

Nontraumatic osteonecrosis of the humeral head

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The humeral head is the second most common site for nontraumatic osteonecrosis after the femoral head, yet it has attracted relatively little attention. Osteonecrosis is associated with many conditions, such as corticosteroid use, sickle-cell disease, alcoholism, dysbarism (or caisson disease), Gaucher's disease, and other systemic conditions. The diagnosis is a clinical and radiographic one, the latter forming the basis for its staging. Treatment depends on the chronicity and severity of symptoms, as well as the degree of clinical and radiographic progression. Surgical treatment includes arthroscopic debridement and core decompression for early osteonecrosis and hemiarthroplasty or total shoulder arthroplasty for more advanced disease. This report reviews osteonecrosis of the humeral head, with an emphasis on current treatment options. (J Shoulder Elbow Surg 2002;11:281-98.)

BACKGROUND

Osteonecrosis, previously termed avascular necrosis or aseptic necrosis, is defined as the in situ death of a segment of bone.⁸¹ The disease may be primary, equivalently spontaneous or idiopathic, in which no clear etiology can be established, or secondary to a variety of known or hypothesized causes.^{81,114} As osteonecrosis continues to be studied, many cases once thought to be idiopathic are now known to result from one or more etiologic factors.

Osteonecrosis can be classified as either post-traumatic or nontraumatic.¹⁵ We limit our discussion to nontraumatic osteonecrosis, because its presenta-

tion and pathogenesis differ significantly from those of post-traumatic osteonecrosis, despite a common pathway of disruption of blood supply to the humeral head and subsequent juxta-articular bone death.

The humeral head is the second most common site of symptomatic osteonecrosis after the femoral head,^{21,23,58} but it has aroused rather limited research interest. Heimann and Freiburger⁴⁹ in 1960 and Cruess et al¹⁹ in 1968 provided initial descriptions of humeral head osteonecrosis. More recent studies have reported on patients with humeral head osteonecrosis* and patients with osteonecrosis at various sites including the humeral head.^{22,23,34} Other outcome studies of prosthetic shoulder arthroplasty have included patients with nontraumatic osteonecrosis,^{1,3,7,8,67,99,130} but the extent to which these patients have been evaluated separately has varied. The aim of this article is to review the pathophysiology of humeral head osteonecrosis and discuss the evaluation and treatment of the disease.

ETIOLOGY

Corticosteroid therapy

The most commonly reported cause of nontraumatic osteonecrosis is corticosteroid therapy.^{55,62,63,101} In 1957 Pietrogrande and Mastro-marino¹⁰⁹ first reported on osteonecrosis after corticosteroid therapy, and in 1968 Sutton¹²⁰ documented 154 cases related to corticosteroid use. The number of cases has risen exponentially since then, paralleling the use of corticosteroids in the treatment of numerous conditions.^{20,72,82,101,114} In 1971 Fisher and Bickel³⁴ described corticosteroid-induced osteonecrosis as a "disease of medical progress." Fortunately, the incidence of osteonecrosis among patients treated with long-term systemic corticosteroids has fallen from more than 25% to less than 5% in recent years,^{81,85,133} owing to judicious steroid use and dosing.⁵⁸

Osteonecrosis typically follows the chronic administration of high-dose steroids,^{58,85,103,133} but it is

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impossible to predict in which patients the disease will develop.⁸¹ The administration of high doses of the corticosteroid methylprednisolone to minimize edema after spinal cord injury prompted concerns over possible increases in the incidence of femoral and humeral head osteonecrosis. However, a prospective cohort study recently concluded that the true incidence of osteonecrosis among patients receiving high-dose steroids was less than 5%.¹³¹ Kenzora⁶⁸ reported that 6000 patients with head injuries were treated with prolonged high-dose steroids at one center without the occurrence of osteonecrosis.

In contrast, others have described osteonecrosis after the administration of corticosteroids in high short-term doses.¹²³ Humeral head osteonecrosis developed after a single 1-week course of corticosteroids for an acute exacerbation of arthritis.⁷² Bilateral shoulder and unilateral hip osteonecrosis developed after a 2-week course of high-dose methylprednisolone for the treatment of septic shock.¹⁰² Osteonecrosis has even been reported after intra-articular injection of the hip and glenohumeral joint.⁷⁵

The interval between corticosteroid administration and the onset of shoulder symptoms is also variable, ranging from 6 to 18 months in one large series.²¹ This is comparable to the interval leading up to the onset of hip symptoms, which ranges from 6 months to 3 years or longer.^{81,85} The incidence of humeral head involvement has not been shown to vary with the underlying indication for steroid use.³⁴

Hemoglobinopathies

Sickle-cell disease and other hemoglobinopathies are the most common cause of osteonecrosis worldwide.²⁶ Chung and Ralston¹¹ were among the first to associate sickle-cell disease with osteonecrosis of the humeral head. The disease is thought to be less progressive than that associated with other etiologies.⁸⁸

Dysbarism (caisson disease)

Decompression sickness, known as dysbarism or caisson disease, typically occurs in deep-sea divers or workers exposed to compressed air environments (caisson workers), such as in tunnels.⁷⁸

Gaucher's disease

Gaucher's disease is an autosomal recessive lysosomal storage disease caused by β -glucosidase deficiency and characterized by abnormal accumulation of glucocerebroside in Gaucher's cells of the reticuloendothelial system.⁶ Gaucher's disease is often seen in Ashkenazi Jews and is an important cause of nontraumatic osteonecrosis in that population.¹¹¹

Alcohol abuse/smoking

Chronic alcohol use is a major risk factor for osteonecrosis, although reports differ on its importance. Hattrup and Cofield⁴⁶ found that only 6% of humeral head osteonecroses could be attributed to alcohol abuse, compared with 56% attributed to corticosteroid use and 20% following trauma. In contrast, Jacobs⁵⁸ attributed 39% of 269 cases of nontraumatic femoral head osteonecrosis to excessive alcohol intake, compared with 28% attributed to corticosteroid use. Studies have demonstrated a strong association between both smoking and excessive alcohol intake and osteonecrosis.^{50,84,115} One study demonstrated an 18-fold increase in risk among individuals consuming more than 1 liter of alcohol per week compared with nondrinkers and a 4-fold increase among smokers compared with nonsmokers,⁸⁴ the latter presumably resulting from local vasospasm.¹¹³

Other risk factors

Some cases of osteonecrosis arise in association with other systemic diseases such as Cushing's syndrome and systemic lupus erythematosus. In many cases, the underlying etiology is confounded by chronic treatment with corticosteroids. Studies in a rabbit model have shown that corticosteroid-induced osteonecrosis occurs only if there is underlying disease.⁸³ Thus, corticosteroids may be only partly responsible for the development of "corticosteroid-induced" osteonecrosis.

Other risk factors for nontraumatic osteonecrosis include chemotherapy, hyperuricemia, hyperlipidemia, myxedema, pancreatitis, peripheral vascular disease, chronic dialysis, and pregnancy.^{21,32,52,55,63,81,82,127} Irradiation and thermal injury have also been implicated in the development of osteonecrosis.^{15,55,63,81} The etiology of osteonecrosis is undoubtedly multifactorial⁶⁵; some of the important risk factors are summarized in Table I. In some patients risk factors cannot be established, so that their osteonecrosis is idiopathic. Despite the different etiologies of nontraumatic osteonecrosis, all cases share remarkable similarities.^{22,93}

PATHOGENESIS

Terms such as "ischemic necrosis" and "avascular necrosis" have been used in the past to describe osteonecrosis, thereby presuming a vascular cause.^{12,114} Much of the literature supports the theory that a common physiologic denominator in the development of osteonecrosis is the interruption of the normal blood supply to bone leading to cell death. This explains post-traumatic osteonecrosis, but other

