a specific etiology[1].

Central to the etiology of osteonecrosis is the compromise of circulation in the vessels supplying the bone. There are four mechanisms for such compromise[1]. Mechanical vascular disruption can result from fracture, dislocation, and fatigue fractures. Arterial occlusion can be caused by thrombosis, embolisms, and abnormally shaped cells. Injury to or pressure on the arterial wall may impair circulation in three different ways; this can occur from within the wall, as with vasculitis, or from within the vessel, due to release of vasoactive substances causing angiospasm. Extravasated blood, fat, or cellular elements may increase extravascular, intraosseous pressure and thus decrease circulation. Finally, occlusion of venous outflow may raise venous pressures over those in the arterial portion of the circulation resulting in compromised circulation to the cells.

Some 90 different bone necroses have been described in literature, all with similar radiologic and histologic findings[3]. Osteonecrosis may affect both epiphyses and apophyses[3]. Examples are Perthes' disease affecting the epiphysis of the femoral head, Osgood-Schlatter's disease affecting the tibial tuberosity, Koehler's disease I affecting the os naviculare pedis, Koehler's disease II affecting the second metatarsal head, Kienboeck's disease or lunatomalacia, and Scheuermann's disease affecting the

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vertebral epiphyses[3]. Adler[3] also classifies osteochondritis dissecans as an epiphyseal osteonecrosis. Subchondral areas are especially prone to osteonecrosis. Here arterioles assume a sinusoid course and are forced to make a 180 degree turn in order to return to the intraosseous circulation[3,4], making them more prone to occlusion. A relatively small number of vascular foramina and a limited collateral circulation will further increase the risk of local ischaemia[2].The femoral head is most frequently affected; next most commonly affected are the proximal humerus, and medial and lateral femoral condyles[5]. The proximal tibia, talus, scaphoid, lunate, and capitellum humeri can also develop osteonecrosis[4,5].

In my clinical practice, I was recently confronted with two patients with therapy-resistant shoulder complaints. I referred both patients back to their respective physicians with requests for further diagnostic testing. Both patients were subsequently diagnosed with osteonecrosis of the humeral head and treated with a hemi-arthroplasty. They both regained nearly full function and reported minimal pain after a course of post-surgical physical therapy. In this article, I review the histopathology, classification, etiology, history and examination findings, and treatment options for osteonecrosis of the humeral head. I will also discuss the pre-operative presentation and clinical course for the two patients mentioned earlier. The intent of this article is to increase the awareness among physical therapists of osteonecrosis of the humeral head as a differential diagnosis for patients with complaints of shoulder pain and decreased range of motion.

## *Histopathology*

Regardless of etiology, the histopathologic events in osteonecrosis are uniform[1]. Necrosis results from tissue ischaemia: a minimum of two hours of complete anoxia is needed for cell death[2]. Initially and for the first few days to a week after vascular compromise has started, there are no histologic changes. During the second week, evidence of cell death is found in the marrow cavity: hematopoietic cells, capillary endothelial cells, and lipocytes become necrotic. Shrinking of osteocytes causes the empty lacunae typical of necrotic bone, while the intramedullary tissues are acidified by release of lysosomes from the necrotic lipocytes. These lipocytes also release free fatty acids, which form an insoluble soap with the calcium released into the marrow cavity. Normal fatty marrow contains little water, but early osteonecrosis causes an increased water content[1]. MRI signal intensity varies based on the fat and water content of tissues[6], making it sensitive to early changes in osteonecrosis.

