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Review Article

Uncommon Osteonecrosis - Diagnosis, Management and Treatment - for (Kienböck; Osteonecrosis of Talus; Preiser; and Osteonecrosis of Humeral Head) Suppl II: Overview

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Abstract

Osteonecrosis is a degenerative bone condition characterized by death of cellular components of bone secondary to disruption of subchondral blood supply, traumatic vascular damage.... Also known as necrosis. avascular, it often affects bone growth at weight-bearing joints. Progressive disease can lead to subchondral collapse, threatening the viability of the involved joint. Therefore, early recognition and treatment of osteonecrosis is essential. This activity discusses the etiology, pathogenesis and treatment of the disease, presentation, and treatment options of uncommon forms of osteonecrosis.

Keywords: *Kienböck's Disease; Osteonecrosis; Osteonecrosis of Talus; Osteonecrosis of Preiser; Osteonecrosis of Humeral Head*

Kienböck's Disease

In 1910, Robert Kienbōck, an Austrian dowser, published radiographic findings of the collapse of the lunate bone, and he named it "lunatomalacia" [1]. In 1920, Müller first observed that repetitive microtrauma from occupational activity can cause Kienböck's disease (KD). The disease is considered to be multifactorial and several mechanical, anatomical and vascular factors are thought to contribute to the pathogenesis of KD.

To date, many hypotheses have been proposed for the etiology of KD over the past 100 years KD or avascular necrosis of the carpal bone lunate is an uncommon disease in young adults. Irisari., *et al*. described two components of KD in children as infantile and adolescent lunatomalacia. Neonate subgroups have been seen in children under 12 years of age with a good prognosis with prudent management. Juvenile lunatomalacia affects children over 13 years of age through skeletal adulthood. Careful treatment leads to good results in the early stages of juvenile molluscum contagiosum. Surgical procedures, usually afferent shortening, may be required to treat severe episodes. Like common KD, the disease is more common in the elderly than in manual workers but may be different in etiology. The elderly group has a low frequency of negative ulnar variance and KD is more common in elderly women than in elderly men. Functional outcomes without surgery have been reported to be good or excellent even in advanced Kienbōck disease in these patients. This may be related to low functional needs in the elderly.

Root cause

Mechanical factors

In 1928, Hulten observed a relationship between the negative ulnar variance KD. [2]. He described an abnormal transmission across the radiosynaptic joint in a negative ulnar variance, which then predisposes to stress fractures in the lunate bone. However, D'Hoore, Nakamura, Stahl S., *et al*. did not find a causal relationship between negative ulnar variance and KD [3,4]. In a meta-analysis, it was revealed that there were insufficient data to support an association between negative ulnar variance and Kienbōck disease. Antuna-Zapico observed a trabecular pattern in different lunate bones (Figure 1).

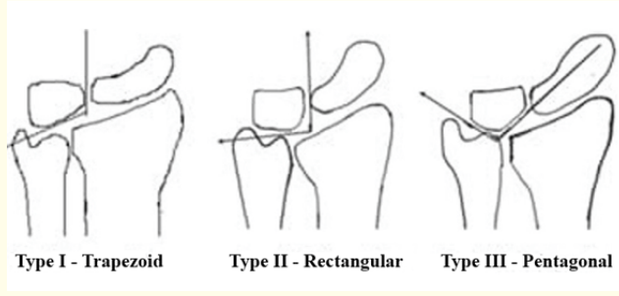

Figure 1: *Antuna-Zapico types of lunate morphology.*

Xiong., *et al*. investigated the trabecular pattern in normal and stage III KD lunates using computed tomography [5]. They detected bony disruption in the palmar or dorsal regions of the distal articular surfaces of diseased tumors because of the compact trapezoidal bone in the central region compared with weaker dorsal and sternal regions. This could be the reason for the rupture and ultimately the collapse of lunate in KD.

Vascular factor

Early studies by Cordes and Stahl suggested a single lunate supply vessel by the foot or dorsal vessels. Lee observed the *in vivo* blood supply pattern of the lunate bone by microscopy and the Spalteholz preparation. He describes three main patterns of blood supply. In 66.5% of cases there was a dual supply of lunate by the palmar and dorsal vessels connecting the intraosseous vessels. In 7.5% of cases there was an unvascular dual blood supply in the bone.

Recently Kim., *et al*. showed wrist position-dependent perfusion of lunate using ultraselective angiography of the radial, central duct, and transverse arteries [6]. They found that maximal blood flow in the neutral wrist position decreased when the wrist extensor muscle contracted. They concluded that prolonged flexion or extension of the wrist in the presence of pain or in splinting, occupation may contribute to the pathogenesis of the disease.

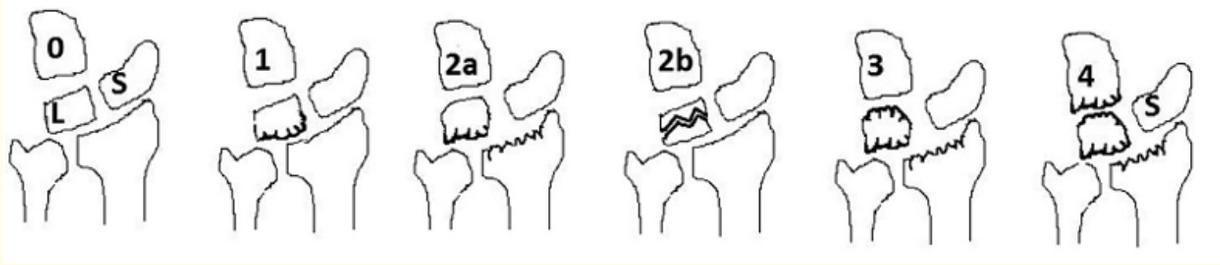

Figure 2: *Bain and Begg arthroscopic classification.*

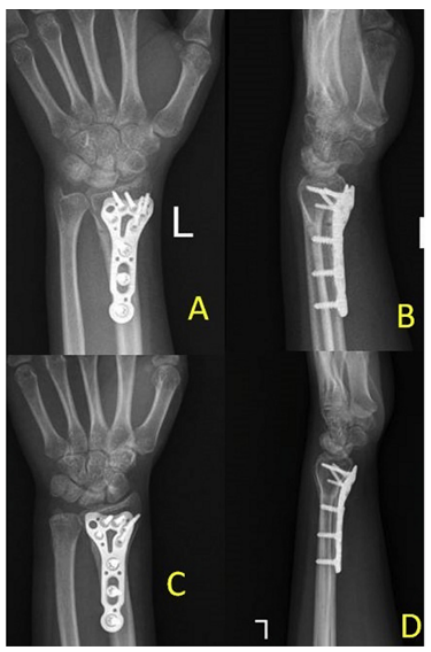

Figure 3: *Immediate postoperative radiographs (A and B) of a 23 years female shows stage II Kienb€ock's disease treated with radial shortening osteotomy. After a 1 year follow-up, PA radiograph (C) and lateral radiograph (D) showed resolution of sclerosis and restoration of lunate height.*

3. Classification

The earlier classifications (Stahl (1947) were based on simple radiographic features [44]. In 1977, Lichtman., *et al*. revised Stahl's original classification and described the four-stage classification (I-IV) of KD [7]. They then introduced stage 0 to represent intermittent ischemia or stress of the lunate bone in MRI negative subjects and stage IIIC in cases of lumbago fractures in the corolla plane regardless of lumen morphology and wrist instability [8].

Lichtman classification [8]:

- Stage 0- Intermittent ischemia
- Stage I- Normal x-ray with positive bone scan
- Stage II- scattered sclerosis on radiograph with normal shape and size
- Stage III- Collapse of A-lunate with wrist alignment and height preserved
- Stage IIIB- Wrist collapse with fixed scleral rotation, quasi-arrested migration
- Stage IIIC- Rupture of the lumen (coronary plane)

- Stage IV- Radiculoarthritis of the wrist.

Bain and Begg proposed a classification of KD based on arthroscopic findings of articular surfaces without lunate function [9]. They determined that the functional joint surface had a normal sparkling appearance with hard bone upon exploration (Figure 2). A nonfunctional joint surface is defined as having any of the following: excessive vibration, cracking, local or widespread cartilage loss, and raised or fractured articular surface. Bursitis is not considered indicative of the extent of Kienbock disease.

Arthroscopy classification Bain and Begg [9] (Figure 2):

- 0 Normal joint surface
- 1 Proximal surface of unusual lunate 2A- Proximal surface of lunate and radius of irregular lunate.
- 2B- Fracture of the frontal bone.
- 3 Lunate fossil radii and proximal and distal surface of the unusual lunate.
- 4 Lunate fossil radii and proximal and distal surfaces of lunates and proximal surfaces of irregular lunates.

Schmitt., *et al*. classification description based on gadolinium-enhanced MRI to characterize the pattern of osteonecrosis in different parts of the lunate bone. It consists of the proximal necrotic zone, the medial comparator region, and the distal normally present lunate bone. Based on signal intensities in different lunate regions on MRI, Schmitt., *et al*. classified the three stages of Kienbock's disease.

Schmitt and Lanz MRI samples [10]:

- N- Normal signal
- A- Marrow edema with intact spine
- B- Early pulp necrosis with fibrovascular-vascular comparison tissue
- C- Necrotic bone marrow with collapse.

Stress ischemia (axial loading of the wrist in an extended wrist) followed by Gadolinium-enhanced MRI will show Schmitt pattern and Lanz type A (bone marrow edema). It is recommended in patients with transient (stage 0) lunate ischemic attacks with negative radiographic or MRI findings.

Treatment algorithm

More recently, Lichtman and Bain proposed a newer classification for Kienbōck's disease that considers the Lichtman osseouse, vasculare Schmitt, and Bain classifications [10]. The current classification considers the patient's age, the condition of the lunate and wrist disease, the surgeon's ability, and the patient's lifestyle preferences. Although comprehensive, the current classification provides new insight into future directions of treatment.

Age

Patients at a younger age have a different natural history than adults. The good revascularization potential and choroidal regenerative potential in skeletally immature patients preclude surgical management as first-line treatment:

- A1 < 15 years of conservative treatment with immobilization.
- A2 16 - 20 years of age apply joint balancing procedures in the absence of response to nonsurgical management of symptoms > 3 months. Most frequent radial shortening osteotomy describe. However, there is a risk of an overgrowth following the procedure. Alternatively, an epigenetic test of distal radius can also be performed.
- A3 > 70 years of age consider surgical management if symptoms persist for more than 6 months From, according to section B or C.
- B1: Lunate intact and lunate protection.

During this period, all articular surfaces of the lunate bone were functional on arthroscopy (Bain 0), without any features of collapse (Lichtman stages 0, I and II) and good perfusion on gadolinium enhanced MRI (Schmitt stage A).

Lunate unloading procedures

Radial shortening osteotomy is the classical technique described to offload the lunate bone by reducing the mechanical forces across it. It is indicated especially in case of ulnar negative variance or neutral. This procedure is well accepted in reducing pain and in improving the function, range of wrist motion, and grip strength. Good results have been reported by combining radial shortening with revascularization procedure. Although reported results have been encouraging in terms of long-lasting symptomatic relief after radial osteotomy, unable to change the natural course of the disease. A systematic review showed that radial osteotomy did not alter the radiological progression of Kienbōck's disease compared with nonoperative treatment in terms of the Lichtman stage but found to be superior in terms of clinical outcomes. Botelheiro., *et al*. have shown long term good clinical results recently with radius shortening osteotomy in Lichtman IIIB stage and concluded that advanced disease with carpal collapse should not be considered as a contraindication to carpal sparing radial shortening osteotomy (Figure 3).