Table I Risk factors for nontraumatic osteonecrosis

Disorder	Trigger*
Corticosteroid administration	Fat embolism
Hemoglobinopathy	Embolism
Alcohol abuse	Fat embolism
Gaucher's disease	Embolism
Dysbarism	Embolism
Connective tissue disorders	Thromboplastin release
Arteritis or vasculitis	Hypersensitivity reaction
Hypercoagulability	Anticoagulant deficiency
Radiation injury	Injury/compression of vessel wall
Pregnancy	Thromboplastin release/fat embolism
Pancreatitis	Fat embolism/proteolytic enzymes
Idiopathic (spontaneous) osteonecrosis	

*Proposed trigger of intravascular coagulation (modified from Mankin⁸¹ and Jones⁶⁵).

explanations have been proposed for the pathogenesis of nontraumatic osteonecrosis.¹¹⁴

Vascular disruption

Phemister and colleagues⁶⁶ in 1939 first implicated vascular obstruction resulting from fat deposition in the development of osteonecrosis. Mankin⁸¹ reasoned that conditions associated with osteonecrosis must compromise humeral head blood supply in 1 of 4 ways: (1) mechanical disruption of blood vessels, (2) injury to or compression of the arterial walls, (3) arterial inflow obstruction such as thrombosis and embolism, and (4) venous outflow obstruction. Conditions associated with osteonecrosis may also be classified by their site of action: intraosseous-extravascular, intraosseous-arterial, extraosseous-arterial, and venous.⁵⁴

The pathogenesis of osteonecrosis of the humeral head is thought to be the same as that of the femoral head, which has been studied far more extensively.^{15,77,127} However, the pathogenesis must be understood in the context of the unique vasculature of the proximal humerus.

The extraosseous and intraosseous major blood vessels supplying the adult humeral head have been studied by Laing,⁷³ who identified a consistent artery on the anterolateral aspect of the humeral head. This ascending branch of the anterior humeral circumflex artery enters the proximal humerus at the upper end of the bicipital groove or by way of its branches into the adjacent greater and lesser tuberosities. Once intraosseous, a single vessel or multiple vessels pursue a tortuous posteromedial course just below the epiphyseal scar; the single vessel has been termed the arcuate artery (of Laing). Although Laing recognized

a rich periosteal vascular network, few anastomoses exist once the artery becomes intraosseous. More recently, Gerber et al⁴⁰ investigated the arterial blood supply to the humeral head in greater detail and reaffirmed the importance of the ascending branch of the anterior humeral circumflex vessel. Together, these studies define the intraosseous distribution of this vessel and confirm its role as the principal arterial blood supply within the humeral head.¹⁵

The subchondral bone of the humeral head is especially vulnerable to thrombotic and embolic phenomenon, because the arterioles in this area become sinusoids that turn 180° to return to the intraosseous circulation.²⁵ The lack of collateral blood flow in the humeral head enhances this vulnerability. Although vascular disruption is implicated in many cases of osteonecrosis, the underlying etiology is multifactorial. The various agents implicated in osteonecrosis appear to act differently to impede blood supply and produce bone cell death.

Corticosteroids

The pathogenesis of corticosteroid-induced osteonecrosis remains controversial. Alterations in fat metabolism have been implicated in 2 theories supported by the observation that patients who are taking corticosteroids demonstrate enhanced lipid production.²⁵ The first theory is based on local changes in bone. Studies into osteonecrosis of the femoral head have noted increases in intraosseous adipocyte size, leading to increased intraosseous pressure and ischemia.^{15,55} The second theory is an embolic one—systemic changes in fat metabolism increase serum lipids and produce fatty changes in the liver.⁶⁰ Fat subsequently embolizes to multiple tissue sites, including bone, resulting in focal osteocyte death.¹⁵ Cadaveric and biopsy studies demonstrate that these emboli localize in subchondral vessels.^{35,60} Other studies using labeled adipocytes showed these to localize in the subchondral bone of both the hip and shoulder.⁹⁵

Hemoglobinopathies

The pathogenesis of osteonecrosis in patients with sickle-cell disease and related blood diseases is thought to be embolic. Deformed erythrocytes disrupt blood flow, causing microinfarcts in the subchondral bone.²⁵ Higher hematocrit levels may increase the risk of osteonecrosis in patients with sickle-cell disease, possibly owing to an increase in blood viscosity.⁸⁸

Dysbarism

Early studies into osteonecrosis resulting from dysbarism implicated intravascular air emboli as causing

congestion and ischemia.⁸⁰ Other theories have been proposed, including the release of vasoactive substances and extravascular compression of vessels.²⁵ Studies implicate secondary injury to adipose tissue from rapidly expanding nitrogen leading to vessel collapse.⁶⁴

Gaucher's disease

Lipid-laden Gaucher's cells in the marrow produce a mass effect, increasing intraosseous pressure and causing extrinsic compression on the intraosseous blood vessels.^{25,55,81,93} Angiospasm caused by factors released from damaged macrophages may produce ischemia and contribute to the development of osteonecrosis.^{25,77,79}

Alcohol abuse

Excessive alcohol use may cause osteonecrosis in a manner similar to that described for corticosteroid use,²⁵ whereby fat produced in the liver embolizes to the subchondral bone. Changes in the bone marrow lead to venous stasis and further necrosis.¹⁰ Increased bone marrow pressure has also been implicated in alcohol-induced osteonecrosis.¹¹⁷

Other risk factors

Systemic diseases that cause bone infarction such as systemic lupus erythematosus and rheumatoid arthritis have been shown to disrupt the vascular supply at the intraosseous arterial level. Direct toxic effects have also been implicated in osteocyte death.¹¹⁴ Although evidence for some mechanisms continues to mount, most remain incompletely understood.

Unifying theories

Unifying theories have been proposed to explain the pathogenesis of osteonecrosis despite the specific underlying etiology. The first theory, proposed by Jones et al⁶¹ in 1974, implicated intravascular coagulation leading to intraosseous thrombosis. The release of nitrogen bubbles, fat emboli, Gaucher's cells, tissue thromboplastin, immune complexes, or bacterial endotoxins was thought to trigger intravascular coagulation.⁶¹ Consequently, in this theory, intravascular coagulation represents an intermediate step or common pathway in the pathogenesis of osteonecrosis, rather than its primary cause.⁶⁵ Some of the causes of secondary osteonecrosis and their postulated mechanisms are listed in Table I.

The accumulated cell stress theory, proposed by Kenzora and Glimcher,^{68,69} is an alternative unifying theory based on the fact that various conditions produce unhealthy bone cells. As these conditions progress or other factors such as corticosteroids are added, cellular homeostasis may be impaired to the

point of cell death. Osteonecrosis occurs once this critical cell stress is reached.⁶⁸

Repair of bone necrosis

Following ischemic bone injury and necrosis, a reactive healing response is mounted. Revascularization occurs by means of blood vessels, termed cutting cones, that enter the necrotic lesion at its margin. Undifferentiated mesenchymal stem cells migrate into the necrotic bone.⁸⁸ Some of these cells proliferate as fibroblasts, which form a layer of fibrous tissue. Macrophages within the fibrous tissue ingest the necrotic trabeculae and cellular debris.⁷⁷ Other cells differentiate into osteoblasts that synthesize new bone onto the remnant necrotic trabeculae.¹²⁸

BIOMECHANICS

The site of the lesion and the local biomechanical factors influence the presentation and progression of osteonecrosis.^{81,126} In contrast to the hip, the shoulder rarely assumes a weight-bearing role. In addition, the glenoid is not as conforming as the acetabulum, so it can withstand greater deformity.²⁵ Finally, preservation of considerable shoulder motion and function is often possible despite glenohumeral joint destruction and stiffness¹²⁸ because of compensatory motion at adjacent joints. These factors help explain why many patients with osteonecrosis of the humeral head do not have symptoms develop until late and have more advanced disease at presentation than those with lesions in the hip. As a result, diagnosis is often delayed and effective conservative treatment is precluded.^{77,128}

The superior central portion of the humeral head is most often the site of flattening and collapse (Figure 1).^{77,82,128} This region is normally in contact with the glenoid at 60° of humeral elevation^{60,77} or, equivalently, 90° of forward elevation,⁵⁷ owing to simultaneous scapulothoracic rotation.¹⁰¹ To limit the repetitive loading of this vulnerable region, patients with early osteonecrosis should avoid overhead activities.