Osteonecrosis may not cause symptoms, especially when it affects only the medullary bone without involving the subchondral plate. The necrotic tissue and especially the saponified fats calcify and may show up as an incidental radiographic finding. Nor is repair always initiated automatically: some small size bone infarcts remain unchanged for life. Repair is only started if the surrounding viable tissues receive some unknown signal indicating that such a process is needed[1]. If the reparative response is in fact initiated, repair processes differ between cancellous and cortical bone[1,5]. In cancellous bone, reactive hyperemia and vascular fibrous repair starts in the adjacent bony tissues. Within a few weeks, necrotic bone is revascularized from this adjacent fibrous tissue. Primitive mesenchymal cells, which accompany these newly formed vessels, differentiate into osteoblasts and osteoclasts. This differentiation may be the result of a combination of bone morphogenic factors released by the necrotic cells, changes in pH, oxygen tension, and mechanical stress[1]. New osteoid is now produced on the scaffolding formed by the necrotic trabeculae. This thickens these trabeculae: appositional new bone on the surface of the necrotic trabeculae may show as increased density on plain radiographs 6-12 months after the onset of necrosis[1,5]. However, this radiographic picture may not reflect the strength of similarly radiodense bone undergoing normal remodeling[1]. Cortical bone is repaired more slowly through the classic sequence of osteoclastic resorption followed by osteoblastic formation: this process is also known as creeping substitution[5]. Osteoclasts are again formed by the differentiation of the mesenchymal cells. The Haversian, but not the interlamellar bone, is first absorbed by the osteoclasts; only after resorption of the majority of the Haversian system do the osteoblasts begin the process of bone formation[1]. Thus, cortical bone becomes osteoporotic first and regains normal density only after it has been repaired; return to normal strength takes at least two years. Resorption of bone temporarily weakens the bone. Pathologic fractures occur between 18-24 months after the onset of osteonecrosis; it takes that long before the slow resorption causes the fracture threshold to be crossed[1].

With osteonecrosis, the cellular component of bone is affected, but not the inorganic and organic matrix of the structural components of the bone[1]. The absence of the cells, however, does not allow the bone to respond adequately to the mechanical effects of continued normal daily activities. Fracture and collapse of the articular surface appear to result from multiple irreparable, fatigue fractures due to repetitive loading during normal patient function. These fatigue fractures seem to occur in areas where the remaining necrotic subchondral trabeculae oriented perpendicular to the joint surface lack the means for repairing microfractures. These fractures are also found at the periphery of the subchondral bone, where osteoclastic resorption has weakened the bone, and at the junction of the repaired denser and the adjacent weaker avascular bone[1].

The crescent sign is an example of this third type of fracture. It is a subchondral fracture found in both the femoral and humeral head that is almost pathognomonic

for osteonecrosis[4]. It usually precedes collapse of the articular surface[4]. This fracture is the result of the difference in repair time needed for cancellous versus cortical bone[5]. Osteoblastic trabecular apposition of new cancellous bone progresses at an equal rate in a straight or crescentic line across the humeral head from distal to proximal[4]. At the same time, the subchondral bone, especially at the anterior, superior, and lateral aspects of the humeral head, undergoes osteoclastic resorption. The junction of weaker subchondral and stronger cancellous bone acts as a stress riser and is subject to a fracture propagating along the subchondral region parallel to the adjacent articular cartilage[4]. The crescentic radiolucent area indicating a subchondral fracture is known as the crescent sign[7].

Another early radiographic finding is an osteolytic subchondral defect of the humeral head, often coinciding with the point of articulation with the glenoid cavity at 90 degrees of abduction, the position in which maximum force is transmitted across the shoulder joint[4]. Subsequent collapse of the articular cartilage is usually found in the superior medial quadrant of the humeral head[7]. Deformity of the articular surface of the humeral head may cause degenerative changes of the glenoid surface; arthritic changes may lead to secondary capsular glenohumeral restrictions[6].

## *Classification*

The modified Ficat-Arlet classification system for staging of humeral head osteonecrosis (Figure 1) is based on radiographic appearance rather than clinical and functional symptoms[6]:

- Stage I is a preradiologic stage characterized by the absence of findings on plain radiographic images. Scintigraphy may show an absence of uptake or, conversely, an increased uptake of radioisotopes, indicating that a reparative response has been initiated[5]. MRI is sensitive to the early changes in intramedullary water content described earlier[1,6]. This stage is labeled Stage I rather than Stage 0, because histopathologic changes are already present[6].
- Stage II is characterized by radiologic evidence of repair. Radiographs may show diffuse osteoporosis, diffuse sclerosis, a mixed osteoporotic and sclerotic appearance, or a localized subchondral osteolytic lesion[5]. Sclerotic changes are often located in the superior central portion of the humeral head[6]. Shape and sphericity of the humeral head are maintained[5,6]. Computed tomography can be used to further define lesions in Stage II and above[6].
- Stage III is differentiated from Stage II by subchondral bone collapse, resulting in a loss of humeral head sphericity. This subchondral collapse is known as the crescent sign discussed earlier. The articular surface may be distorted[6]; sometimes there is a mild flattening of the articular surface[8], but it has not collapsed[6].
- Stage IV is characterized by an extensive collapse of the subchondral bone causing severe deformity of the humeral head. An osseocartilaginous fragment may become intra-articular causing symptoms consistent with those of a loose body inside the joint[5,6].
- Stage V is differentiated from Stage IV by the osteoarthritic changes observed in the glenoid fossa[6]. Despite its lack of clinical and functional relevance, this classification system is used to establish the most appropriate treatment for patients with humeral head osteonecrosis.

