Kienbock's Disease and Other Avascular Necrosis

9

Luc De Smet

Keywords
Kienböck's disease • Lunatomalacia • Lunate • Preisser's disease • Scaphoid • Johnson's disease • Capitate • Avascular Necrosis • Bone Necrosis • Carpus • Arthritis • Wrist • Leveling procedure • Proximal row carpectomy • Vascularised bone graft

Avascular (bone) necrosis can be caused by trauma, intake of (high doses) corticosteroids, alcoholism and various connective tissue disorders. However, in the majority of cases no clear aetiology can be found. In the wrist, avascular necrosis (AVN) of carpal bones involves mostly the lunate, scaphoid and sometimes the capitate. These bones have what is called a "vascular pattern-at-risk" as described by Gelberman and Gross [1]. AVN of other carpal bones has been reported, although these are even rarer. New imaging techniques, especially magnetic resonance imaging (MRI), have improved the early diagnosis of osteonecrosis, (as radiographs may be normal in the initial stages) although the ultimate diagnosis is histological.

L. De Smet, MD, PhD
Department of Orthopaedic Surgery,
University Hospitals Leuven, Weligerveld 1,
Pellenberg 3210, Belgium
e-mail: Luc.desmet@uzleuven.be

Kienbock's Disease

Introduction

The radiological appearances of avascular osteonecrosis of the lunate were first described by Kienbock in 1911 as "lunatomalacia" and since then this condition has been named after him [2]. The necrotic lunate becomes soft and flattens under the constant load of the wrist. This disturbs the normal architecture and kinematics of the carpus, with the ultimate development of secondary osteoarthritis. Generally, however, this progression is slow. Unfortunately with Kienbock's much is still unknown, specifically with regards to aetiology, the natural course of the disease and most importantly, treatment. There is a lack of evidence based treatment protocols. Most published series are simply case series.

History and Examination

Kienbock's disease mainly affects young males (third and fourth decade), although it can occur in

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 3: Inflammation, Arthritis and Contractures*, DOI 10.1007/978-1-4471-6557-6_9,

women, teenagers and sometimes older people. It is usually unilateral and isolated. A history of trauma is often mentioned, although this is often minor. A wrist subjected to repetitive trauma is also a common feature. Insurance companies however, usually reject the association between work and AVN. It is slowly progressive and the symptoms and signs are not very specific. Diffuse dorsal wrist pain, weakness and restricted motion are the first and often the only symptoms. On physical examination there is tenderness over the dorsal aspect of the wrist, often well localised over the dorsal side of the lunate. Later, swelling due to synovitis, loss of wrist motion and decreased grip strength occurs [3]. Early signs of synovitis include a restricted anteroposterior drawer sign at the wrist (pseudoinstability), as well as the obliteration of the anatomical snuffbox. In the anteroposterior drawer test, the examiner grasps the forearm of the patient, just proximal of the wrist joint with one hand and with the other hand holds the patient's hand just distal of the wrist joint; the joint itself being held in full pronation. The patient's hand is then moved in a dorso-palmar direction. Normally there is some translation possible, but not when there is synovitis or pain. Watson's scaphoid shift test is also usually painful. Sometimes, lunate collapse is detected on a routine wrist x-ray in an otherwise asymptomatic patient.

Investigations

The diagnosis and staging of Kienbocks disease is based on the x-ray findings [3, 4] (Table 9.1). In the early stages, plain radiographs can be normal, with the diagnosis being made by an abnormal bone scan or MRI. For the latter, an inversion of the signal on T1 and T2 weighted images is seen. Upon injection of Gadolinium, the lunate does not enhance. Schmitt et al. [3] have proposed a classification based on the MRI findings (Table 9.2).

With further progression, the typical sclerosis of the lunate appears on the anteroposterior radiographs, followed by collapse and fragmentation of the lunate. This causes a loss of the normal carpal height with rotation of the scaphoid. This change in configuration can lead to altered force transmission across the wrist, with the development of secondary osteoarthritis. During this process it is postulated that new bone can be laid down at the lunate fossa, giving the impression of lengthening of the radius, compared to the ulna. Added to that and usually seen on a CT scan, a subchondral fracture of the lunate in the coronal plane can be seen. This radiological evolution forms the basis of the classification system proposed by Lichtman et al. [4]: (Fig. 9.1).

Table 9.1 Lichtman's classification

Stage 1. The radiographic appearance of the lunate is normal, bone scintography is positive, MRI is abnormal.
Stage 2. The lunate exhibits increased density, but its size and shape are normal.
Stage 3. The lunate has collapsed, allowing the capitate to migrate proximally. In stage IIIA the scaphoid maintains a normal position relative to the rest of the carpus; In stage IIIB there is rotatory subluxation, leading to a scaphoid "ring" sign.
Stage 4. Secondary osteoarthritis of the radiocarpal joint

This classification has been found to be reliable and reproducible.

Another way of staging the disease is by arthroscopy. Baine and Begg [5] described four different stages of damage to the articular surfaces (Table 9.3).

In comparison to the new approach, this gives a more detailed view on the different articular surfaces, predicting the possible outcome after (surgical) treatment.

Aetiology and Evolution

The pathogenesis in Kienbocks disease is unknown. The blood supply of the lunate is variable and is thought to be the key factor in the pathogenesis of the disorder. The extra-osseous and intra-osseous vascular anatomy has been well studied [6]. According to Gelberman and Gross [1], all scaphoids and capitates and 8–20 % of lunates have a vascular pattern called "at risk" or type 1. These are carpal bones, which are mostly supplied by only

Table 9.2 Classification by MRI scan findings

	Pattern A	Pattern B	Pattern C
Gaudolinium enhanced	Increased	Mixed signal (inhomogeneous)	Increased (homogeneous)
T1	Decreased or normal	Decreased	Decreased
T2	Increased	Decreased	Decreased

Pattern A: marrow oedema and viable bone trabeculae
Pattern B: bone marrow necrosis
Pattern C: necrotic bone marrow and collapse

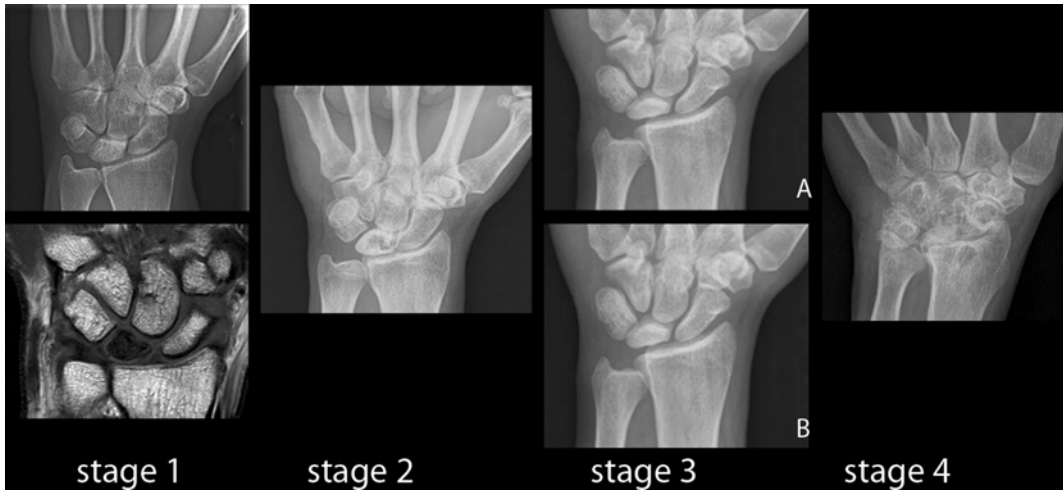

Fig. 9.1 Lichtman stages of Kienbock's disease. *A* and *B* are parts of the classification system

Table 9.3 Classification by wrist arthroscopy findings

0: Normal articular surfaces
1: Only the proximal surface of the lunate is damaged
2A: Distal and proximal articular surfaces of the lunate are involved
2B: Fracture in the coronal plane
3: Involvement of both articular surfaces of the lunate and the lunate fossa of the distal radius
4: Finally, also the head of the capitate is damaged

one vessel without additional anastomoses, therefore occlusion of this vessel would lead to necrosis. Besides arterial insufficiency, venous stasis has also been suggested as a possible cause of (carpal) bone necrosis. Extrinsic factors, such as fractures or repetitive minor trauma, can damage the intra-osseous blood supply leading to osteonecrosis, although the evidence for this is poorly documented. Even in lunate and perilunate dislocations the lunate seldom, if ever, undergoes avascular necrosis. The presence of a fracture therefore seems to be more a consequence rather than a cause of the condition.

Hulten [7] reported on the association between Kienbock's disease and an ulnar minus variance. Several authors have reported a change in ulnar variance with age, sex and position of the wrist, as well as in osteoarthritis due to Kienbock's disease [8, 9]. Other authors have confirmed this finding. However, more recently an alternative explanation has been put forward [10, 11] involving a (pseudo) lengthening of the radius occurring due to bone apposition on the lunate facet of the distal radius (Fig. 9.2). In our personal series, we found that a negative ulnar variance did not predispose to Kienbock's disease, although there were indications that a negative ulnar variance could predispose to a more rapid progression of the disease process [12]. A recent biomechanical study, using finite element modelling, demonstrated that ulnar minus variance was instrumental to further collapse of the lunate [13].