As the osteonecrosis becomes more advanced, thickened trabeculae form at the periphery of the lesion,¹¹⁹ surrounded by regions of osteopenia resulting from bone resorption. The rate of resorption may exceed the rate of remodeling and repair, resulting in weak subchondral bone unable to withstand normal joint stresses.^{60,77} Two years or longer may be needed for enough bone resorption to cause a pathologic fracture,¹⁰⁵ but eventually microfractures occur and the underlying cancellous bone slowly collapses. Without structural support, the articular surface fatigues and deforms. The humeral head becomes flattened, the joint becomes incongruous, and over time

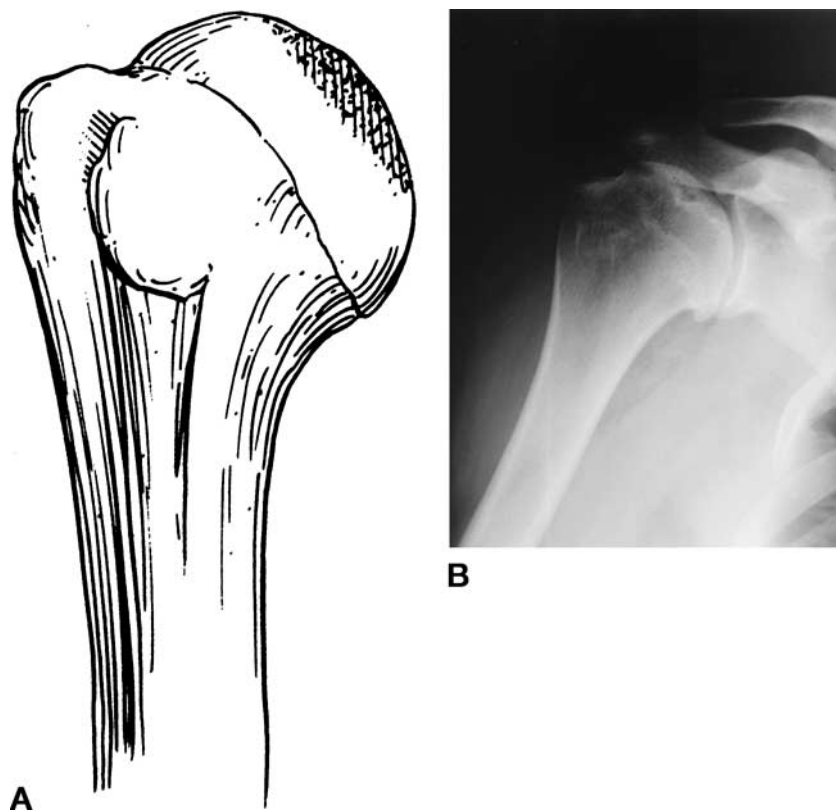


Figure 1 Line drawing (A) and radiograph (B) illustrating juxta-articular sclerosis of the superior central portion of the humeral head. This corresponds to stage II osteonecrosis, according to the modified Ficat-Arlet classification.

the degenerative changes may progress to involve the glenoid articular surface.

The size of the lesion and extent of the articular incongruity may influence the progression of osteonecrosis. Small femoral condyle lesions may heal with fibrocartilage following limited debridement.^{96,117} Articular surface incongruities of 2 mm or greater have been demonstrated in 85% of shoulders with osteonecrosis,⁷² but these have not been strongly associated with a poor outcome.

EPIDEMIOLOGY

The incidence of humeral head involvement, either in absolute terms or relative to involvement at other sites, is difficult to determine accurately because osteonecrosis of the humeral head often follows an indolent course. Matsen et al⁸² reported that nearly 5% of patients with glenohumeral arthritis had underlying osteonecrosis of the humeral head.

In an early retrospective review of 77 patients in whom osteonecrosis occurred after corticosteroid use, Fisher and Bickel³⁴ noted involvement of 127 femoral heads and 11 humeral heads; only 4 other sites were documented. In a series of 95 patients with corticosteroid-induced osteonecrosis, Cruess²⁰ described in-

volvement of the femoral head in 91 patients, the humeral head in 18, the distal femur or proximal tibia in 18, the talus in 6, and the capitellum in 1.

Patients with humeral head disease typically have involvement at other sites, especially the hip. Neer¹⁰¹ demonstrated concomitant hip involvement in 10 of 26 patients, and Cruess²³ noted bilateral hip involvement in 19 of 22 patients with nontraumatic osteonecrosis of the humeral head. In a recent study by L'Insalata et al,⁷² 76% of patients with osteonecrosis of the humeral head had involvement at other sites, including 90% of those with corticosteroid-induced osteonecrosis. Overall, 69% of patients had hip involvement, which was bilateral in two thirds of the cases.⁷² Thus, most patients with corticosteroid-induced osteonecrosis of the humeral head have osteonecrosis of the hip, often bilateral. In contrast, only 20% of patients with corticosteroid-induced hip disease have concomitant involvement of at least one humeral head. In extreme cases, patients with multifocal osteonecrosis involving 10, 17, and 18 distinct sites, including one or both humeral heads, have been described.^{20,76,87}

The epidemiology of humeral head osteonecrosis is perhaps best understood for patients with sickle-cell

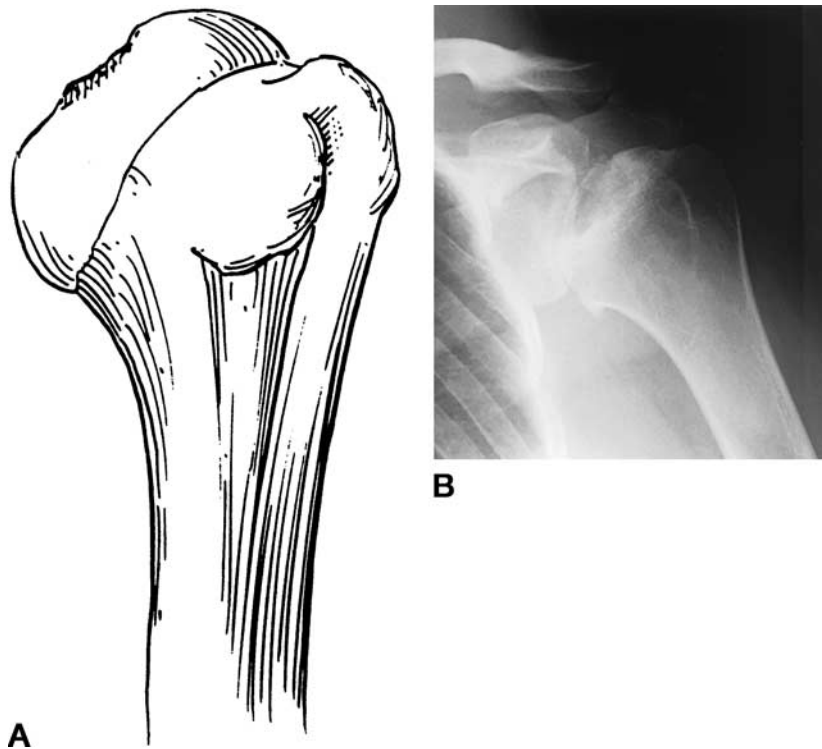


Figure 2 Line drawing (A) and radiograph (B) illustrating subchondral collapse and fracture (crescent sign) representative of stage III osteonecrosis.

disease. Early studies reported that nearly one third of patients with sickle-cell disease had osteonecrosis of the shoulder develop.^{11,26} A more recent prospective multicenter study of over 2500 patients with sickle-cell disease followed up for over 5 years⁸⁸ found that the overall incidence of osteonecrosis was roughly 5%. The prevalence ranged from 3% of patients younger than 25 years old to 20% of those older than 35 years old. Moreover, the prevalence varied with the severity of the underlying disease, ranging from 1% of young patients with sickle-cell disease to 3% of patients with sickle-cell anemia.⁸⁸

Some studies into dysbaric osteonecrosis suggest that the humeral head may be more frequently involved than other sites,^{38,43} whereas others report that lesions of the humeral head comprise about 30% of those among divers.^{27,38,78} In one study the prevalence of osteonecrosis among professional divers and compressed-air workers was estimated to be 6% and 24%, respectively.⁴³ The prevalence may be decreasing, owing to improved education on proper decompression.

