■ ASPECTS OF CURRENT MANAGEMENT

Kienböck's disease

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Kienböck's disease is a form of osteonecrosis affecting the lunate. Its aetiology remains unknown. Morphological variations, such as negative ulnar variance, high uncovering of the lunate, abnormal radial inclination and/or a trapezoidal shape of the lunate and the particular pattern of its vascularity may be predisposing factors. A history of trauma is common. The diagnosis is made on plain radiographs, but MRI can be helpful early in the disease. A CT scan is useful to demonstrate fracture or fragmentation of the lunate. Lichtman classified Kienböck disease into five stages. The natural history of the condition is not well known, and the symptoms do not correlate well with the changes in shape of the lunate and the degree of carpal collapse. There is no strong evidence to support any particular form of treatment. Many patients are improved by temporary immobilisation of the wrist, which does not stop the progression of carpal collapse. Radial shortening may be the treatment of choice in young symptomatic patients presenting with stages I to III-A of Kienböck's disease and negative ulnar variance. Many other forms of surgical treatment have been described.

In 1910, Robert Kienböck, an Austrian early pioneer of radiology, published his classic description of lunatomalacia.¹ He believed that it was caused by traumatic rupture of the ligaments and vessels around the lunate, causing aseptic necrosis, softening, and progressive collapse of the bone.² In 1928, Hultén³ noted that 74% of his 23 patients with Kienböck's disease showed an ulnar-minus variant compared with only 23% in the general Swedish population. He advocated treatment by shortening the radius, and later, Persson⁴ described lengthening the ulna. Almost 100 years after the initial description by Kienböck, the precise aetiology of lunatomalacia is still not known.

Relevant anatomy

The lunate articulates proximally with the radius and with the triangular fibrocartilage (TFC). The thickness of the TFC is proportional to the negative ulnar variance: the shorter the ulna, the thicker the TFC.⁵ Distally, in approximately one-third of wrists, the lunate articulates only with the capitate. In the other cases, a second distal joint surface is present, 1 mm to 6 mm in size, articulating with the hamate (Fig. 1).⁶⁻⁸ Lee⁹ found that 26% of lunates had an arterial supply from either the palmar or the dorsal non-articular

surfaces alone, by means of a single major vessel, 8% had palmar and dorsal vessels without intra-osseous anastomoses, and the remainder had a similar arrangement but with anastomoses. Other authors^{10,11} reported that in between 7% and 20% of normal lunates only palmar vessels vascularised the whole bone, placing the lunate at risk of traumatic interruption of its vascular supply. Pichler and Putz¹² studied the venous drainage of the carpal bones. They observed the existence of delicate periosteal dorsal and palmar venous plexuses, which were smaller for the lunate owing to its semicircular shape.

Relevant biomechanics

Investigation of the transmission of force through the normal wrist using discrete element analysis models revealed that, on average, 90.3% of the total radio-ulnocarpal force was transmitted to the radius, with 61.0% through the radioscaphoid joint and 39.0% through the radiolunate joint.¹³ A mean of 9.7% was dissipated through the TFC. The proximal pole of the scaphoid was constantly transmitting most of the scaphoid load, with a radioscaphoid *versus* radiolunate peak pressure ratio of 1.6, and a radio- *versus* ulno-lunate peak pressure ratio of 4.0. The load applied to the lunate was a function of the

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Fig. 1a

Fig. 1b

a) Pre-operative radiograph showing stage III-B Kienböck's disease. Note the existence of a distal medial facet on the lunate articulation with the hamate, and b) the profile view.

amount of the bone that was not covered by the distal radius, and, to a lesser extent, to the ulnar variance.¹³

Iwasaki et al¹⁴ studied the effects of lunate collapse and demonstrated that in the early stages of the disease (stages II and III-A), the normal position of the scaphoid to some extent prevented transmission of excessive forces to the lunate. However, as the scaphoid assumed its flexed position, in stage III-B, the loads across the lunate were increased, thereby accelerating the process of collapse and fragmentation.