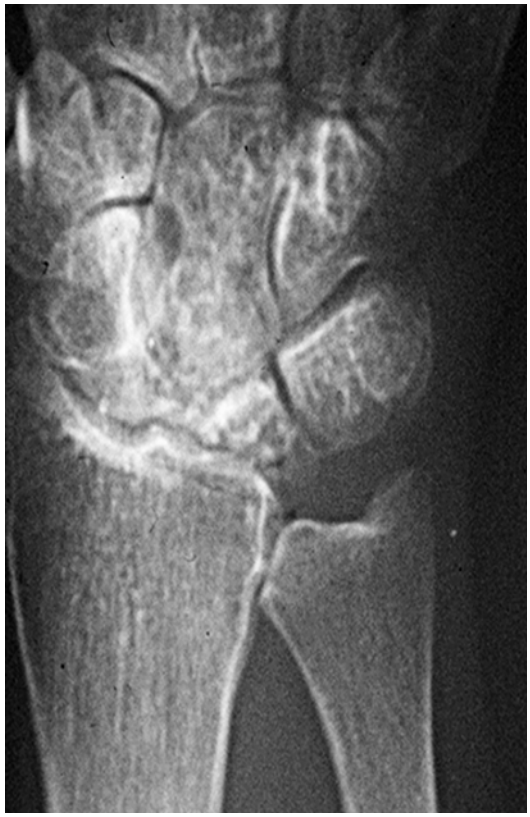

Fig. 9.2 Pseudolengthening of the distal radius due to bone apposition on the lunate fossa facing the necrotic lunate

Other morphological factors of both the distal radius and the lunate may also play a role in the aetiology of Kienbocks [14]. Comparing the contralateral wrist with an age and sex matched control group of patients with Kienbocks, the lunates were smaller and had more of a radial tilt than the controls and the radial slope of the distal radius was less. Antuna-Zapico [15] distinguished different types of lunate, one of which was more prone to necrosis.

There are also different patterns of evolution once the lunate becomes necrotic. The general pattern was described by Lichtmann et al. [4] who modified the initial grading by Stahl [16]. The lunate itself can undergo different types of collapse. The lunate can also fracture, usually in a coronal plane. Subsequently and in some incidences the proximal row remains stable. However, in most cases, with collapse of the lunate the normal architecture of the carpus is lost. Generally this results in flexion of the scaphoid. Depending on other factors, this will result in either dorsal or volar angulation of the collapsed lunate. This carpal collapse ultimately leads to (painful) degenerative changes: Kienbock related advanced collapse (KRAC) – wrist.

Management

A thorough evaluation of the involved wrist is necessary, prior to treatment. Not all wrists need treatment. When the pain is mild there is no need to recommend surgery. Acute pain may also disappear rapidly with rest and analgesics. Only when symptoms are long standing (more than 1 year) and are affecting the quality of life of the patient, should surgery be considered. With regard to the surgery itself, treatment is very much decided by the stage of the disease process. If the lunate itself has not collapsed, or there is little evidence of carpal collapse, then some form of reconstructive surgery, by way of a combination of both decompression and the insertion of a local vascular graft, is recommended. If, however, the lunate is fragmented and there is severe carpal collapse then salvage surgery, by way of either arthrodesis or excision (proximal row carpectomy), is more suitable.

Not all patients with Kienbock's disease need surgical intervention.
The need for surgery is based on the level of symptomatology, particularly pain.
If surgery is required, the procedure undertaken is dictated by the staging of the disease process.

Other patient factors are equally important; including, age and demands of the patient, profession and hobbies, smoking habits, their views on surgery and acceptance of the risks. The clinical condition of the wrist, particularly any synovial swelling or rigidity, also has a bearing on decision making. Obviously, it is ill advised to operate on an acutely inflamed wrist, and a wrist that is already stiff will become no more mobile following arthrodesis.

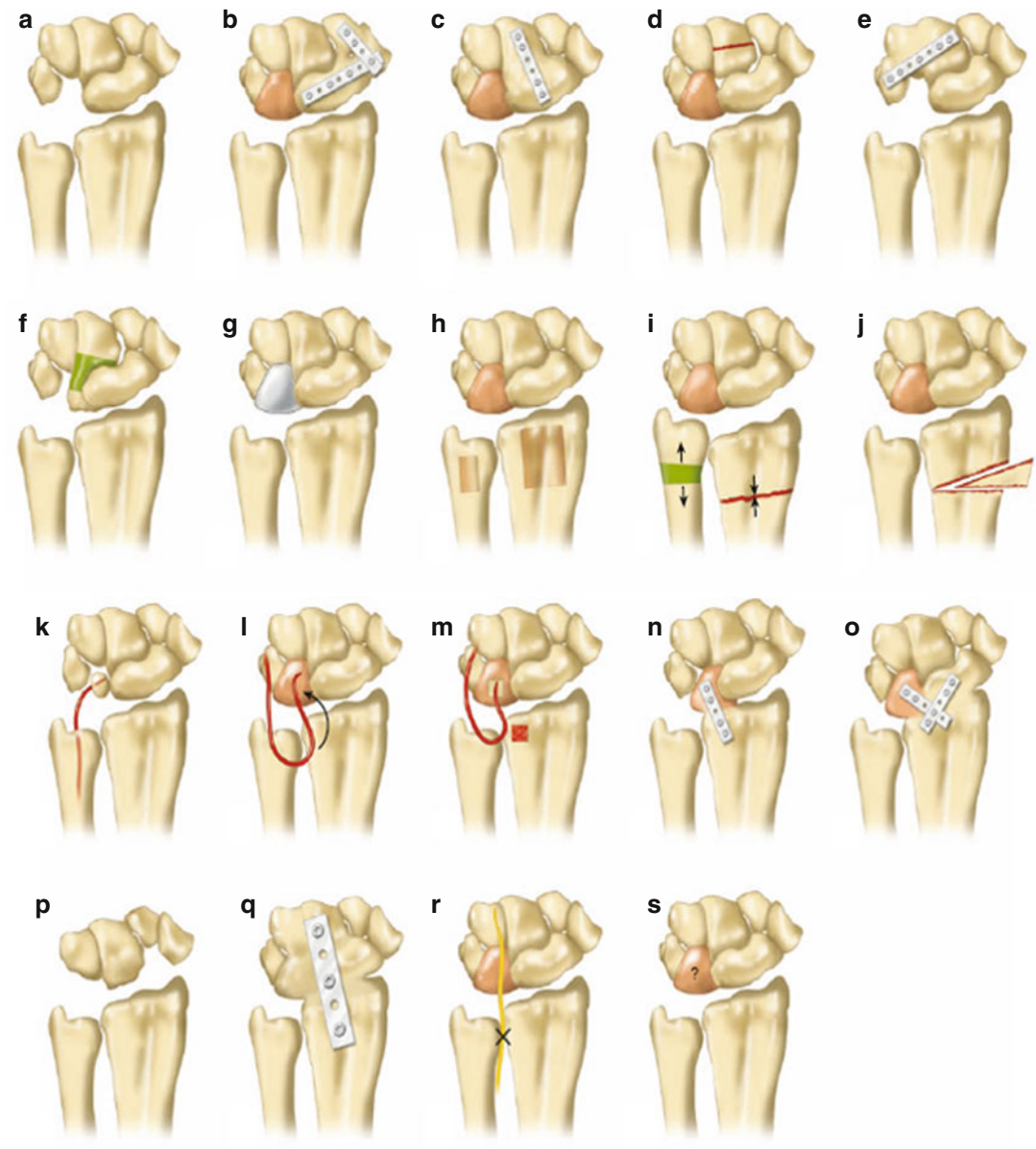

Fig. 9.3 Overview of the most popular procedures to treat Kienbock's disease. (**a**) Resection of the lunate. (**b**) STT arthrodesis with or without lunate resection. (**c**) Scapho-capitate arthrodesis with or without lunate resection. (**d**) Capitate shortening. (**e**) Lunate resection with ulnar column arthrodesis. (f) Lunate resection and capitate lengthening. (**g**) Lunate prosthesis. (**h**) Metaphyseal osteotomy (Core decompression). (**i**) Ulna lengthening (*arrows*) and/or radial shortening (*arrows*). (**j**) Wedge (opening or closing) osteotomy. (**k**) Lunate resection and substitution with the vascularised pisiform. (**l**) Direct vessel implantation. (**m**) Vascular bone graft (VBG). (**n**) Radio-lunate (Chamay) arthrodesis. (**o**) Radio-lunate-scaphoid arthrodesis. (**p**) Proximal row resection. (**q**) Full wrist arthrodesis. (**r**) Denervation. (**s**) whatever the future brings

Treatment options include both conservative and operative methods. As a rule, surgical treatment is preferred, since it generally leads to quicker improvement in symptoms and better outcomes. Many operative treatments have been devised for the various stages of Kienbock's disease. Generally, however, they can be grouped into three broad categories (Fig. 9.3):

(i) Direct revascularisation by vessel implantation or a vascularized bone graft (VBG) (stage 1 and 2).
(ii) Joint decompression. This not only relieves the symptoms, but it allows spontaneous revascularisation of the lunate, whilst diminishing the compressive forces acting on it (stage 1, 2 and 3A). This can be done by levelling the distal radioulnar joint (DRUJ), intracarpal arthrodesis (scaphotrapezialtrapezoidal (STT) or scaphocapitate arthrodesis) or capitate shortening. Restoring the normal carpal height is the key element in preventing osteoarthritis.
(iii) Salvage procedures for pain relief. These include wrist denervation, (resection) arthoplasty or radiocarpal arthrodesis (stage 3B and 4).

