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Journal of ISAKOS

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Current Concepts Review

Diagnosis and treatment of avascular necrosis of the humeral head: Current concepts

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ARTICLE INFO

Keywords:

Shoulder
Humeral head
Avascular necrosis
Osteonecrosis
Management

ABSTRACT

Avascular necrosis (AVN) of the humeral head is an uncommon clinical entity which can result in significant morbidity for patients. There is a paucity of literature concerning humeral head AVN, which may be due to the relatively rarity of the condition and poorly understood nature. Despite being first described decades ago, the underlying pathophysiology leading to humeral head AVN is still poorly defined. While the staging of humeral head AVN is well described, not much is known about prognosticating factors to predict the eventual course. Most of the management options are based on that of femoral head AVN, and even so, there is a paucity of good quality clinical trials in the literature. This current concepts paper describes what is known about humeral head AVN and proposes a management algorithm to guide clinicians.

Diagnosis and Treatment of Avascular Necrosis of the Humeral Head: Current Concepts

- There are numerous causes of humeral head avascular necrosis (AVN), most commonly involving corticosteroids and haemoglobinopathies like sickle cell disease.
- Humeral head AVN is usually insidious in onset and often presents in the later stages.
- Diagnosis and classification of humeral head AVN relies on plain radiographs and magnetic resonance imaging.
- Management of humeral head AVN depends on the symptoms and stage of disease.

Diagnosis and Treatment of Avascular Necrosis of the Humeral Head: Future Perspectives

- While the classification of humeral head avascular necrosis (AVN) is relatively well described, there should be more focus on elucidating prognosticating factors for future progression to humeral head collapse.
- Good quality randomised controlled trials comparing the efficacy of the various treatment methods, especially for early stage AVN, are needed.

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<https://doi.org/10.1016/j.jisako.2022.11.002>

Received 12 June 2022; Received in revised form 26 October 2022; Accepted 15 November 2022

Available online xxxx

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Introduction

Background

Osteonecrosis is the in-situ bone cell death following disruption to the blood supply [1]. Juxta-articular osteonecrosis can cause arthritis, resulting in significant morbidity for patients [1,2]. The humeral head is the second most common site of atraumatic avascular necrosis (AVN) after the femoral head [1]. To date, there is no clear guideline on the management of this condition.

Blood supply to the humeral head

The blood supply to the humeral head comes from the anterior circumflex humeral artery (ACHA) and the posterior circumflex humeral artery [3] (PCHA). The ACHA, which gives off an ascending anterolateral branch, was thought to be the main blood supply of the humeral head.

In recent years, the PCHA has been recognized as a critical source of blood supply to the subchondral region of the proximal humerus, with recent studies showing that blood supply to majority of the humeral head comes from the PCHA [4–6]. The PCHA runs posterior to the humeral head, giving off branches that enter at the bone–cartilage border and greater tuberosity [3].

The circumflex arteries form an intra-osseous network within the humeral head [4]. Of note, the superior aspect of the humeral head has been found to have the poorest blood supply [3].

Etiology

Traumatic causes of AVN include fractures and dislocations of the proximal humerus [1], with a 10–33% risk of AVN after a fracture [3]. There are also reports of AVN following rotator cuff repair [2,3].

The most common non-traumatic aetiology of humeral head AVN is steroid use (see Figs. 1 and 2). Corticosteroid is given as treatment for a wide variety of diseases [7,8], including Severe Acute Respiratory Syndrome (SARS) [9,10]. While prolonged use of high dose corticosteroids has been correlated with an increased risk of AVN [7], there have been reports of AVN with small doses, such as intra-articular injections [1]. In addition, the diagnosis of AVN post-steroid use is variable, ranging from 6 to 24 months [1,8].

Sickle cell disease is an autosomal recessive haemoglobinopathy where red blood cells deform under low oxygen states, potentially occluding capillaries. The reported incidence of AVN in sickle cell disease is 5.6% [11]. While humeral head AVN secondary to corticosteroids may undergo spontaneous resolution in the early stages, the area of AVN in sickle cell disease tends to increase [12], suggesting repeated bone infarcts from micro-emboli.

