ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 8| Issue: 12| December 2022|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2022: 8.205 || ISI Value: 1.188

KIENBÖCK'S DISEASE, AVASCULAR NECROSIS OF THE LUNATE

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Article DOI: https://doi.org/10.36713/epra12085
DOI No: 10.36713/epra2085

SUMMARY

Introduction: *Avascular necrosis of the lunate bone was described in 1910 by the Austrian radiologist Robert Kienböck, which is named after him. A mixture of different factors such as mechanical, vascular and genetic predisposition may be related to the pathogenesis of this disease. As for the natural evolution of the disease, failure in early diagnosis and early treatment may lead to a gradual evolution from stage I to stage IV, causing discomfort to the patient.*

Objective: to detail the current information related to avascular necrosis of the lunate, description, etiology, classification, imaging presentation and management of Kienböck's disease.

Methodology: *a total of 32 articles were analyzed in this review, including review and original articles, as well as clinical cases, 20 bibliographies were used because the other articles were not relevant for this study. The sources of information were PubMed, Google Scholar and Cochrane; the terms used to search for information in Spanish, Portuguese and English were: Kienböck, avascular necrosis of the lunate and lunatomalacia.*

Results: *it is the second most frequent cause of avascular necrosis of the carpal bones and generally affects males between 20 and 40 years of age. Nuclear magnetic resonance has a greater contribution due to greater sensitivity and detection of radiographically occult cases, computed tomography also has a good specificity at the time of diagnosis. Radiography at the beginning of the disease does not present evident changes and nuclear scintigraphy presents non-specific findings. In the first stage, the treatment is based on immobilization with a plaster cast or splints. When incomplete necrosis is evidenced in the second stage, conservative treatment can be performed, however with complete necrosis or in the third and fourth stage, it requires "joint leveling" surgery and probably vascular bone grafting or transfer of branches of adjacent arteries. Stage IIIA usually merits lunate restoration, in stage IIIB and Lichtman IV wrist arthrodesis can be used.*

Conclusions: *Kienböck's disease presents with unilateral pain over the dorsal aspect of the wrist, weakness and limited wrist motion, in addition to functional impotence, decreased grip strength, wrist edema, sensory disturbances in the median nerve territory and synovitis, depending on the stage. It is related to the following variables such as ulnar minus or ulnar negative variation, vascular contribution of the lunate bone, morphology of the lunate, radial inclination angle. The diagnosis is clinical and imaging where Lichtman's classification is useful. Treatment will depend on the cause and also on the stage of the disease.*

KEY WORDS: *Kienböck, avascular necrosis, lunatomalacia, lunate.*

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INTRODUCTION

The lunate bone is the central bone in the proximal row of the carpus, it articulates with the scaphoid, large bone, pyramidal and occasionally the hooked bone. It is involved in much of the motion of the wrist and is one of the bones that constitute the radiocarpal joint, as well as articulating with the ulna by means of the triangular fibrocartilage complex(1).

Avascular necrosis of the lunate bone, also known as lunatomalacia, was first described in 1910 by the Austrian radiologist Robert Kienböck, which is why it is also known as Kienböck's disease(1-3). A mixture of different factors such as mechanical, vascular and genetic predisposition may be related to the pathogenesis of this disease. As for the natural evolution of the disease, failure in early diagnosis and early treatment may lead to a gradual evolution from stage I to stage IV(4).

METHODOLOGY

A total of 32 articles were analyzed in this review, including review and original articles, as well as cases and clinical trials, 20 bibliographies were used because the information collected was not important enough to be included in this study. The sources of information were Cochrane, PubMed and Google Scholar; the terms used to search for information in Spanish, Portuguese and English were: Kienböck, avascular necrosis of the lunate lunate and lunatomalacia.