There is a risk of DRUJ incongruity after shortening or wedge osteotomy of the radius. Various modifications have been described later on.

Very distal radius wedge osteotomy is a newer modification where osteotomy is done with its apex distally contrary to convention closing wedge osteotomy. It reduces the radial inclination angle and increases the lunate covering ratio without affecting DRUJ congruity.

Selective shortening wedge osteotomy of the radius (Camembert osteotomy) facing the lunate can be done. They decompress the lunate bone without decompressing the scaphoid as seen in whole radius shortening osteotomy. It is performed in conjunction with Sennwald's ulnar shortening osteotomy in cases with positive ulnar variance or if there is distal radioulnar joint (DRUJ) discontinuity after shortening.

Treatment of Kienbōck's disease (Table 1).

Osseous (Lichtman)	Vascular (Schmitt)	Principle	Technique
0 I, II, IIIA	A, B	Unload Radiolunate	Immobilization, Joint leveling procedures Radial shortening osteotomy, Ulnar lengthening, capitate shortening
		Revascularization	Vascularized bone graft core decompression, Radius Metaphyseal core decompression (MCD)

IIIB	B	Unload Radiolunate Radial shortening osteotomy Lunate excision+Bypass disease column/or proximal shift of capitates (restore lunate function) fuse disease column	Radial shortening osteotomy Lunate excision Bypass disease column/or proximal shift of capitates (restore lunate function) SC fusion, STT fusion capitate osteotomy and transposition fuse disease column RSL fusion proximal row carpectomy
IIIC	C	Lunate Salvage Lunate excision Luate excision+Bypass disease column/or proximal shift of capitates (restore lunate function) fuse disease column	Internal fixation Lunate replacement and interposition (tendon, silicon, pyrocarban prosthesis) SC fusion, STT fusion capitate osteotomy and transposition RSL fusion proximal row carpectomy
IV	C	Wrist Salvage	Wrist arthrodesis, wrist arthroplasty

Table 1: *Treatment of Kienbōck's disease.*

Metaphyseal core decompression (MCD) of the radius is an induction-based extrapolar curettage of myocardium from the distal radius of the physiological fracture healing response that enhances wrist perfusion.

Lunate forage is a minimally invasive option based on the concept of reducing intravascular hypertension involving endoscopically assisted clavicle drilling combined with bursectomy.

Vascular bone graft - Differential vascular graft 4 + 5 elongated compartment arteries (ECA) Petiole-based bone graft, Second or third metacarpal base bone graft, free vascularized pelvis or pisiform graft or grafts from the described volar radius procedures for intact revascularization 4+5-chamber elongation artery-based bone grafts (ECA), commonly used because of the wide pedicle diameter than. Vascular bone grafting has also been described in combination with a temporary trapezoidal stapler with a k-wire to bypass mechanical stress through the lumen until it becomes vascularized.

B2-Lunate compromised (Lichtman stage IIIA, Schmitt stage B and Bain grade 1)

Lunate reconstruction is the treatment of choice in this stage of proximal articular pole of the lunate.

Vascular trochlea femoral transplant: This is particularly helpful in cases of proximal lunate necrosis. A free femoral graft from the femur can be used to replace the proximal necrotic articular surface of the lunate bone.

Near-aligned lens cutting (PRC)

PRC is an alternative procedure that can be performed provided that the joint surfaces are of lunate radius and duty cycle. This procedure is relatively contraindicated for inpatients under 35 years of age and who still wish to engage in demanding activities. While it's technically easier, it does carry the risk of developing arthritis between the head and the radius of the capitol.

B3-Lunate not reconstructable: Lunate salvage (Lichtman stage IIIC, Schmitt stage C and Bain grade 2b)

Lunate excision and its replacement with ribbed, silicon, pyrocarbon, polyethylene-based prosthetics have been described. Titanium lunate arthroplasty (TLA) has also shown promising long-term results. If the junction of the caps and lunate faces is functional, PRC can be performed.

Recently, fractures of the auricle have been saved by internal fixation provided that the coronal shell is intact in the absence of fracture, sclerosis or collapse and without radiation arthritis and incapacitated joints on computed tomography.

C1-Combined center column coupling (Lichtman stage IIIA or C, Schmitt stage B and Bain grade 2a, 3 or 4)

- C1a- Compromised Radiolunate Joint: If the joint is incapacitated, radiocapholunate can be performed at this stage.
- C1b - Peripheral radiating joints and wrist medial joints: With the involvement of both radiolunate and capitolunate coupling, scaphocapitate fusion can be performed but the prerequisite is that the radioactive coupling is intact.

C2-Intact carpal-arthritis (Lichtman stage IIIB, Schmitt stage B and Bain grades 2-4)

With further collapse and degeneration of the central column, the joints of typhoid are initially functional. Therefore, encapsulation is a good surgical option (Figure 6). In addition, arthritis can also be treated by tomography (STT)).

Lunate resection, osteotomy, and its proximal transposition in conjunction with the wrist fusion (Graner procedure) have also been described for advanced disease.

Recent changes include vascular capillary transposition with a combination of iliac or distal radial grafts in lieu of transpositional heads with good results in stage III.

C3-Unrecoverable wrist (Lichtman stage 4, Schmitt stage C and Bain grade 4)

When the joint is collapsed or further degraded, it is not possible to reconstruct the wrist and chiropractic or chiropractic can be performed depending on the patient's needs.

What can the surgeon provide (D) and what does the patient want? (E) and working setting, different surgical options can be offered to patients with their pros and cons. Therefore, surgical options should be offered according to the patient's needs and activities.

Author's preferential treatment

The treatment algorithms proposed by Lichtman and Bain are very comprehensive and useful. We try to follow the above mentioned treatment protocol with our own modifications. At presentation, we assessed the following: (1) intensity of pain (2) patient's active range of motion (3) how it affected the patient's lifestyle (3) disease duration (4) patient age (5) ulnar variance and (6) radiographic classification as suggested by Lichtman classification. The disease was explained to all patients and possible future procedures. Very young children (under 15 years of age) were placed with Paris Plaster (POP) for 8 - 12 weeks and they were reevaluated. Usually, these children relieve pain. They rarely require surgery, if the pain persists or the stage of the disease worsens then surgery is recommended. Radial shortening in the positive or neutral variance of ulnar is the most common, recommended procedure for these patients, regardless of disease stage. The outcome of this surgery ranges from good to excellent in these patients. In the young (20 - 40 years), all patients were advised to have surgery and wrist arthroscopy was included as an essential tool for these patients. Patients with severe pain have

bursitis around the lunate, which reacts with dead bone. Wrist arthroscopy not only helps to evaluate the articular surface of the carpal bones, but bursectomy is performed uniformly in all cases. Removal of the bursa around the lunate and luno-capitate areas provides early relief. Patients with early presentation and Lichtman stage 1 or 2 with positive ulnar variance are recommended afferent shortening after performing arthroscopic carpal bursectomy. The radiographic findings can be variable and sometimes, the lunate's blood vessels return (Figure 3). In the literature, the best result is radial shortening in cases of positive variance ulnar. If there is a negative ulnar variance, then shorten as specified. The ICSRA-based 4.5 graft procedure was reserved for stage 1 Lichtman patients with neutral or negative ulnar variance (Figure 4). Vascular osteotomy requires violation of the wrist capsule and sometimes this leads to fibrosis and limited range of motion of the wrist. We do scapho-capitate pinning with a temporary K-wire to unload the lunate. Pins will be removed after four months. Usually, in patients with limited range of motion, they have a careful prognosis, regardless of the procedure performed. These patients often present late and they are already in the advanced stages of the disease.

Osteonecrosis of Talus

Osteonecrosis of the talus is difficult to diagnose and treat because the anatomical position of the henna means it is obscured and its blood supply is precarious. Osteosarcoma of the nail is not always clinically symptomatic, and the patient should be observed until revascularization and consolidation are completed. There is no consensus on the pathophysiology or natural course of the disease. Many surgical treatments and techniques have been tried, but very few long-term results have been published.

The incidence of talar osteonecrosis is increasing with the increasing incidence of high-energy trauma. Damage to the talisman requires great force; Falls from significant heights and motor vehicle crashes are the main causes of this condition. Survivors of a high-speed motor vehicle crash often have distal extremity injuries. The use of highly developed imaging techniques has led to an increase in the number of patients diagnosed with early nail lesions.

Provides anatomy and blood vessels

Understanding the unique anatomy of the nail and its blood supply is important for understanding osteonecrosis of the nail. The direction of the neck is different from the direction of the body in both horizontal and inclined planes. In the horizontal plane, the neck moves in an intermediate direction with deflection. In the sagittal plane, the neck is deviated. This complex shape can make it difficult to accurately determine the reduction on radiographs. The nail has seven joint surfaces, accounting for nearly 60% of its surface, and fixing with screws using the anteromedial method is complex (Figure 4). These nails are most stable in the mortise of the dorsiflexion because it is wider anteriorly than posteriorly. The bone is depressed due to flexion at the base of the nail, which is the most common site of nail fracture, especially during increased flexural strength with axial loads.

Figure 4: *Schematic drawings showing the important anatomic features of the talus. A: Posterior view, B: Inferior view, C: Lateral view, D: Superior view, E: Medial surface of the talus.*

The talus has no tendons or muscles. The entire blood supply comes from several methods of direct vascular insertion, and understanding each contribution is important to avoid iron-induced vascular injury. The posterior tibial, posterior tibial, and iliac arteries are the three major accessory arteries supplying the scapula (Figure 5). Posterior tibial artery contributes the main blood supply of the Talus stem through the tarsal and deltoid arteries. The carpal tunnel artery arises from the posterior tibial artery in the deltoid ligament below the medial tibia, and it passes between the sheath of the biceps muscle and the flexor hallucinogen to enter the tarsal canal. The deltoid artery, which passes between the deep and superficial deltoid ligaments and arises near the origin of the carpal tunnel, is an important source of extracorporeal circulation to the base of the nail. Preservation of the deltoid artery is important during stabilization or reduction of the neck and trunk of the nail. The dorsal sinus artery is formed from the anatomical loop between the dorsal pedicle and the peritoneal arteries and it fuses with the carpal tunnel artery. Together these arteries feed most of the neck and tip of the nail.