Non Operative Treatment

The rationale for this approach lies in the observation that spontaneous recovery can occur in some early onset cases. Cases of asymptomatic patients, with longstanding radiographic evidence of Kienbock's have also been reported. Previous reports suggest that the pain can subside within a few years. Kristensen et al. reported good results after short periods of immobilisation [17]; whereas Mikkelsen and Gelineck noted poor outcomes after non-operative treatment [18]. The debate continues, with some authors advocating a conservative approach, whereas others have observed progressive clinical and radiographic deterioration [19], or have demonstrated better outcomes with surgery [20]. A "watch and wait" policy, or, in a very painful wrist, a temporary short period of immobilisation with casting or bracing, however, can result in an asymptomatic wrist. A non-operative approach is also preferred in younger patients (Fig. 9.4). What we and others have observed in these, albeit rare, cases, is that there is some adaptation of the carpus through remodelling, rather than collapse.

Revascularisation by Vascularised Bone Implantation

Direct revascularisation, or replacement of necrotic bone by well-vascularised bone, is a logical approach to the problem. The basic principles involve revascularisation of the lunate and temporary stabilisation of the carpus (to prevent collapse during the revascularisation period), which preserves the integrity of the lunate and the architecture and kinematics of the wrist. Historically, the lunate has been replaced by the pisiform bone, which is pedicled on the ulnar artery [21]. This procedure is usually called Saffar's technique. Other authors have removed the necrotic bone by curettage, followed by cancellous bone grafting and vessel implantation, using the posterior interosseous artery. There is some evidence that the bone grafts were revascularised by this technique. We found only one paper using this technique in 11 wrists [22]. They reported stabilisation of the lunate with good pain relief in nine patients. More recently Jones et al. described one case of vessel implantation, in combination with Bone Morphologic Protein (BMP) [23].

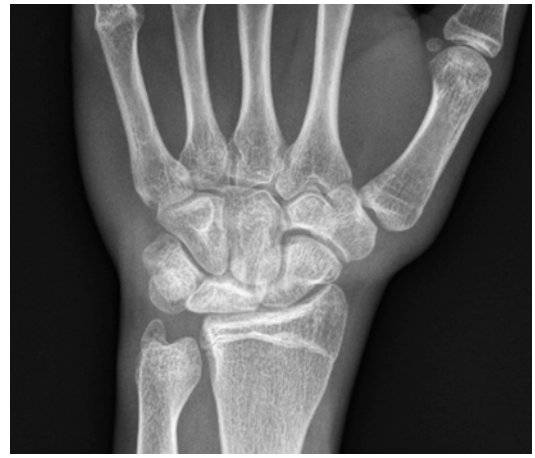

Fig. 9.4 Juvenile Kienbock's disease

Vascularised bone grafts (VBG) have the advantage of immediate implantation of viable bone, which simplifies matters by substituting the bone defect for a healing fracture. One does not have to wait for the secondary revascularisation of a cancellous bone graft (creeping substitution) and it avoids the period of temporary weakening that occurs with non-vascularised bone grafts.

The Mayo group studied the vascularity of the distal radius. Based on anatomical studies

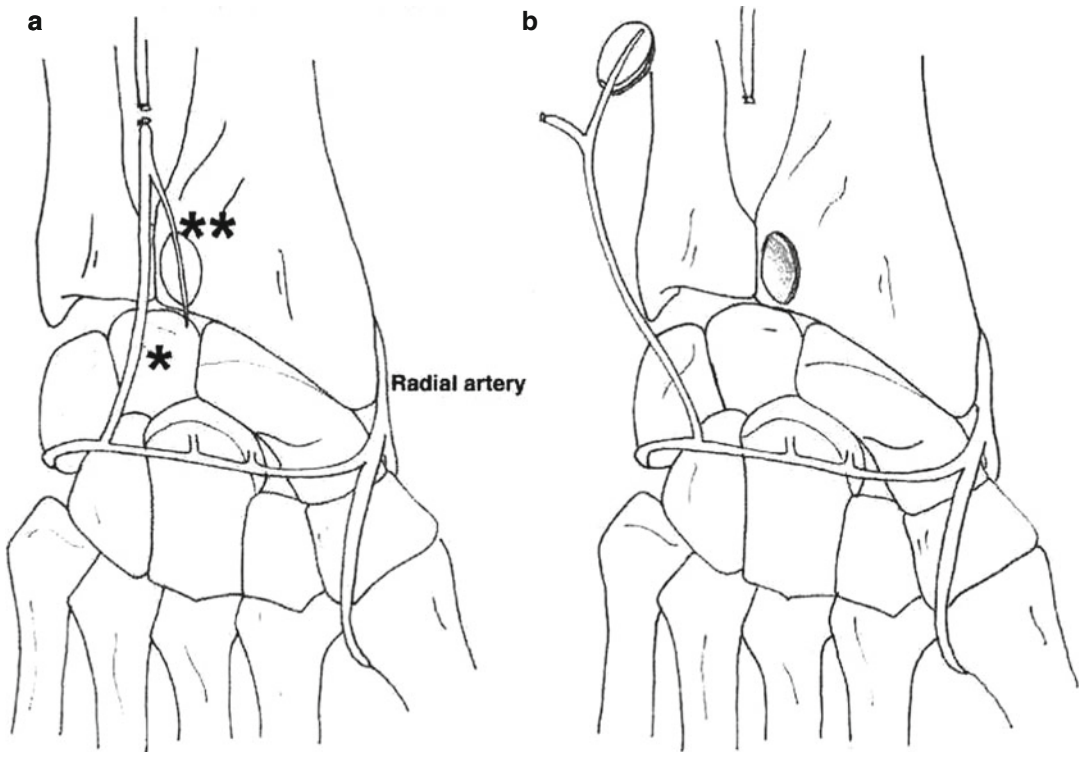

Fig. 9.5 Schematic presentation of the VBG. (**a**) Anatomy of the fifth extensor compartment artery (ECA) (*) and the fourth ECA (**). (**b**) Ligation of interosseous artery and harvesting of the graft. Retrograde vascularisation of the graft from the fifth ECA towards the fourth ECA

and animal experimentation they developed the use of the so-called 4, 5 extensor compartment vascularised bone graft [24]. In their clinical series of 26 patients they reported pain relief in 92 % and a significant improvement in grip strength and maintenance of carpal height in 77 %. We have also applied this technique in the treatment of early Kienbock's. Contraindications to VBG would include smoking, the older patient, a patient with a stiff wrist and a patient where the lunate is fractured.

Surgical Technique of the Dorsal VBG (Fig. 9.5)

A straight skin incision is made over the dorsal aspect of the wrist, from the third metacarpal base to the distal forearm. The fifth dorsal extensor compartment is exposed and incised. The extensor tendons are retracted radially. The fifth extensor compartment artery (ECA), which originates from the dorsal branch of the anterior interosseous artery, is identified. The fourth extensor compartment artery originates from the fifth ECA or anterior interosseous artery and supplies nutrient branches to the dorsal aspect of the radius within the fourth extensor compartment. A bone graft proximal to the radiocarpal joint and overlying the fourth ECA, including the nutrient vessels, is outlined. The dorsal wrist capsule is opened, according to Berger's ligament sparing incision. The lunate is then exposed and the necrotic part is removed through a dorsal cortical window. The anterior interosseous artery is ligated proximal to the fourth and fifth ECA. The vascularised bone is now mobilised (Fig. 9.6) and is placed into the gap in the lunate. In patients with stage IIIa lunatomalacia, an STT-fusion is also performed at the same time, in order to control the rotary subluxation of the scaphoid and to prevent carpal collapse. Postoperatively, the patients are immobilised in a below-elbow splint for 6 weeks.

The metacarpal head of the index ray is another donor site for VBG. It was first described

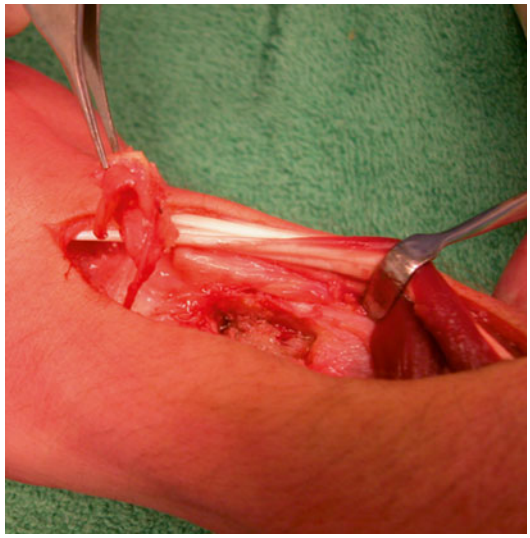

Fig. 9.6 Intra-operative view: the VBG is pre-elevated and will be moved distally

by Bengoechea et al. (one case) [25] and then by Zafra et al. (five cases) [26]. All cases obtained a satisfactory outcome, but all had an additional procedure on the radius (shortening or wedge osteotomy).

Finally, free vascularised bone grafts from the iliac crest have also been used [27, 28]. Both authors mentioned above performed this procedure in 18 wrists and both observed good bony integration in 16 wrists.