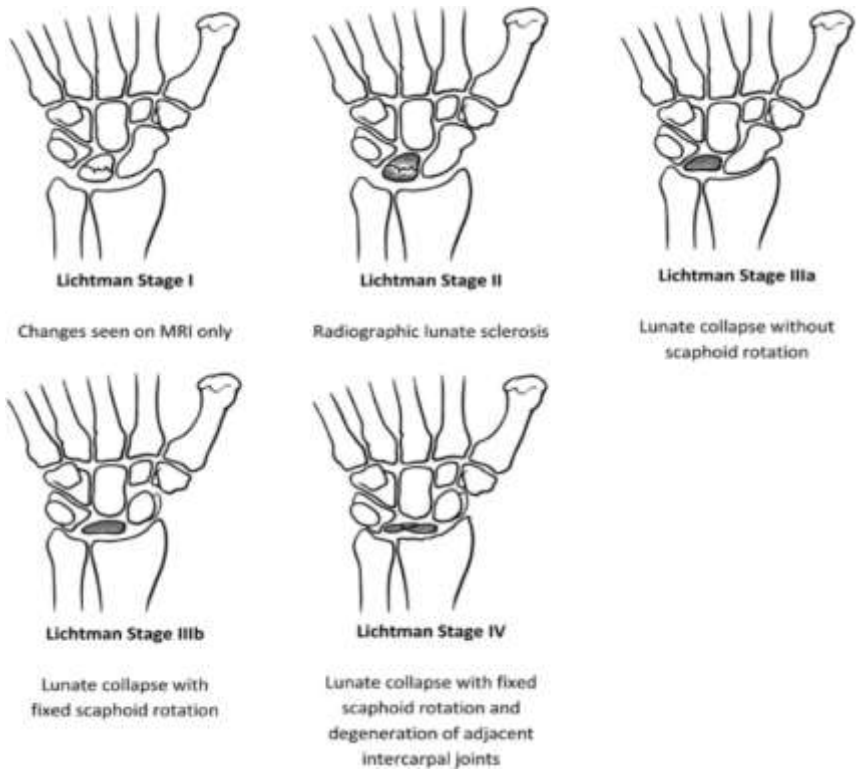
The choice of the bibliography exposes elements related to avascular necrosis of the lunate; in addition to this factor, etiology, presentation, evaluation of Kienböck's disease are presented, as well as the diagnosis and management of the disease.

DEVELOPMENT

Kienböck's disease or lunatomalacia is the second most frequent cause of avascular necrosis of the carpal bones, only after scaphoid avascular necrosis. It generally affects males between 20 and 40 years of age and the condition rarely presents bilaterally or with the presence of trauma. Among the most frequent signs and symptoms we can find unilateral pain on the dorsal side of the wrist, weakness and limited movement of the wrist, functional impotence and decreased grip strength, as well as wrist edema, sensory disturbances in the territory of the median nerve and synovitis. In the final stages, pain becomes more moderate and stiffness and loss of strength predominate. Axial loading and wrist extension usually worsen the pain(1,5,6).

Avascular necrosis of the lunate has a common prevalence of 0.5% and increases to 1.1%-2% in groups exposed to vibration such as those working with jackhammers(3).

Figure 1. Lichtman classification



Source: Caso Radiol Rep. 2022 jun; 17(6): 2115–2119(3)

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Table 1. Lichtman classification for Kienböck disease.

Lichtman Classification	
STAGE I	- Normal radiograph - Linear fracture lines - Decreased uniform signal on T1 MRI images - Bone scans are positive but nonspecific
STAGE II	-Single radiographs show sclerosis of the lunate - Fracture lines - No evidence of lunate collapse.
STAGE IIIA	- Semilunar collapse, maintaining alignment and height of the carpus.
STAGE IIIB	- Semilunar collapse plus one of the following: ● Loss of carpal height ● Proximal migration of the large bone ● Scaphoid rotated and flexed
STAGE IV	- Stage IIIB + radiocarpal degenerative changes

Source: The Authors.