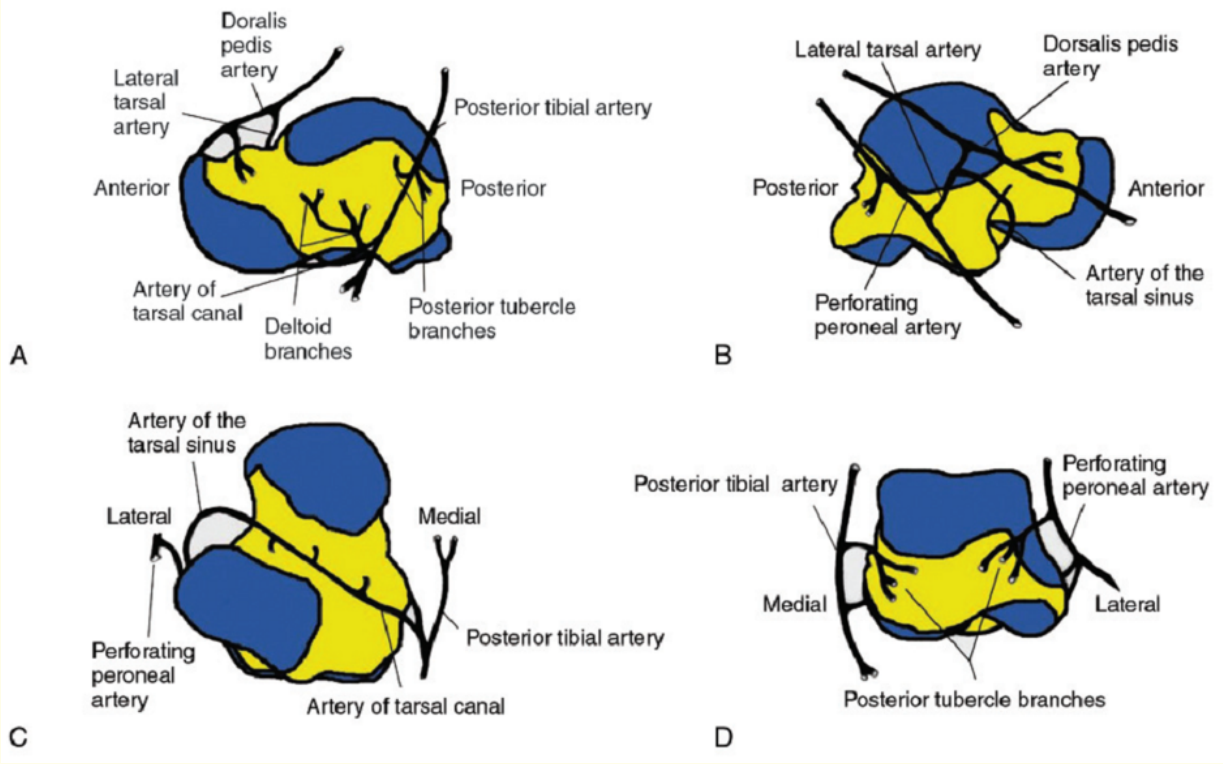

Figure 5: *Schematic drawings showing the blood supply of the talus. A: The medial talar blood supply. The first branches of the posterior tibial artery are the posterior tubercle branches. More distally, the posterior tibial artery gives off the tarsal canal artery with its deltoid branches. This artery courses through the tarsal canal. B: The lateral talar blood supply. The lateral tarsal artery connects the dorsalis pedis artery to the perforating peroneal artery and branches to form the tarsal sinus artery. C: The inferior talar blood supply. The tarsal sinus artery and the tarsal canal artery form an anastomotic loop within the tarsal canal. D: The posterior talar blood supply. The posterior tubercle branches of the posterior tibial artery and perforating peroneal artery supply the medial and lateral tubercles.*

Etiology and incidence

Osteoma of the nail has three main causes. Approximately 75% of patients have a history of trauma including neck or body fractures. Fifteen percent of patients had a non-traumatic medical condition as well as a history of steroid use (regardless of dose or duration of use). Some of these patients were alcohol dependent, sickle cell disease, dialysis, hemophilia, hyperuricemia or lymphoma. The remaining 10% of patients have idiopathic nail necrosis with no identifiable traumatic or medical cause.

The Hawkins grading system for talus neck fractures classifies future risk of osteonecrosis based on fracture displacement and joint correspondence. The risk after breaking roof of Taluy type I is 10%; after fracture type II, nearly 40%; and after type III fracture, about 90%. Type IV implies the development of osteonecrosis to a greater extent than type III. Talismanic trunk and neck fractures were not significantly different in the risk of development.

Death of hematopoietic cells, capillary endothelial cells and adipocytes can usually be confirmed microscopically 1 to 2 weeks after circulatory injury. Fat cells release lysosomes that acidify the tissue, the osteoclasts begin to shrink and the water content of the bone increases. As a result of bone breakdown, fat saponification or bone replacement occurs, that is, the gradual replacement of necrotic tissue with new bone-forming tissue, followed by bone formation. Without the ability to repair itself, the vascular disordered bone eventually collapses, appears patchy, and hardens. This process is accelerated by micro-additives, which can occur during unprotected weight bearing with shock.

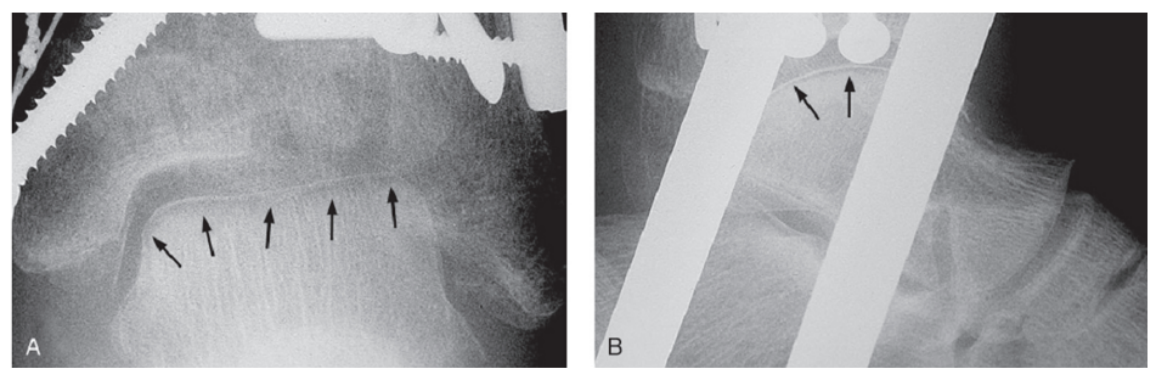

Figure 6: *The Hawkins sign in a woman who had undergone external and internal fixation of a complex pilon fracture. Mortise view (A) and lateral (B) radiographs of the ankle reveal striking subchondral radiolucency (arrows), indicating talar viability.*

Symptoms and diagnosis

Pain is the most common symptom of Talar bone necrosis and is closely related to the loss of joint integrity. Before the joint surface is collapsed, the patient may have no symptoms. With bone necrosis under the cartilage, collapse under the cartilage can occur due to lack of structure support against pressure from body weight on the joint surface. This string is considered a fracture under cartilage, and it can cause pain as well as mechanical symptoms.

Although MRI and bone scanning are useful to detect early taluy bone necrosis, the assessment should start with simple ankle-shaped ankle. Early changes in sclerosis, cystic changes and progressive changes associated with epidural collapse can be seen on simple X -ray

films. The Hawkins signs can provide evidence of vascular re -vascular and are thought to be a reliable initial sign of the ability of blood vessels to exist, with few false negative results (Figure 6). Hawkins is a contrast strip under the cartilage of the foundation can be seen on the AP X -ray film of ankle from 6 to 8 weeks after fracture and on the X -ray film on the next 10 to 12 weeks later When broken. The collapse under the cartilage often has no symptoms, and rarely detects damage to the pure X -ray film in the early stages of fungal bone necrosis. MRI is considered a major diagnostic tool in the first stage. MRI is used to diagnose and quantify the degree of bone necrosis because its sensitivity to fat cell signal changes. The bone marrow is mainly composed of fat components responsible for strong T1 images, and bone marrow necrosis accompanied by edema then an early part of bone necrosis. Artericated materials show the density of water, revealed with high signal intensity on T2W image. MRI can also be used to check the advanced stages of Talar bone necrosis. To minimize signal interference, use titanium screws to fix the taluy roof break. Titanium implants are preferred than stainless steel implants because their magnetic properties are not magnetic. Scanning of Technetium TC 99M is also helpful to diagnose the early stages of Talar necrosis; Often it is made from 6 to 12 weeks after fixing the fracture of the taluy roof and shows a reduction in the absorption in the taluy roof. X -ray detections are often used with ficat and arlet classification systems to determine the degree of talar bone necrosis (Table 2).

Nonsurgical treatment

Many treatments for Talar osteonecrosis have been described, but some long -term or important results have been published, and there is no consensus on the best treatment. Non -surgical treatment is given priority for Talar osteoarthritis in Ficat and Arlet phase I, II or III. Some of the initial studies found benefits to avoid carrying weight until the circuit was completed. Most people who are not treated (are defined as not weighing less than 3 months).

Stage	Radiographic Appearance
I	Normal
II	Cystic and sclerotic lesions Normal talar contour
III	Crescent sign Subchondral collapse
IV	Narrowing of the joint space Secondary changes in the tibia

Table 2: *The Ficat and Arlet classification of the radiographic appearance of talar osteonecrosis.*

The number and time of weight should be determined for each patient based on the location of the lesion and its symptoms. There is no need to limit the ambition to carry weight if there is still enough bone structure to support weight. [59 11] However, regardless of weight, it is important to preserve ankle motion, especially bending and expanding, and protecting the ankles from the tension Varus and Valgus using braces Tailed tea cakes or walking machines (Figure 4).

The use of oral or intravenous bisphosphonates can be tested for patients with osteoarthritis. [60]. Bisphosphonates are anti -infection agents that inhibit the impact of adult bone cells on the bone, thus changing the balance between the reabsorption and bone deposits to allow more deposition. Bisphosphonates seems to temporarily stimulate the proliferation of Pro-Osteoblast cells, increasing their differentiation, increasing the production of osteoprotegerin protein against cancer against bone cells and reducing edema at the position of the disease. Osteoporosis. Although bisphosphonates have been widely used for the bone patients of the femur, the use of them for talar osteoporosis is outside the brand and controversial.

Ultrasound bone stimulants can also be used to regenerate bone low -intensity ultrasound that have been found to enhance the differentiation of the bone of the medium stem cells, stimulating the difference and the difference and the difference The proliferation of bone activity, inhibits the activities of osteoarthritis, and improves blood. And speed up the healing of stress cracks.

Surgical treatment

Core extract

The increase in pressure in the bone area is thought to be the result of edema related to cell death. This repair process causes additional damage by increasing local compartment pressure and inhibiting vascular reinhibition. The purpose of the core extract is to reduce the core pressure, use many drilling processes and thus enhance the re -virtue. Extract is also effective in relieving pain.

The exercises with a diameter of 1.5 to 4 mm are often used to extract talk lesions. Two to 10 holes are performed on bone damage using a traditional rear method, one side or intermediate method, depending on the location of the lesion.

Bone transplant

Autograph bone transplant method can be used before the rescue process is considered Autografts without blood vessels from the pelvic tops are the most widely used bone pieces, followed by Allograft. Autograph's advantages compared to the Allograft include biological compatibility and alliance quickly; Disadvantages include pain and limitations of sponsor's website on the quantity and size of the graft bone. Self-bone transplant is usually done with the removal or scraping of the necrotic area (Figure 7).

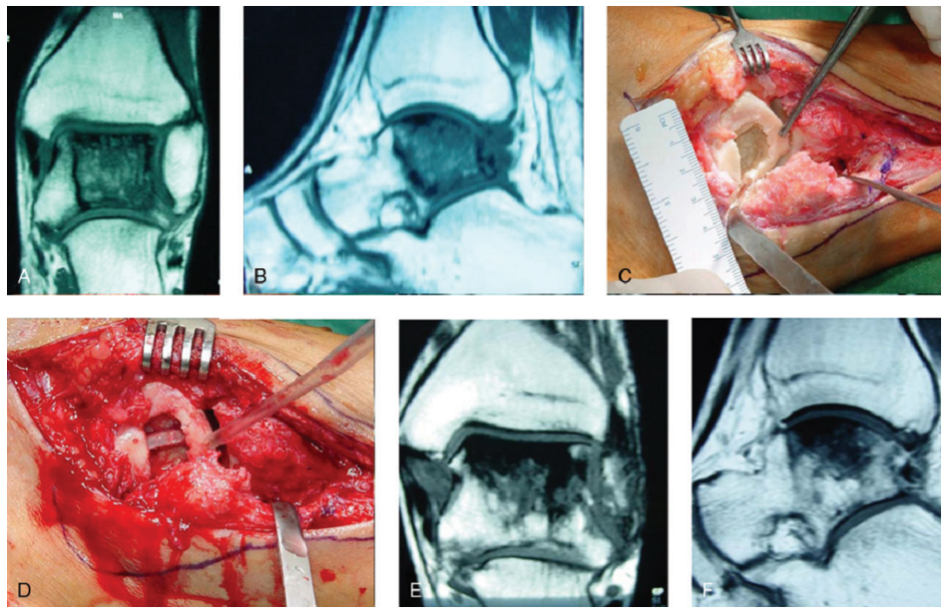

Figure 7: *A and B: Preoperative MRIs showing wide osteonecrosis, which was revealed with low signal intensity. Intraoperative photographs showing removal and curettage of necrotic tissue. (C) And insertion of autologous tricortical bone block and cancellous bone grafting. (D), (E) and (F) MRIs taken at 2-year follow-up show a well revascularized talus.*

If the lesions are large and mainly related to the joint surface, the allograft structure can be a good choice. Allograft osteochondral can be reviewed a part of the joints in a young patient with localized damage (Figure 8).

