
Osteonecrosis and Osteochondrosis

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Abstract

This succinct chapter on osteonecrosis and osteochondrosis in the wrist and hand describes the aetiology and typical bones involved in these conditions. A description of the available of imaging modalities including plain radiographs, computed tomography, scintigraphy and Magnetic resonance imaging are described with their strengths and weaknesses. Detailed individual description and classification of carpal bone osteonecrosis and osteochondrosis including Kienbock's disease of the lunate and the rarer Preiser's disease of the scaphoid are included.

1 Introduction

Osteonecrosis or avascular necrosis of the carpal bones is a common cause for chronic wrist pain, limiting movement and weakening grip strength. In the wrist osteonecrosis most frequently occurs as a result of disruption to the osseous blood supply as a result of trauma. While the scaphoid is the most frequently affected bone in the wrist, osteonecrosis of any of the carpal bones can occur and multiple carpal bone involvement is described (De Smet et al. 1993; Humphrey et al. 2006; Telfer et al. 1994; Zafra et al. 2004; Garcia and Vaca 2006).

The osteochondroses refer to a heterogenous group of conditions with a tendency to affect the immature skeleton. A variety of possible mechanisms have been proposed for these conditions, but those affecting the wrist and hand are characterized by features of osteonecrosis thought to occur through a process of microtrauma.

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The most well recognized of these conditions in the hand and wrist is Kienböch's disease affecting the lunate.

Osteonecrosis from any cause can result in late complications, most commonly osteoarthritis and although surgical options meet with varied success, early diagnosis may limit these complications. Conventional radiographs are often the first line imaging investigation but it should be appreciated that the changes seen are relatively late in the disease process. MR imaging is extremely well suited to the detection of the early phases of avascular necrosis, permitting diagnosis before collapse of the carpal bones has occurred (Golimbu et al. 1995).

2 Aetiology

The term osteonecrosis by definition implies bone necrosis or bone death. Originally infection was thought to be the underlying cause but histopathological studies showed that the bone is aseptic and also avascular, implicating vascular insufficiency as the underlying aetiology. A variety of insults may lead to osseous vascular impairment and carpal bones are particularly vulnerable to such insults as they have a poor collateral circulation. Notably osteonecrosis occurs most often in the scaphoid, lunate and capitate bones which have particularly tenuous vascularity (Botte et al. 2004; Gelberman and Gross 1986).

Vascular impairment and therefore osteonecrosis may result from a variety of underlying causes including radiation, vascular, metabolic and systemic conditions. In the wrist the most common cause is trauma, with the scaphoid and capitate most frequently affected. A clear cause for osteonecrosis is not always established. This situation most commonly arises in the lunate where it is termed Kienböch's disease. Despite the absence of a history of trauma it is generally believed that the aetiology of these conditions is traumatic in nature, rather than relating to a primary vascular event (Resnick 2002). Other osteochondroses described in the hand and wrist are Preiser's disease (scaphoid), Thiemann's disease (phalanges) and Dieterich's disease (metacarpal head) (Botte et al. 2004; de Smet 2000; Ferlic and Morin 1989; Herbert and Lanzetta 1994; Tashjian et al. 2004).

The different cellular elements within bone (including osteocytes, osteoblasts and osteoclasts) differ in their susceptibilities to anoxia. After physical

disruption of blood flow, the haemopoietic elements are generally acknowledged to be the first to undergo anoxic death followed by bone cells and subsequently bone marrow fat cells (Crues 1986; Glimcher and Kenzora 1979a, b, c). This means that temporary anoxia can result in varying degrees of cell death potentially affecting the haemopoietic elements without necessarily involving the other bone cells. It is generally recognised that once marrow fat cell death occurs the involved segment of bone can be labelled as infarcted. Marrow involvement is critical to the detection of bone infarction on MRI examination.