CLASSIFICATION

Experience with osteonecrosis of the femoral head has shown the value of a staging system based upon the radiographic appearance of bony changes.¹⁵

Because this appearance varies little from one bone to the next, regardless of the underlying etiology,¹²⁸ osteonecroses of the humeral and femoral heads are staged similarly. The Ficat-Arlet classification system, initially described in 1968 and revised in 1980, is the most common classification for osteonecrosis of the femoral head.^{2,32,33} The adaptation of this classification to the humeral head by Cruess²¹ has become the most widely used.¹¹⁴

Stage I osteonecrosis signifies the absence of radiographic changes and requires magnetic resonance imaging (MRI) or radionuclide imaging for identification. Stage II is the first stage with radiographic changes (Figure 1). It is characterized by sclerosis of the superior central portion of the humeral head. The sclerosis may be wedge-shaped¹⁴ or mottled and diffuse.⁷⁷ Focal subchondral osteolysis without fracture may also be identified.¹²⁸

The "crescent" sign is the hallmark of stage III disease (Figure 2).⁷⁰ It represents a gently curved subchondral fracture through necrotic and partially revascularized bone that extends into the joint. The articular surface becomes depressed a few millimeters¹⁵ so that the round contour of the head is compromised⁹² without becoming significantly deformed.⁷⁷ The fractured fragment is often sclerotic and is best viewed on an anteroposterior radiograph

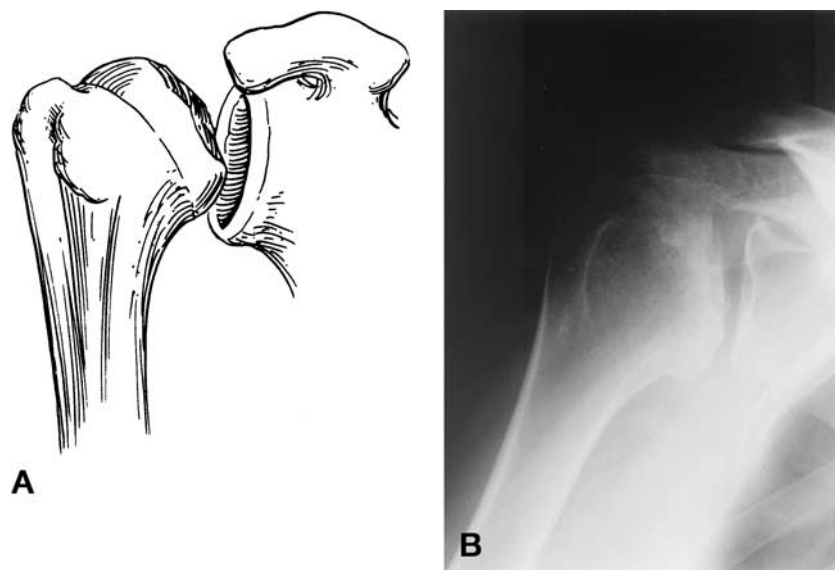


Figure 3 Line drawing (A) and radiograph (B) illustrating degenerative changes on the humeral side only. This corresponds to stage IV osteonecrosis.

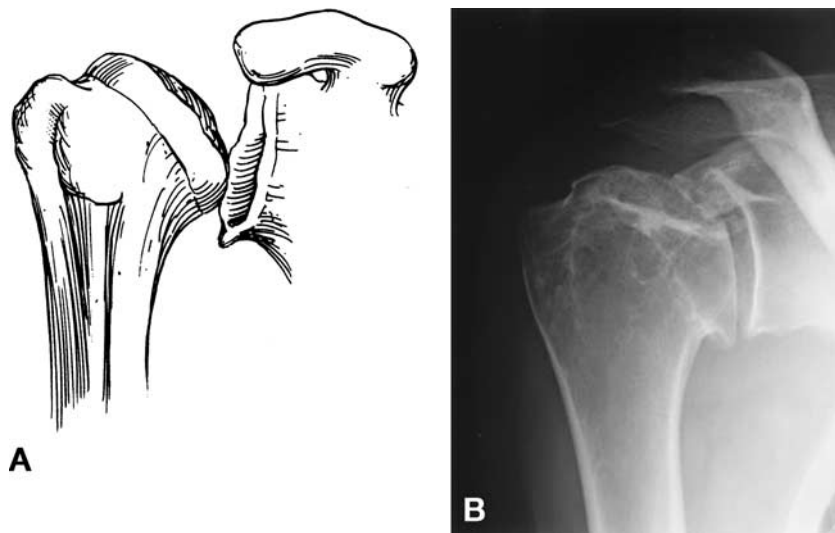


Figure 4 Line drawing (A) and radiograph (B) illustrating glenohumeral joint destruction representative of stage V osteonecrosis.

with the arm in external rotation or on an axillary radiograph.¹⁵ Occasionally, a flap of cartilage and subchondral bone becomes elevated from its underlying necrotic bed. This may assume the radiographic appearance of a sequestrum⁹² and cause mechanical symptoms.¹⁵

Stage IV disease is characterized by widespread collapse of the subchondral bone into the adjacent, soft necrotic bone and extensive destruction of the underlying trabecular pattern⁹² leading to secondary arthritis¹⁵ (Figure 3). The collapsed bone is usually confined to the superior central portion of the humeral

head. Stage V is the final stage of osteonecrosis, in which the glenoid is also involved, leading to an incongruous glenohumeral joint (Figure 4).^{15,72}

NATURAL HISTORY

In general, the clinical progression of nontraumatic osteonecrosis of the humeral head appears to be slow, and thus many patients have advanced radiographic changes at presentation. However, the natural history varies greatly, so it is difficult to predict which patients will have severe arthrosis develop.

Patients with sickle-cell disease tend to have the most benign course. In the large study cited previously, only 21% of patients reported symptoms of pain and limited motion at presentation, and only a single patient out of 2500 required arthroplasty.⁸⁸ In another study only 2 joint replacements were performed in 138 patients with sickle-cell disease.²⁶

Excluding patients with sickle-cell disease, those taking corticosteroids tend to fare better than those with other risk factors.^{20,22,23,72} Hatrup and Cofield⁴⁶ found that frank degenerative changes, extensive head involvement, and a history of trauma were associated with a poor prognosis. In their series, 43% of patients with corticosteroid-induced osteonecrosis required shoulder replacement at 3 years after diagnosis, compared with 80% of patients with post-traumatic osteonecrosis. Patients taking corticosteroids often have a relatively painless shoulder, despite glenohumeral stiffness and advanced radiographic stage. This may be because many patients receiving long-term corticosteroid therapy have multiple comorbidities that overshadow their shoulder condition.⁷²

Patients with humeral head osteonecrosis have also been stratified by severity of symptoms and radiographic stage. Cruess²² divided patients with corticosteroid-induced disease into 3 groups. The first group had minimal deformity and symptoms—their lesions appeared to stabilize without collapse. Patients in the second group had significant deformity and functional deficits but led lives that did not require extensive use of their shoulders. Corticosteroids were continued in these patients, and the condition did not progress. The third group had severe pain and stiffness necessitating hemiarthroplasty.