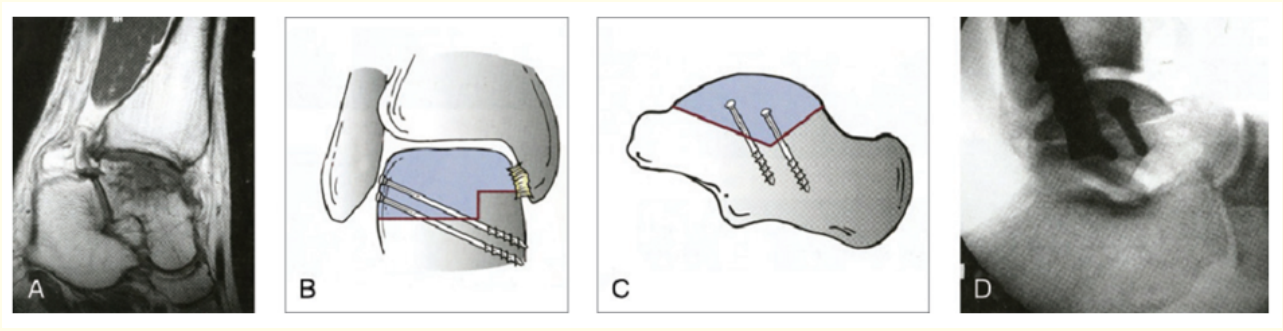

Figure 8: *Segmental osteochondral allograft resurfacing for an ankle with focal talar osteonecrosis. A: Preoperative MRI showing large segmental involvement. B and C: Schematics showing preoperative planning for partial joint arthroplasty. D: Lateral radiograph showing placement of prepared allograft with screws from a transmalleolar approach.*

A piece of blood vessel bone graft from the pelvis has been used to regenerate blood vessels in a 16 -year -old patient with osteoarthritis after injury. The tongue graft can also be used, although this technique is required in each specimen, the horizontal pedal of the nearby Tarsal artery and provides shapes, about 4.1 cm long and can be Medial Malleolus (Figure 9).

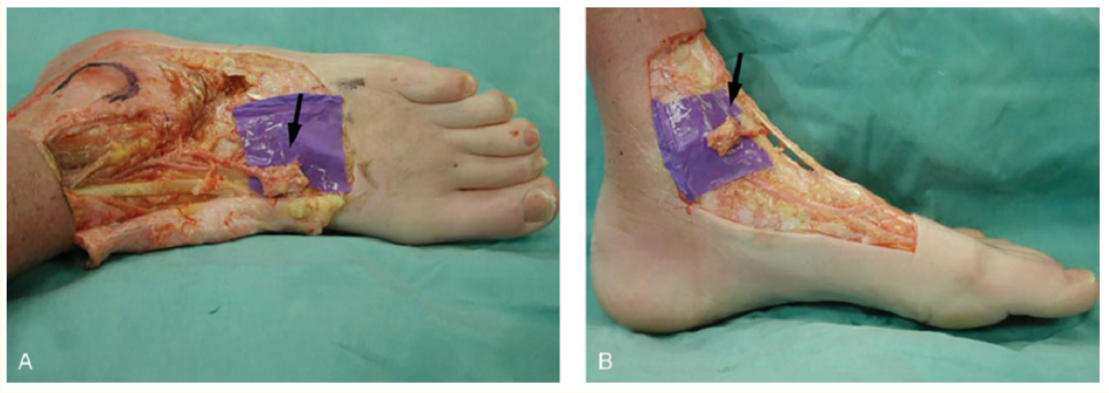

Figure 9: *Photographs showing the transverse pedicle branch of the proximal lateral tarsal artery with the cuboid pedicle (arrow) (A) and its range (arrow), which can be rotated even to the medial malleolus (B).*

Arthrodesis

Arthrodesis can be considered as one of the final options if other treatments are unsuccessful or in patients with Talar osteoporosis phase IV. The arising under the skin was used in an effort to push blood vessels into Talus and thus avoid osteoporosis and patients later with the fracture and collapse of the Talar body.

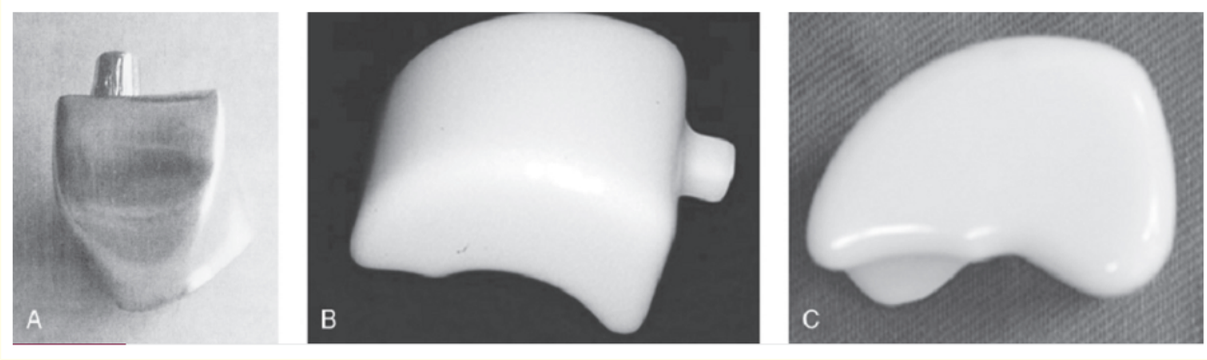

Figure 10: *Photographs showing a stainless steel talar prosthesis (A), a first-generation ceramic talar prosthesis with a peg for an intact talar neck (B), and a second-generation implant without the peg (C).*

Large cannulated screws, blades, locking sheets, locked sticks or non -Multan sterilized substances have been widely studied for the synthetic ankle reaction in patients with Talar osteoarthritis. The technical selection should be based on the patient's condition, the location and scope of the lesions, and the hobby of the surgeon.

Mini opening technique, similar to laparoscopic technique, uses a small wound expansion at the same position as an endoscopy portal. Curettage or removing necrotic area during ankle joint can cause shortening. The risk is at least in the early stages of osteoporosis if the area is small and shallow.

Talar body parentheses and total ankle arthroplasty

A fake Talar body has been used in an effort to avoid the disadvantages of joints or surgery to remove all in a large area of osteoarthritis and to save Talus function. The difficulties of the use of the Talar body fake have shrink, loosen, unstable due to the loss of ligament attachments, inaccurate prosthetic shapes and costs. Fourteen of 16 patients with favorable results related to pain and function after receiving the Talar body fake part made of stainless steel (Figure 10A). A fake part of Alumina ceramic and the second -generation ceramic body has been designed (Figure 10B and 10C). In the following fake part, the latch to fix on the neck of the alive Talus has been removed to avoid the loosening found with the first generation false part, which seems to be the result of concentrated tension. The total number of ankle joints for a patient with Talar osteoporosis was first reported more than 35 years ago. A few published studies are available, all reporting unsatisfactory results as well as some modifications to Arthrodesis [12] (Table 3). Arthrodesis is a treatment for end -stage talar osteoporosis. The surgeons rarely use all ankle endoscopic surgery in Talar bone patients because of their poor bone invasion and lack of supportive bone structure.

Study (Year)	Number of Patients	Follow-Up (Years)	Implant (Manufacturer)	Results
Newton (1982)	3	3	Scandinavian Total Ankle Replacement (Small Bone Innovations)	Collapse in two patients, conversion to fusion in one Persistent pain in one patient
Buechel., *et al.* (1988)	2	2	Buechel-Pappas (Endotec)	Complex regional pain syndrome in one patient
Buechel., *et al.* (2003)	2	5	Buechel-Pappas	Collapse in one patient Complex regional pain syndrome in one patient
Takakura., *et al.* (2004)	2	2	TNK Ankle (Japan Medical Materials)	Collapse in both patients, conversion to fusion

Table 3: *Studies of cementless total ankle arthroplasty for talar osteonecrosis.*

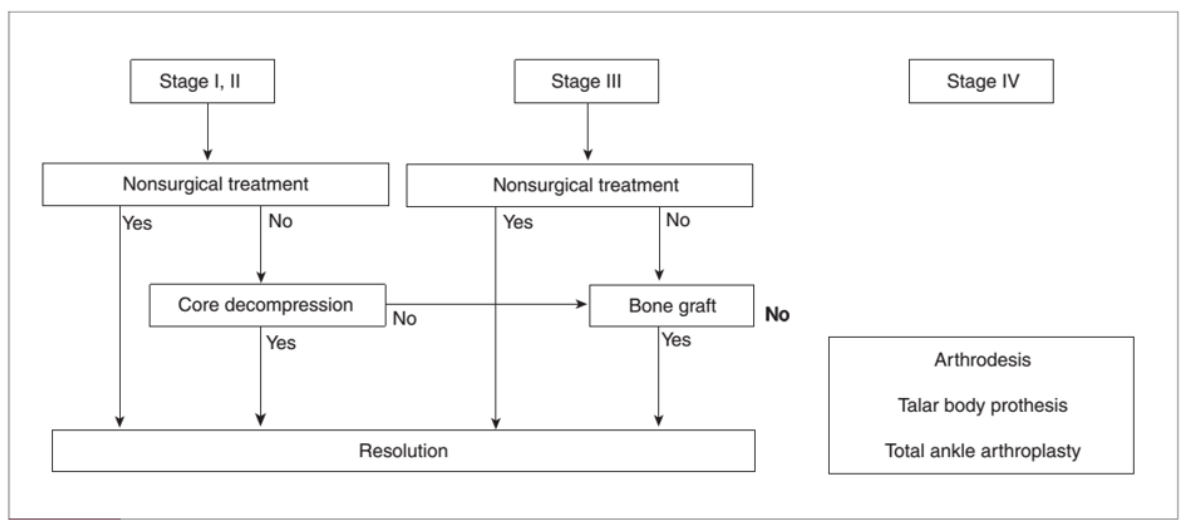

Figure 11: *Treatment algorithm for osteonecrosis of the talus.*

Treatment algorithm

The treatment of Talar bone is marked with a lot of controversy and less consensus. The proposed algorithm starts with the use of ficat and ARLE classification (Figure 4). Successful treatment is determined to relieve pain or terminate joint collapse. An area of necrotic expansion on MRI or sclerosis expanding on a simple X -ray does not mean failure of treatment. If the collapse of the joint surface has been stopped, even with the progression of necrosis, the surgeon may wait for the vascular re -ventilation and resolve the damage. Non -surgical treatment is completely indicated for patients with osteoarthritis stage I or II. If not effective, the core extract may be considered or bone grafting. A patient with stages of stages of stages with the collapse of the joint surface will not benefit from the core extract; Bone grafting is the only possible procedure. A rescue procedure like Arthrodesis can be done if there are joint changes.