3 Imaging Techniques

3.1 Radiography

In most cases the X-ray findings lag behind the clinical manifestations, hence the initial radiographs may appear normal. The onset of fat necrosis brings with it vasodilation in the still vascularised surrounding bone resulting in an influx of inflammatory cells. Dead bone starts to undergo removal by osteoclasts and this means that potentially viable bone will become relatively osteopaenic. This comparatively radiolucent bone will be adjacent to non-viable necrotic bone which becomes relatively sclerotic (Fig. 1). As the process progresses increasing sclerosis followed by collapse and fragmentation of the avascular bone occurs.

3.2 Computed Tomography

Computerised tomography is rarely used in the primary assessment of carpal bone osteonecrosis. However the complex 3-D anatomy of the carpal bones means it can be useful for providing information relating to the extent of volume reduction of the affected bone and the detection of early subchondral collapse, along with the detection of secondary changes such as osteoarthritis. It can also prove more sensitive to the detection of an associated un-united fracture and sclerosis in the avascular bone.

The base of the hook of hamate is at a relatively high risk for avascular necrosis following fracture (Telfer et al. 1994). The complex anatomy of this bone means avascular necrosis is often best detected at CT.



Fig. 1 Early avascular necrosis 8 weeks following a scaphoid fracture. **a** Ulnar deviated PA film with tube angulation and **b** Anterior Oblique shows a fracture line. The difference in

density can be appreciated between the increased density of the proximal pole, accentuated by the relative osteopaenia of the distal pole

CT changes seen in osteonecrotic bone reflect the plain film findings of sclerosis, appreciated as increased attenuation and subsequent collapse and fragmentation (Fig. 2). Cystic change may be seen along the margins of an un-united fracture (Bush et al. 1987).

3.3 Magnetic Resonance Imaging

Magnetic resonance is the imaging modality of choice for the early detection of osteonecrosis, depicting marrow signal changes before changes are apparent using plain films and CT (Imhof et al. 1997). Although scintigraphy may offer similar sensitivity it shows less specificity when compared with MRI. The signal abnormality on MRI depends on alteration of the fat cells in the bone marrow, initially seen as oedema-like change within the marrow, appreciated as a reduction in the normal fat signal on T1 weighted imaging and increased signal on water sensitive sequences (Imhof et al. 1997; Mitchell et al. 1989) (Fig. 3). As the process progresses areas of reduced signal intensity on T2 weighted imaging emerge, surrounded by areas of increased signal intensity representing the interface between non-viable bone and reparative granulation tissue. At this stage MRI may show a characteristic serpiginous double line at



Fig. 2 Scaphoid fracture with early avascular necrosis. The sagittal CT reformat shows increased attenuation to the proximal pole of the scaphoid reflecting osteonecrotic bone with relative osteopaenia in the distal pole

the interface between the necrotic area and the fibrovascular granulation tissue. The double line represents the granulation tissue adjacent to reactive