Table 1 provides a non-exhaustive list of possible aetiologies of humeral head AVN. The common link is disruption to the humeral head blood supply [13]: Extra-osseous vascular (trauma resulting in transection of blood supply), intra-osseous extra-vascular (hypertrophic adipocytes and/or bone oedema increasing intra-osseous pressure and leading to venous stasis), intra-osseous intra-vascular (intra-capillary fat or air emboli, haemoglobinopathies).

Presentation

Symptoms and signs

Patients with humeral head AVN have an indolent course, with majority already being in the later stages at initial presentation. Unlike the hip, the shoulder joint is not usually a weightbearing joint [45] and the glenoid is less encompassing compared to the acetabulum. Furthermore, the scapulothoracic joint allows for compensatory movement. Nevertheless, symptomatic patients often have a painful click accompanying certain shoulder movements, resulting from joint incongruity or a



Fig. 1. X-rays of Cruess stage III-IV. This 82-year-old male with type 2 diabetes mellitus, hypertension, and STEMI 10 years prior with persistent heart block, presented with left shoulder pain. Ultrasound showed rotator cuff tendinosis. A subacromial steroid injection was performed, which initially relieved the pain. Several years later, he presented with intractable left shoulder pain. X-rays showed sclerosis of the humeral head with flattening of the humeral head and erosive changes. The glenoid is spared, making this Cruess stage III-IV. Acknowledgement to Dr Lim Chee Yeong for providing the images.

cartilage flap [8,46]. This can progress to rest pain and reduced range of motion (ROM), resulting in limitation of daily activities.

Disease staging

AVN often begins at the superomedial aspect of the humeral head. This region articulates with the glenoid when the arm is in 90 degrees of abduction, where the greatest amount of stress is exerted on the humeral head [8]. This correlates with the area of poorest blood supply.

Plain radiograph is effective in evaluating osseous pathology but may be unable to detect the pre-radiographic stage of AVN. Magnetic resonance imaging (MRI) of the shoulder is useful in detecting early AVN and for staging. The most widely known staging system would be Cruess, comprising of five stages (Table 2).

While disease staging helps to guide management, perhaps greater importance should be placed in prognosticating the risk of humeral head collapse for those in earlier stages, so there can be timely and sufficient intervention to minimize progression to later stages. This is critical since patients with humeral head AVN are usually young and those with radiological progression tend to have poorer outcomes [14].

The degree of subchondral involvement on diagnosis may predict the likelihood of future progression to humeral head collapse. Sakai et al. [15] found that if the necrotic angle measured on MRI was greater than 90° at the time of diagnosis, the chance of future collapse was up to 92%, whereas those less than that were unlikely to progress. Similarly, lesions involving less than 15% of the humeral head were less likely to collapse compared to those who had moderate (15–30% of humeral head) or severe (more than 30% of humeral head) involvement initially [7,12].

Aside from the degree of humeral head involvement on diagnosis, the presence of shoulder pain is another possible indicator for humeral head collapse. Between 49 and 60% of asymptomatic patients progressed to humeral head collapse [7], compared to 81–86% of symptomatic patients [7,12].