Patients with stage I disease frequently present with intermittent, non-specific wrist pain accompanied by synovitis that may resemble a wrist sprain. Plain radiographs may appear normal or small linear compression fractures through the lunate may be seen. There is no evidence of collapse, sclerosis or increased radiodensity of the lunate. On MRI there is evidence of uniform signal decrease on T1 and T2 images indicating osteonecrosis of the lunate. If revascularization is performed, after a surgical intervention, an increase of the signal in T2 can be observed(7,8).

Stage II is clinically characterized by increased edema, progressive pain and variation on stiffness. Radiographs show sclerosis of the lunate with or without the presence of linear compression fractures. The lunate is more radiodense, however, there is no collapse and the height is maintained. The rest of the carpal structures remain without degenerative changes(7,8).

Stage III presents continuous sclerosis and collapse of the lunate. It is subdivided into two stages depending on its alignment and relationship to the carpus. Attention should be paid to the scapholunate angle and height of the carpus. A decrease in height may be represented as a collapse or osteoarthritic changes within the carpus. Carpal height is the measurement between the distal articular surface of the large bone to the lunate fossa of the distal radius. The degree of carpal collapse is the height of the carpus divided by the length of the third metacarpal which is approximately 0.53(7).

Stage IIIA is characterized by collapse of the lunate with preservation of carpal height and intercarpal alignment. Stage IIIB is characterized by collapse of the lunate and characteristic changes of the large bone and scaphoid. The large bone migrates proximally and there is a decrease in carpal height. Symptomatology progresses from vague pain and synovitis to symptoms of instability, radial and ulnar deviation, progressive pain and decreased grip strength(7).

Stage IV is characterized by progressive carpal collapse, leading to radiocarpal degenerative changes. Radiographs show decreased joint space, subchondral sclerosis, degenerative cysts and osteophyte formation. Symptoms progress to stiffness, constant pain and edema(7).

Regarding the main causative factor of Kienböck's disease, there is no clear consensus. However, it is related to the following variables:

Ulnar minus or ulnar negative variation: this is when the ulna is excessively shortened in relation to the radius which can cause increased mechanical stress. In addition the comparatively longer radius can cause repetitive microtrauma on the lunate. It can be found in 78% of Kienböck's cases(1).

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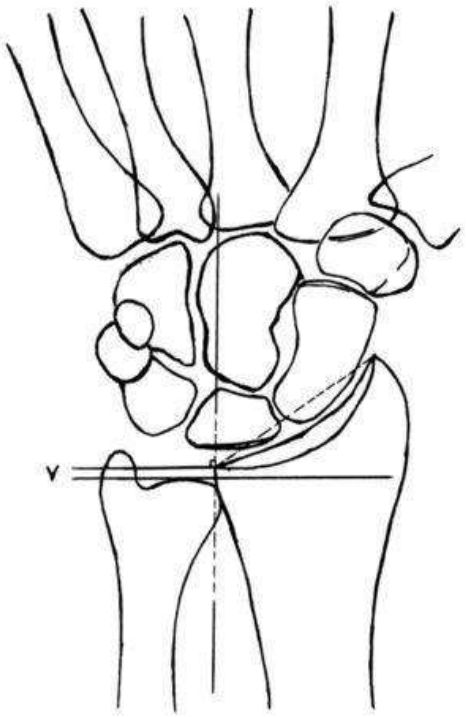
Figure 2. Measurement of ulnar variance with technique described by Quenzer et al.



Source: Rev Esp Cir Ortopédica Traumatol. 2005;49(2):106-11(9).

Vascular supply of the lunate bone: the lunate bone accepts blood supply through the palmar and dorsal penetrating arteries, which are branches of the dorsal and palmar radiocarpal and intercarpal arches. They have almost no intraosseous collaterals. There are clinical studies that show that approximately 20% of the lunate crescents present only one vascular blood tributary. The lower the number of penetrating arteries, the higher the probability of developing Kienböck's disease(1,10).

Morphology of the lunate: the smaller the size of the lunate bone, the greater the risk of Kienböck's disease, because it bears more axial load. The shape of the lunate is varied, it can be triangular due to the lack of the medial articular facet (type I), square (type II) or rectangular (type III). The triangular shape presents a less strong trabecular pattern and is therefore a risk factor for the progression and development of the condition(1).