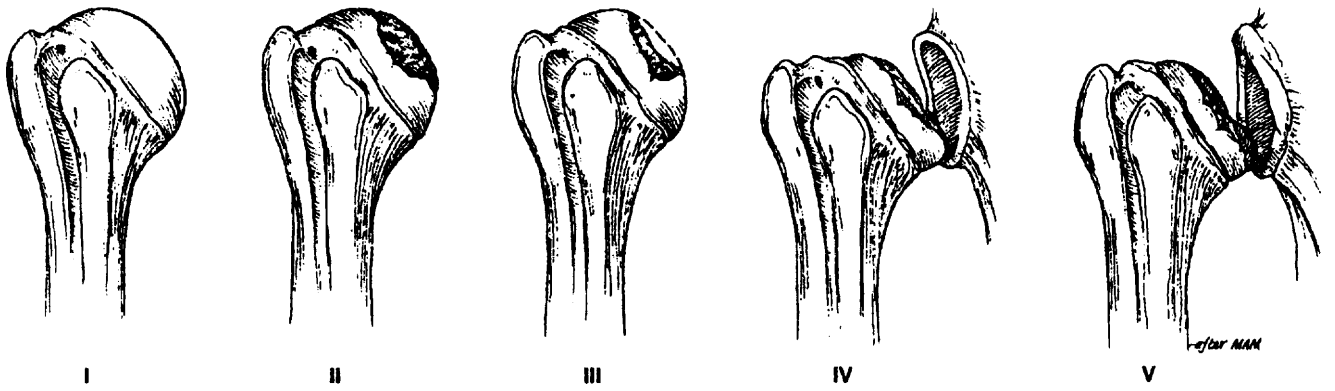


*Fig. 1: Stages of osteonecrosis of the humeral head. Stage I changes are not visible on plain radiographs, nor are they discernible on gross examination. Stage II is marked by sclerotic changes and evidence of bone remodeling, but the shape and sphericity of the humeral head are maintained. Stage III is differentiated from stage II by the presence of subchondral bone collapse or fracture, resulting in loss of humeral-head sphericity. In stage IV, the humeral head has an area of collapsed articular surface; the fragment may become displaced intra-articularly. In stage V, there are osteoarthritic changes in the glenoid fossa. ©1997 American Academy of Orthopaedic Surgeons. Reprinted from the Journal of the American Academy of Orthopaedic Surgeons, Volume 5 (6), pp. 339-346 with permission.*

## *Etiology*

Above are described the four mechanisms postulated for the circulatory compromise central to the etiology of osteonecrosis. A number of medical diseases are known to cause osteonecrosis through one or more of these mechanisms. Sometimes the only presenting symptom for this diagnosis is the patient mentioning one of the causative medical diseases when asked about his or her medical history[6]. The following is a discussion of the medical diseases and conditions associated with osteonecrosis of the humeral head.

## *Trauma*

The anterolateral ascending branch of the anterior humeral circumflex artery runs lateral to the tendon of the long head of the biceps. It enters the head of the humerus at the junction of the proximal end of the intertubercular sulcus and the greater tuberosity. This intraosseous branch, called the arcuate artery, perfuses almost the entire head of the humerus. The posterior humeral circumflex artery vascularizes only the posterior portion of the greater tuberosity and a small posteroinferior part of the humeral head[9]. A four-part fracture of the proximal humerus is associated with a high prevalence of osteonecrosis because it separates the humeral head from both its principal (anterior circumflex) and its secondary (posterior circumflex) blood supply[6]. Fracture fragments may lacerate the anterolateral ascending branch[8]. Surgical fixation may restore shoulder function but can also add further vascular insult[6]. Intramedullary pinning and abrasion of the periosteum may damage vessels[3]. Gerber et al[9] note that techniques used for internal fixation of complex fractures of the proximal humerus are likely to injure the anterolateral branch of the anterior humeral circumflex artery, compromise its anastomoses, or even damage both. The incidence of osteonecrosis after four-part fractures of the humeral head has been reported as a complication in one-eighth to one-third of all patients[6].