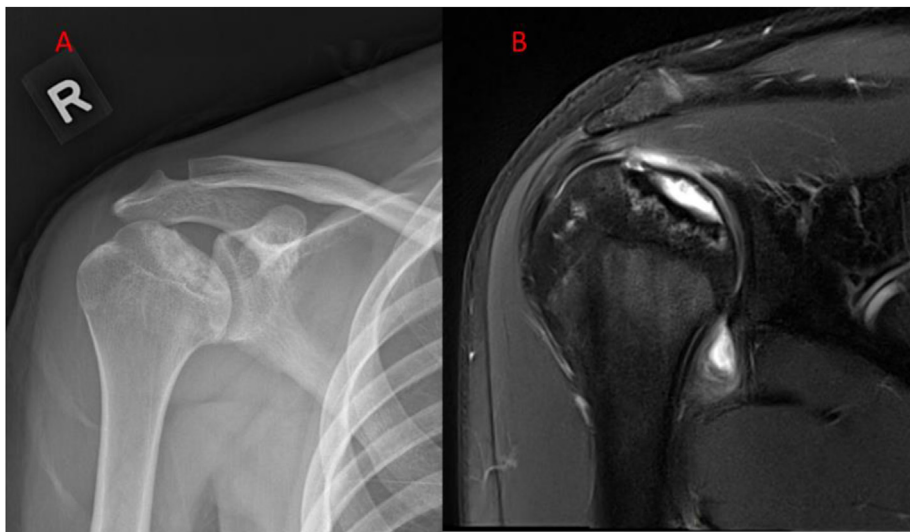


Fig. 2. X-rays and MRI of AVN Cruess stage IV. This patient is a 38-year-old female who presented with pain and weakness of her right shoulder. She had an 8-year history of SLE, with three relapses in the past year. She has been on high dose steroids since her diagnosis. Examination of the right shoulder demonstrated crepitus and weakness of rotator cuff and deltoid muscles. X-rays and MRI (A, B) showed sclerosis and collapse of the superomedial aspect of the humeral head with loss of congruity (Cruess stage IV). Hemiarthroplasty has been offered. Acknowledgement to Dr Lim Chee Yeong for providing the images.

Management

Conservative therapy

Conservative management includes lifestyle modification, avoiding excessive active shoulder abduction and flexion, while preventing stiffness through passive ROM. Modifiable aetiologies such as alcohol and steroid use should be avoided where possible. Analgesia such as non-steroidal anti-inflammatory drugs (NSAIDs) and opioids may be considered for pain control. Conservative management has varying degrees of success, especially with regards to ROM and performing activities of daily living (ADLs) independently [8,16]. The consensus is that conservative management for stage III or more tend to have poorer outcomes [13,17].

Bisphosphonate is a class of anti-resorptive medication that inhibits osteoclast activity, thereby increasing bone density. Bisphosphonates may reduce pain and disease progression in early stage femoral AVN [18, 19]. In contrast, in a study [20] consisting of five patients with early stage humeral head AVN treated with bisphosphonate therapy, 80% progressed within four years. However, the sample size was small, and it remains unclear whether bisphosphonate therapy will be effective in humeral head AVN, especially for stage I.

Surgical intervention

Joint-preserving surgeries include arthroscopic debridement and core decompression with or without bone grafting. Joint-replacing options

Table 1

Etiologies for humeral head AVN.

Traumatic	Non-traumatic
1. Non-iatrogenic <ul style="list-style-type: none"> • Fracture • Dislocation • Fracture-dislocation 	1. Medications <ul style="list-style-type: none"> • Corticosteroids
2. Iatrogenic <ul style="list-style-type: none"> • Rotator cuff repair 	2. Systemic diseases <ul style="list-style-type: none"> • Hemoglobinopathies: Sickle cell disease • Connective tissue/autoimmune diseases: Systemic lupus erythematosus, psoriasis • Metabolic diseases: Alcoholism, Gaucher disease • Hormonal diseases: Cushing disease
	3. Others <ul style="list-style-type: none"> • Dysbarism • Hemophilia (see Fig. 3) • Electrical/thermal injury • Radiation therapy • Septic arthritis • Extra-corporeal shockwave therapy

consist of humeral head re-surfacing, hemiarthroplasty (HA), and total shoulder replacement (TSA).

Arthroscopic debridement

The use of isolated arthroscopic debridement for humeral head AVN is mainly limited to case reports in the literature. Hardy [21] and Hayes [22] have reported doing arthroscopic debridement for stage III and above. Both studies had improved UCLA (University of California Los Angeles) scores and ROM post-operatively. Arthroscopic debridement accords the advantages of low morbidity, rapid improvement in symptoms, and joint preservation. However, its utility in treating humeral head AVN aside from the presence of locking symptoms attributable to loose bodies or cartilage flaps remains to be seen.