Leveling Procedures of the DRUJ: Radial Shortening or Ulna Lengthening

Based on Hulten's finding [7] that Kienbock's disease was more frequent in ulna minus variant wrists, levelling the ulna to the radius by shortening the radius or lengthening the ulna has been proposed. This is one of the oldest and most accepted techniques for the treatment of Kienbock's disease. Radial shortening is now preferred to ulna lengthening, as the latter has a high incidence of non-union. Several large series have been reported using these procedures [20, 24–39] (Tables 9.4 and 9.5). The basic mechanism seems to be an unloading of the lunate and prevention of further collapse. More sophisticated procedures include lateral closing and opening wedge osteotomies of the distal radius.

Table 9.4 Results of radial shortening

Author	Year	N	% good
Axelson	1973	19	100
Rosemeyer	1976	19	69
Schattenkerk	1987	20	70
Nakamura	1990	23	83
Weiss	1991	29	87
Gomis	1994	28	85
Siala	2000	31	80

Table 9.5 Results of ulna lengthening

Author	Year	N	% good
Armistead	1982	20	90
Sundberg	1983	19	95
Quenzer	1993	64	90
Trail	1996	20	100

Most classical papers and handbooks still recommend an osteotomy for stages one and two and sometimes for stage 3, provided there is an ulna minus variance. The morphology of the sigmoid notch and the ulnar head must be evaluated prior to any joint levelling procedure. Morphological studies of the DRUJ [40, 41] have distinguished three different types of sigmoid notch, which creates the potential for DRUJ incongruity or impingement following a change in the length of one of the forearm bones. Other types of decompressive procedure should be considered in these cases. These would include various intracarpal procedures [42, 43].

Surgical Technique of the Radial Shortening Osteotomy

A standard volar approach is used, radial to the flexor carpi radialis. The distal one-third of the radius is exposed. A six hole dynamic compression plate (DCP) or a three hole distal radial fracture fixation plate is applied on the volar surface. Markings are made to determine the level of the osteotomy and the correct rotation of the radius. The drill holes for the proximal three holes are made and the plate is provisionally fixed with one screw, but not tightened. The plate is rotated over 90° and the osteotomy is performed. A 2 mm slice of bone is removed. This is sufficient to decompress the lunate. Next, the plate is swung

back and the distal part of the radius is brought into close contact with the proximal part and the plate is fixed under compression, according to the standard techniques of osteosynthesis. Immediate postoperative mobilisation is allowed.

Adequate pain relief has been obtained with levelling procedures (radial shortening and ulna lengthening). The fate of the lunate in these procedures is, however, not clear. Weiss [38] found no progression, whilst Wada et al. [44] and our previous study found further progression of the disease process.

Proponents of more sophisticated procedures, including lateral closing or opening wedge osteotomies and medial closing wedge osteotomies of the radius claim that the osteotomy changes the morphological aspects of the distal radius [44, 45]. In general the results are also good. However, it is felt that radial opening osteotomies produce a better decompression than the closing wedge osteotomy [46]. The clinical outcomes, however, between the two were not significantly different.

Some authors believe that the biological effect of the osteotomy is responsible for the pain relief. Based on this, Illarramendi et al., proposed a simple fenestration of the distal radius and ulna, rather than changing the length or orientation [47]. Their series consisted of 22 patients, of which 16 were pain free and 4 had only moderate pain. Schultz et al. confirmed these findings in his series of 10 patients [48].

Intracarpal Procedures

One of the most important consequences of lunate necrosis and the subsequent collapse, is the disturbance of the overall carpal architecture, in particular rotational subluxation of the scaphoid. Based on the work by Watson et al. on the treatment of scapholunate ligament tears, some authors have reported good results with an STT arthrodesis for treating Kienbock's disease [49–51] (Fig. 9.7). It has also been proven that this procedure unloads the lunate to a similar degree as a joint levelling procedure. The STT arthrodesis can also be combined with a VBG or other revascularisation procedures (in Lichtman's stage 3a). In earlier stages, temporary fixation of the scaphoid to the capitate, using K wires, for a period of 6–8 weeks, rather than a formal arthrodesis is a good alternative.

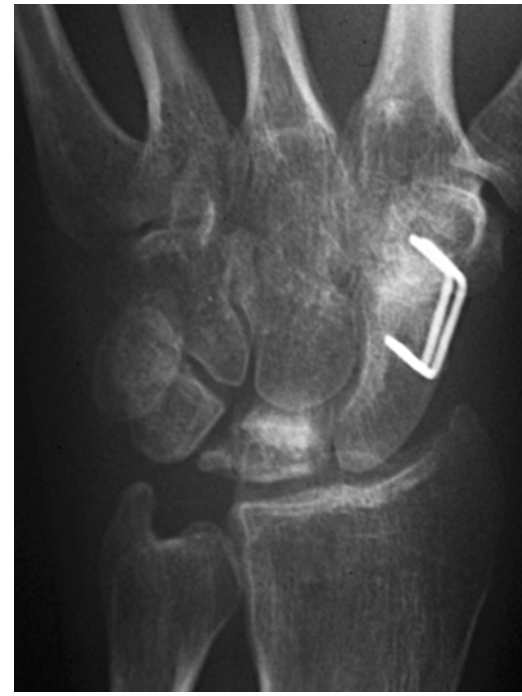

Fig. 9.7 STT arthrodesis with the use of Shapiro staples. The geometry of the lunate remain unchanged with time

Other intracarpal procedures include scaphocapitate arthrodesis, a capitate shortening osteotomy and triquetro-capito-hamate fusion combined with, or without, a lunate resection.

Pre-existing wrist stiffness is a relative contraindication for a partial arthrodeses. A stiff wrist rarely gets better with a fusion. The main complications of these procedures include non-union, hardware failure and, last but not least, impingement between the radial styloid and the scaphoid, with the development of osteoarthritis. This complication is hard to deal with, since it appears to progress even when a radial styloidectomy has been undertaken.

Surgical Technique of the STT

We prefer a palmar Russe approach. The FCR tendon sheath is incised on its radial side and the tendon retracted ulnarly. The wrist joint is accessed through the bed of the FCR sheath. The STT joint is exposed distally and the capsule is

incised transversally. The scaphoid is reduced by manual pressure and fixed to the capitate with a K-wire. The cartilage and subchondral bone of the distal pole of the scaphoid and the proximal side of the trapezium and trapezoid are removed. The gap is filled with cancellous bone from the iliac crest, or the distal radius. The STT joint is fixed with K-wires, staples or a Herbert screw (Fig. 9.7). Immobilisation in a cast is continued for up to 6 weeks, followed by wrist motion exercises.

Most authors [52, 53] recommend this procedure as a primary treatment. The outcomes are generally reported as good, with Mayo wrist scores between 62 and 66 and a DASH score of 24.8. However, some authors have reported no significant differences between an STT fusion and radial shortening, although the outcome was better than a full radiocarpal fusion [53].

With regard to capitate shortening, whilst this does decompress the lunate, the clinical and radiological results so far have been reported as poor.

Scaphocapitate fusion has a similar biomechanical effect as an STT fusion. Sennwald and Uferrast identified satisfactory results in 10 of their 11 cases [54]. Capitohamate fusion also resulted in an excellent outcome in the series of Oishi et al. (45 patients, 42 painfree) [55].

A more sophisticated procedure was proposed by Wilhelm et al. [56]. After resection of the lunate, a transverse osteotomy of the capitate is performed and, by callus distraction, brought into the empty space. They performed this in 14 patients. Lu et al. had a larger series (30 patients) and reported reasonable results [57]. However, we do not see any advantage over a simple proximal row carpectomy.

Salvage Procedures

Once osteoarthritis is present (stage 4), or in stiff wrists (stages 3B), a reliable salvage procedure is preferred. As with other joints, the solutions are either arthrodesis, excision arthroplasty or denervation. The outcome with total wrist fusion is less satisfying, with a high percentage of complications and unsatisfactory results. Our own follow-up studies have demonstrated that preservation of some wrist motion is preferred by patients, although the amount of movement is not crucial. Partial – radiolunate and radioscapholunate fusions – have been proposed to overcome the poor results of full wrist arthrodesis. Kilgus et al. found an acceptable result in five cases of radioscapholunate fusion [58]. Conversely the Wrightington group had 50 % failure of their radiolunate fusions [59].

Full radiocarpometacarpal arthrodesis resulted in 55 % pain reduction, a DASH of 51.4 and 70 % return to previous occupations in the large series of Sauerbier et al. [60]. This was confirmed in another smaller series by Tambe et al. [53].

Arthroplasty, by using a silicone spacer, was initially proposed by Swanson in 1993 [61]. However, silicone synovitis, with the appearance of intraosseous cysts all over the carpus and distal radius, is a major potential complication. Kaarela et al. had to remove 12 of the spacers in 39 patients [62]. Partial denervation of the wrist joint, by sectioning the posterior interosseous nerve, is a common adjunctive procedure. An isolated full denervation of the wrist joint is, however, also possible and indicated in limited situations i.e. with older patients, those with moderate pain and in patients who wish to retain as much range of motion as possible.

For most clinicians, however, the first choice is proximal row carpectomy (PRC), particularly for stiff wrists with stages 3 and 4 of disease. Despite the theoretical possibility of damage to the lunate fossa, proximal row carpectomy has led to very satisfactory outcomes in most series and also in our hands [63–65].

PRC, however, is not indicated if there is severe damage to the cartilage of the lunate fossa, ulnar translocation of the wrist (seen sometimes following previous radial styloidectomy), damaged capitate head or following previous intracarpal arthrodeses.