Talus's osteoporosis is not a common disease, but if not treated, it can lead to Talus collapse and progressive pain with joint changes. After fracture of the neck neck, up to 90% of patients may be at risk of osteoarthritis, depending on the fracture model, because the vascular structure is vulnerable to the damage around the Talar bone. Specifically, osteoarthritis in a young patient can lead to end -stage arthritis that requires joints or later joint surgery in life. Only a few comparative results of Talus bone treatment have been published. Careful assessment and treatment decision are extremely important. X -ray should be used with CT, bone scanning or MRI, this is the most useful image method for the disease. Determining the circuit of circuit using bone scanning or MRI is essential to determine the best treatment plan. Before the collapse of the joint surface, non -surgical treatment is designed to protect the joints to the limited axle, which should be used until the circuit is re -connected. For a patient who has undergone unsuccessful surgical treatment or the collapse of the dome, the treatment options may include core extract and bone transplant. A rescue procedure should be considered the final method or for a patient with joint changes in the final stage.

Preiser Disease

AVN of paratyphoid has been reported in patients with no history of significant trauma but complaining of worsening afferent wrist pain and spontaneous onset. Physical examination reveals pain around the dorsal side of the wrist and swelling around the wrist joint.

To date, although the etiology and pathology of this disease have not been determined, idiopathic vascular insufficiency of the wrist and repetitive mechanical stress on the wrist are considered to be its causes. Most cases have been associated with systemic steroid use for a systemic disease, such as autoimmune hemolytic anemia, systemic lupus erythematosus, or kidney transplantation. Another possible etiology is the use of topical steroids for a localized disease such as deQuervain's disease. In addition, many cases are associated with risk factors such as smoking, alcoholism, and/or infections.

Preiser first used the term sterile squamous gangrene in 1909. In 1910, Preiser described five people with a history of wrist injury, who later developed aseptic gangrene. In 1911, during a congress in Hamburg, Preiser [13] was attributed to the loss of blood vessels in the fascia due to trauma that ruptured the vessels inside the fascia ligament as he was unable to identify any signs of a fracture. Some have suggested that Preiser's cases were caused by ichthyosarcoma fractures that were not apparent using the lower resolution radiographs available in the early 1900s.

Patients with Preiser's disease present with wrist pain with or without a history of wrist injury. The examination is likened to that of Kienbock disorder: wrist pain, swelling, loss of wrist motion, and reduced grip strength.

Vascular anatomy/biomechanics

Regardless of the title used to describe typhoid, the underlying pathological process of an inadequate blood supply for typhoid remains the common denominator. How typhoid loses its blood supply is still less controversial and at best speculative. In 1980, Gelberman and Menon [14] suggested that the AVN of the proximal pole of post-traumatic typhoid may be due to the vascular pattern that supplies typhoid. In postmortem studies, they demonstrated that 70% to 80% of proximal psoriatic individuals receive their blood supply from radial arterial branches that enter through the spine (Figure 12). Waist or proximal pole fractures can certainly disrupt this single vascular supply to the proximal choroids and eventually lead to AVN. In a follow-up study, Panagis and colleagues grouped carpal bones into categories based on the size and location of the nutrient vessels, the presence of internal vessels, and the number of bones that depend on a vessel. nutrition. Group I carpal bones, including the carpal bones, have a large area dependent on a single vessel with few internal vents.

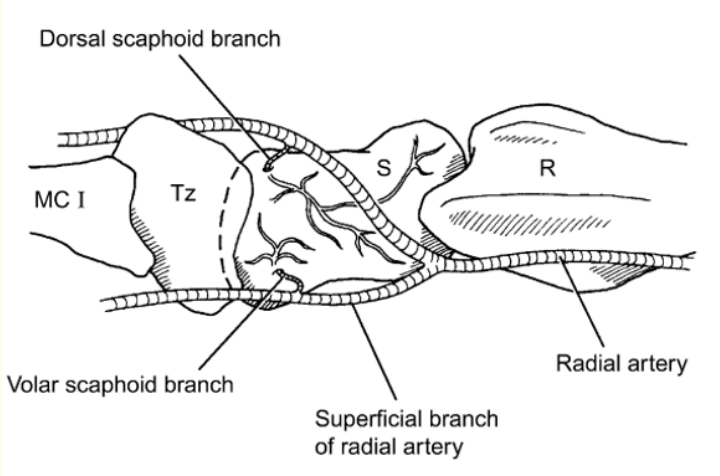

Figure 12: *Illustration of the scaphoid demonstrating that the main blood supply enters on the dorsal and radial aspects. MCI: First Metacarpal; R: Radius; S: Scaphoid; Tz: Trapezium. (From Trumble TE. Principles of hand surgery and therapy. Philadelphia: W.B. Saunders; 1999. p. 94; with permission).*

Acting as a mechanical link between the proximal and distal wrist rows, the choroid is important for the maintenance of normal wrist kinematics (their details are beyond the scope of this article). To maintain wrist relationships, fish scales have 5 joints (trapezoid, trapezoid, cap, lunate, and distal radius). Thus, despite its small size, this carpal bone is almost entirely covered with cartilage (Figure 13).

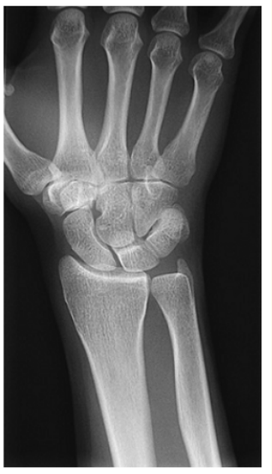

Figure 13: *Plain radiograph demonstrating the five articulations of scaphoid: trapezium, trapezoid, capitate, lunate, and distal radius.*

This correlates with the previously mentioned findings of Gelberman and Menon, who found only two entry perforations at ligament attachment sites not covered by articular cartilage.

Further support for the hypothesis that the idiopathic osteosarcoma of typhoid is derived from surgical pathology is the fact that typhoid is a load-bearing bone with little space to allow nutrient vessels to enter. The spaces available for the entrance of blood vessels, areas without hyaline cartilage, are often shared by ligamentous attachments. Importantly, Taleisnik and Kelly [15] have noted that some of the vessels leading to the choroid follow pathways consistent with the carpal ligaments. Based on these findings, many authors have speculated that repetitive trauma and microtrauma to surrounding wrist structures may lead to vascular disruption and Preiser's disease in susceptible patients. In fact, Herbert and Lanzetta [16] hypothesized that a certain proportion of patients must receive some blood supply through the scapholunate ligament complex (SLIL) (Figure 14). From clinical observations, these authors noted ''many cases'' in which proximal fragments persisted when their only remaining attachment was to SLIL. In contrast, the authors also reported patients with proximal extremity necrosis after SLIL interruption.

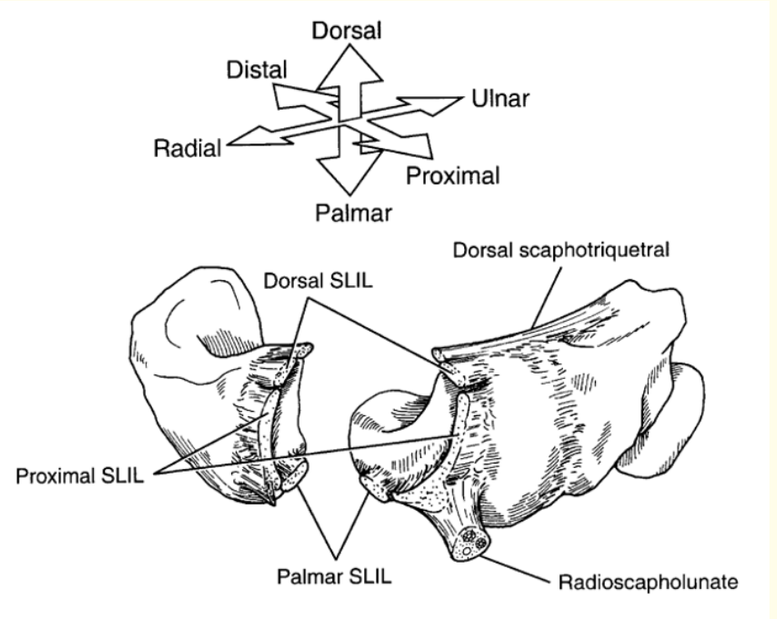

Figure 14: *Illustration demonstrating some of the ligamentous attachments of the scaphoid. Note the three segments of the SLIL, including the strong dorsal portion and the weaker membranous and palmar sections. This ligamentous attachment site may represent an area through which some patients receive blood flow.*

Authors have also investigated wrist loading characteristics, specifically ulnar variance, and its relationship to Preiser's disease (Figure 15). Herbert and Lanzetta [16] noted that seven out of eight of their patients who had idiopathic avascular necrosis of the scaphoid had ulnar positive variance, whereas studies by Kalainov and colleagues [17], Vidal and coworkers [18], and De Smet [19] found no relationship between variance and idiopathic osteonecrosis of the scaphoid. It should be noted, however, that from the report by Herbert and Lanzetta [16] it is difficult to ascertain how the radiographs were taken and how variance was measured. Thus, at this time the data remain inconclusive regarding ulnar variance and its effect on scaphoid vascularity.

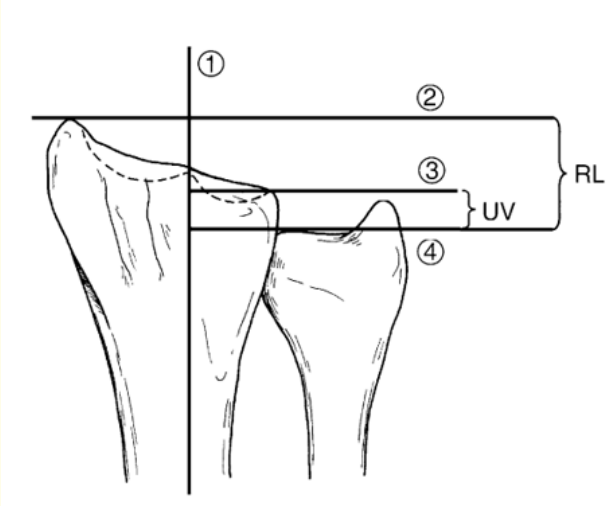

Figure 15: *Ulnar variance is measured as the distance between lines 3 and 4. When the ulna is shorter than the radius it is termed negative ulnar variance. Positive ulnar variance occurs when line 4 is more distal than line 3. RL: Relative Length of the Radius; UV: Ulnar Variance.*

Etiology

The etiology of PD is hypothesized to be related to repetitive trauma, steroid use, chemotherapy, trauma, connective tissue disorders, and alcohol intake, all of which are thought to be potentially disruptive blood supply to typhoid patients. Ulnar variance does not appear to be a risk factor. Schmitt., *et al.* [20] using contrast-enhanced MRI, discovered a "3-layer region" for PD: the proximal pole joined first, followed by the lumbar and finally the distal pole.