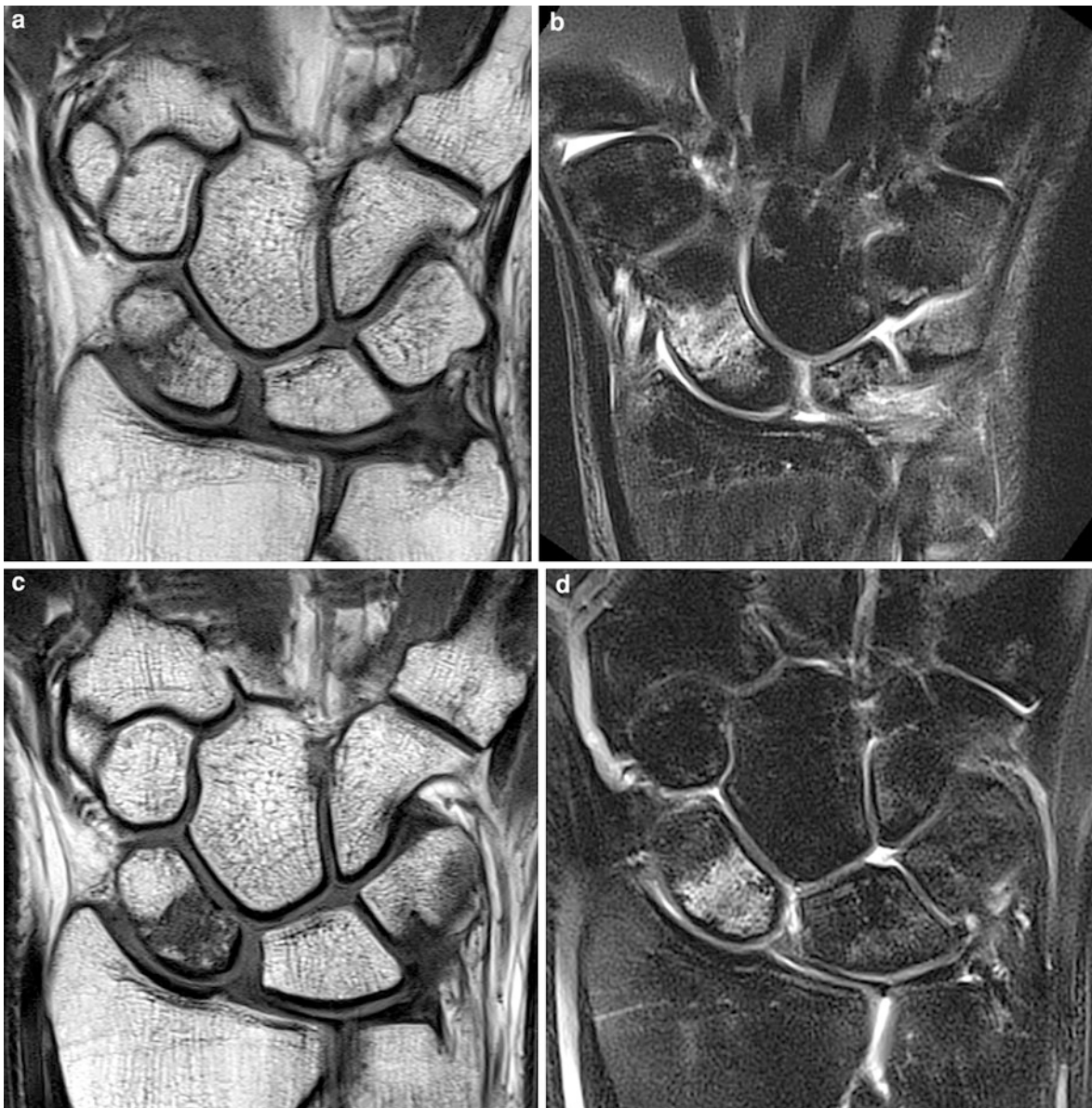


Fig. 3 **a** Coronal T1 and **b** Coronal Oblique T2 (fat sat) MR images obtained 18 days following scaphoid fracture. On the T1 the fracture line interrupts the marrow signal with patchy signal reduction around the fracture line seen as increased T2 signal (oedema) on the T2 image. Note the normal marrow

signal at the proximal pole. Five weeks later (**c**) Coronal T1 MR image shows diffuse low signal and (**d**) Coronal T2 (fat sat) image shows diffuse increased signal in the proximal pole with normal signal in the distal pole. The appearances represent early AVN

bone formation (Imhof et al. 1997; Mitchell et al. 1989). While the two lines may not always be distinguished a sharp line of demarcation is still a typical and helpful feature (Fig. 4). Contrast-enhanced imaging can be performed and may help distinguish viable from non-viable bone but should be interpreted with caution. See Sect. 4.1.

3.4 Bone Scintigraphy

The relative insensitivity of radiographs for detecting the early stages of avascular necrosis has led to the increased use of MRI for this role. Bone scintigraphy is also very sensitive to the early detection of osteonecrosis although it is relatively non-specific and