Rutherford and Cofield¹¹² have related the natural history of humeral head osteonecrosis to its stage. In an early report, with a mean follow-up of 4.5 years, 9 of 11 patients with stage II or III disease did not require additional treatment, whereas all 5 patients with stage IV or V disease underwent prosthetic arthroplasty. More recently, Hatrup and Cofield⁴⁶ evaluated 200 shoulders in 151 patients with osteonecrosis attributed to various factors including corticosteroid use (56%), trauma (20%), alcohol abuse (6%), and idiopathic factors (15%). Excluding 38 shoulders lost to follow-up, 97 shoulders underwent prosthetic shoulder arthroplasty, 5 underwent other procedures, and 60 had not required surgery at the time of most recent follow-up. Using Kaplan-Meier survivorship analysis, the authors found that 42% of shoulders with stage II disease, 29% with stage III disease, 55% with stage IV disease, and 79% with stage V disease had required shoulder replacement 3 years after diagnosis. At a mean follow-up of 8.6 years, 75% of those treated nonoperatively had only

occasional shoulder discomfort and no significant pain.⁴⁶

L'Insalata et al⁷² reported more discouraging results in 65 shoulders with osteonecrosis, 80% of which were related to corticosteroid use. Within 2 years of initial presentation, 35 shoulders (53%) had required prosthetic arthroplasty. Only 15 of the remaining shoulders treated nonoperatively had satisfactory results. Patients with stage III or more advanced disease had the worst outcomes.

CLINICAL EVALUATION

Insidious shoulder pain with movement is a frequent presentation of nontraumatic osteonecrosis of the humeral head. Pain at night may or may not be common,²³ but one study showed that over 70% of patients had difficulty sleeping.⁷⁷ Symptoms prevent normal work in nearly 80%. At presentation, patients often have a painful click accompanying certain shoulder movements, resulting from joint incongruity, a cartilage flap, or a large loose body.^{23,48}

Patients with osteonecrosis of the humeral head are younger than most patients with primary or other types of osteoarthritis. Matsen et al⁸² determined that patients with nontraumatic osteonecrosis presenting for treatment had a mean age of 51 years. The mean age of 26 patients treated with shoulder arthroplasty by Neer¹⁰¹ was 39 years (range, 21-64 years). Patients with sickle-cell disease have osteonecrosis develop very early in life⁸⁸—the mean age in one series was 26 years.²⁶

A thorough medical and social history must be obtained to determine predisposing factors for the development of osteonecrosis such as corticosteroid use, smoking, or excessive alcohol use.¹¹⁷ In a patient with mild symptoms and equivocal radiographic features, identifying the risk factor may be the key to diagnosis. Interventions that eliminate the risk factor may not influence disease progression in the involved joint, but they may diminish future risk to other joints.⁷⁷

Physical examination may reveal local tenderness, but active and passive motion is often preserved until the late stages of the disease.²¹ The discomfort may be greatest when the arm is abducted or elevated 90°, corresponding to the position of maximum glenohumeral loading. Other authors have concluded that painful glenohumeral rotation with the arm at the side distinguishes advanced osteonecrosis from rotator cuff disease, which is associated with painful abduction.¹¹⁷

Laboratory tests are used to identify associated conditions and eliminate other causes of shoulder pain. Depending on the history and physical examination, useful tests may include complete blood count, erythrocyte sedimentation rate, and C-reactive pro-

tein to help rule out infection, as well as specific serology for rheumatoid arthritis and related inflammatory conditions. Gaucher's disease is characterized by elevated serum acid phosphatase, but the diagnosis should be confirmed by enzymatic and mutational analysis.⁶ In many cases the history, physical examination, and laboratory tests serve only to raise the suspicion of osteonecrosis.

IMAGING AND OTHER STUDIES

Since the first radiographic diagnosis of osteonecrosis by Sweetman et al,¹²¹ in 1960, plain radiographs have become essential for the diagnosis and classification of osteonecrosis. True anteroposterior and axillary radiographs are usually obtained,¹² and 40° posterior oblique radiographs in internal and external rotation may also be helpful.⁴⁷ Although the radiographic findings used in staging osteonecrosis of the humeral head are usually diagnostic, the differential diagnosis of a lucent lesion includes bone infarct, bone cyst, benign or malignant bone neoplasm, and infection.⁴¹ Radiographs can also be used to quantify the size of the lesion through use of the necrotic angle,⁷⁴ which was originally described for the hip.⁷¹ The necrotic angle is the sum of the angle subtended by the involved articular surface on both anteroposterior and axillary radiographs.⁷⁴

When radiograph findings are normal, as in stage I, other imaging is required for diagnosis. MRI depicts a clear demarcation between live and dead bone¹⁰ and has been used to detect osteonecrosis that cannot be detected by plain radiographs or scintigraphy.^{16,91,107} Osteonecrosis alters normal marrow signal, which is uniformly high on T₁-weighted images, owing to its predominantly fatty content.¹⁰⁷ In the hip, stage I osteonecrosis appears on MRI as linear bands of low signal on both T₁- and T₂-weighted images that represent reactive bone at the margins of the infarct.¹⁰⁷ The MRI findings in osteonecrosis of the humeral head have recently been described.^{108,134} A double-line sign, previously described in the femoral head, representing bands of alternating high and low signal intensity, was observed in over 50% of patients.¹⁰⁸ The bright band, corresponding to hyperemic granulation tissue, lies within a dark band, corresponding to fibrosis or sclerosis.¹³⁴ In later stages, fragmentation and articular collapse are seen, often accompanied by joint effusion.^{107,134} Necrotic areas appear as regions of low signal intensity. Spin-echo sequences, surface coils, and special techniques such as the short T₁ inversion recovery (STIR) technique may accentuate regions of marrow edema or identify subtle articular degeneration and collapse.¹³⁴

The scintigraphic appearance of early osteonecrosis is a photopenic zone corresponding to the necrotic

segment. Increased radionuclide uptake accompanies revascularization and repair, producing a "doughnut" sign consisting of a central photopenic zone surrounded by a ring of increased activity.^{29,125} Either increased or decreased uptake may be demonstrated on radionuclide imaging for stage I disease. Scintigraphy becomes less specific when the increased activity accompanying revascularization obliterates the central photopenic zone.¹¹⁷ Single photon emission computed tomography bone scintigraphy uses sequential tomographic images to distinguish the photopenic zone from adjacent zones of activity, thereby enhancing specificity.^{17,45,117}

The sensitivity and specificity of radionuclide imaging and MRI for detecting osteonecrosis have been determined for the femoral head but not for the humeral head. The sensitivity and specificity of radionuclide imaging in detecting lesions in the femoral head have been reported to be as high as 92% and 100%, respectively.¹²⁵ The sensitivity of MRI in the detection of osteonecrosis of the femoral head has been reported to be even higher,^{37,90,91} approaching 100% in several series.^{16,30,36,39,42,89,122} In general, MRI is more sensitive in the detection of early disease,¹³⁴ and radionuclide imaging is less sensitive for the transition from stage I to stage II disease, during which the central photopenia may become obscured. In contrast to the variable patterns of radionuclide uptake, the appearance of osteonecrosis on MRI is relatively constant.¹³⁴

Computed tomography (CT) is used when other diagnoses are being considered. The CT scan correlates well with radiographs and may be used to quantify the extent and severity of late disease.¹³⁴ A lesion may appear on CT as a sharply circumscribed lucent lesion with a density similar to fat, surrounded by an irregular, calcified border.⁴¹ Cofield¹⁵ has previously advocated trispiral tomography to demonstrate subchondral fracture, but CT is rapidly replacing this technique.

Bone marrow pressure measurements have enhanced our understanding of the pathophysiology of osteonecrosis. Because the increase in intraosseous pressure occurs in stage I disease, prior to the appearance of radiographic changes,²⁴ its measurement is thought by some to have clinical utility. Measurement of bone marrow pressure has been advocated by Ficat and Arlet³² and by Hungerford and Zizic⁵² at the time of core decompression. Venography has demonstrated additional evidence of osteonecrosis in the hip, such as poor visualization of the major efferent veins, delayed drainage of contrast, and reflux of contrast into the femoral head and diaphysis,¹⁵ but this technique has not been applied to the humeral head.