Core decompression






Core decompression (CD) aims to decrease intra-osseous pressure and promote revascularization [23] and can be performed open [24,25], arthroscopic-assisted [26] or even percutaneously [27]. CD has achieved good results especially for stage I and II humeral head AVN, where 88–100% [24,25,27] of patients showed good post-operative UCLA scores. In fact, a proportion of patients with early stage AVN who underwent CD showed no radiological progression after 5 years of follow-up. However, the efficacy of CD in the later stages decreases significantly, with good results obtained in only 70% and 14–16% of stage III and IV AVN, respectively. L'Insalata [14] found that CD for stage III did not prevent clinical or radiological progression.

In contrast to steroid consumption, patients with humeral head AVN secondary to sickle cell disease who underwent CD often progressed to stage III and beyond [28]. The proposed explanation is that sickle cell disease is unmodifiable; therefore, repeated vaso-occlusion will undermine any attempts at joint preservation. This is especially so if the patient is homozygous for the haemoglobin S trait (Hb SS) [12,28].

Grafting procedures

Various methods of grafting have been described, such as cartilage allograft [29] or autograft [30,31] to repair the chondral defect, deltoid muscle-pedicled bone graft [35] or vascularized scapular graft [35,29] and bone marrow grafting (often taken from iliac bone crest) [32–34]. The literature is heterogenous and includes stage III and above humeral head AVN [29–32] although most seem to report improvements in pain and ROM. These procedures are often paired with CD to enhance the chances of success. Hernigou et al. found that mesenchymal stem cell therapy injected percutaneously after core decompression was more effective in preventing progression for early stage (I/II) AVN, and the

Table 2
Cruess radiological classification of humeral head AVN.

Stage	Plain radiograph (X-ray)	MRI	Illustration
I	No changes	Hyperintense signal	
II	Sclerosis of humeral head	Hyperintense signal at subchondral bone	
III	Osteochondral lesion (crescent sign) with no collapse of the humeral head	Double contour sign	
IV	Collapse of the humeral head with loss of joint congruity	Same as radiograph	
V	Osteoarthritis of the humeral head, with extension to the glenoid	Same as radiograph	

addition of cell therapy significantly reduced the chance of collapse (10% vs. 74%) over a follow-up period of 7 years [33]. Similar to isolated CD, the addition of bone marrow grafting does not seem to halt radiological progression in patients with sickle cell disease [34].

Arthroplasty

Arthroplasty remains the treatment of choice for humeral head AVN in the arthritic stage (stage IV and V). The main categories for arthroplasty include partial re-surfacing (PS), HA, and TSA. An absolute contraindication to arthroplasty is ongoing infection.

PS involves replacing the defect in the cartilage of the humeral head with a metal implant. There are a few common criteria for PS [35,36], including (1) a well-defined area of cartilage loss rather than diffuse arthritis, (2) area of cartilage loss is not excessively large (size of available metal implant is limited), (3) no glenoid wear, and (4) good bone stock. Bearing these in mind, PS is usually used for stage III or IV AVN. If careful patient selection is done, PS has shown to provide pain relief and improvements in ROM [35–37] at 2.5–3 years follow-up. The need for revision surgery in the reported literature is minimal although the follow-up duration is short. Advancements in re-surfacing implants have resulted in the development of hemi-resurfacing or total re-surfacing, which have comparable results [38,39].

PS presents several advantages due to minimal resection of the native bone stock [35–37,40]. These include preservation of remaining healthy cartilage, maintaining the original biomechanics of the shoulder and easier conversion to other forms of arthroplasty if needed since most of the humeral head and neck remain intact. The operative duration and blood loss are also lower, with reduced risk of intra-operative peri-prosthetic fracture [35,36,40]. However, careful placement of the metal implant is required to prevent any offset in the articular cartilage, which would present an opportunity for the implant to be caught and levered out [40].