As Kienböck's disease could be the result of excessive bone stress, studies have been conducted to understand how it might be possible to unload the lunate. Radial shortening or ulnar lengthening appear to redistribute some of the radiolunate load to the radioscapoid and to the ulnocarpal joints.¹⁵⁻¹⁷ Provided that the radius is not shortened more than 4 mm, the procedure does not seem to affect the transmission of force through the distal radioulnar joint.^{18,19} Some intercarpal fusions also reduce the loading across the radiolunate joint, as is the case with scapho-trapezotrapezoidal fusion,^{15-17,20,21} scaphocapitate fusion,^{17,21} and capitate-hamate fusion when there is associated shortening of the capitate.^{17,22} Conflicting results have been published regarding the effects of changing the radial inclination.²³⁻²⁵ It is likely that the lateral closing-wedge osteotomy does not actually decrease the total radiolunate load, but, by allowing better covering of the lunate by the distal radius, enlarges radiolunate joint contact, thereby reducing its dangerous peak pressures.²⁶

Clinical features

Kienböck's disease usually affects adults between 20 and 40 years of age, who are predominantly male manual workers. The condition is typically unilateral. Both sides are equally affected.^{27,28} The history of a specific traumatic event is frequent, months or years before the diagnosis. The symptoms of dorsal wrist pain and diminished grip strength usually occur at the time of carpal collapse, probably related to the progressive alteration in carpal architecture and function rather than to bone necrosis.²⁹ Carpal tunnel syndrome may complicate the course of the disease.³⁰

Imaging

The diagnosis is usually made on plain radiographs which form the basis for staging, treatment and evaluation of the results. The classification of Lichtman et al,³¹ which is a modification of the previous classifications of Ståhl² and of Decouls et al,²⁷ has good reliability and reproducibility.^{32,33} In stage I, the density of the lunate and its shape are normal. The diagnosis may be made by MRI. Stage II is characterised by densification of the lunate, without significant alteration of its shape. Stage III, the most common stage at initial presentation, is defined by collapse of the lunate and is subdivided into stage III-A, without carpal collapse, and stage III-B with diminished carpal height and fixed palmar flexion of the scaphoid. The distinction between stages III-A and III-B can be difficult. Goldfarb et al³² proposed classification of any patient with a radioscapoid angle $> 60^\circ$ as stage III-B. In stage IV, there are extensive carpal degenerative changes.

Fig. 2

Pre-operative CT scan of the same patient as in Figure 1, demonstrating fragmentation of the necrotic lunate.

A CT scan will demonstrate a coronal fracture of the lunate, splitting the bone into volar and dorsal fragments, or the existence of lunate fragmentation, not always clearly seen on plain radiographs (Fig. 2).

MRI is helpful early in the disease, when the radiographs are still normal in stage I. T₁-weighted images will then demonstrate decreased signal intensity. These images, which are not specific,³⁴ should be interpreted with care as subchondral oedema, seen for example in ulnocarpal impaction or after trauma of the wrist, may give similar images.

Aetiology

Although the precise aetiology is not known, the final results of lunate fragmentation and collapse are secondary to osteonecrosis, as demonstrated histologically. The loss of blood supply may be the consequence of either primary circulatory problems or subchondral fractures resulting in progressive collapse with secondary vascular impairment. As noted above, a proportion of normal lunates are at risk of interruption of their vascular supply. Jensen³⁵ and Schiltenswold et al³⁶ measured high intra-osseous pressures in the lunate of patients with Kienböck's disease. In particular, Schiltenswold et al³⁶ found that dorsiflexion of the wrist markedly increased the pressure, sometimes to above the systolic pressure. They also measured the intra-osseous

pressure in normal lunates and found higher pressures than in the neighbouring capitate. These observations suggest that necrosis of the lunate might be the consequence of impaired venous outflow. Also in favour of the circulatory theory is the observation that some lunates may be revascularised by implantation of a vascular pedicle or a vascularised bone graft.^{29,37-39} Kienböck's disease may exceptionally complicate sickle-cell anaemia, causing bone infarction,⁴⁰ or may be seen in some patients with cerebral palsy who have an abnormally flexed wrist.⁴¹