Radial tilt angle: The smaller the radial tilt angle, the higher the risk of Kienböck's disease. This angle can be found between the horizontal and a line drawn from the ulnar tip of the radial articular surface to the tip of the radial styloid(1).

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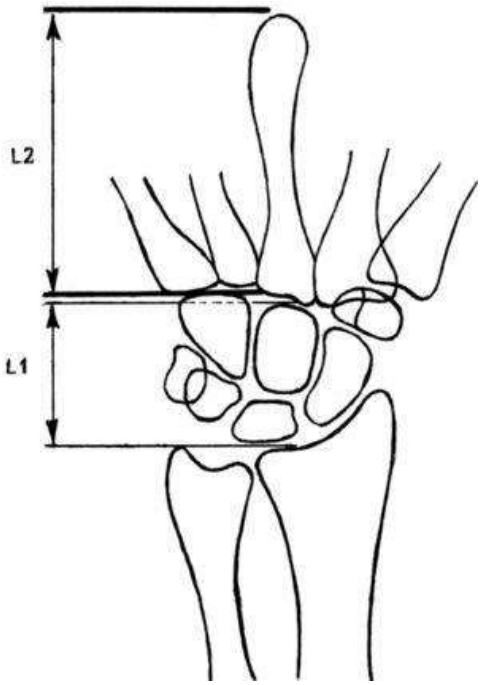
Figure 3. Degree of carpal collapse by Salmon et al. through the ratio of carpal height (L1) and third metacarpal length (L2).



Source: Rev Esp Cir Ortopédica Traumatol. 2005;49(2):106-11(9).

Avascular necrosis of the lunate most of the times leads to the destruction of the joint in 3 to 5 years if it is not treated in time and it is a characteristically progressive disease(3,5,6). This disease alters the biomechanics of the wrist resulting in early arthritis and degenerative changes(11).

A clinical study evaluating 27 patients over 50 years of age diagnosed with Kienböck's disease suggests that the option of surgery should be thoroughly evaluated in this age group, as the disease may have a naturally benign radiographic and clinical course(12).

The diagnosis of Kienböck's disease is clinical and by imaging. Nuclear magnetic resonance has a greater contribution due to greater sensitivity and detection of radiographically occult cases, computed tomography also has a good specificity at the time of diagnosis(1).

The following is a brief description of the different imaging tests used in Kienböck's disease:

MRI: In addition to assessing the integrity of the articular cartilage, MRI shows a decreased signal from the crescentic bone marrow on T1-weighted images which is characteristic of the disease. Both T2 and short TI inversion recovery images change with the progression and extent of osteonecrosis(1).