Surgical Technique of the Proximal Row Carpectomy

A standard longitudinal dorsal approach is used. The dorsal retinaculum is opened over the third compartment and the dorsal capsule is incised according to Berger by a ligament sparing incision [24]. The carpal bones are then inspected. If the status of the cartilage on the lunate fossa of the radius and on the capitate are found to be satisfactory, the procedure can continue. Usually, the carpal bones are removed piecemeal; “en bloc” resection is rarely possible. Capsule and

retinaculum are then repaired anatomically. Postoperatively, the wrist is immobilised in a cast for 4 weeks, although this is not always necessary.

Although most reported series are small, proximal row carpectomy in Kienböck's disease mostly results in a favourable outcome. We assessed the outcomes in 21 patients (mean age 39 years) with advanced Kienbock's disease treated by resection of the proximal carpal row [50]. Thirteen patients had little or no pain, 3 had moderate and 5 had severe pain. Begley and Engber also reported satisfactory results in 14 patients, with decreased wrist pain in all patients, grip strength of 72 % of the contralateral side, unchanged range of motion or slight improvement in 12 of the 14 patients after surgery and a return to former employment for all patients [66].

Conclusion

Kienbock's disease is a progressive and chronic wrist disorder, which occurs as a result of avascular necrosis of the lunate, ultimately leading to osteoarthritis of the wrist. It usually affects young and active adults.

Non-operative treatment is recommended for children and juvenile patients. In older patients with mild symptoms, a conservative approach is also recommended, at least in the first instance.

Otherwise surgical treatment is undertaken. The chosen procedure depends on the stage of the disease, the ROM of the wrist, the length of the ulna, the shape of the sigmoid notch and the presence or absence of a fracture of the lunate.

For stage 1 and 2 we prefer a VBG, with temporary scapho-capitate fixation. An alternative is a radial shortening osteotomy, provided that the ulna is short and that the geometry of the DRUJ can accommodate this.

For stage 3a we currently recommend a VBG with definitive STT arthrodesis, provided that the ROM is acceptable and there is no fracture of the lunate. However, the situation is fluid and we are not convinced that an STT arthrodesis is always required.

For stage 3b and for stage 3a with a fracture of the lunate, with acceptable ROM, we believe a salvage procedure, particularly PRC, gives better results.

Similarly for stage 4 and stiff stage 3b, where there is acceptable cartilage on the radius and the head of the capitate, a PRC is an excellent procedure.

For those in which a PRC would be ill advised, due to severe damage to the lunate fossa of the distal radius, either a full wrist arthrodesis, or a denervation for the older patient, is recommended. Finally, prosthetic replacement of the wrist is possible, although experience is limited.

Stage 1, 2 & 3A – preferred treatment is some form of decompression, either radial shortening or an STT fusion with a local vascular bone graft.
Stage 3B – proximal row carpectomy and sometimes formal arthrodesis
Stage 4 – with significant cartilage involvement, wrist arthrodesis
For patients with stiff wrists, proximal row carpectomy is probably the preferred option.
Finally, denervation is an option for patients with mild pain who wish to retain full range of motion, although results are unpredictable.

Preiser's Disease: Non-Traumatic Necrosis of the Scaphoid?

In medical literature, the eponym "Preiser" has been used for the idiopathic avascular necrosis of the scaphoid bone [67]. In contrast to Kienbock's disease, the scaphoid is rarely involved in idiopathic bone necrosis, despite the high incidence of necrosis of the proximal pole following scaphoid fractures and the perilous nature of its blood supply [68, 69]. Of the cases reported by Preiser in 1910, all had previously reported an incident of acute trauma and it is not unreasonable to suppose that his cases were scaphoid fractures with post-traumatic pseudarthrosis and bone necrosis. Since then, controversy exists about the actual existence of this disease.

As expected, the aetiology remains unknown. Most authors, however, agree that the common pathway is impaired blood supply to the scaphoid.

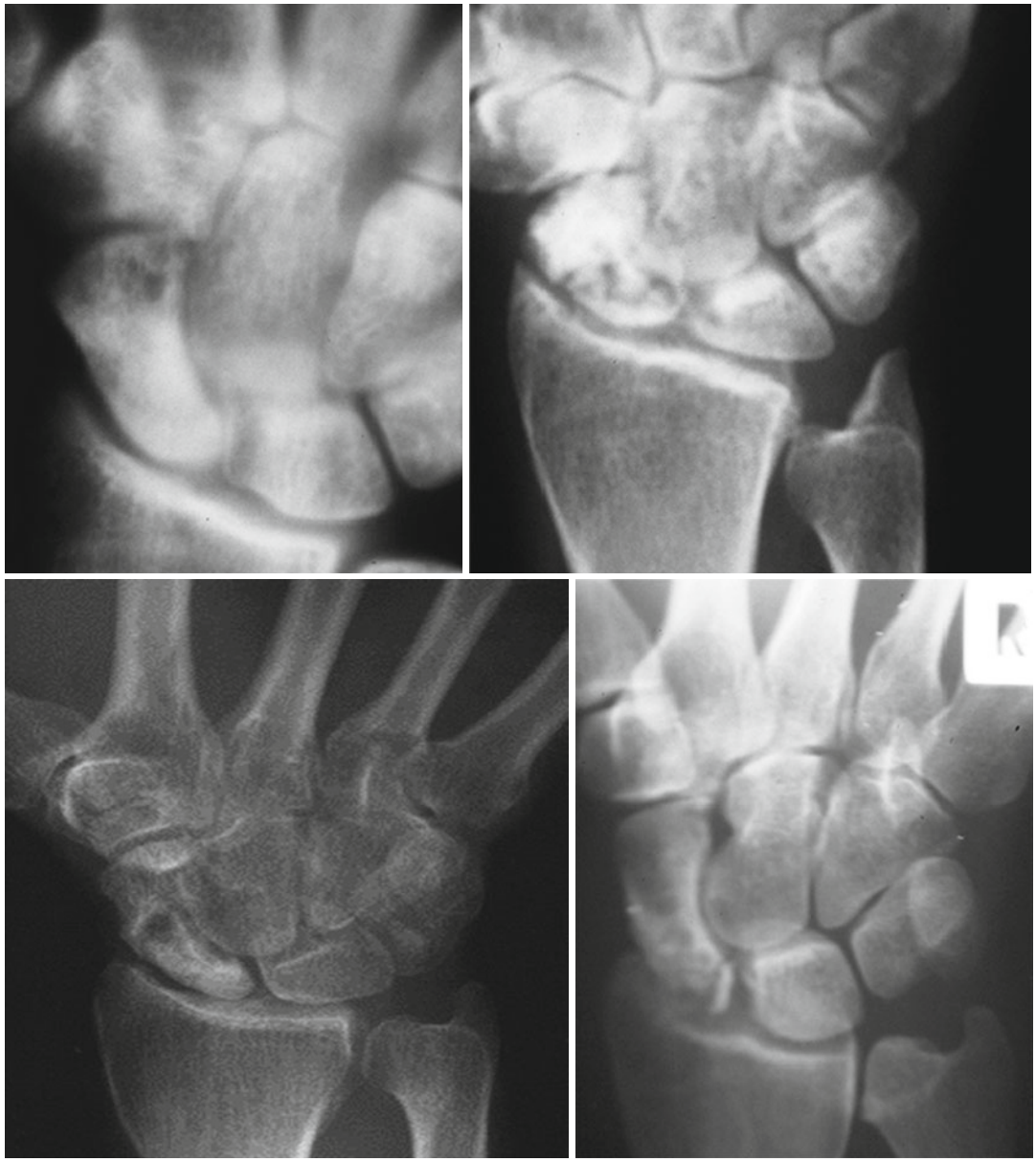

Fig. 9.8 Preiser's disease or AVN of the Scaphoid: different aspects

Taleisnik and Kelly [69], Gelberman and Menon [68] and Gelberman and Gross [1] have studied the vascularity of the carpus extensively. They were able to demonstrate a blood supply at risk. Specifically, the proximal pole receives its blood supply from a distal branch of the radial artery entering through the dorsal ridge of the scaphoid and running retrogradely. According to these authors, these scaphoids have a type I intraosseous blood supply, meaning that only one vessel is responsible for supplying the larger part of the scaphoid. This branch traverses an intra-articular membrane. It is proposed therefore, that when intra-articular pressure rises, compression and occlusion of this branch can occur. This is not only the case when there is intra-articular synovitis, but also when the extensor carpi radialis brevis exerts pressure on the scaphoid's surface when the wrist is flexed and ulnarly deviated. Repetitive micro-traumas are also held responsible for occlusion of

the nutrient branches. The distal pole has supplementary vascularisation and is better protected against bone infarction (Fig. 9.8).

Most publications are case reports or limited series. Since the 1990's, however, the interest in this disease has increased. Vidal et al. in 1991 [70] and Herbert and Lanzetta in 1993 [71] published some larger series (resp. 9 and 8 cases). However, 1 had a mal-united fracture of the scaphoid. Kaleinov et al. distinguished two patterns of the disease: type I (diffuse ischemia and necrosis) and type II (localised necrosis) based on a series of 19 cases [72]. We previously reported a series of 21 cases. On several occasions the condition has been related to systemic disease and/or steroid ingestion, chemotherapy [70], hypoplasia of the scaphoid [70, 73], ulna minus [73] and ulna plus variance [71].