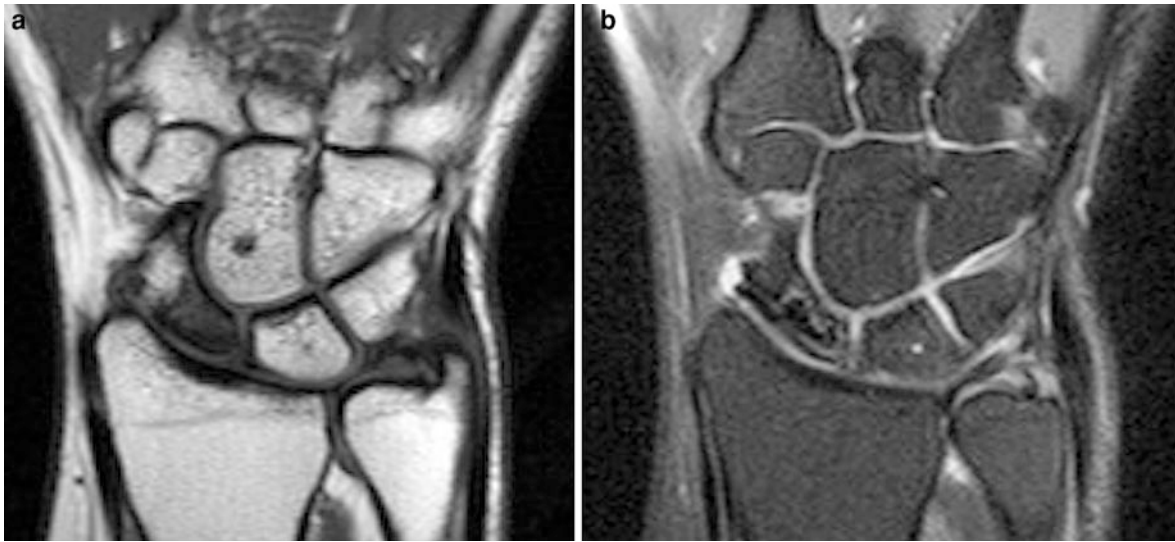


Fig. 4 **a** Coronal T1 W shows abnormal low signal within the proximal scaphoid bone **b** PD (fat sat) image also shows abnormal low signal delineated by a high signal serpiginous line. There is surrounding abnormal low signal within the

proximal scaphoid bone demarcating the dead bone within the proximal pole and representing granulation tissue. There was no history of trauma in this case, which represents the partial form of Preiser's disease

should always be interpreted alongside conventional radiographic findings. In the early stages of avascular necrosis increased metabolic activity in the bone is seen as higher uptake in bone scintigraphy on early and late phase scanning. Later bone scintigraphy typically indicates an area of decreased radio-tracer uptake; a cold spot or photopenic region. This in isolation is not diagnostic of osteonecrosis as other conditions including myeloma, some skeletal metastases, irradiation, haemangiomas and infection can also result in reduced or impaired tracer uptake.

4 Scaphoid

The scaphoid is the most common site for osteonecrosis to occur in the wrist. This is because of its high susceptibility to trauma and precarious blood supply. The proximal pole of the scaphoid receives its blood supply in a retrograde fashion from small dorsal ridge vessels entering the waist of the scaphoid from the radial artery. These vessels provide around 70–80% of the internal vascularity of the bone, in particular to the proximal pole. A separate vascular supply from the palmar aspect supplies the remaining internal vascularity, all to the region of the distal pole (Botte et al. 2004). The consequent tenuous blood

supply to the proximal pole makes it vulnerable to osteonecrosis following fracture. There is a reported frequency of proximal pole osteonecrosis of between 11 and 65% following scaphoid fracture (Mazet and Hohl 1963). However this varies according to the site of the fracture, with fractures of the proximal third having the highest incidence of osteonecrosis and a reducing incidence with more distally located fractures. Osteonecrosis has been reported as occurring in the distal pole of the scaphoid following fracture, but this is extremely rare (Sherman et al. 1983).

Plain radiographs of established cases show an increased bone density in the affected scaphoid seen at between 4 and 8 weeks and associated with non-union (Fig. 1). Later reduction in size, fragmentation and secondary degenerative changes can be seen (Fig. 5).

MR imaging is now the modality of choice for the detection of early avascular necrosis. Typically, MR will show low signal intensity within the bone marrow of the proximal pole on both the T1 and T2 weighted images. It has been suggested that high signal on T2 implies viability of bone but this is not the case (Cerezal et al. 2000) (Fig. 6a). Contrast enhancement significantly increases the detection of non-viable bone but the use of dynamic enhancement studies does not appear to improve sensitivity (Cerezal et al. 2000; Megerle et al. 2011; Donati et al.