Glenohumeral dislocations may also cause vascular damage[6,10]. The anterior and posterior circumflex arteries form anastomoses with each other and with the vessels of the humeral diaphysis, the thoracoacromial artery, the suprascapular artey, the subscapular artery, and the deep brachial artery; the posterior circumflex artery also anastomoses with the circumflex scapular artery[9]. Gerber et al[10] demonstrated the importance of these anastomotic connections in a patient who did not develop osteonecrosis, despite a rupture of both circumflex arteries close to their origin from the axillary artery after an anterior subcoracoid dislocation of the glenohumeral joint.

Unrelated fractures can also put the patient at risk for humeral head osteonecrosis: fat from the exposed medullary cavity may embolize and occlude circulation in the head of the humerus[2].

## *Corticosteroids*

Patients may receive corticosteroid medications for collagen vascular diseases, dermatologic disorders, hematologic disorders, rheumatoid arthritis, and gout, or following organ transplantation[4]. Stress fractures through osteoporotic bone, hypercoagulability, and steroid-induced vasculitis have all been implicated as contributing to steroid-related osteonecrosis[5,6]. Corticosteroids also cause significant alterations in fat metabolism[4-6]; this systemic alteration in the metabolism results in a fatty liver and hyperlipidemia. This may cause fat embolisms which occlude circulation. Increased intraosseous fat cell size may raise intraosseous pressure and compromise circulation[4-6]. Cruess[5] mentions massive accumulation of intracellular lipids in osteocytes as a possible cause of osteocyte necrosis.

## *Alcoholism*

Together with steroid-induced and idiopathic osteonecrosis, alcohol-induced necrosis accounts for the majority of cases of non-traumatic osteonecrosis[1]. Alcohol abuse can increase endogenous cortisol levels[6]. It is also responsible for alterations in fat metabolism resulting in fatty changes in the liver with associated fat embolisms and raised intraosseous pressure as a result of increased fat cell size[5,6].

## *Sickle cell disease*

Sickle cell disease is a generic term for a group of autosomal recessive disorders characterized by an abnormal form of hemoglobin (hemoglobin S or HbS) within the erythrocytes[11]. In sickle cell disease, the red blood cells have their normal biconcave disc shape when they are oxygenated, but they assume a crescent or sickle cell shape during deoxygenation. Sickle cell disease has two main pathophysiologic features, both of which can contribute to circulatory compromise. Repeated cycles of sickling and unsickling damage the red blood cells; this causes a chronic hemolytic anemia, and the bone marrow expands to compensate for this increased erythrocyte hemolysis[11]. This bone marrow hyperplasia increases intraosseous pressure compromising intraosseous circulation[6]. Sickled red blood cells lose their vital ability to deform and squeeze through tiny blood vessels. Subsequently, they clog these small vessels depriving the tissues of an adequate blood supply. This occlusion results in hypoxia, which causes more erythrocytes to deoxygenate and sickle. Accumulation of sickled cells can totally obstruct blood vessels and thus cause anoxia and osteonecrosis[11].

Milner et al[7] studied the prevalence of humeral head osteonecrosis in 2524 patients with sickle cell disease. Overall prevalence at entry into the study was 5.6%. Prevalence increased with age: 2.7% of patients younger than 25 had osteonecrosis, increasing to 9.7% in pa-

tients aged 25-34, and to 19.8% in patients over the age of 35. The high prevalence of 3.8% in the 15-24 year old age group is worth noting. Of the 1655 patients without necrosis at study entry, 149 developed osteonecrosis of the humeral head. In 125 of these patients, sufficient data were available to evaluate both shoulders: 100 of 125 developed bilateral osteonecrosis. Osteonecrosis is not uniformly distributed over all subtypes of sickle cell disease: incidence was found highest in patients with sickle cell anemia and concomitant alpha-thalassemia[7]; this may be related to higher hematocrit values with thalassemia, causing increased viscosity of blood[6].