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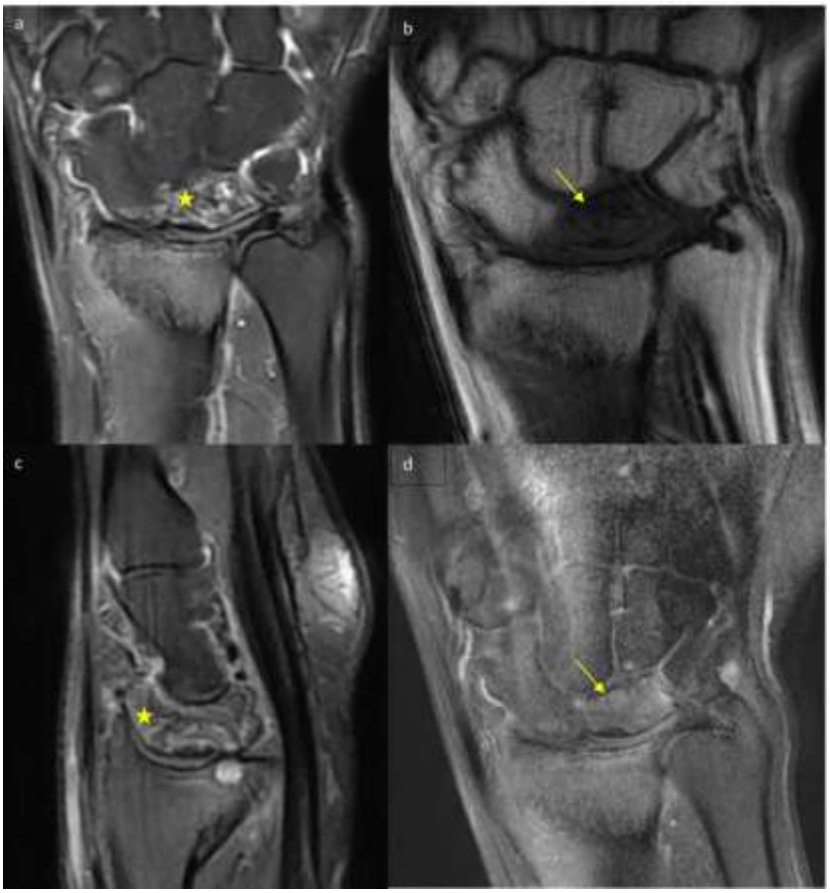
Figure 4. a. MRI of left hand coronal DP Fat Sat sequence: collapsed lunate with heterogeneous signal. b. MRI of left hand coronal T1-weighted sequence: collapsed lunate in hypointense in T1. c. MRI of left hand sagittal DP Fat Sat sequence: collapsed lunate with heterogeneous signal. d. MRI of left hand sagittal Fat Sat sequence: collapsed lunate with heterogeneous signal. MRI of the left hand T1-weighted coronal sequence with fat suppression and gadolinium injection: collapsed lunate without significant enhancement.



Source: Caso Radiol Rep. 2022 jun; 17(6): 2115–2119(3)

Radiography: at the beginning of the disease, no evident changes are observed, so they usually appear normal, according to the progression of the disease it may present: cystic changes, diffuse semilunar sclerosis, collapse of the articular surface and of the carpus, secondary arthrosis of the middle carpus and/or radiocarpal. Coronal fractures may occur in type I lunate fractures (6,13).