The features of each radiographic stage of osteonecrosis along with its MRI and clinical correlates are

Table II Staging of humeral head osteonecrosis

Stage	Radiographic	MRI	Clinical
I	Normal	Low signal bands	Normal
II	Mottled, sclerotic bone	Uniform loss of signal	Normal
III	Crescent sign	Crescent sign	? Pain with activity
IV	Extensive collapse	Effusion	Pain with activity
V	Glenoid degenerative changes	Extensive collapse, glenoid changes	Pain at rest

summarized in Table II. Although MRI and radionuclide imaging are important in the early diagnosis of humeral head osteonecrosis, MRI offers no advantage over standard radiographs once the diagnosis has been made.¹⁵ Consequently, despite the high accuracy of these studies, their findings must be correlated with the radiographic appearance, and thus, the latter remains the most effective staging tool.

Once a lesion has been identified, radiographs of the contralateral shoulder should be obtained so that subclinical disease may be identified early enough for the patient to benefit from conservative or minimally invasive treatment. If the radiograph findings are negative, MRI of the asymptomatic shoulder should be considered. Alternatively, radionuclide imaging can be performed, which offers the added benefit of excluding disease in remote joints. It will become clear in the following sections that any treatment plan short of prosthetic arthroplasty is predicated on the early diagnosis of osteonecrosis.⁷⁷

NONOPERATIVE TREATMENT

Successful treatment should preserve shoulder function, halt the progression of disease, and decrease symptoms.⁷⁷ Nonoperative treatment begins with patient education and addressing known risk factors. Offending habits such as smoking and alcohol use should be discontinued, corticosteroids should be taken judiciously, and alternative treatments should be sought.

Specific treatment is tailored to each patient according to symptoms and functional limitations, but staging aids in the decision making. Nonoperative treatment, consisting of physical therapy and activity modification, may be used initially for stage I and stage II osteonecrosis,¹²⁸ especially when symptoms are relatively mild. Exercises to preserve shoulder motion are instituted, and activities requiring overhead elevation are restricted.^{11,21,74}

Analgesic medications may provide some symptom relief,^{47,77} but studies into the role of pharmacologic agents in the treatment of humeral head osteonecrosis are lacking. A cortisone injection into the glenohumeral joint may provide relief and delay arthroplasty, but its use must be weighed against the potential for disease progression and for infection, especially in the immunocompromised patient. Elec-

trical stimulation has been used,¹⁵ but its role remains unknown.

Evidence of either stage III or more advanced disease or radiographic progression of disease has been found to be a strong predictor of poor outcome following nonoperative management.⁷² Cruess²² believed that nonoperative treatment was appropriate for patients with minimal deformity and symptoms and for patients with limited demands and minimal symptoms despite significant deformity and dysfunction—the first 2 groups he described. Persistent symptoms signal the failure of conservative treatment and the need for surgical intervention.¹²⁸

ARTHROSCOPIC DEBRIDEMENT

Johnson⁵⁹ was the first to report on arthroscopic debridement and loose body removal in patients with humeral head osteonecrosis. More recently, 3 case reports have specifically addressed the arthroscopic treatment of humeral head osteonecrosis. One describes the successful arthroscopic excision of a large osteochondral fragment and debridement of a complementary humeral head defect in a patient with corticosteroid-induced osteonecrosis.⁴⁸ The second report suggests that arthroscopy can be used to excise chondral and osteochondral lesions and debride the articular surfaces as an adjunct to decompressing stage I through stage III lesions.⁴ The authors speculate that arthroscopy might aid the placement of osteochondral grafts in the shoulder, much in the same way it is used in the knee, but actual results were not provided. The third report describes the arthroscopic-assisted reduction and bone grafting of an osteochondral lesion in a patient with idiopathic stage IV osteonecrosis of the humeral head.⁹⁷ A flap of articular cartilage was removed arthroscopically, a bone tunnel through the greater tuberosity to the lesion was made, the tunnel was filled with iliac crest autograft, and the retrieved flap of cartilage was replaced arthroscopically. At 2-year follow-up the shoulder was pain-free and could be elevated to 160°.⁹⁷

Several other studies have reported improvement in pain and function after arthroscopic debridement for glenohumeral arthritis,^{9,31,59,98} but patients with osteonecrosis were not discussed specifically. In addition, long-term follow-up on patients undergoing debridement is lacking and any reported efficacy

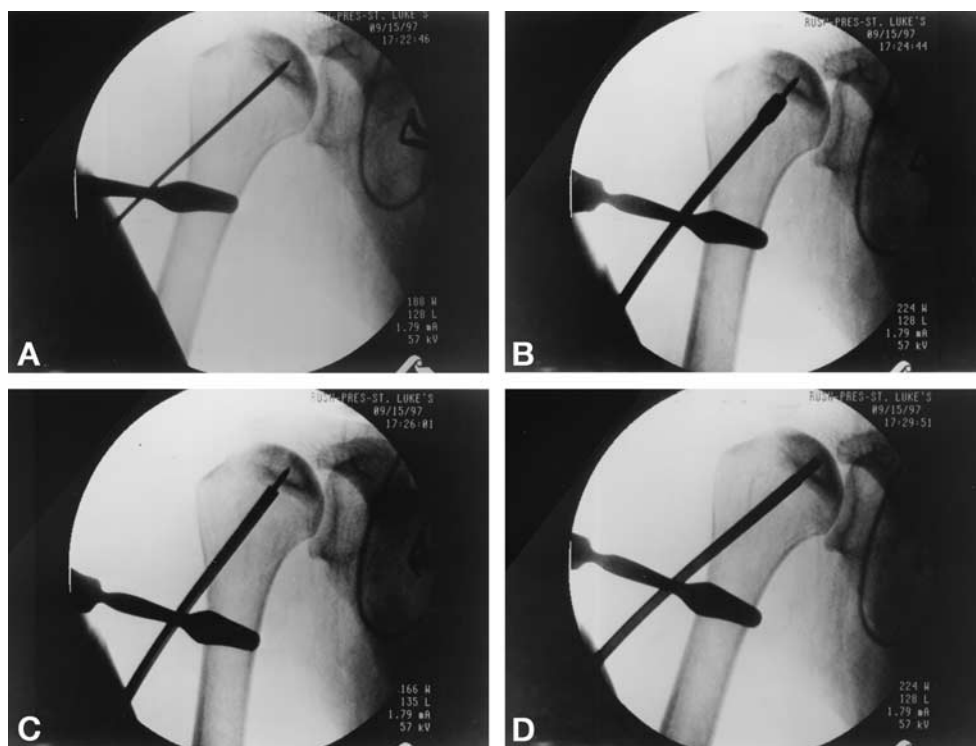


Figure 5 Core decompression of the proximal humerus. **A**, A guide pin is inserted through the lateral humeral cortex and advanced into the center of the osteonecrosis site. **B**, An 8-mm anterior cruciate ligament (ACL) bone tunnel reamer is then advanced to the base of the lesion. **C**, The bone biopsy device is advanced over the guide pin to the base of the lesion. **D**, The guide pin is removed, and several core biopsy specimens are manually extracted.

may relate to procedures performed concurrently such as capsular release. Clearly, the role of arthroscopy⁷⁷ and related minimally invasive techniques in the treatment of humeral head osteonecrosis remains unknown. However, the low morbidity, rapid return of comfort and function, and preservation of bone stock afforded by arthroscopic debridement, as well as concerns about the effectiveness of core decompression and the durability of shoulder arthroplasty in the young patient, will likely fuel the popularity of arthroscopy for the treatment of osteonecrosis in the future.