Parkinson et al. suggested that a negative ulnar variance could also be a predisposing factor for avascular necrosis of the scaphoid [73]. This was rejected by Vidal et al. [70] and De Smet et al. [74]. In the larger series of Herbert and Lanzetta [71], 7 of the 8 cases even had an ulnar plus. The distribution of ulnar variance in our series was also not significantly different to a control group. The diagnosis is obvious in most cases, although strict criteria have not been established. We propose the following criteria:

1. absence of trauma (even minor) and/or surgical procedures to the wrist
2. radiological alterations (condensation, cysts, collapse) involving at least 80 % of the bone
3. MRI changes involving the whole scaphoid (with the exception of the distal tubercle)
4. histological examination indicating bone necrosis in the distal part of the scaphoid.

For the full diagnosis, the first two criteria would be suggested and the latter two confirmative.

Treatment is still controversial. Conservative measures (NSAID's, rest and splinting) are recommended initially, although the outcome is variable. Scaphoid excisions, partial or total, with or without replacement have been reported. Several so called salvage procedures can be undertaken, including proximal row carpectomy, a full wrist fusion, a four-part bone fusion (lunate-capitate-triquetrum and hamate) with scaphoid resection or a denervation of the wrist. Proximal row carpectomy was reported as giving good results by Alnot et al. [75]. Recently, Moran et al. reported a small series of revascularisation of the scaphoid using a vascularised bone graft taken from the distal radius with promising results [76].

Avascular Necrosis of the Capitate: Jönsson Disease

Despite the fact that the capitate has a similar vascular supply to the lunate and the scaphoid, cases of idiopathic AVN of the capitate are rare and in the literature only single cases are reported. This condition has been named after Jönsson [77] (Fig. 9.9), although there are only 24 cases identified in the literature. AVN of the capitate can be seen after fractures of the capitate, with or without perilunate dislocation (Fenton's Syndrome or trans-scaphoid, trans-capitate perilunate dislocation or capitonavicular Syndrome). Dereudre et al. described three distinct aetiological factors; micro-trauma, hyperlaxity or carpal instability and vascular factors [78]. We also think that metabolic causes should be included. There have also been reports of AVN of the capitate in hyperuricaemia steroid intake and Gaucher's disease. Usually it is found in young adults (athletes), the symptomatology is non-specific and the keystone of diagnosis is clinical suspicion. The diagnosis relies on radiographic appearance and MRI findings. Milliez described 3 types: I. the head of the capitate only, II. the body of the capitate only and III. total involvement [79]. Treatment is still a matter of discussion. Conservative measurements have been proposed in children. Recently, vascularised bone grafts have been prosposed [80]. A partial prosthesis of the head of the capitate (pyrocarbon) seems to be logical, but no long term series has been reported. Finally, an arthrodesis between the capitate, scaphoid and lunate, or a full midcarpal arthrodesis are good salvage options.

Necrosis of the Other Carpal Bones

- Since the other carpal bones do not have an 'at risk' vascular supply, the incidence of avascular necrosis is rare. The diagnosis is

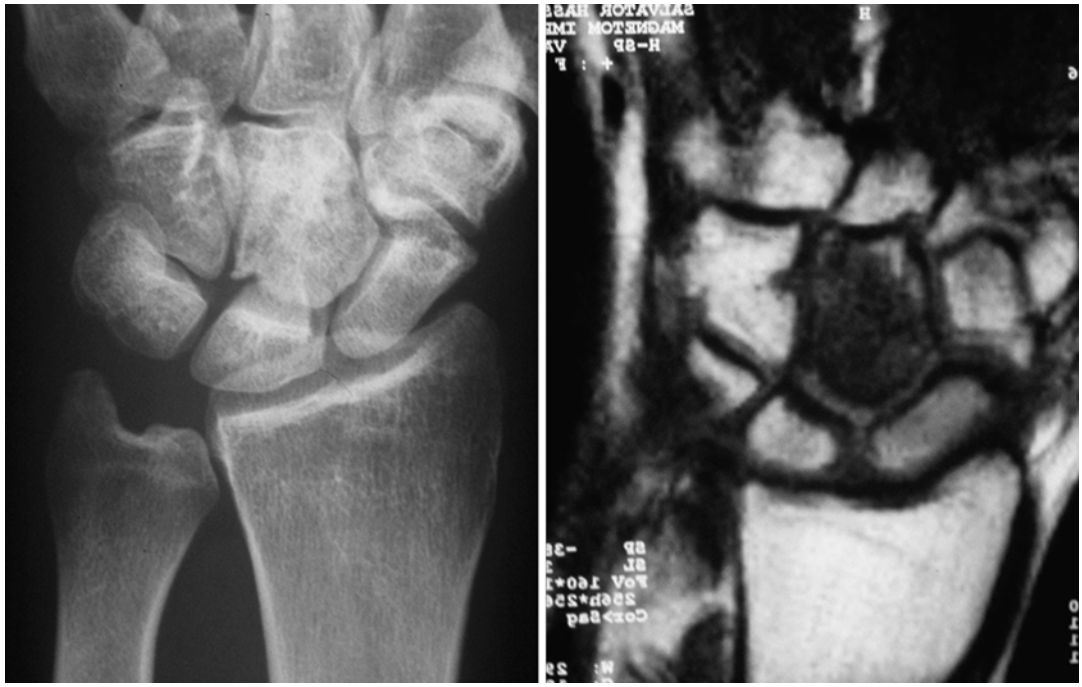

Fig. 9.9 AVN of the Capitate

often made after a long period of complaints and multiple examinations. Since the availability of MRI, the diagnosis has become more reliable. Osteonecrosis, or AVN of the hamate, (Fig. 9.10) is very rare and only a few cases have been reported. The hamate has a type 2 vascular supply, without intraosseous anastomoses. Although the natural evolution of necrotic carpal bones is not known, certainly for those bones involved, pain is the major reason to treat these patients. One can also assume that these carpal bones will ultimately collapse, resulting in articular incongruity and subsequently osteoarthritis. As a consequence, a more aggressive approach has been recommended. Two of the previously reported cases underwent curettage and conventional grafting associated with a capitohamate fusion. Both resulted in good pain relief, although the range of motion was reduced. The recent development of vascularised pedicle bone grafts from the distal radius

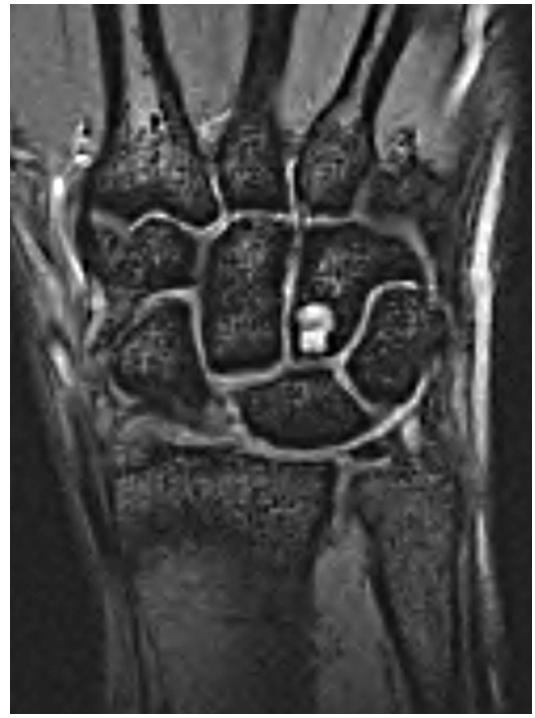

Fig. 9.10 AVN of the Hamate

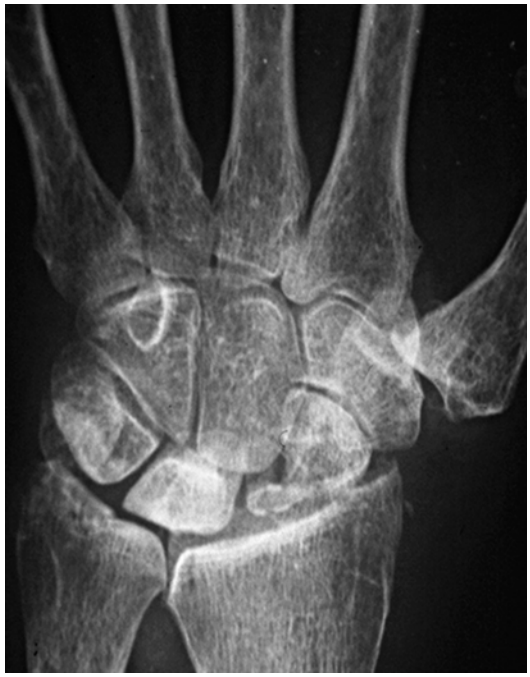

Fig. 9.11 AVN of the scaphoid, lunate and hamate

Table 9.6 Case reports of avascular necrosis in other carpal bones

Triquetrum:	1 case [80]
Pisiform:	2 cases [81, 82]
Trapezium:	1 case [83]
Trapezoid:	1 case [84]
Multiple bones:	3 cases [85]

seem to have had excellent early clinical and radiological success, as described by Moran et al. [76]. We used the fifth extensor compartment VBG in a personal case.