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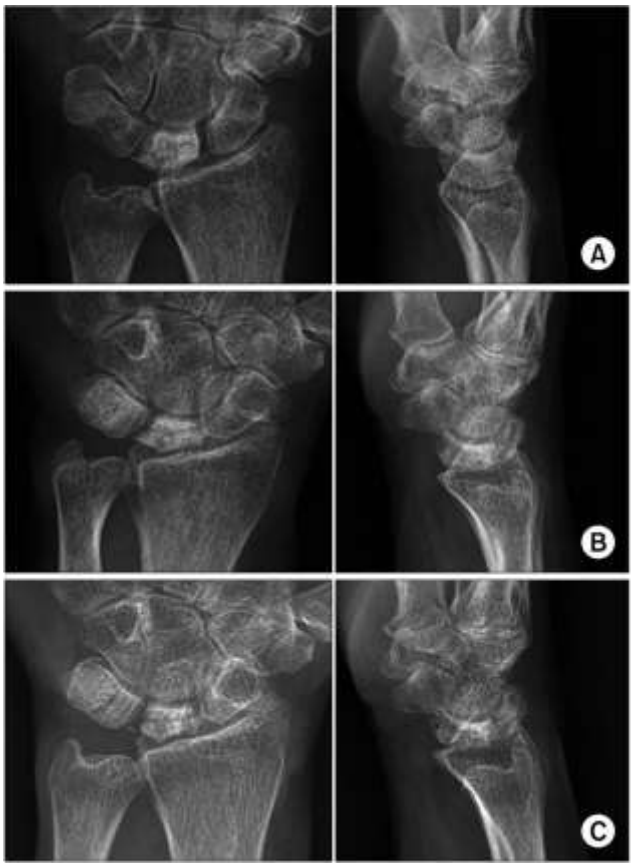
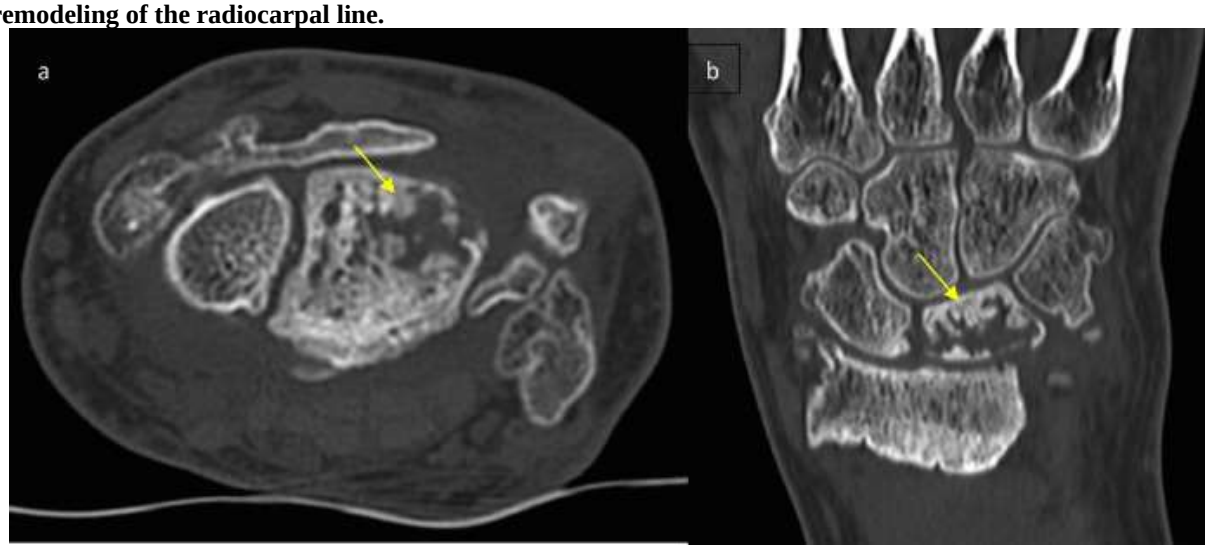
Figure 5. Plain radiographs of Kienböck's disease and its evolution.



Source: Clin Orthop Surg. 2022 Sep; 14(3): 450–457(12).

Computed tomography: it is more sensitive than radiographs to find mild subchondral fractures, fragmentation, carpal instability, fractures of the lunate in the coronal section and the degree of trabecular rupture. It is frequently used for surgical planning (1).

Figure 6. Axial and coronal CT of the left wrist with fragmented and agglomerated semilunate aspect of the lunate with arthritic remodeling of the radiocarpal line.



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Nuclear scintigraphy: it presents non-specific findings, previously it was used as a complementary diagnosis(1).

The differential diagnosis of the disease can be made with: arthritis, ulnar impaction syndrome, osteoid osteoma, semilunar intraosseous ganglion, bone contusion, enostosis/bone island(1,13).

Pain relief is the main goal of treatment in Kienböck's disease, in addition to preserving motion and grip strength. Treatment will depend on the cause and also on the stage of the disease(1).

In the first stage, treatment is usually based on immobilization with plaster or splints, conservative treatment is recommended; in the second stage it is also possible to treat conservatively when there is incomplete necrosis, however with complete necrosis, or in the third and fourth stage it will require "joint leveling" surgery and probably vascular bone grafting or transfer of branches of adjacent arteries. The main objective in the treatment of stage II is revascularization, unloading and decompression of the lunate(1,14).

Subsequent stages presenting with lunate collapse and secondary degenerative arthrosis of the wrist may require proximal row carpectomy or intercarpal arthrodesis. To unload the lunate in cases with coexisting ulnar negative variance, radial shortening osteotomy is most often used (1).