### *Gaucher's disease*

Gaucher's disease is another autosomal recessive disorder, which may cause osteonecrosis. It is a rare glycolipid storage disease, commonly seen in Ashkenazi Jews[12]. Glucocerebroside, a neutral glycolipid, accumulates in the macrophages throughout the reticulo-endothelial system as a result of a genetically determined absence of the enzyme beta-glucocerebrosidase[12,13]. These macrophages filled with cerebroside are known as Gaucher's cells[1]. Such glycolipid-laden cells accumulate in the marrow cavity and, by their mass effect, increase intraosseous pressure[1,5,6]. Substances released from the damaged macrophages may also injure the cells directly or indirectly by producing angiospasm[6]. Rodrigue et al[12] identified splenectomy, male gender, and a higher platelet count as significant univariate risk factors for developing humeral osteonecrosis in patients with Gaucher's disease. They hypothesize that removal of the spleen, which is a common therapeutic procedure in these patients, decreases the total body reservoir for storage of glycolipids resulting in increased accumulation of Gaucher's cells in the marrow space.

### *Decompression sickness*

Decompression sickness, or Caisson's disease, occurs in individuals exposed to compressed air environments, especially deep-sea divers[6]. Dysbaric osteonecrosis is at least partly caused by nitrogen bubbles coming out of solution in the blood as a result of a rapid drop in ambient barometric pressure[1]. Non-embolic factors are hypothesized to play a role in dysbaric osteonecrosis such as the release of vasoactive substances from damaged osteocytes, promotion of thrombosis, thickening of the intima of blood vessels, and compression of blood vessels by extravascular nitrogen bubbles[6,8]. Non-ischemic contributory factors may include a gas-induced osmotic shift of fluids due to rapid pressure changes, auto-immune changes, and increased oxygen tension. This increased oxygen tension is toxic to the collagen component of bone, limiting its ability to withstand mechanical deformation[6].

### *Other diseases*

Irradiation of bone most likely leads to osteonecrosis as a result of radiation damage to the vessel walls[1,6]. Especially the earlier, weaker forms of radiation therapy were prone to cause necrosis because the bone proportionately absorbed more radiation[3]. Vessel wall damage and osteonecrosis have also been described after electrocution injuries[6]. Vasculitis may result from systemic disease, such as systemic lupus erythematosus and rheumatoid arthritis[6]. Pancreatitis[2], oral contraceptives[2], (familial) hyperlipidemia[1,5], and obesity[2] can cause osteonecrosis by way of fat embolisms. Gout causes elevated serum uric acid levels; this hyperuricemia can cause precipitation and embolization of sodium urate crystals[2]. Other conditions associated with osteonecrosis are diabetes mellitus, Cushing's disease, pregnancy, peripheral vascular disease, hemophilia, smoking, and chronic renal failure associated with dialysis[2,6].

### *History and examination findings*

We have discussed above diagnostic imaging findings in the different stages of osteonecrosis of the humeral head. We have also discussed how identification of a causative medical condition may be the only information leading the practitioner to suspect osteonecrosis[6]. Osteonecrosis can occur asymptomatically, especially when it only affects the medullary and not the subchondral bone[1]. Revascularization or calcification can take place, or the bone can even remain avascular and necrotic without clinical problems, only producing an incidental radiographic finding[1,5]. Conversely, microfractures, even prior to complete fracture and articular surface collapse, may elicit pain when sufficiently mechanically stressed[1]. Milner et al[7] note that only 38.4% of patients with radiographic evidence of sickle cell-induced osteonecrosis of the humeral head were symptomatic about the shoulder. Because the glenoid is much more shallow and less conforming than the acetabulum, and because the shoulder is less of a weightbearing joint than the hip, greater degrees of deformity as a result of osteonecrosis are tolerated in the shoulder than in the hip[4,14]. Glenohumeral motion may be lost, but scapulothoracic motion is maintained, sometimes preserving functional range of motion[4]. Patients are likely to present later in the course of the disease as a result of the unique anatomy of the shoulder[4].