- The case of a 66-year old female patient with hyperlipidaemia, corticosteroid induced osteoporosis and obstructive lung disease, with avascular necrosis of the proximal row of the carpus (Fig. 9.11) and the hamate is described. No other sites of avascular bone necrosis were identified. This patient, however, similar to the previously described one, had several contributing factors for avascular necrosis.
- For other isolated carpal bone AVN's, please see Table 9.6:

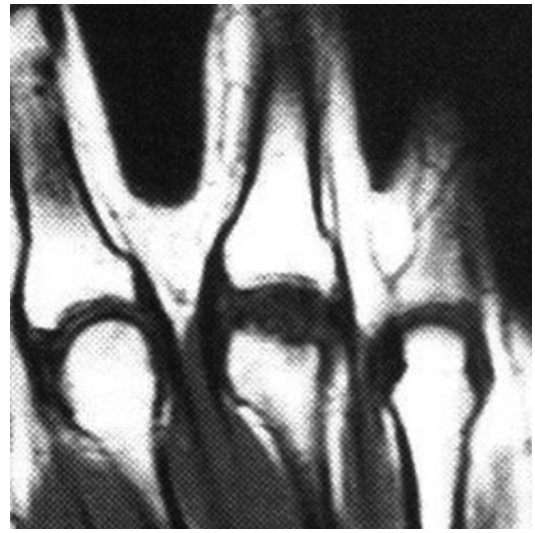

Fig. 9.12 AVN of head of the metacarpal

Avascular Necrosis of the Head of the Metacarpal: Dieterich Disease

Avascular necrosis of the metacarpal head, or Dieterich's disease, is also extremely rare and has been associated with trauma, systemic lupus erythematosis (SLE), congenitally short digits and steroid use [86] (Fig. 9.12).

It has been reported in all the metacarpal heads, but appears to involve most frequently the long finger, followed by the index, ring and small fingers. The thumb is least commonly involved [87].

Wright and Dell, who studied the vascularity of the metacarpals, found that in 35 % of the specimens, the main artery to the distal epiphysis was absent, making these metacarpal heads solely dependent on small circumferential peri-capsular vessels [88]. This pattern is even more frequent in the middle finger (60 %). Blunt trauma to the metacarpal head, with joint effusion, may compress the periosteal blood vessels, causing necrosis. In SLE, avascular necrosis occurs as a result of vasculitis involving these vessels.

If the diagnosis is not obvious on standard radiographs and there is only slight flattening of the metacarpal head or disruption of the trabecular pattern, further investigation is necessary.

A technetium bone scan will show an area of increased uptake at the involved MCP joint. At the same time, it is useful to screen the other bones in patients using steroids. Recently, MRI has proven to be very useful in establishing the diagnosis of avascular necrosis. It also quantified the extent of the necrotic zone and any involvement of the overlying cartilage. Usually MRI demonstrates a hypointense zone on T1-weighted images and a hyperintense zone on T2-weighted images, both suggestive of avascular necrosis.

Several treatment options, ranging from rest to surgery, exist. The type of surgery depends on the quality of the cartilage layer. If this is still good, subchondral debridement and cancellous bone grafting gives excellent results. If the cartilage is destroyed however, arthroplasty or arthrodesis may be necessary.

References

1. Gelberman R, Gross M. The vascularity of the wrist. Clin Orthop. 1986;202:40–9.
2. Wagner JP, Chung KC. A historical report on Robert Kienböck (1871–1953) and Kienböck's disease. J Hand Surg. 2005;30A:1117–21.
3. Schmitt R, Fellner F, Obletter N, Fiedler E, Bautz W. Diagnosis and staging of lunate necrosis. A current review. Handchir Mikrochir Plast Chir. 1998;30:142–50.
4. Lichtman DM, Alexander AH, Mack GR, Gunther SF. Kienböck's diseas–pdate on silicone replacement arthroplasty. J Hand Surg Am. 1982;7A:343–74.
5. Baine GI, Begg M. Arthroscopic assessment and classification of Kienbock's disease. Tech Hand Up Extrem Surg. 2006;10:8–13.
6. Gelberman RH, Bauman TD, Menon J, Akeson WH. The vascularity of the lunate bone and Kienböck's disease. J Hand Surg Am. 1980;5A:272–8.
7. Hulten O. Uber anatomische variationene der Handgelenckknochen. Acta Radiol. 1928;9:155–69.
8. Epner R, Bowers W, Guilford W. Ulnar variance the effect of positioning on roentgen filming technique. J Hand Surg Am. 1982;7:298–305.
9. Palmer AK, Glisson RR, Werner FW. Ulnar variance determination. J Hand Surg Am. 1982;7:376–9.
10. Weiss AP. Negative ulnar variance is not a risk factor for Kienböck's disease. J Hand Surg. 1994;19-A:1057–8.
11. Kristensen SS, Thomassen E, Chistensen F. Ulnar variance in Kienböck's disease. J Hand Surg. 1986;11-B: 258–60.
12. Goemine S, Degreef I, De Smet L. Negative ulnar variance has a prognostic value in the progression of Kienböck's disease. Acta Orthop Belg. 2010;76:38–41.
13. Ledoux P, Lamblin D, Wuilbaut A, Schuind F. A finite-element analysis of Kienbock's disease. J Hand Surg. 2008;33 Eur:286–91.
14. Mirabello SC, Rosenthal DI, Smith RJ. Correlation of clinical and radiographic findings in Kienböck's disease. J Hand Surg. 1987;12-A:1049–54.
15. Antuna-Zapico JM. Malacia del Semilunar. Doctoral thesis, Valladolid, 1966.
16. Stahl F. Lunatomalacia. Acta Chir Scand Suppl. 1947;95:126.
17. Kristensen SS, Thomassen E, Christensen F. Kienböck's disease–late results by non-surgical treatment. A follow-up study. J Hand Surg. 1986;11 Br: 422–5.
18. Mikkelsen SS, Gelineck J. Poor function after nonoperative treatment of Kienböck's disease. Acta Orthop Scand. 1987;58:241–3.
19. Keith PP, Nuttall D, Trail I. Long-term outcome of nonsurgically managed Kienböck's disease. J Hand Surg. 2004;29 Am:63–7.
20. Salmon J, Stanley JK, Trail IA. Kienböck's disease: conservative management versus radial shortening. J Bone Joint Surg. 2000;82 Br:820–3.
21. Saffar P. Replacement of the semilunar bone by the pisiform. Description of a new technique for the treatment of Kienboeck's disease. Ann Chir Main. 1982;1: 276–9.
22. Moneim MS, Duncan GJ. Kienbock's disease: treatment by implantation of vascular pedicle and bone grafting. Iowa Orthop J. 1998;18:67–73.
23. Jones NF, Brown EE, Vögelin E, Urist MR. Bone morphogenetic protein as an adjuvant in the treatment of Kienbock's disease by vascular pedicle implantation. J Hand Surg Eur Vol. 2008;33Eur: 317–21.
24. Moran SL, Cooney WP, Berger RA, Bishop AT, Shin AY. The use of the 4 + 5 extensor compartmental vascularised bone graft for the treatment of Kienböck's disease. J Hand Surg. 2005;30A:50–8.
25. Bengoechea-Beeby MP, Cepeda-Uña J, Abascal-Zuloaga A. Vascularized bone graft from the index metacarpal for Kienböck's disease: a case report. J Hand Surg [Am]. 2001;26:437–43.
26. Zafra M, Carrasco-Becerra C, Carpintero P. Vascularised bone graft and osteotomy of the radius in Kienböck's disease. Acta Orthop Belg. 2005;71:163–8.
27. Arora R, Lutz M, Deml C, Krappinger D, Zimmermann R, Gabl M. Long-term subjective and radiological outcome after reconstruction of Kienböck's disease stage 3 treated by a free vascularized iliac bone graft. J Hand Surg. 2008;33 Am:175–81.
28. Gabl M, Lutz M, Reinhart C, Zimmerman R, Pechlaner S, Hussl H, Rieger M. Stage 3 Kienböck's disease: reconstruction of the fractured lunate using a free vascularized iliac bone graft and external fixation. Hand Surg. 2002;27 Br:369–73.
29. Armistead RB, Linscheid RL, Dobyns JH, Beckenbaugh RD. Ulnar lengthening in the treatment of Kienböck's disease. J Bone Joint Surg. 1982;64 Am:170–8.