CORE DECOMPRESSION

Core decompression, with or without bone grafting, aims to decrease intraosseous pressure⁵³ and promote revascularization, and its use has been reported extensively for the treatment of osteonecrosis of the femoral head, with success rates ranging from 40% to 90%.^{51,86,124} Core decompression of the femoral head remains controversial, but it generally appears to be efficacious for pre-collapse (stage I and II) disease.^{32,93} In contrast to the femoral head, ex-

perience with core decompression of the humeral is limited.⁴⁷

Mont and colleagues^{74,92} have described the operative technique of core decompression of the humeral head. A small incision is made in the anterior axillary fold just above the pectoralis major tendon and extended bluntly within the deltopectoral interval to the proximal humerus.⁷⁴ A cannulated reamer is used over a guidewire to ream into the proximal humeral metaphysis (Figure 5, A and B). The reamer is inserted lateral to the bicipital groove to avoid damaging the ascending branch of the anterior humeral circumflex artery. The reamer is advanced, under fluoroscopic guidance, to a point inferior and lateral to the lesion and replaced by a coring device that is manually inserted to extract the core biopsy (Figure 5, C and D).

The results of core decompression in 20 patients (30 shoulders) were reviewed at greater than 5 years' follow-up.⁹² Fourteen shoulders had stage I or II disease, and the remainder had stage III or IV disease. The mean UCLA shoulder score at fol-

low-up was 28.5 (out of 30 points), compared with 14 points preoperatively.¹²⁷ All patients reported immediate pain relief after decompression. At follow-up, good to excellent results were obtained for all shoulders with stage I or II disease and 7 of 10 shoulders with stage III disease but for only 1 of 6 shoulders with stage IV disease. The remaining 8 shoulders required shoulder arthroplasty, which was performed within 1 year after core decompression for shoulders with stage IV disease and within 4 to 6 years for shoulders with stage III disease.⁹² The study has recently been expanded to include 63 shoulders in 43 patients followed up for 2 to 18 years (mean, 8.6 years) after core decompression.^{74,94} Fifteen shoulders with fair or poor UCLA shoulder scores at the most recent follow-up eventually underwent arthroplasty. The remaining shoulders had good to excellent scores, including 15 of 16 with stage I disease, 15 of 17 with stage II disease, 16 of 23 with stage III disease, and only 1 of 7 with stage IV disease. The authors reiterated that core decompression provides lasting pain relief and improved function in nearly all patients with stage I and II disease and the majority of patients with stage III disease. A review of 178 patients with osteonecrosis of the humeral head associated with sickle-cell disease reported good shoulder function in the 12 patients who underwent core decompression.¹⁰⁶

L'Insalata et al⁷² described humeral head drilling in 5 shoulders with stage III disease, 2 of which also underwent arthroscopic debridement. All shoulders had clinical progression of disease, and 4 required arthroplasty within 1 month to 3 years after drilling. The authors concluded that core decompression was ineffective in preventing clinical or radiographic progression of stage III osteonecrosis of the humeral head. This echoed the conclusion of Neer,¹⁰¹ who reported on the failure of humeral head drilling and bone grafting in 2 patients.

In a recent review, Zuckerman and colleagues⁷⁷ reported moderate success with core decompression. They cautioned that the greatest difficulty remains the inability to identify most patients early enough in the disease process to benefit from the procedure. In summary, it has not been shown definitively that core decompression is significantly more effective than conservative treatment alone.¹⁵ The results of core decompression for stage I and stage II disease suggest a possible role for this technique.⁴⁷ However, studies by L'Insalata et al,⁷² Hungerford,⁵³ and Mont and colleagues^{74,94} suggest that the outcome after core decompression for patients with stage III osteonecrosis of the humeral head may not differ substantially from the natural history of the disease. Once patients have symptomatic stage IV or stage V disease, an alternative to core decompression should be considered.

BONE GRAFTING

Deltoid muscle pedicled bone has been grafted into a stage III humeral head lesion.¹¹⁰ At 18-month follow-up, shoulder function had improved and there was radiographic evidence of graft incorporation. More recently, autogenous iliac bone grafting in 2 patients with symptomatic stage III and stage IV osteonecrosis of the humeral head has been reported.¹²⁹ The lesions were debrided and the graft fashioned and held in place with screw fixation. Both patients demonstrated radiographic union and a good clinical result approximately 3 years after surgery. Bone grafting with or without arthroscopic assistance remains experimental, and further study is needed before it can be recommended.

PROSTHETIC SHOULDER ARTHROPLASTY

The most reliable and durable treatment for osteonecrosis of the humeral head remains prosthetic shoulder arthroplasty. The indications are similar to those for other diagnoses, namely significant pain and dysfunction refractory to alternative treatment. Because radiographs often correlate poorly with symptoms, early consideration of arthroplasty may be appropriate for patients with severe symptoms and poor prognostic factors despite relatively early disease.⁴⁷

The only absolute contraindication to prosthetic arthroplasty is active infection, but relative contraindications include loss of deltoid and rotator cuff function, neuropathic arthropathy,⁴⁷ and severe brachial plexus injury. Age is an important consideration because patients with osteonecrosis are often young. Several recent studies have raised concerns over the long-term durability of prosthetic arthroplasty and the need for multiple revisions in young patients.^{18,56,118,132} Sperling et al¹¹⁸ demonstrated 15-year survival rates of 84% and 73% for total shoulder arthroplasty and hemiarthroplasty, respectively, in patients aged 50 years or younger. Functional outcome in 6 patients with osteonecrosis was not specifically reported, but 3 had an unsatisfactory result. It follows that young patients undergoing prosthetic shoulder arthroplasty should be carefully counseled regarding the importance of joint protection and activity modification.⁴⁷ Arthrodesis is infrequently accepted but remains an option for patients unwilling to give up strenuous use of their shoulders.

The technique for shoulder arthroplasty has been described extensively.^{44,47,82} Careful attention to component positioning and soft-tissue release is essential to the success of the procedure. The decision of whether to cement or press-fit the humeral component is a controversial one. Neer¹⁰¹ demonstrated that young patients with osteonecrosis had thick cortices, so a good press fit could be obtained. Others have

Table III Results of prosthetic arthroplasty for osteonecrosis

Study	No.	Total	Type	% Pain relief	Active elevation
Amstutz et al ¹	3	56		100	160°
Bade et al ³	8	38	NT	100	170°
Boyd et al ⁷	11			100	119°
Boyd et al ⁸	6			83	—
Cruess ²⁰	5	—	NT	100	101°
Dines et al ²⁸	3	20	PT	100	128°
Kay and Amstutz ⁶⁷	3	15		100	143°
Neer ⁹⁹	3	12	PT	100	Good-excellent
Neer ¹⁰¹	24	—	NT	91	—
Orfaly et al ¹⁰⁴	21	21	8 PT, 13 NT		123°
Rutherford and Cofield ¹¹²	17	17	NT	94	157°
Warren et al ¹³⁰	5			100	116° gain

Modified from Hattrup.⁴⁷

Total, Overall number of arthroplasties; NT, nontraumatic; PT, post-traumatic.

repeated these recommendations, reserving cement for situations in which a good press-fit cannot be obtained.^{13,14,47}

The decision of whether glenoid resurfacing should be done remains a controversial one as well. Most surgeons resurface the glenoid only when there is extensive wear corresponding to stage V disease^{116,128} and an intact or reparable rotator cuff.⁴⁷ However, the incidence of glenoid resurfacing has varied among surgeons. Rutherford and Cofield¹¹² reported that 37% of their patients underwent total shoulder arthroplasty. In contrast, Zuckerman and colleagues⁷⁷ have not found glenoid resurfacing to be necessary in the majority of patients with osteonecrosis. Occasionally, a patient who has had a humeral head replacement alone will require late conversion to a total shoulder arthroplasty.¹⁵

Many reports of prosthetic shoulder arthroplasty include patients with osteonecrosis, but their number is usually so small that their outcome is not evaluated separately.^{15,47} An early study by Neer et al¹⁰⁰ of 261 unconstrained total shoulder arthroplasties included only 2 patients with osteonecrosis. More recent series suggest that patients with nontraumatic osteonecrosis represent approximately 5% of all patients undergoing prosthetic shoulder arthroplasty.^{82,101,118} This proportion may be increasing as a result of more accurate diagnosis of osteonecrosis and expanding indications for shoulder arthroplasty.