Patients typically present with poorly localized shoulder pain, which often occurs even at rest and at night, and is aggravated by activity[6]. The pain is usually gradual in onset[4]. Abduction is the earliest movement affected[7]. As discussed earlier, this may be related to the greater forces transmitted over the shoulder in a position of abduction[4]. Pain with active range of motion (AROM) is particularly severe with abduction and external rotation[4]. Flexion and extension AROM may be little restricted, even with se-

vere destruction of the humeral head[7]. At times an audible or palpable click is present with AROM[4]; this may be related to the intra-articular osseocartilaginous loose body sometimes found during later stages of the disease[5,6]. Passive range (PROM) is preserved until the later stages of the disease[4]. Boissonnault[2] notes that a sudden worsening of complaints followed by a dramatic sudden loss of range should alert us to the possibility of an osteonecrotic fracture in patients at risk for this disease.

Looking at a patient with humeral head osteonecrosis from the perspective of a physical therapist rather than that of a medical doctor, we can expect the patient to present with an inflammatory type pain related to the injury and repair process. This may produce pain at rest and at night, as mentioned above. Superimposed upon this inflammatory pain, there is likely to be a mechanical pain; this pain will be aggravated by mechanical stress sufficient to produce nociception as a result of deformation of mechanically weaker affected bone tissue or of differently loaded healthy bone tissue. The location of an osteolytic lesion in the area where the humeral head articulates with the glenoid at 90 degrees of abduction[4] may cause pain with active and especially resisted movement in this range, even before the occurrence of subchondral fracture and deformity. In Stage IV and V humeral head osteonecrosis, the deformity of the head and (in Stage V) the glenoid cavity can be expected to result in joint inflammation, including capsulitis. In these patients, we can expect to find a capsular pattern restriction of the glenohumeral joint during AROM and PROM. The endfeel during PROM will depend on the stage of the disease[15]. Muscular spasm indicates acute inflammation; a soft capsular, boggy endfeel points towards intra-articular effusion; a hard capsular endfeel is a sign of capsular fibrosis. Linear translation as part of passive accessory motion (PAM) testing may reveal changes in joint surface contour and associated crepitus. PROM and PAM testing may produce inconsistent results due to the presence of a loose body changing its intra-articular location.

## Treatment

The choice of treatment intervention in humeral head osteonecrosis depends on the stage of the disease and the possible presence of contra-indications to surgical treatment. Conservative therapy is considered appropriate for Stage I and II osteonecrosis[6]. It is also used in more advanced stages of the disease, if surgical options are not desired or contra-indicated[6]. Contra-indications to arthroplasty include loss of both deltoid and rotator cuff function, the presence of an active infectious process, or a neuropathic arthropathy[8]. If the specific causative medical condition can be identified, the first step is to remove the offending agent. This may mean discontinuation of alcohol abuse, removing the patient from a pressurized oxygen environment, or stopping corticosteroid therapy, if alternative treatments are available[6]. Hormone replacement therapy may be helpful in preventing osteonecrosis in patients with Gaucher's disease[12]. Goodman[11] mentions a number of stressors, relevant to physical therapy, that can precipitate a vaso-occlusive crisis in sickle cell disease patients: overexertion, dehydration, and the therapeutic use of cryotherapy. Hattrup[8] notes that analgesics, activity modification, and "unspecified" physical therapy modalities may be beneficial in early stages of the disease. Usher and Friedman[4] recommend using pendulum exercises to maintain range of motion and avoiding activities requiring extensive shoulder elevation. Based on the histopathologic processes discussed earlier, excessive compressive and shear forces need to be avoided during physical therapy interventions to prevent fracture and subsequent articular surface collapse. This excludes strong muscular contractions about the shoulder, especially at 90 degrees of abduction, and passive angular long-lever arm stretching. However, even forces parallel to the joint surface and applied close to the joint line, as used in glenohumeral manual mobilization, may introduce excessive shear forces. Attempting to get therapeutic benefit from high repetition, low-load exercises rather than the contra-indicated low repetition, high-load exercises may cause fatigue (micro) fractures in structurally weakened bone and, therefore, also seem contra-indicated.