30. Axelson R. Niveau Operationen bei Mondbein nekrose. Handchirurgie. 1973;5:187–96.
31. Rosemeyer B, Artmann M, Viernstein K. Kienbock's disease. Follow up studies and therapeutic considerations. Arch Orthop Unfallchir. 1976;85(1):119–27.
32. Gomis R, Martin B, Idoux O, Chammas M, Allieu Y. Kienboeck disease: treatment by shortening osteotomy of the radius. Rev Chir Orthop Reparatrice Appar Mot. 1994;80:196–204.
33. Nakamura R, Imaeda T, Miura T. Radial shortening for Kienböck's disease: factors affecting the operative result. J Hand Surg. 1990;15 Br:40–5.
34. Quenzer DE, Dobyns JH, Linscheid RL, Trail IA, Vidal MA. Radial recession osteotomy for Kienböck's disease. J Hand Surg. 1997;22 Am:386–95.
35. Siala A, Ben Ayeche ML, Frikha R, Ghannouchi G, Moula T. Results of diaphyseal shortening of the radius in the treatment of Kienböck's disease: a series of 31 cases. Rev Chir Orthop Reparatrice Appar Mot. 2000;86:151–7.
36. Sundberg SB, Linscheid RL. Kienböck's disease. Results of treatment with ulnar lengthening. Clin Orthop Relat Res. 1984;187:43–51.
37. Trail IA, Linscheid RL, Quenzer DE, Scherer PA. Ulnar lengthening and radial recession procedures for Kienböck's disease. Long-term clinical and radiographic follow-up. J Hand Surg. 1996;21 Br: 169–76.
38. Weiss AP. Radial shortening. Hand Clin. 1993;9: 475–82.
39. Schattenkerk ME, Nollen A, van Hussen F. The treatment of lunatomalacia. Radial shortening or ulnar lengthening? Acta Orthop Scand. 1987;58(6):652–4.
40. De Smet L, Fabry G. Orientation of the sigmoid notch of the distal radius. Acta Orthop Belg. 1993;59: 269–72.
41. Sagerman SD, Zogby RG, Palmer AK, Werner FW, Fortino MD. Relative articular inclination of the distal radioulnar joint: a radiographic study. J Hand Surg [Am]. 1995;20:597–601.
42. Horii E, Garcia-Elias M, Bishop AT, Cooney WP, Linscheid RL, Chao EY. Effect on force transmission across the carpus in procedures used to treat Kienböck's disease. J Hand Surg. 1990;15 Am:393–400.
43. Werner FW, Palmer AK. Biomechanical evaluation of operative procedures to treat Kienböck's disease. Hand Clin. 1993;9:431–43.
44. Wada A, Miura H, Kubota H, Iwamoto Y, Uchida Y, Kojima T. Radial closing wedge osteotomy for Kienböck's disease: an over 10 year clinical and radiographic follow-up. J Hand Surg. 2002;27 Br:175–9.
45. Garcia-Elias M, An KN, Cooney WP, Linscheid RL. Lateral closing wedge osteotomy for treatment of Kienböck's diseas. A clinical and biomechanical study of the optimum correcting angle. Chir Main. 1998;17:283–90.
46. Watanabe K, Nakamura R, Horii E, Miura T. Biomechanical analysis of radial wedge osteotomy for the treatment of Kienböck's disease. J Hand Surg. 1993;18A:686–90.
47. Illarranmendi AA, Schulz C, De Carli P. The surgical treatment of Kienböck's disease by radius and ulna metaphyseal core decompression. J Hand Surg. 2001;26 Am:252–60.
48. Schulz C, De Carli P, Anetzberger H, Illarramendi A. Stress osteotomy of the distal radius- and ulna metaphysis (Illarramendi procedure): an alternative treatment method in lunate necrosis. Handchir Mikrochir Plast Chir. 1998;30:188–95.
49. Watson HK, Hempton R. Limited wrist arthrodesis. 1 The triscaphoid joint. J Hand Surg Am. 1980;5:320–7.
50. Sauerbier M, Tränkle M, Erdmann D, Menke H, Germann G. Functional outcome with scaphotrapeziotrapezoid arthrodesis in the treatment of Kienböck's disease stage III. Ann Plast Surg. 2000;44:618–25.
51. Watson HK, Monacelli DM, Milford RS, Ashmead IV D. Treatment of Kienböck's disease with scaphotrapeziotrapezoid arthrodesis. J Hand Surg. 1996;21A:9–15.
52. Minami A, Kimura T, Suzuki K. Long-term results of Kienböck's disease treated by triscaphe arthrodesis and excisional arthroplasty with a coiled palmaris longus tendon. J Hand Surg. 1994;19 Am:219–28.
53. Tambe AD, Trail IA, Stanley JK. Wrist fusion versus limited carpal fusion in advanced Kienbock's disease. Int Orthop. 2005;29:355–8.
54. Sennwald GR, Ufenast H. Scaphocapitate arthrodesis for the treatment of Kienböck's disease. J Hand Surg. 1995;20 Am:506–10.
55. Oishi SN, Muzaffar AR, Carter PR. Treatment of Kienbock's disease with capitohamate arthrodesis: pain relief with minimal morbidity. Plast Reconstr Surg. 2002;109:1293–300.
56. Wilhelm K, Hierner R, Brehl B. Callus distraction for progressive lengthening of the capitate bone after resection of the lunate bone in stage III lunate malacia. Surgical technique and 1 year results. Handchir Mikrochir Plast Chir. 1997;29:10–9.
57. Lu L, Gong X, Liu Z, Zhang Z. Capitate transposition to replace necrotic lunate bone with a pedicle for Kienbock's disease: review of 30 cases. Chin Med J (Engl). 2003;116:1519–22.
58. Kilgus M, Weishaupt D, Künzi W, Meyer VE. Radioscapholunate fusion: long-term results. Handchir Mikrochir Plast Chir. 2003;35:317–22.
59. Tambe A, Ali F, Trail I, Stanley J. Is radiolunate fusion a viable option in advanced Kienböck disease? Acta Orthop Belg. 2007;73:598–603.
60. Sauerbier M, Kluge S, Bickert B, Germann G. Subjective and objective outcomes after total wrist arthrodesis in patients with radiocarpal arthrosis or Kienböck's disease. Chir Main. 2000;19:223–31.
61. Swanson AB, de Groot Swanson G. Implant resection arthroplasty in the treatment of Kienböck's disease. Hand Clin. 1993;9:483–91.
62. Kaarela OI, Raatikainen TK, Torniainen PJ. Silicone replacement arthroplasty for Kienböck's disease. J Hand Surg. 1998;23 Br:735–40.
63. De Smet L, Robijns P, Degreef I. Proximal row carpectomy in advanced Kienbock's disease. J Hand Surg. 2005;30 Br:585–7.

64. Iwasaki N, Minami A, Oizumi N, Suenaga N, Kato H, Minami M. Radial osteotomy for late-stage Kienböck's disease. Wedge osteotomy versus radial shortening. J Bone Joint Surg. 2002;84 Br:673–7.
65. Nakamura R, Horii E, Watanabe K, Nakao E, Kato H, Tsunoda K. Proximal row carpectomy versus limited wrist arthrodesis for advanced Kienböck's disease. J Hand Surg. 1998;23 Br:741–5.
66. Begley B, Engber W. Proximal row carpectomy in advanced Kienböck's disease. J Hand Surg. 1994;19 Am:1016–8.
67. Preiser G. Eine typische posttraumatische und zur Spontanfraktur fuhrende Osteitis des Naviculare carpi. Fortschr Geb Roentgenstr. 1910;15:189–97.
68. Gelberman R, Menon J. The vascularity of the scaphoid bone. J Hand Surg Am. 1980;5:508–13.
69. Taleisnik J, Kelly P. The extraosseous and intraosseous blood supply of the scaphoid bone. J Bone Joint Surg. 1966;48 Am:1125–37.
70. Vidal M, Linscheid R, Amadio P, Dobyns J. Preiser's disease. Ann Hand Up Limb Surg. 1991;10:227–36.
71. Herbert TJ, Lanzetta M. Idiopathic avascular necrosis of the scaphoid. J Hand Surg. 1994;19 Br:174–82.
72. Kaleinov D, Cohen M, Hendrickx RW, Sweet S, Culp R, Osterman A. Preiser's disease: identification of two patterns. JHand Surg. 2003;28 Am:767–8.
73. Parkinson R, Noble J, Bale R, Freemont AJ. Rare abnormalities of the scaphoid in association with congenital radial ray defects of the hand. J Hand Surg. 1991;16 Br:208–11.
74. De Smet L, Aerts P, Walraevens M, Fabry G. Avascular necrosis of the carpal scaphoid: Preiser's disease. Report of 6 cases and review of the literature. Acta Orthop Belg. 1993;59:139–42.
75. Alnot JY, Frayman JM, Bocquet L. Les ostéonécroses aseptiques primitives totales du scaphoïde. A propos de 3 cas. Ann Chir Main Membre Super. 1990;9: 221–5.
76. Moran S, Cooney W, Shin A. The use of vascularized grafts from the distal radius for the treatment of Preiser's disease. J Hand Surg. 2006;31 Am:705–10.
77. Jönsson G. Aseptic necrosis of the os capitatum. Acta Radiol. 1942;23:562–4.
78. Dereudre G, Kaba A, Pansard E, Mathevon H, Mares O. Avascular necrosis of the capitate: case report and a review of the literature. Chir Main. 2010;29:203–6.
79. Milliez PY, Kinh Kha H, Allieu Y, Thomine JM. Idiopathic aseptic osteonecrosis of the capitate bone. Literature review apropos of 3 new cases. Int Orthop. 1991;15:85–94.
80. Murakami H, Nishida J, Ehara S, Furumachi K, Shimamura T. Revascularization of avascular necrosis of the capitate bone. Am J Roentgenol. 2002;179: 664–6.
81. Por Y, Chew W, Tsou I. Avascular necrosis of the triquetrum. Hand Surg. 2005;10:91–4.
82. Match R. Non-specific avascular necrosis of the pisiform bone. J Hand Surg. 1980;5 Am:341–2.
83. Olah J. Bilateral aseptsche Nekrose des Os Pisiforme. Z Orthop Ihre Grenzgeb. 1968;140:590.
84. Garcia-Lopez A, Cardoso Z, Ortega L. Avascular necrosis of the trapezium bone: a case report. J Hand Surg. 2002;27 Am:704–6.
85. Struzegger M, Mencarelli F. Avascular necrosis of the trapezoid bone. J Hand Surg. 1998;23B:550–1.
86. De Smet L. Avascular necrosis of multiple carpal bones. Ann Chir Main Memb Super. 1999;18:202–4.
87. Dieterich's H. Subchondrale Herderkrankung am Metacarpale III. Arch Klin Chir. 1932;171:555–67.
88. Wright T, Dell P. Avascular necrosis and vascular anatomy of the metacarpals. J Hand Surg. 1991; 16-Am:540–4.