The reported outcomes after prosthetic arthroplasty for osteonecrosis of the humeral head are summarized in Table III. A few small studies have specifically evaluated the outcome of shoulder arthroplasty for osteonecrosis, and several larger studies have stratified the outcome of shoulder arthroplasty by underlying diagnosis. For the most part, these studies fail to provide details on functional results and complications and include both patients with post-traumatic

osteonecrosis and patients with nontraumatic osteonecrosis, even though the former tend to have more modest gains after prosthetic arthroplasty.^{3,47}

As shown in Table III, nearly all patients reported dramatic pain relief and improved motion. Neer¹⁰¹ reported excellent results with near normal motion and without evidence of loosening in 24 shoulders that underwent prosthetic shoulder arthroplasty for nontraumatic osteonecrosis, 15 of which were hemiarthroplasties. Cruess²⁰ reported pain relief and improved range of motion at 1 to 6 years after humeral head replacement for the most severely involved 5 shoulders in his series. Kay and Amstutz⁶⁷ reported similar improvements after 3 hemiarthroplasties for osteonecrosis of the humeral head. Rutherford and Cofield¹¹² reported on 17 osteonecrotic shoulders in 13 patients. Ten shoulders with stage IV changes and a single shoulder with stage V changes and insufficient glenoid bone stock underwent hemiarthroplasty, and 6 shoulders in 5 patients with stage V changes underwent total shoulder arthroplasty. Postoperatively, 16 of 17 shoulders had either no or mild pain. Shoulder abduction and external rotation averaged 161° and 77°, respectively, in patients who underwent hemiarthroplasty, and 150° and 67°, respectively, in patients who underwent total shoulder arthroplasty.¹¹²

A recent prospective study examining the outcome after prosthetic arthroplasty for osteonecrosis demonstrated significant improvements in pain and function using various self-assessment instruments.¹⁰⁴ Forward elevation, internal rotation, and external rotation improved considerably, but forward elevation did not approach normal. Although patients with nontraumatic osteonecrosis had greater pain and functional deficits preoperatively than those with post-traumatic osteonecrosis, both groups had comparable results. However, the results in both groups were somewhat

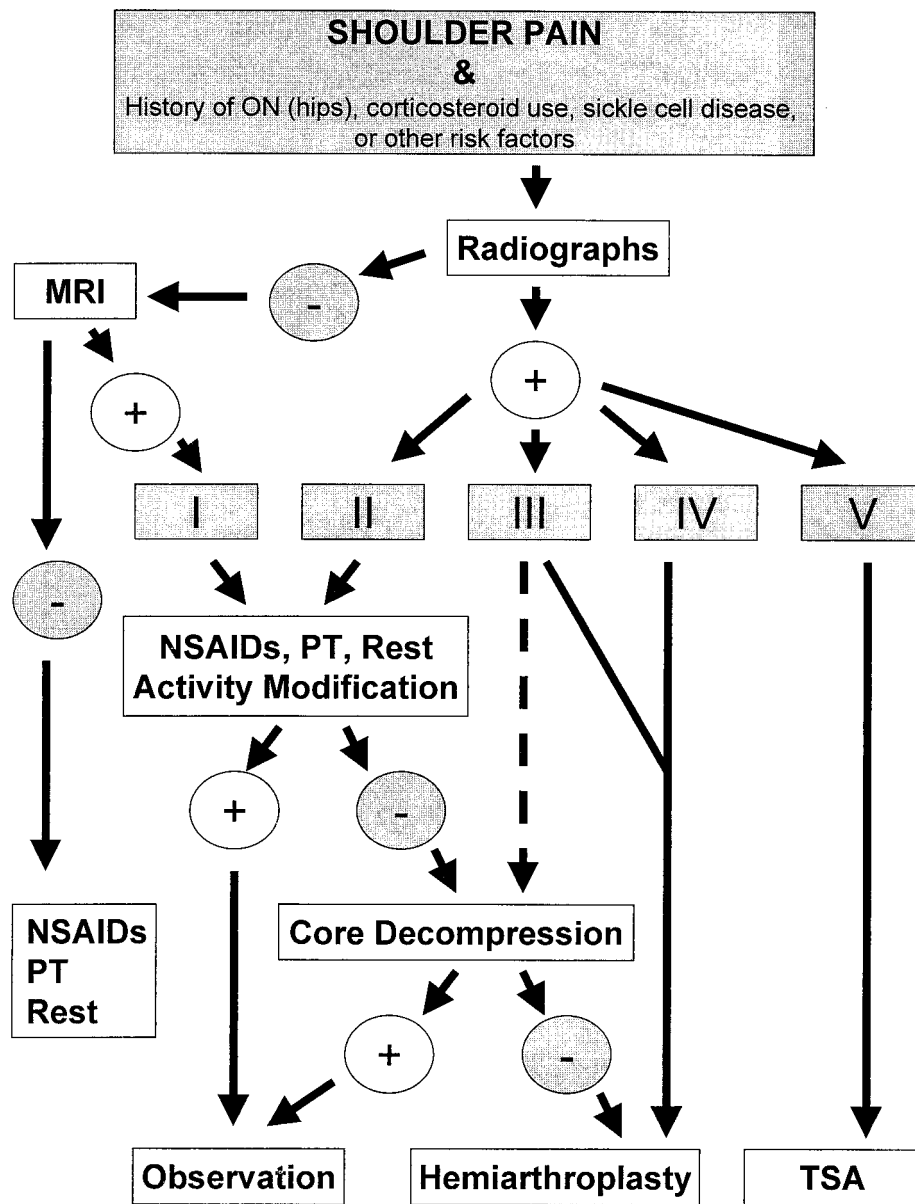


Figure 6 Algorithm for the treatment of nontraumatic osteonecrosis of the humeral head. ON, Osteonecrosis; NSAIDs, nonsteroidal anti-inflammatory drugs; PT, physiotherapy; TSA, total shoulder arthroplasty.

inferior to those in a concurrent group of patients with osteoarthritis.¹⁰⁴

The results of prosthetic arthroplasty for nontraumatic osteonecrosis are generally good, in part because the condition affects neither the integrity of the capsule nor the integrity of the rotator cuff. Furthermore, significant contracture and bone deformity do not occur until late in the disease.^{47,101} These factors

enhance the opportunity for improved shoulder comfort and function.

Complications after arthroplasty for osteonecrosis are infrequently noted and often relate to the underlying systemic condition.⁷⁷ Cruess²³ noted a single infection in his series. Rutherford and Cofield^{15,112} identified rotator cuff tears in 2 patients who underwent total shoulder arthroplasty, one of whom underwent further

surgery. Kay and Amstutz⁶⁷ noted superior instability in 2 of 3 shoulders after hemiarthroplasty. Postoperative brachial plexus injury and the need for additional surgery such as distal clavicle excision have also been noted.^{47,67}

SUMMARY

Nontraumatic osteonecrosis of the humeral head has become recognized as a debilitating, often progressive disease. The success of nonoperative or minimally invasive treatment is predicated on the early diagnosis of osteonecrosis.⁷⁷ A current algorithm for the treatment of nontraumatic osteonecrosis is presented in Figure 6. Nonoperative treatment designed to maintain mobility and comfort is often useful in the early stages of the disease. Core decompression is a minimally invasive treatment that has demonstrated effectiveness for early osteonecrosis, but frequently, patients are not identified in time to benefit from this treatment. Fortunately, when conservative treatment or core decompression fails, humeral head replacement provides excellent pain relief and improved motion and function.

Earlier identification of osteonecrosis should facilitate earlier intervention in the future. Studies are needed to evaluate the efficacy of imaging modalities such as scintigraphy and MRI in diagnosing asymptomatic humeral head osteonecrosis in patients with symptomatic hip or contralateral shoulder disease. Studies are also needed to define the role of prophylactic interventions such as core decompression. A more complete understanding of the underlying pathophysiology and epidemiology should enable an earlier diagnosis of nontraumatic osteonecrosis. In turn, an earlier diagnosis should enhance the outcome of treatments that are less invasive than joint replacement.

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