Fig. 5 Established proximal pole scaphoid osteonecrosis 6 months following fracture. There is fracture non-union with dense sclerosis and volume loss in the proximal pole. Cystic change is also seen about the fracture line

2011) (Fig. 6b). While studies do indicate that MRI provides a good assessment of the vascularity of the bone as assessed with punctate bleeding at the time of surgery; the general picture that is emerging suggests that MRI, even with gadolinium enhancement, is less good at predicting outcome of surgery in terms of union and revascularization (Cerezal et al. 2000; Megerle et al. 2011; Schmitt et al. 2011; Singh et al. 2004; Dawson et al. 2001).

4.1 Preiser's Disease

Preiser's disease is a rare condition which results in ischaemia of the scaphoid bone without a history of trauma. Rarely it can occur bilaterally and has also been described with Kienböck's disease (de Smet 2000; Ferlic and Morin 1989; Herbert and Lanzetta 1994) (Fig. 7). Risk factors may include excessive alcohol intake, corticosteroid administration, chemotherapy and other disease processes.

Histologically, there are empty bony lacunae, necrotic marrow and partly degenerated articular cartilage. Clinically, pain along the dorsal and radial aspects of the wrist is typical.

Conventional radiographic images and CT show the expected sclerosis, volume loss and cystic change which, with time progresses to collapse and fragmentation (Fig. 7 and 8). Using MRI two patterns can be identified, a partial or focal form with ischaemia of the proximal half of the scaphoid (Fig. 4) and a diffuse form with complete ischaemia and necrosis (Fig. 7) (Kalainov et al. 2003). The former, partial type has a better prognosis. Isolated non-traumatic osteonecrosis of the distal pole of the scaphoid has also been described (Garg et al. 2011).

Treatment protocols for Preiser's disease include wrist immobilization and cortisone injections. Surgical options include partial excision with replacement by non-vascularised or vascularised bone graft, proximal row carpectomy, scaphoid excision with midcarpal fusion and total wrist fusion.

5 Lunate

5.1 Kienböck's Disease

Kienböck's disease is most commonly observed in patients between the ages of 20 and 40 years, with a predilection for the right hand in persons engaged in manual labour. Some patients have an antecedent history of trauma but the majority of cases are idiopathic. There is a strong association between ulna minus variance and Kienböck's disease, which has led to the suggestion that abnormality in loading is an important aspect in the aetiology, although 40% of cases are seen in neutral ulna variance. Additionally, negative ulnar variance is usually bilateral and Kienböck's disease is rarely bilateral. Other theories that have been postulated include medialisation of the lunate, horizontalisation of the radial epiphysis or abnormal morphology of the lunate. In some individuals there is a variation in the blood supply of the lunate; a single dominant branch (8%) rather than a dual supply from volar and dorsal branches with an anastomosis potentially increasing the risk of osteonecrosis (Gelberman and Gross 1986).

5.1.1 Imaging

The appearances of established lunate osteonecrosis on conventional radiographs is initially sclerosis followed by progressive collapse of the lunate and fragmentation (Figs. 9 and 10). Associated findings such as ulnar negative variance may also be seen and in the later stages degenerative change may occur.

