

Commentary

Kienböck's Disease May Be Due to a Wrist-Joint Tamponade

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Kienböck's disease may be due to a mechanism (similar to a muscle compartment syndrome) that reduces the lunate perfusion. Arthrocentesis at an early stage can probably prevent the disease.

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Introduction

The etiology of Kienböck's disease (osteonecrosis of the lunate bone) has been debated for a century now. Kienböck asserted that it was first a nutritional disturbance of the lunate bone followed by a fracture of the weakened bone^[1]. Most studies have focused on anatomical risk factors (such as ulnar variance, radial inclination, and lunate morphology), but the cause of the osteonecrosis is still considered enigmatic. Current hypotheses include suggestions of localized venous obstruction and interstitial edema ("compartment syndrome of the bone") due to stress microfractures^[2], reduced arterial blood flow to the lunate bone due to an inappropriate wrist position^[3], and intra-osseous venous thrombosis due to an inflammatory disorder^[4]. Hence, it seems justified to call attention to an explanation that seems more consistent with the clinical evidence.

Scaphoid fractures, femoral neck fractures, and Kienböck's disease

Scaphoid fractures and femoral neck fractures are etiologically interesting in this context: partly because they are associated with a high risk of osteonecrosis; and partly because the proximal fragment of a scaphoid fracture, the femoral head of a femoral neck fracture, and the lunate bone all have the characteristics of being drained via capsular veins, which are directly exposed to intra-articular pressure.

After an undisplaced scaphoid fracture, an intra-articular pressure of 10 kPa (76 mm Hg) due to hemarthrosis (*wrist-joint tamponade*) was recorded^[5] – more than enough to cause osteonecrosis of the proximal fragment – and arthrocentesis (3 mL blood) in this case brought immediate pain relief.

It is well documented that hemarthrosis in the hip joint can cause osteonecrosis of the femoral head because of increased intra-articular pressure (*hip-joint tamponade*) after an undisplaced femoral neck fracture^{[6][7][8]}. By contrast, when the joint capsule ruptures in a displaced femoral neck fracture, no joint tamponade occurs because the intra-articular bleeding is drained.

The fact that Kienböck's disease often (about 50% of cases) has a history of prior trauma to the wrist^[9], but is rare after intercarpal dislocations and fracture-dislocations (none observed in 59 cases)^[10], suggests that, similar to undisplaced femoral neck fractures, Kienböck's disease may result from a wrist-joint tamponade—an effect that does not occur when the joint capsule ruptures. Kienböck's disease has also been associated with several medical disorders (such as rheumatoid arthritis, gout, juvenile idiopathic arthritis), which are likely to have episodes of severe joint effusion^[11]. Thus, at least some of these non-traumatic cases appear to be explained by wrist-joint tamponade.

Conclusions

Wrist-joint tamponade may explain both traumatic and non-traumatic cases of Kienböck's disease and may also explain why the disease rarely occurs after intercarpal dislocations and fracture-dislocations. The proposed mechanism is similar to a muscle compartment syndrome (but is confined by a joint capsule instead of a muscle fascia). A joint tamponade increases the pressure in the capsular veins draining the lunate bone, reduces the arteriovenous pressure gradient, and thus decreases lunate perfusion, which may result in osteonecrosis. After spontaneous revascularization and osteolysis, anatomical risk factors have a mechanical impact on the weakened lunate bone that can subsequently collapse^[12]. Wrist arthrocentesis of a tense joint effusion at an early stage (if given the opportunity, e.g., shortly after a trauma) can probably prevent osteonecrosis, thereby avoiding the development of Kienböck's disease.

References

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Declarations

Funding: No specific funding was received for this work.

Potential competing interests: No potential competing interests to declare.