HA and TSA are the mainstay of treatment for osteoarthritis secondary to AVN. HA is generally used for stage IV AVN, whereas TSA is performed when there is concurrent glenoid arthritis (stage V). The outcomes between the two procedures are similar [41–43] at a mean follow-up of 8–10 years, providing comparable pain relief and ROM. Glenoid wear is the most common complication following HA, with the need for revision surgery ranging from 2 to 10% [41–43]. Loosening of the glenoid component of TSA requiring revision is a relatively common complication, with a reported incidence of 5–21% [41–43].

The underlying aetiology for AVN may affect the eventual outcomes for TSA. Burrus et al. [44] found that compared to a control group of TSA performed for patients without AVN, TSA performed for traumatic AVN had a significantly higher risk of post-operative complication including infection, dislocation, revision surgery, and stiffness. Similarly, TSA for AVN secondary to steroid use had increased risk of infection, revision surgery, and fracture compared to the control group.

Management algorithm

The advent of MRI allows easier detection of early stage AVN. Therefore, there is an increasing need to focus treatment on earlier

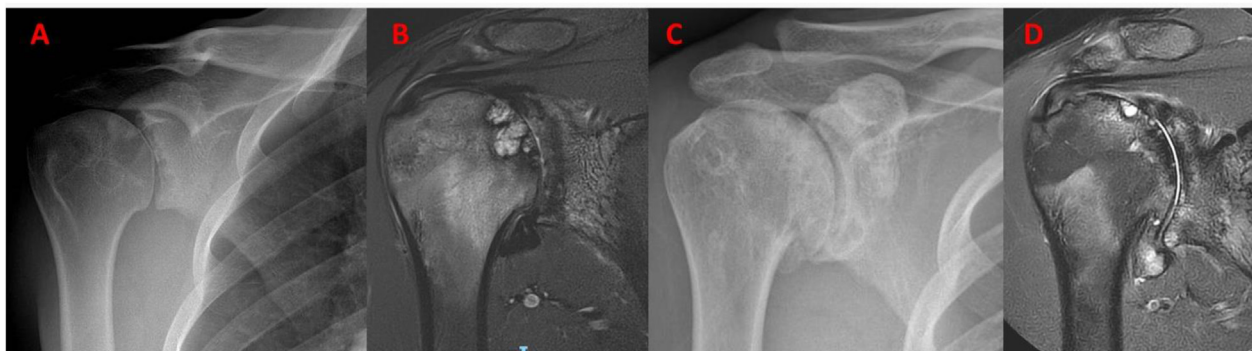


Fig. 3. X-rays and MRI of progressive AVN of the humeral head. The patient is a 31-year-old lifeguard with a background of hemophilia A, presenting with several years of right shoulder pain. X-ray (A) showed subchondral sclerosis of the superomedial humeral head, with subchondral cysts noted on MRI (B) at the same region (Cruess stage II). Core decompression was initially offered but the patient elected to proceed with conservative treatment. Nine years later, the patient's X-ray and MRI (C, D) showed extensive glenohumeral arthritis (Cruess stage V), and despite his age, arthroplasty was offered. However, the patient deferred surgical intervention.