According to the mechanical theory, necrosis of the trabeculae of the lunate is the consequence of progressive collapse of the bone under excessive loads, causing repeated microfractures. There would be a 'nutcracker' effect between the capitate and the relatively rigid radius on the radial side and the relatively elastic fibrocartilage on the ulnar side.^{4,42} Some lunates may be predisposed to collapse because of their particular anatomy causing an uneven internal distribution of the bone stresses. The initial lesion might be a fracture caused by minor trauma. Interruption of the intra-osseous vessels may cause localised trabecular necrosis.⁹ The anatomical factors that would create a significant risk are disputed in the literature. Since the initial observation of Hultén,³ many authors,^{30,31,42-45} but not all,^{8,46-49} have found a statistical relationship between negative ulnar variance and Kienböck's disease. The condition has been rarely reported in patients with a congenital^{50,51} or post-traumatic⁵² ulnar-minus deformity, although it should be recognised that Kienböck's disease is not seen after operations for ulnar shortening. In some patients with advanced disease the negative ulnar variance is secondary, resulting from the apposition of bone on the ulnar margin of the distal radius in response to the collapse of the lunate.⁵³ The possible relationship between ulnar variance and Kienböck's disease seems to vary in different parts of the world: there appear to be more normal subjects with ulnar-plus variants in black and Asian populations.^{44,49,54} In East Asia, Kienböck's disease has been seen in patients with positive ulnar variance, a very rare observation in Caucasians.^{37,49,55} Other anatomical factors could be important, such as uncovering of the lunate by the distal radius,⁵⁶ the shape of the lunate (trapezoidal),^{8,57} the existence of a midcarpal facet on the lunate to articulate with the hamate (Fig. 1),⁷ and the radial inclination of the distal radius.^{8,45,46} In favour of the mechanical theory is the fact that many patients recall initial trauma to the wrist,^{2,27,30,42,45,58,59} occasionally with an identifiable fracture line^{2,30,60} or even with scapholunate dissociation.⁶¹ Many necrotic lunates are fragmented, as demonstrated by CT scans (Fig. 2). Unpublished finite element analysis data from the authors support the view that in unfavourable anatomical situations, where the lunate rests on the radial side on a relatively rigid distal radius and on the ulnar side on a very elastic structure, a particularly thick fibrocartilage, the lunate may undergo uneven high internal loads, with stress concentration predisposing to fracture. After an

Fig. 3

Radiograph of the same patient as in Figures 1 and 2 showing Graner-Wilhelm progressive lengthening of the capitate. After resection of the lunate via a dorsal approach, 2 mm external mini fixation pins are implanted into the head and body of the capitate. An osteotomy is then performed at the neck of the capitate. Contrary to Wilhelm et al⁸⁴ we do not believe it necessary to temporarily transfix the scaphocapitate or scapho-trapezio-trapezoid joint.

initial fracture, the internal stresses would become even higher, causing further stress fractures and progressive collapse.

Treatment

There is still little evidence to support any particular form of operative treatment, or to indicate its superiority over conservative measures.⁶² Indeed, the surgical option may be even worse, with many patients unable to resume heavy professional occupations after surgery.⁶² However, several retrospective studies have suggested better results when radial shortening is performed compared with conservative treatment.^{63,64} In long-term studies of conservative management, a progressive radiological deterioration is observed in most patients, but the severity of changes in the lunate and of carpal collapse does not always correlate with symptoms.^{30,31,62,65,66} Many patients derive benefit from temporary immobilisation of the wrist.^{2,30} Some maintain excellent function, even in stage IV. We agree with Amadio and Moran⁵⁷ that surgery is indicated only for patients with symptomatic disease after an adequate trial of immobilisation and anti-inflammatory medication.

Many surgical procedures have been proposed. We now outline our current approach according to the stage of the disease.

Stages I and II. There is an opportunity to revascularise the bone in order to heal the lesion and to prevent collapse of the lunate. If there is negative ulnar variance, particularly in association with marked uncovering of the lunate, we aim to restore a more favourable biomechanical situation, either by shortening the radius^{48,56,58-60,63,67-70} or by lengthening the ulna.^{28,42,71,72} Several studies have suggested that the clinical outcome of radial shortening is better than that of ulnar lengthening, which has a higher rate of nonunion and problems with the distal radioulnar joint.^{28,58,72,73} Radial shortening is performed through a radiopalmar approach, with osteosynthesis using a 3.5 mm dynamic compression plate. Eccentric compression through the oval holes allows progressive closure of the osteotomy. Care must be taken to avoid excessive shortening, which could affect pronation-supination or even lead to ulnocarpal impaction. We have no experience of an oblique shortening osteotomy, nor of a distal metaphyseal osteotomy.^{56,58} The latter may be more favourable in terms of bony union, but presents a higher risk of failure of the internal fixation.

Assessment of the different published studies^{58-60,67-70} indicates that radial shortening offers durable pain relief and may improve grip strength, but does not offer a cure. At best, it can slow down its development. Several authors have indicated that the factor which most affects the outcome is age, with less satisfactory results in patients over the age of 30.^{74,75} In the youngest patients, some evidence of lunate revascularisation may be seen years after the osteotomy.⁶⁹

With neutral ulnar variance, or in the exceptional case of an ulnar-plus variant, revascularisation of the lunate may be indicated, although it has been suggested that revascularisation might precipitate collapse of the lunate.⁷⁶ It is attempted by implantation of a dorsal metacarpal arteriovenous pedicle,^{29,37,38} or by a vascularised bone graft, which is usually taken from the dorsal aspect of the radius via pedicles from the fourth and fifth extensor compartments.³⁹ In order to protect the lunate during the early period of revascularisation, temporary unloading of the wrist using external fixation is advisable.²⁹ Signs of revascularisation may appear after 18 to 36 months, but only in approximately half the cases.²⁹