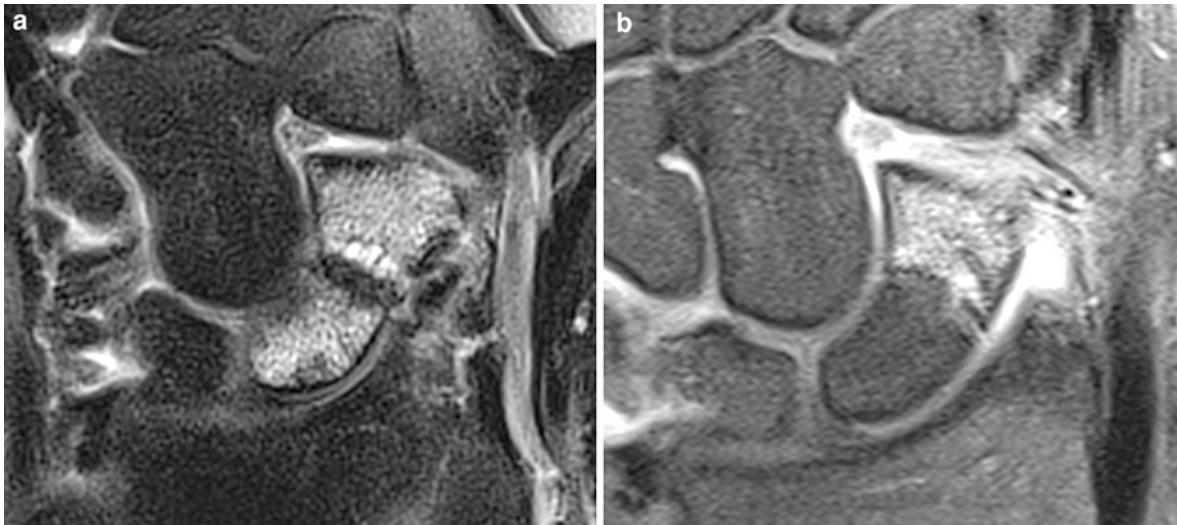


Fig. 6 **a** Coronal T2 W (fat sat) shows scaphoid fracture non-union. High signal oedema is seen in the marrow in both the proximal and distal fracture fragments. **b** Coronal T1 W (fat sat) post intravenous gadolinium shows oedema-induced

enhancement in the distal pole with absent enhancement of the oedematous but avascular proximal pole despite the presence of oedematous change on T2 W imaging



Fig. 7 Bilateral Preiser's disease. **a** Reformatted CT. There is sclerosis, fracturing and fragmentation with volume loss in the scaphoid of this patient without trauma. **b** T1 weighted and **c** T2 weighted (fat sat) MR images from the contralateral wrist

show the condition is bilateral, with diffuse signal abnormality throughout the scaphoid with a serpiginous boundary between representing the interface between necrotic bone and granulation tissue Courtesy of Dr Phillip Tirman

Radiographs are important in differentiating osteonecrosis from other causes of wrist pain such as ulna-lunate abutment, fractures, carpal malalignment, instability and arthritis.

CT of the wrist is more sensitive than plain films for demonstrating structural change including sclerosis, compression and fractures of the lunate. Fractures are more common in Kienböck's disease than previously reported (Fig. 11). Earlier detection of fractures by CT, before collapse of the lunate occurs, may allow treatment to prevent the collapse (Friedman et al. 1991).

As with osteonecrosis at other sites, MRI and scintigraphy provide earlier evidence of Kienböck's disease and can help establish the diagnosis in the presence of normal conventional radiographs. Two patterns of abnormality have been defined on MRI, one involving only focal involvement of the radial half of the lunate, the second being a diffused reduction in T1 signal throughout the bone marrow (Sowa et al. 1989 Nov) (Fig. 12). In common with osteonecrosis elsewhere, marrow signal appears low on T2 imaging in the later stages of the disease, but initially high signal may



Fig. 8 Preiser's disease. The scaphoid shows sclerosis and volume loss due to osteonecrosis. There was no history of trauma in this 25-year-old male presenting with wrist pain and successfully treated with a vascularised graft Courtesy of Kimberly K. Amrami MD. Mayo Clinic, Rochester Minnesota, USA

be seen, which may be focal or patchy in distribution. Cystic change and collapse/fragmentation may be seen in the later stages of the disease (Fig. 13).

5.1.2 Staging of Kienböck's Disease

Although many classifications have been proposed they are quite similar, four stages are recognised in the commonly used Lichtman classification (Lichtman et al. 1977).

Stage I: Pre-radiological disease.

The plain radiograph is normal except for possible predisposing factors such as negative ulnar variance. Detection of stage 1 disease is by MRI where there is typically internal bone marrow signal change with low signal on T1 W and low or patchy high signal on T2 W imaging. Stage 1 disease can also be detected by bone scintigraphy.