In stage IIIA it usually requires restoration of the lunate, in stage IIIB it is customary to perform a partial arthrodesis of the wrist. As for rescue procedures, such as wrist arthrodesis, they are mainly limited to Lichtman IV(14).

Partial shortening of the large bone is a decompression technique for the treatment of lunatomalacia with positive or neutral ulnar variance. The large bone shortening technique done by means of a smaller cut takes less time compared to radial shortening and leads to an advantage in stage II(15,16).

It is possible to compare the results between the combined technique of large bone/hook osteotomy with the isolated large bone shortening osteotomy, however, both present good postoperative results(15).

In a clinical study it was possible to confirm that the height and dimensions of the lunate can be restored when performing the shortening of the large bone combined with neutral ulnar variation in stage IIIA, improving the final results and thus evidencing a low failure rate. In stage II patients, there was no evidence of superiority of large bone shortening combined with large bone shortening alone. An important prognostic factor in treatment results is the lunate height index(17).

In a systematic review by Wang et al. a significant decrease in joint functionality, specifically in the flexion-extension arc of motion, could be found when comparing conservative management with 89°, non-rescue treatment such as lunate reconstruction and radial osteotomies with 95°; and rescue procedures such as intercarpal and radiocarpal arthrodesis, proximal row carpectomy, total wrist arthroplasty with 73° ($p = 0.0001$). Meanwhile, all treatments brought improvements such as pain relief, ability to return to their activities and grip strength. However, there was no significant difference in grip strength ($p = 0.28$)(18).

The scapho-semilunar fusion technique shows promising results in the management of advanced Kienböck's disease without the need for excision of the lunate lunate(19,20).

CONCLUSIONS

Kienböck disease is the second most frequent cause of avascular necrosis of the carpal bones, generally affecting males between 20 and 40 years of age, presenting unilateral pain on the dorsal aspect of the wrist, weakness and limited movement of the wrist, as well as functional impotence, decreased grip strength, wrist edema, sensory alterations in the territory of the median nerve and synovitis, depending on the stage.

It is related to the following variables such as ulnar minus or ulnar negative variation, vascular contribution of the lunate bone, morphology of the lunate, radial inclination angle.

In stage I they frequently present intermittent and non-specific wrist pain. Plain radiographs may be normal or small linear compression fractures through the lunate may be seen. There is no evidence of collapse or sclerosis. Stage II is characterized clinically by increased edema, progressive pain and variation in stiffness. Radiographs show sclerosis of the lunate with or without the presence of linear compression fractures. Stage III presents continuous sclerosis and collapse of the lunate. It is subdivided into two stages depending on its alignment and relationship to the carpus. Stage IIIA is characterized by collapse of the lunate with preservation of carpal height and intercarpal alignment. Stage IIIB is characterized by collapse of the lunate and characteristic changes of the large bone and scaphoid, in addition there is a decrease in the height of the carpus. Stage IV is characterized by progressive collapse of the carpus, leading to radiocarpal degenerative changes.

The diagnosis of Kienböck's disease is clinical and imaging. Magnetic resonance imaging has a greater contribution due to greater sensitivity and detection of radiographically occult cases, computed tomography also has a good specificity at the time of diagnosis. Radiography at the beginning of the disease does not present evident changes and nuclear scintigraphy presents non-specific findings.

Treatment will depend on the cause and also on the stage of the disease. In the first stage, treatment is based on immobilization with plaster cast or splints. When incomplete necrosis is evidenced in the second stage, conservative treatment can be performed, however with complete necrosis or in the third and fourth stage, it requires "joint leveling" surgery and probably vascular bone grafting or transfer of branches of adjacent arteries. In stage IIIA it usually requires restoration of the lunate, in stage IIIB it is customary to perform a partial arthrodesis of the wrist. As for salvage procedures, such as wrist arthrodesis, they are mainly limited to Lichtman IV.

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Conflict of interest statement:
The authors report no conflicts of interest.
Funding:
The authors report no funding by any organization or company.