Symptomatic stage III. Although shortening of the radius remains an option, we recommend restoration of the carpal height in order to prevent subsequent degenerative arthritis. Twenty years ago, one of the authors (FS) published a technique of excision of the lunate and scapho-trapezio-trapezoid fusion. The restored carpal height was maintained during bone healing by transarticular distraction using external fixation.⁷⁷ We did not consider it necessary to interpose an implant or a soft-tissue autograft at the site of resection of the lunate. More recently we have tended to favour scaphocapitate arthrodesis,^{78,79} which is technically easier. The goals of intercarpal fusion are to preserve carpal height, to maintain the scaphoid in its proper position, to prevent degenerative arthritis, and to relatively unload the

Fig. 4a

Fig. 4b

Progressive distraction is applied after seven to ten days at the rate of 1 mm/day without wrist or hand immobilisation in the same patient as in Figures 1 to 3.

Fig. 5a

Fig. 5b

Fig. 5c

Radiographs of the same patient as in Figures 1 to 4 at a) 32 days, b) 46 days and c) 142 days after the operation, showing solid healing of the capitate and restoration of carpal height.

lunate if it is preserved.^{16,20,80} Because intercarpal fusion causes marked loss of movement^{15,80,81} and may be complicated by nonunion, other complications or late arthritis,^{82,83} we now use progressive capitate lengthening after excision of the lunate in order to restore carpal height. This modification of Graner's procedure has been described by Wilhelm, Hierner and Brehl⁸⁴ (Figs 1 to 5).

Symptomatic stage IV. With advanced degenerative arthritis, the available salvage options include wrist denervation, proximal row carpectomy if the articular surfaces of the capitate and radius are preserved, or total wrist fusion. Sometimes there is an associated carpal tunnel syndrome, which is managed by simple decompression.

Other surgical options. Partial or total excision of the lunate can reduce the symptoms but does not prevent carpal collapse.^{28,85,86} Silicone implants are contraindicated as they induce particulate 'siliconitis'.^{65,87,88} The implant may sublux or dislocate into the carpal tunnel.⁶⁵ Saffar⁸⁹ has described lunate replacement by a vascularised transfer of the pisiform. Other authors^{26,55,64,90-92} have recommended a lateral closing-wedge osteotomy of the distal radius, a procedure that might be indicated in the rare patients with neutral or positive ulnar variance but may cause restriction of forearm rotation.⁵⁵ Comtet et al⁹³ proposed hemiepiphyseal lengthening of the ulna. Illarramendi, Schulz and De Carli⁹⁴ performed radial and ulnar metaphyseal 'decompression', without osteotomy,

and reported excellent results at a follow-up of ten years. We have no experience of capitate shortening with or without capitate-hamate arthrodesis,⁹⁵ simple capitate-hamate fusion,⁹⁵ nor of the original Graner procedure which combines capitate lengthening with intercarpal arthrodesis.⁹⁶

Conclusions. The aetiology of Kienböck's disease remains poorly understood. Biomechanical factors are important at a certain stage of its evolution, as suggested by the significant changes in mineralisation of the distal radius.^{97,98} The natural history is not well known and the symptoms do not necessarily correlate with the radiological appearance. Surgical treatment has not been shown to be superior to conservative measures. Comparative retrospective studies should be interpreted with care, as patients with more advanced lesions are more likely to have undergone surgery. In European countries, a manual worker who complains of persistent wrist pain and weakness after an operation is at high risk of losing his job, and it is difficult to relocate manual workers into less physically demanding professions. For these reasons, radial shortening probably remains the best surgical option in symptomatic stages I to III-A in young manual workers with ulnar-minus variance, because in this procedure the carpus is left undisturbed, preserving its residual mobility. Bone healing should be obtained with a reasonable time span, and nonunion should be avoided. We advise autologous bone grafting at the site of the osteotomy. Excision of the lunate and progressive lengthening of the capitate may be an excellent solution in stage III-B. In stage IV, arthrodesis of the wrist has given us acceptable results, allowing many patients to return to their previous occupations with a painless ankylosed wrist.

Kienböck's disease occurs infrequently in children. A non-operative approach is recommended with prolonged immobilisation in a cast as there is great potential for healing and remodelling at this age.^{74,99} Radial shortening may exceptionally be indicated in teenagers.^{50,57}

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