Conservative therapy has shown poor results in patients with more advanced disease (Stage III or higher)[8]. The use of core decompression as a therapeutic intervention is based on the hypothesis discussed earlier that increased intra-osseous pressure may be responsible for compromising local circulation[6]. With core decompression of the humeral head, a 5 mm diameter coring device is driven into the proximal humeral metaphysis just lateral to the bicipital groove[14]. Mont et al[14] retrospectively reviewed the results after 2-14 years (average 5.6 years) for 30 shoulders in 20 patients who had undergone core decompression for symptomatic humeral osteonecrosis. Using the UCLA shoulder rating system, they found that all Stage I and II shoulders (n=14) had good or excellent results, and that 7 of 10 Stage III shoulders had excellent results. Three Stage III shoulders were treated later with arthroplasty. Only one of the five Stage IV shoulders had a good result; the other four shoulders were treated with follow-up arthroplasty. Core decompression seems most effective in the early stages and less effective in the later stages of humeral osteonecrosis[6]. Cushner and Friedman[6] describe a case in which a vascularized bone graft from the distal humeral diaphysis with a posterior deltoid muscle pedicle was placed into a core decompression tunnel in a patient with Stage III post-traumatic osteonecrosis; results were good but might have been the same with core decompression alone. They also report on a case in which the humeral head

lesion and an osseocartilaginous loose body were arthroscopically debrided[6]. Arthroplasty is the most appropriate treatment in patients with Stage IV and V osteonecrosis[6]. Studies limited to researching the results of arthroplasty in patients with humeral head necrosis report 83-100% pain relief and 119-170 degrees of post-operative active elevation[8]. In Stage IV patients a glenoid component is usually not necessary: hemi-arthroplasty is the procedure of choice[6]. Most Stage V patients, due to extensive cartilage loss and glenoid deformity, will require a glenoid component and a total shoulder arthroplasty is indicated[6]; arthrodesis is a salvage procedure and may be indicated in this patient population in case of persistent infection and concomitant brachial plexus lesions[8].

## *Case study #1*

A 61 year old female patient presented with constant right shoulder and lateral upper arm pain, which started seven weeks earlier when reaching into extension to grab groceries out of a shopping cart. She reported hearing a cracking noise at that time. Pain increased with reaching overhead, getting dressed, lying on the right shoulder, and even holding a coffee cup. Pain was present also at night. No easing positions, activities, modalities, or medications could be identified. The patient noted crepitus in the shoulder and decreased AROM in abduction, internal rotation (IR), and external rotation (ER). The patient denied any inflammatory symptoms about the shoulder. The only neurologic abnormality noted was occassional numbness in the fourth and fifth fingers of the right hand. Medical history seemed non-contributory. Patient was a heavy smoker.

Observation showed a forward head posture, increased thoracic kyphosis, and right scapular winging. AROM of frontal plane abduction-elevation was bilaterally 160 degrees with a 90-160 degree painful arc on the right. PROM frontal plane abduction-elevation was 180 degrees on the right and 160 degrees on the left with a normal endfeel. PROM of glenohumeral abduction was 80 degrees bilaterally. PROM ER was limited to 30 degrees on the right with a spasm endfeel versus 70 degrees on the left. With IR the patient could reach to the spinous process of T6 on the left and T9 on the right with an empty endfeel in the right shoulder. Manual muscle testing (MMT) revealed a grade of grossly 4/5 bilaterally about the shoulder and elbow. The Hawkins-Kennedy and horizontal adduction tests for subacromial impingement were positive on the right. The empty can test was 4-/5 and painfree bilaterally. The apprehension test (crank test) reproduced pain on the right, but the augmentation test was negative. Sulcus sign, labral tests, acromioclavicular (AC), and sternoclavicular (SC) tests were negative as well. Posterior translation of the humeral head showed restriction.

A physical therapy diagnosis was made of subacromial impingement syndrome in the right shoulder, posterior glenohumeral capsular tightness, and decreased rotator cuff muscle endurance. Decreased sensation in the fingers was attributed to thoracic outlet compression syndrome related to muscular guarding about the right shoulder. A treatment program of dexamethasone-iontophoresis to decrease inflammatory symptoms in the right subacromial space and low-load high-repetition active assisted, active, and resisted exercises in non-impingement provoking positions to increase rotator cuff strength and maintain range of motion was started. The patient did not tolerate manual mobilization aimed at stretching the posterior capsule. The home program consisted of posterior capsule stretching and rotator cuff exercises.

Re-evaluation one month later showed a decrease in AROM and PROM of the right shoulder, decreased strength of grossly 3+/5 bilaterally without pain, and a positive Hawkins-Kennedy impingement test on the right. Patient was referred back to physician with request for further diagnostic testing.