The lack of a consistent definition for PD has added to the difficulty of studying this rare entity. In the literature, PD is referred to interchangeably as "avascular necrosis of paratyphoid". "Idiopathic idiopathic typhoid necrosis" and "idiopathic typhoid necrosis in the absence of obvious fractures". Preiser [13] reported 5 cases, all of which had prior significant wrist trauma, 3 of which subsequently developed radiographically visible fishbone fractures [13]. Attempts to distinguish between traumatic AVN and idiopathic AVN may be futile, as the proximal AVN of typhoid fever from undetected and healed fracture is indistinguishable from idiopathic AVN. Because all of the early cases of Preiser were associated with fractures or prior trauma, Ferlic and Morin proposed defining PD as traumatic avascular necrosis, although this is not yet universally accepted Pebble. In addition, PD is extremely rare, with reports limited to case reports or a small disease series; Consequently, there is still no consensus on optimal diagnosis and management, as well as a lack of understanding

of the natural history. Based on their clinical experience, Sokolow., *et al*. [21] believe that stages 1 and 2 of the Herbert and Lanzetta classification [16] last 1 or 2 years, and stage 3 lasts 2 to 8 years, followed by the start of match changes.

Diagnosis

By definition, a patient with Preiser's disease will have no history of significant wrist injury or prior fracture or surgery; however, patients will frequently report a history of prior wrist hypertension or heavy labor. Clinically, the vast majority of patients will present with dull, increasingly severe pain. The pain is often present for months to years before manifesting itself, and it is often localized to the radial aspect of the wrist.

On examination, some patients will experience mild to moderate swelling on the dorsal or radial side of the wrist. This swelling is often aggravated by increased load and use of the wrist. Range of motion and grip strength will generally be reduced, often severely, when compared with the lateral wrist, which is probably normal. As reported by Kalainov., *et al.* [17], the disease occurs about two-thirds of the time in the dominant wrist. Although Preiser's disease almost exclusively affects adults, there is one case report of a 10-year-old boy who developed exudative and necrotic plaques with no history of trauma or steroid use.

Image analysation

Standard plain radiographs may be normal or show changes ranging from patchy sclerosis to fragmentation with atelectasis and radicular inflammation. Changes can include all or only part of the scanned image. These investigators suggested that the changes in avascular necrosis of secretory squamous cells progressed in 4 stages: stage I: radiologically normal, bone computed tomography positive; stage II: increased density of the proximal pole, systemic osteoporosis; stage III: proximal pole fragment with or without pathological fracture; and stage IV: wrist collapse with osteoarthritis.

If the radiograph is normal, a bone scan can help identify problems with the radial carpels; however, bone computed tomography cannot adequately differentiate between tumors, AVNs, or fractures in terms of temporal morphology.

Furthermore, after a positive bone scan, the patient is often sent for an MRI evaluation, which not only identifies the location of the pathology on imaging but also determines the nature of the pathology. Therefore, the authors avoided bone scans and went straight to MRI (Figure 16). MRI is a fundamental step in the diagnosis of Preiser's disease. In the early stages of the disease, there is a decrease in signal intensity in the T1 sequence that is indicative of osteonecrosis. In the later stages of the disease, altered T1 signaling is due to bone remodeling. This finding may lead to the false impression that the entire wound is necrotic.

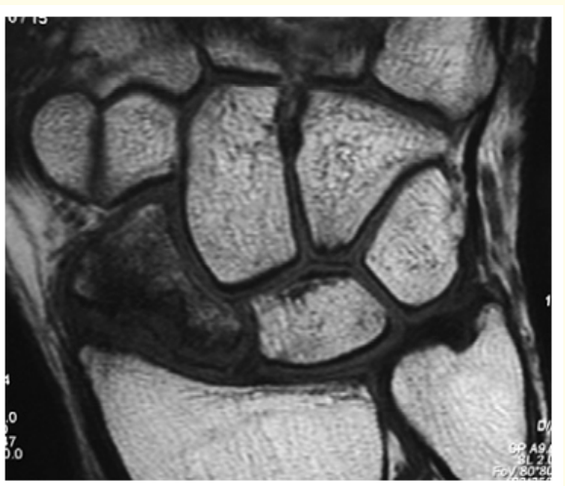

Figure 16: *Coronal section of an MRI demonstrating diffuse necrosis of the scaphoid in a patient with Preiser's disease.*

Bone scan

The use of bone scanning is mentioned throughout the literature on Preiser's disease. As in most conditions, it is a sensitive, but non-specific, imaging modality. It has been superseded by the availability of magnetic resonance imaging (MRI).

Tomography

The use of computed tomography can improve the detection of bone fragments, cysts, and deformities. The addition of arthroscopy can help detect sternum fractures.

Classification

The most commonly cited radiographic classification for Preiser's disease is the 4-stage classification proposed by Herbert and Lanzetta [16] in 1994. They reported 8 patients with "aseptic necrotizing typhoid fever" apartment". In this series, all cases involved proximal portion only. Cases were radiographically classified as: (1) normal radiograph with positive bone scan, (2) proximal densitometry, (3) proximal slim and (4) collapsible wrist pattern. For early-stage disease, the authors suggest conservative treatment or vascular bone grafting to potentially change the course of the disease.

In 2003, Kalainov., *et al*. [17] classified patients into 2 categories, based on MRI features, in a retrospective study of 19 patients. Type 1 has diffuse meningitis involvement on MRI and has a worse outcome and increased deterioration on radiographs, whereas type 2 has limited involvement at the proximal pole and a more favorable outcome. In addition, type 1 cases were often associated with steroid use (not statistically significant), and type 2 cases had a statistically higher rate of prior wrist injury. However, the clinical significance of this classification is unclear.

Treatment

A variety of treatment options have been proposed for early-stage PD, including immobilization, cortisone injections closed coronal cartilage resection and non-vascular and vascular bone grafts [17].

Moran., *et al*. [22] reported 8 cases of PD treated with vascular bone grafting. The three patients with stage 2 and 5 were stage 3 according to the Herbert and Lanzetta classification, and all were patients with diffuse type 1 ischemia as defined by Kalainov., *et al*. This procedure provides analgesia and incomplete revascularization of the proximal pole as assessed on MRI.

In 2011, Hayashi., *et al*. [23] reported 4 cases of PD who were treated with a closed cleft palate. Two patients were stage 2 and stage 2 was stage 3 according to the Herbert and Lanzetta classification [16]. This procedure resulted in significant pain relief in 3 out of 4 patients. However, the longest follow-up time was 1 year. It is not clear why recirculation occurs when the radial meniscus is closed.

Lenoir., *et al*. [24] reported 10 new cases and reviewed the literature on 126 cases in 29 articles. Nonsurgical treatment is reported to be ineffective in the early stages and the disease is nearly always progressive, while vascular bone grafting appears to be effective in the early stages.

For end-stage disease (stages 3 and 4), treatments reported in the literature include arthroscopic excision of the exudate, replacement of inert, flexible silicone elastomers [16] and excision. proximal row [17], scabbing with and without quadriceps meniscus [17] and total carpal vertebrae [17]. Menth-Chiari and Poehling [25] reported excellent analgesia with arthroscopy alone for a case of stage 3 PD, with no worsening at 30 months of follow-up. De Smet., *et al*. [19] reported 4 cases of PD treated with proximal row resection, of which 3 had "excellent" outcomes and 1 required wrist surgery.

Serum sickness is characterized by osteoclast psoriasis, and multiple treatment options have been described in the literature. However, few advances have been made in the past decade in the understanding of PD. The etiology, natural history, diagnosis and optimal treatment of this entity remain unclear. A specific and uniform definition for PD that would facilitate the study of these unusual cases is essential for further investigation. A multicenter study looking at not only treatment but also natural history would be interesting. Without understanding the natural history of this entity (spontaneous resolution versus progression to fragmentation), treatment options may not be better than placebo.

Osteonecrosis of Humeral Head

Osteonecrosis of the tip of the bone is an uncommon condition that occurs when the vascular system is impaired. Vascular insufficiency may be related to arterial or venous circulation of the humeral head. Osteoma of the humeral head occurs at approximately the same rate as in the knee but lower in the hip. After the hip, osteonecrosis of the humeral head is the second most common site of noncancerous osteonecrosis. However, only a few studies have been published regarding head osteonecrosis since the report of Heimann and Frieberg [26] in 1960 and Cruess., *et al.* [27] in 1968. Etiology Multifactorial is characterized by loss of bone blood circulation, which eventually leads to cell death. This circulatory disruption may be of traumatic or traumatic origin. Posttraumatic osteonecrosis usually occurs after a pharyngeal fracture. Here, we focus on atraumatic disease and provide an evidence-based approach to the diagnosis and treatment of osteonecrosis of the humeral head. Despite the clear differences, the mechanism, assessment, and management of femoral head necrosis have been studied mostly in relation to femoral head necrosis.

Humeral head blood supply

The major external and internal blood vessels supplying the humeral tip have been investigated, and an ascending branch of the anterior circumferential artery at the anterior aspect of the humeral head has been identified to provide a consistent supply to the humeral tip. This ascending branch of the anterior circumference, termed the arcuate artery by Laing [28], enters the proximal ganglion at the superior end of the bifacial sulcus or by its branches entering the larger and smaller narrow ducts. adjacent. Once the arcuate artery becomes abnormal, it will pursue a postoperative tortuous process just below the perineum. The humeral tip is also perfused, to a lesser extent, by a posterior circumferential artery (Figure 17).

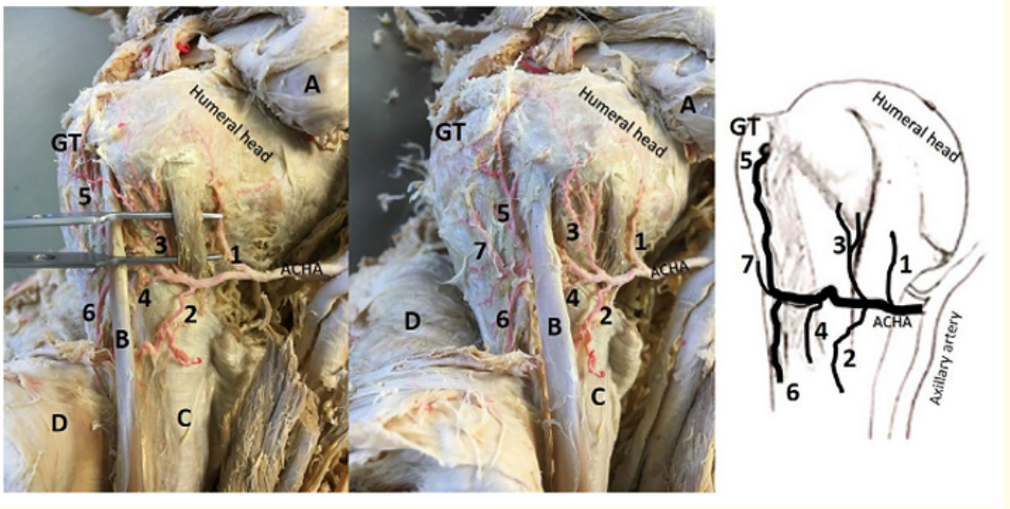

Figure 17: *Anterior view of same left proximal shoulder (second image: slightly rotated) with exposure of the branches of the anterior circumflex humeral artery (ACHA; ethics approval: University of Pretoria 70/2017)-most common course, red latex injected. A: Reflected pectoralis minor; coracobrachialis and short head of the biceps brachii; B: Long head of the biceps brachii tendon; C: Latissimus dorsi tendon; D: Reflected deltoid; GT: Greater tubercle; Pre-tubercular branches-1: Branch that supplies inferior glenohumeral capsule, subscapularis and possible lower part of lesser tubercle; 2: Medial descending branch (latissimus dorsi tendon and lower long head of biceps tendon); 3: Medial ascending branch (enters medial edge of intertubercular groove); 4: Branch in intertubercular groove. Post-tubercular branches-5: Anterolateral (lateral ascending) branch; 6: (Lateral) descending branch; 7: Possible "transverse branch".*

Because the arterioles in this area zigzag, rotating 180 times before returning to the visceral circulation, the blood supply to the subchondral bone is highly susceptible to thrombus formation and embolism. Overall, the existence of a single major artery and the tortuousness of the subchondral arterioles make the vascular system of the humeral head particularly susceptible to trauma and thromboembolic events.