Stage II: Sclerosis of the lunate bone without collapse (Fig. 9).

Stage III: Lunate fragmentation and collapse.

As a consequence of collapse there may be associated scapholunate dissociation and rotatory subluxation of the scaphoid. This has led to subdivision of stage III into stage IIIA: no rotatory subluxation and IIIB: associated scapholunate dissociation and rotatory subluxation.



Fig. 9 Plain radiograph showing sclerosis of the lunate typical features of Kienböck's disease. Note the presence of ulnar negative variance, a typical but not exclusive association with Kienböck's disease

Stage IV: Arthritis of the radiocarpal and mid carpal joints.

Although there is no difficulty in establishing the diagnosis of Kienböck's disease with MRI, many therapeutic options exist. Along with conservative management there are surgical options, including revascularisation of the lunate and lunate unloading by ulnar lengthening or radial shortening. However there is no strong consensus on the indications and results are variable (Kristensen et al. 1986; Weiss et al. 1991).

6 Capitae

Osteonecrosis of the capitae is recognised following fracture or more commonly following midcarpal fracture dislocations (scaphocapitate syndrome) (Tashjian et al. 2004). Although idiopathic osteonecrosis without a history of antecedent trauma is described it is rare (Kutty and Curtin 1995; Lapinsky and Mack 1992). The vascular anatomy of the capitae shares similar features



Fig. 10 Kienböck's disease at a more advanced stage than shown in Fig. 8, with subchondral fracturing and collapse at the proximal pole, again with ulnar minus variance



Fig. 12 Kienböck's Disease. Coronal T1 W image shows diffuse low signal change in the lunate, but there are focal regions of increased and intermediate signal intensity which are often observed in bone necrosis. Courtesy of Dr Muhammed Mubashar, University Hospital South Manchester, UK



Fig. 11 Sagittal CT reformat shows advanced Kienböck's disease with sclerosis, fracture and fragmentation. Early secondary degenerative change at the radioulnar joint can be appreciated as irregular joint space loss and cystic change in the subchondral bone of the radius

to those of the scaphoid with a retrograde internal flow pattern which explains the predominant proximal pole pattern of necrosis.

The changes described on X-ray are of initial proximal pole osteopaenia followed by sclerosis. This may progress to complete fragmentation and collapse. MRI will show typical features of osteonecrosis as previously described.

Treatment often involves excision of the capitate bone with carpal fusion, revascularisation has also been successfully achieved (Murakami et al. 2002).

7 Hamate

Avascular necrosis of the hamate is relatively uncommon and in the cases reported follows trauma. Post-traumatic osteonecrosis of both the hook of hamate and body have been described (Telfer et al.



Fig. 13 Coronal T2 W image showing advanced Kienböck's disease. High signal change is seen associated with cystic change and volume loss

1994; Failla 1993). It has been noted that like the scaphoid and capitate the proximal pole is vascularised via a retrograde circulation and, as would be expected, the proximal pole seems to be more vulnerable (Botte et al. 2004; Tashjian et al. 2004).

8 The Hand

Osteonecrosis has been reported in a variety of bones in the hand, most commonly in the metacarpal heads and about the proximal interphalangeal (PIP) joints of the phalanges. Thiemann's disease is reported as a rare autosomal-dominant form of avascular necrosis affecting the PIP joint (Tashjian et al. 2004). The condition has also been termed juvenile osteochondropathy of the fingers and multiple phalangeal epiphysitis. It usually presents as progressive enlargement of the PIP joints, frequently starting with the middle finger before involving the index and ring fingers. There is often sparing of the little PIP and thumb IP joints. The condition is usually painless, but contractures, finger shortening and deformity are said to be features (Rubinstein 1975). As expected, radiographic features include flattening, sclerosis and fragmentation of the epiphyses.

Osteonecrosis of the metacarpal heads (Dieterich's disease) has been described in association with trauma, systemic lupus erythematosus and steroid use. Radiographs show sclerosis and fragmentation of the metacarpal head which may show features resembling periarticular erosion (Tashjian et al. 2004).

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