## *Case study #2*

A 63 year old man was referred to physical therapy with a diagnosis of adhesive capsulitis of the right shoulder. The patient reported pain in the whole right shoulder and lateral upper arm, which was aggravated by shoulder flexion, abduction, and forearm supination. Pain was also present at night. It was relieved by a counter-irritant rubbing ointment, application of heat, and by using NSAIDs. The patient reported that the right shoulder felt warm to the touch. Patient denied a neurologic deficit. Pain and decreased range in the shoulder started insidiously over the previous two months. Radiographs were negative. An intra-articular infiltration with cortizone somewhat increased motion but gave no relief of pain. Previous medical history revealed an adhesive capsulitis of the left shoulder, which required manipulation under anaesthesia.

Observation showed a right convex thoracic kyphoscoliosis, scapular winging right, and atrophy of the right supraspinatus. AROM in frontal plane abduction-elevation was grossly 160 degrees on the left and 130 degrees on the right with a painful arc between 90 and 130 degrees right. PROM abduction-elevation was 160 left and 130 right, both with a hard capsular endfeel. PROM glenohumeral abduction was 90 degrees on the left and 60 degrees on the right. PROM ER was limited to 45 degrees on the right versus 75 degrees left, both with a hard capsular endfeel. On PROM IR, the patient was able to reach T8 on the left and T11 on the right with an empty endfeel. MMT was grossly 5/5 bilateral except for ER and abduction right, which were 4-/5. Impingement tests were

negative, except for pain on the right empty can test. AC and SC joint tests were negative. A physical therapy diagnosis of capsular fibrosis of the right glenohumeral joint with decreased strength in abduction, ER, and scapular stabilizers was made. A treatment plan of manual glenohumeral mobilization and low-load high repetition rotator cuff and scapular stabilizers strengthening program was started.

At re-evaluation after 3 weeks, PROM in glenohumeral abduction had increased to near-normal, leaving the patient with a non-capsular pattern. Excessive ventral translation was found in a position of 90 degrees of abduction and maximal ER. Questioning the original diagnosis of adhesive capsulitis, I referred the patient back to the physician. The patient returned with a request for further therapy. I concentrated on contract-relax techniques to address what appeared to be a muscular restriction to ER, leaving the rest of the program the same. A week after this re-evaluation, the patient reported a "gun-shot like sound" in his shoulder while performing his normal daily activities. Patient was referred to physician and received a manipulation of the right glenohumeral joint the next week. Re-evaluation showed minimal increase in AROM but no changes in PROM. MMT was 3+/5 in abduction and ER right. Despite what appeared to be appropriate therapy of glenohumeral manual mobilization and rotator cuff and scapulothoracic strengthening, the patient continued to complain of constant deltoid and lateral upper arm pain. A final re-evaluation showed negative impingement, instability, and labral signs. At a loss to explain the patient's continued complaints I referred the patient back to the physician asking for his opinion on the patient's lack of a consistent pattern of evaluative findings indicating an identifiable causative pathology amenable to physical therapy treatment.

## Conclusion

Osteonecrosis of the humeral head does not cause a consistent pattern of signs and symptoms by which we can identify this diagnosis. As illustrated by the two case studies, the evaluation can seem to indicate other diagnoses, such as subacromial impingement syndrome and glenohumeral capsular fibrosis. Osteonecrosis often can only be suspected based on the patient's reporting a medical condition that is known to increase chances of developing osteonecrosis. In the case of these two patients, the only indicator in the history was the smoking habit of the female patient. Both patients reported a sudden increase in symptoms associated with a loud noise about the shoulder during a normal daily activity. This may have been the instant that a subchondral fracture occurred. The fact that both patients presented with constant pain without a consistent musculoskeletal pattern of signs and symptoms that could explain the continued pain and the fact that neither patient responded to what appeared to be the appropriate treatment based on evaluation and re-evaluations prompted me to send these patients back to their respective physicians.

This article may help the physical therapist in identifying patients at risk for humeral osteonecrosis, allowing them to not only appropriately but also expediently refer patients. It should also be instrumental in physical therapy management of those patients with an established diagnosis of early stage osteonecrosis of the humeral head.

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