Etiology and pathogenesis

The origin of humeral head necrosis may be posttraumatic or nontraumatic. Although the presentation and pathogenesis are different, both share a common pathway that leads to disruption of the articular head blood supply and subsequent para-articular death.

Traumatic

Traumatic conditions, such as fractures, dislocations, or shoulder surgery, can lead to lateral head necrosis when the outflow of the lateral cephalic artery is disrupted, including when it enters the bone through the lateral vertebrae. Arterioles and capillaries are smaller and in the endothelial canal. Anatomical neck fractures of the humeral head put the circulation of the head at risk. Among all proximal hip fractures, quadriceps fractures had the highest incidence of osteonecrosis, ranging from 15% to 30%, possibly due to disruption of both coronary artery thickness. anterior and posterior (i.e. the distance between the fracture and the articular surface of the head) results in less mechanical stress during healing and a smaller area of revascularization. Furthermore, the surrounding soft tissue of the humeral head is well-vascularized, providing better healing in the hip.

Corticosteroids

Corticosteroid treatment is the most commonly reported cause of noncancerous osteonecrosis. Osteonecrosis after cortisone induction was first reported in 1957 [29]. Since then, several investigators have reported a series of cases of osteonecrosis associated with corticosteroid use. Although the correlation between corticosteroids and osteonecrosis is well established, the pathogenesis remains controversial. Two hypotheses have been proposed to explain the mechanism by which changes in fat metabolism following corticosteroid administration may lead to osteonecrosis. One hypothesis is that an increase in the size of fat cells in the cartilage leads to increased pressure in the cartilage, which in turn may lead to ischemia causing osteonecrosis of the femoral head.

Alcohol abuse

Alcohol abuse has been reported to be associated with osteonecrosis of the humeral head in 6% to 39% of non-traumatic cases. The pathogenesis appears to be similar to that of corticosteroids, in that fatty vessels produced by the liver block vessels in the subchondral bone. Changes in the bone marrow following alcohol abuse can also lead to venous stasis, increased pressure, and bone death.

Hemoglobinopathies

Several hemoglobinopathies are common causes of osteonecrosis, especially sickle cell disease. The pathogenesis is mainly related to embolization, but deformed erythrocytes also seem to cause microvascular foci in the subchondral bone. Higher hematocrit leads to higher blood viscosity and increases the risk of osteonecrosis. In addition, bone marrow hyperplasia due to chronic hemolytic anemia can increase bone marrow pressure, leading to osteonecrosis.

Caisson's disease (incest)

Caisson's disease, also known as diver's disease or decompression sickness, occurs in deep-sea divers or those who work in compressed air [30]. The pathogenesis appears to be vascular occlusion, embolism, and ischemia by air bubbles, although rapidly expanding nitrogen may cause secondary damage to adipose tissue and atelectasis.

Gaucher disease, systemic diseases and other risk factors

The accumulation of lipid-rich Gaucher cells in the bone marrow can either increase intramedullary pressure or exert direct pressure on blood vessels, leading to osteonecrosis. Osteonecrosis is also associated with other systemic diseases, such as lupus erythematosus, rheumatoid arthritis, and Cushing's syndrome. Other risk factors include chronic dialysis, chemotherapy, hyperlipidemia, myoedema, pancreatitis, pregnancy, electric shock, radiation, and thermal trauma.

Stage/classification

The stage of osteonecrosis is important because treatment and outcome are largely determined by the severity of the disease. X-rays are often used to determine the advanced stage of the disease. The most frequently used staging system for osteonecrosis of the shoulder is that of Cruess [27] who modified the Ficat and Arlet system [31] to classify osteonecrosis of the femoral head. This system is based on progression to collapse, from stage I (no radiographic changes) to stage V (head collapse and arachnoid involvement) (Figure 18). In cases where staging is difficult due to non-standard imaging findings, arthroscopy may be helpful. MRI has been shown to fail to identify some of the soft tissue tears and subsequent osteoblastic degeneration noted during the arthroscopic investigation.

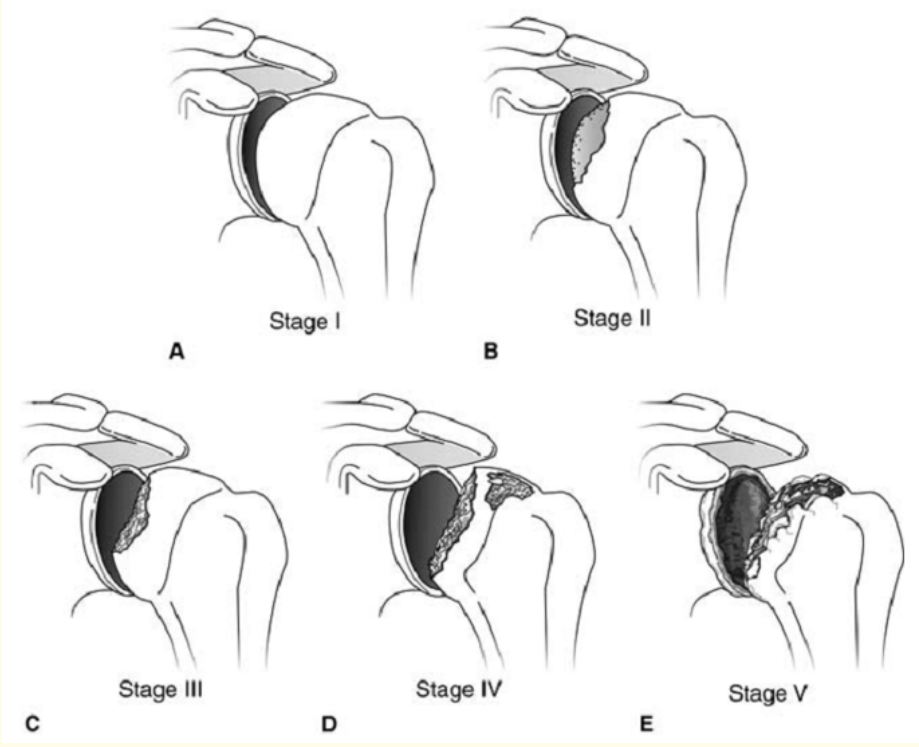

Figure 18: *Illustration demonstrating the progression from precollapse lesion to late stage disease and humeral head collapse in the osteonecrotic humeral head. A: Stage I, no radiographic evidence of shoulder osteonecrosis. The humeral head appears normal, with no sclerosis, and curvature is maintained. B: Stage II, signs of mottled sclerosis appear, but the curvature of the humeral head remains intact. C: The presence of a crescent sign is indicative of subchondral fracturing and defines stage III disease. The humeral head may become less spherical as the early stages of collapse are seen. D: Stage IV, progression to collapse of the subchondral bone. E: Stage V is defined by the onset of degenerative changes in the glenoid cavity.*

Diagnose

History and clinical signs A thorough examination of the medical and social history is important to determine if there is an underlying disease or risk factor. It has been shown that even small amounts of cortisone can cause disease, making past drug treatment necessary.

In the early stages, shoulder pain that radiates even at rest may be the only sign. Thereafter, symptoms worsened with nocturnal attacks, limited activity, and reduced range of motion. In the later stages, clicks may or may not occur with or without pain on examination because of arthritis or the presence of loose osteochondral bodies. Physical examination usually reveals only local pain and reduced range of motion in the later stages.

Pain is often dull, and because the glenoid is not as fit or weight-bearing as acetabulum, it can tolerate greater deformity. Therefore, in most cases, patients develop symptoms at a later stage than in femoral head necrosis. As a result, diagnosis is often delayed and effective conservative treatment is excluded.

Imaging

Radiographs are useful for diagnosis in all stages except stage I. Anterior, axillary, and radial radiographs are usually sufficient to diagnose and assess the extent of the lesion. Care must be taken to distinguish osteonecrosis from other medical conditions, such as isolated bone infarctions, cysts, benign or malignant tumors, and infections. For early detection, MRI is essential. Because the intensity of the MRI signal is based on the water content of the tissues, it is easy to detect the early congestion that occurs in osteonecrosis. In later stages, MRI can demonstrate the actual size of the lesion and confirm the diagnosis. The diagnostic findings of osteonecrosis are similar to those observed in femoral head osteonecrosis. Double line sign was observed in 50% of patients with humeral head necrosis. In the later stages, subchondral fractures and collapse are characteristic of osteonecrosis. The sensitivity of MRI in detecting early or late stages of osteonecrosis was 100% in some studies. This sensitivity is higher than that of scintigraphy or CT. Scintigraphy offers an advantage in detecting osteonecrosis of other parts of the body in a study. When osteonecrosis of one end of bone has formed, radiographs or MRI of the bilateral shoulder and hip should be performed to rule out their early involvement.

Treatment

Although the clinical symptoms and etiology of necrosis play an important role in the treatment of each patient, stage is the most objective criterion for determining the most appropriate treatment. Conservative treatment can be effective in stages I and II (primary stage), when the patient's symptoms are relatively mild. Cruess [27] advocates nonsurgical treatment for patients with minimal deformity and symptoms or those with limited physical needs. When symptoms persist, and there are signs of deterioration despite conservative treatment, surgical intervention is recommended. Advocated surgical options include arthroscopic surgery, core decompression, vascular bone graft, and shoulder arthroplasty.

Non-surgical treatment

Nonsurgical treatment includes patient education and management of known risk factors, such as smoking and alcohol consumption, for discontinuation. Corticosteroids must be replaced with alternative medicine or reduced in dose, and high-altitude activities or dynamic manual work should be limited. To prevent stiffness from inactivity, physical therapy should include total passive range-of-motion and pendulum exercises without the need for active overhead exercises. Using nonsteroidal anti-inflammatory drugs and analgesics can ease the patient's pain and increase comfort in daily activities. Intra-articular cortisone injections may provide some pain relief, but its use must be weighed against the potential for disease progression. In general, the goals of conservative treatment are to relieve pain, restore normal range of motion, and delay disease progression.

Arthroscopy

The use of arthroscopy as a minimally invasive modality for the treatment of osteonecrosis of the shoulder was first reported by Johnson in 1986 [32]. Since this initial description, additional case reports and Small case series have further described the role of arthroscopy in the treatment of osteoarthritis of the shoulder, noting improved function and pain relief at short-term follow-up. The advantage of arthroscopy is that it can be used not only for diagnosis and staging, but can also be used to remove body laxity and resect jaw flaps.

Arthroscopic core decompression

Arthroscopy core decompression could theoretically provide the benefits of both procedures. Dines., *et al.* [33] described a technique in which diagnostic arthroscopy is used to first evaluate areas of bone necrosis.

The authors then performed a decompression using the anterior cruciate ligament guided through the anterior or posterior portal to pinpoint the area of necrosis. Chapman., *et al*. [34] also reported the same using pre-platelet image intensity to confirm the exact location of the guide. The anterior position of the guide bar was confirmed arthroscopically. The guide pin was over-ground with a 7 mm reamer under direct arthroscopic imaging and imaging to avoid perforation of the guide pin. All three patients treated with this technique experienced immediate pain relief in the study of Dines., *et al* [33]. The patient in Chapman., *et al*. [34] had an unstable postoperative course, with full return of mobility (ROM) 3 days after surgery.