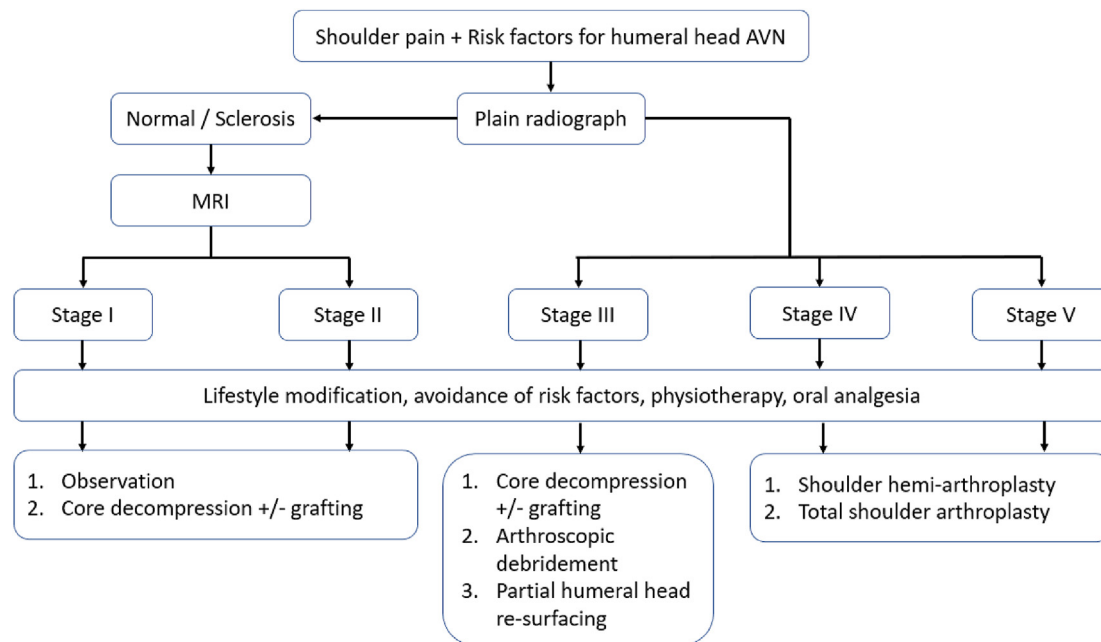


Fig. 4. Management algorithm for humeral head AVN

stages, since patients are often younger. Based on the review of literature, the following management algorithm is proposed (Fig. 4).

The authors suggest that MRI be done for patients with shoulder pain and underlying risk factors for humeral head AVN with unremarkable or early stage AVN radiographs. This allows early stage (I/II) AVN to be detected and some form of prognostication of future progression. Conservative therapy (rest, physiotherapy, analgesia) should be pursued initially. The eventual management will then be guided by the stage of AVN on diagnosis and eventual progression.

For stage I and II AVN, aside from conservative therapy, CD with or without grafting may be considered, especially when there are risk factors such as pain or a large area of involvement on MRI. This is because such procedures may help the patient symptomatically and potentially delay progression to later stages.

Stage III AVN straddles the boundary between early and late stage AVN, as there is disruption in the articulating surface with no gross arthritis. CD with or without grafting may be attempted although it is likely a temporizing measure to delay the need for joint replacement. CD is useful in patients who either wish to avoid a larger surgery such as PS, or have multiple co-morbidities where a smaller operation may be more advisable. Arthroscopic debridement can be performed in the same setting or in isolation if there are identifiable cartilage flaps or loose bodies, that contribute to patients' symptoms. Finally, PS can be considered in patients who meet the criteria described earlier although there is a need to inform patients of the possibility of revision surgery in the future. For patients who do not meet the criteria for PS (for example area of cartilage defect is not well-defined), CD with or without grafting may be more suitable.

The management of stage IV and V AVN is generally not contentious as there is gross arthritis of the glenohumeral joint, with the main differentiating factor being the presence of glenoid arthritis where TSA is usually more suitable. Where possible, HA should be performed patients with stage IV AVN as compared to TSA due to its lower risk for revision surgery.

Overall, patient involvement in the decision-making process is important as patients are young and regardless of the treatment selected, there is always a chance of disease progression or need for additional surgeries. This is especially if there are known risk factors for poorer outcomes such as underlying non-modifiable risk factors like sickle cell anaemia.

Future research could be done to determine risk factors that prognosticate the likelihood for progression to humeral head collapse from

the time of diagnosis. This will allow healthcare professionals to better counsel patients with regards to their expected disease course and the options for treatment. High quality randomised controlled trials should be conducted where possible to compare the efficacy of the various treatment methods.

Conclusion

Humeral head AVN can lead to destruction of the glenohumeral joint and result in significant patient morbidity. The current literature is not sufficiently robust to definitively prove which treatment method is more appropriate for a given stage of AVN. However, with careful patient selection taking into account the patient's symptoms, stage of disease, and underlying aetiology, the authors propose the aforementioned management algorithm to help guide the decision-making process.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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