Bone grafting

Rindell [35] described the use of pedicle grafts in an attempt to revascularize the head in a patient with stage III humeral head necrosis. Core decompression was performed, and a vascular bone graft was inserted into the distal canal with the posterior deltoid pedicle into the canal. After 18 months of follow-up, the patient had significant pain relief and shoulder mobility had improved. There is also radiographic evidence of graft fusion. In another study, two patients were treated with resection and pelvic transplantation. After 3 years of follow-up, transplantation and clinically successful outcomes were observed.

Shoulder arthroplasty

Indications for shoulder orthopedics in osteoarthritis are similar to those for degenerative joint disease, i.e. significant pain and dysfunction unresponsive to nonsurgical treatment. Shoulder arthroplasty appears to be the most reliable treatment for osteonecrosis of the tip of the bone. Age is a major concern in shoulder arthroplasty, and some surgeons have questioned the durability of prosthetic implants, emphasizing the need for multiple shoulder modifications in patients with arthroplasty. young person. In patients 50 years of age and younger, Sperling., *et al*. [36] reported success rates of 84% and 73% for total blepharoplasty and osteotomy, respectively, during follow-up. follow up 15 years. There is only a small series in the literature regarding shoulder replacement for osteonecrosis of the humeral head. In some studies, results were not reported separately for posttraumatic and nontraumatic osteonecrosis. Rather, the results were combined with those of degenerative joint disease. It is suggested that 5% to 7% of patients undergoing shoulder arthroplasty have bilateral head necrosis.

Cruess [27] reported pain relief and good range of motion in five cases of severe non-traumatic osteonecrosis in his series. Small differences were noted between humeral head replacement and total shoulder replacement. No or mild pain was observed in 77.3% of patients while subjective improvement was noted in 79.5% of patients. Poorer functional outcomes have been observed in posttraumatic osteonecrosis, and higher outcomes have been observed in steroid-induced osteonecrosis.

In a recent study of shoulder arthroplasty in patients with non-traumatic osteonecrosis of the head, Parsch., *et al.* [37] reported that only 4 out of 13 patients had a good outcome or color after 30 months of follow-up. The authors found that patients 65 years of age and older had worse functional outcomes than younger patients. They also note that progressive glenoid erosion or preoperative glenoid destruction is one of the main causes of failure in the application of angioplasty.

Resurfacing

The Humeral head resurfacing technique preserves bone stock and maintains natural head tilt, version, deviation, and angle, facilitating any future corrective surgery. Indications for resurfacing include failed core decompression and arthroscopy or initial presentation with complete or near complete loss of articular cartilage, with sufficient epithelial bone remaining to permit safe fixation. whole implant. One of the disadvantages of this technique is the difficulty of accessing the arachnoid space. Indications for resurfacing include complete or near complete loss of articular cartilage, with sufficient epithelium remaining to permit solid fixation of the implant. This technique can be applied to treat cartilage loss due to osteonecrosis. In 2001, Levy and Copeland [38] reported their experience with shoulder resurfacing in 94 patients with a median follow-up of 6.8 years (range 5 to 10 years). This study included four patients with osteonecrosis. The adjusted mean Constant score for the osteonecrosis population improved from 11 points preoperatively to 74 points postoperatively. ROM in this population improved from 63° to 133° in forward elevation, 49° to 118° in abduction, and −3° to 81° in external rotation; Improves internal rotation from buttock reach to L1 vertebra.

Conclusion

Many conditions have been linked as risk factors for ON. However, it is difficult to determine the true etiological status when anecdotal or retrospective methods are used, regarding the etiology of the low prevalence, especially as many studies report idiopathic ON. accounts for 10 to 15% of cases. When possible, we relied on reports that used prospective cohorts, longitudinal or analytic, to identify strong associations between risk factors and ON. This does not mean that we rule out other possible conditions as risk factors for ON, but it does require strong evidence of a cause.

Closely etiologically related risk factors for ON seem to converge through mechanisms of vascular impairment to produce subtissue or segmental ischemia and osteocyte death. Subsequent repair often results in subchondral bone loss exceeding formation, resulting in structural damage and fracture. Understanding the risk factors and pathophysiology has therapeutic implications as several treatment regimens are available to optimize crest and epiphyseal circulation, prevent bone resorption and preserve subchondral bone. In traumatic ON, acceptable fracture location and timing, technique and method of immobilization are important. In the case of noncancerous ON, specific prophylaxis is available to prevent lipodystrophy (by limiting alcohol intake and taking corticosteroid doses), using statins, and treating Glaucoma with enzyme replacement. The ON associated with the hypercoagulable syndrome has been prevented with warfarin and enoxaparin. Bisphosphonates have been used to rebalance the rate of digestion and formation of subchondral bone with the expectation of maintaining the mechanical integrity of the subchondral bone and minimizing the risk of fracture and non-healing.

Bibliography

1. Kienböck's R. "Concerning traumatic malacia of the lunate and its consequences: joint degeneration and compression". *Fortsch Geb Roentgen* 16 (1910): 77-103.

2. Hult en O. "Concerning anatomical variations of the carpal bones". *Acta Radiologica* 9 (1928): 155-168.

3. D'Hoore K., *et al*. "Negative ulnar variance is not a risk factor for Kienböck's disease". *The Journal of Hand Surgery* 19A (1994): 229-231.

4. Stahl F. "On lunatomalacia (Kienböck's disease): a clinical and roentgenological study, especially on its pathogenesis and the late results of immobilization treatment". *Acta Chirurgica Scandinavica* 95 (1947): 3-73.

5. Xiong G., *et al*. "Microstructural study of the lunate in stage III Kienböck's disease with micro-computed tomography imaging". *The Journal of Hand Surgery: British and European* 42.1 (2017): 71-77.

6. Kim S., *et al*. "Superselective angiography of the wrist in patients with Kienböck's disease". *BMC Musculoskeletal Disorders* 20 (2019): 143.

7. Lichtman DM and Degnan GG. "Staging and its use in the determination of treatment modalities for Kienböck's disease". *Hand Clinics* 9.3 (1993): 409-416.

8. Lichtman DM., *et al*. "The classification and treatment of Kienböck's disease: the state of the art and a look at the future". *The Journal of Hand Surgery: European* 35.7 (2010): 549-554.

9. Bain GI and Begg M. "Arthroscopic assessment and classification of Kienböck's disease". *Techniques in Hand and Upper Extremity Surgery* 10.1 (2006): 8-13.

10. Schmitt R and Kalb K. "Imaging in Kienböck's disease [in German]". *Handchir Mikrochir Plast Chir* 42.3 (2010): 162-170.

11. Comfort TH., *et al*. "Long-term results of displaced talar neck fractures". *Clinical Orthopaedics and Related Research* 199 (1985): 81-87.

12. Takakura Y., *et al*. "Ankle arthroplasty using three generations of metal and ceramic prostheses". *Clinical Orthopaedics and Related Research* 424 (2004): 130-136.

13. Preiser G. "Zur frage der typischen erna¨hrungssto¨rungen der kurzen hand und fusswurzelknochen". *Forschr geb Rongenstrahlen* 17 (1911): 360-362.

14. Gelberman RH and Menon J. "The vascularity of the scaphoid bone". *The Journal of Hand Surgery* 5.5 (1980): 508-513.

15. Taleisnik J and Kelly P. "The extraosseous and intraosseous blood supply of the scaphoidbone". *Journal of Bone and Joint Surgery* 48 (1966): 1125-1137.

16. Herbert TJ and Lanzetta M. "Idiopathic avascular necrosis of the scaphoid". *The Journal of Hand Surgery* 19 (1994): 174-182.

17. Kalainov DM., *et al*. "Preiser's disease: identification of two patterns". *The Journal of Hand Surgery* 28.5 (2003): 767-778.

18. Vidal MA., *et al*. "Preiser's disease". *Annales Chirurgiae et Gynaecologiae* 10 (1991): 227-236.

19. De Smet L. "Avascular nontraumatic necrosis of the scaphoid: Preiser's disease?" *Chirurgie de la Main* 19 (2000): 82-85.

20. Schmitt R., *et al*. "Idiopathic osteonecrosis of the scaphoid (Preiser's disease)-MRI gives new insights into etiology and pathology". *European Journal of Radiology* 77.2 (2011): 228-234.

21. Sokolow C., *et al*. "Preiser's Disease". *Journal of the American Society for Surgery of the Hand* 4 (2004): 103-108.

22. Moran SL., *et al*. "The use of vascularized grafts from the distal radius for the treatment of Preiser's disease". *The Journal of Hand Surgery American* 31.5 (2006): 705-710.

23. Hayashi O., *et al*. "Closed radial wedge osteotomy for Preiser's disease: a report of four cases". *The Journal of Hand Surgery* 16.3 (2011): 347-352.

24. Lenoir H., *et al*. "Idiopathic avascular necrosis of the scaphoid: 10 new cases and a review of the literature. Indications for Preiser's disease". *Orthopaedics and Traumatology: Surgery and Research* 98.4 (2012): 390 -397.

25. Menth-Chiari WA and Poehling GG. "Preiser's disease: arthroscopic treatment of avascular necrosis of the scaphoid". *Arthroscopy* 16.2 (2000): 208-213.

26. Heimann WG and Freiberger RH. "Avascular necrosis of the femoral and humeral heads after high- dosage corticosteroid therapy". *The New England Journal of Medicine* 263 (1960): 672-675.

27. Cruess RL., *et al*. "Aseptic necrosis following renal transplantation". *Journal of Bone and Joint Surgery* 50 (1968): 1577-1590.

28. Laing PG. "The arterial supply of the adult humerus". *Journal of Bone and Joint Surgery* 38 (1965): 1105-1116.

29. Rietrogrande V and Mostromarino R. "Osteopatia da prolungoto trattamento cortisonico". *Ortopaedia e Traumatologia deli Apparato Motore* 25 (1975): 791-810.

30. MacCallum RI., *et al*. "Bone lesions in compressed air workers. With special reference to men on the Clyde Tunnels 1958 to 1963". *The British Editorial Society of Bone and Joint Surgery* 48 (1966): 207-235.

31. Ficat RP. "Idiopathic bone necrosis of the femoral head: Early diagnosis and treatment". *The British Editorial Society of Bone and Joint Surgery* 67 (1985): 3-9.

32. Johnson LL. "Historical and Pathological Perspectives of Abrasive Arthroscopy: Current Status". *Arthroscopy* 2 (1986): 54-69.

33. Dines JS., *et al*. "Arthroscopic-assisted core decompression of the humeral head". *Arthroscopy* 23 (2007): 103.

34. Chapman C., *et al*. "Arthroscopically assisted core decompression of the proximal humerus for avascular necrosis". *Arthroscopy* 20 (2004): 1003-1006.

35. Rindell K. "Muscle pedicled bone graft in revascularization of aseptic necrosis of the humeral head". *Annales Chirurgiae Et Gynaecologiae* 76 (1987): 283-285.

36. SperIirg JW., *et al*. "Neer hemiarthroplasty and Neer total shoulder arthroplasty in patients fifty years old or less: long-term results". *Journal of Bone and Joint Surgery* 80 (1998): 464-473.

37. Parsch D., *et al*. "Shoulder arthroplasty in nontraumatic osteonecrosis of the humeral head". *Journal of Shoulder and Elbow Surgery* 12 (2003): 226-230.

38. Levy O and Copeland SA. "Cementless surface replacement arthroplasty of the shoulder: 5- to 10-year results with the Copeland mark-2 prosthesis". *The British Editorial Society of Bone and Joint Surgery* 83 (20011): 213-221.

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