

Kienbock's disease following pellet injury to the lunate: A case report

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ABSTRACT

A case of a 32-year-old male soldier with a history of pain in left wrist 15 months after the patient received pellet injuries in a grenade attack firearm injury is presented. Radiography revealed avascular necrosis (AVN) of the left lunate (Kienbock's disease) with a small metallic pellet embedded in the lunate. We postulate that the AVN may be either due to disruption of the vasculature, mechanical overload or metallosis. To our knowledge, this is the first time a firearm injury resulting in a retained pellet in the lunate has been implicated in Kienbock's disease.

KEY WORDS: Kienbock's disease, lunate necrosis, pellet injury

INTRODUCTION

Kienbock's disease (lunatomalacia), first described by Robert Kienbock as ligamentous trauma to the lunate resulting in the interruption of internal arterial supply to the bone [1], is defined as a specific form of avascular osteonecrosis affecting the lunate. Kienbock's disease is characterized by sclerosis, cystic changes, fragmentation, and articular surface collapse as present on plain radiographs [2,3]. Kienbock's disease is a rare condition with an unknown etiology [2,3], seen more in manual laborers and athletes. Due to its unknown etiology the management is highly controversial with both conservative and operative management alleviating pain, improving function, and limiting the progression of the disease [2,3]. It can resemble a scapholunate sprain, lunate fracture, or lunate dislocation, which can make differentiating this condition from other wrist conditions a challenge for many healthcare practitioners. In our case, the etiology could possibly be ascribed to a pellet embedded in the lunate following a grenade blast. This has not been reported in the literature till date.

CASE REPORT

A 32-year-old male soldier reported with pain in left wrist of about 8 months duration, mostly while doing heavy work or

physical training, especially while doing pushups. There was a past history of being involved in a firefight with militants. He had been wounded by a grenade and had suffered multiple pellet injuries, one of which was to the wrist. At that time, he had been examined by his military doctor and was given first aid and analgesic and antibiotic medications. He was alright after a week's rest and subsequently went back to his duty. 15 months after the injury, he started getting pain in the wrist on vigorous activity. Patient was again seen by his military doctor and put on non-steroidal anti-inflammatory drugs, which gave him some relief. However, he was referred for a second opinion to our hospital. Physical examination did not reveal any significant abnormality. There was no restriction of the range of motion of the wrist, in flexion and extension of the digits [Figures 1 and 2]. Plain radiographs of the left wrist [Figure 3] revealed features of avascular necrosis (AVN) of the lunate (Kienbock's disease) with a pellet embedded in the lunate. The patient was given a wrist splint and analgesics, advised to avoid overusing the wrist and strenuous activity and counseled on the need for surgery in the future. He has since been transferred out of the state.

DISCUSSION

Kienbock's disease is lunatomalacia due to AVN of the carpal lunate [1]. The real etiology is not very well understood [2,3]



Figure 1: Full palmar flexion



Figure 2: Full dorsiflexion

since it is commonly observed without any history of antecedent trauma. Mostly, the patients comprise males in their third and fourth decade, physically active, usually manual laborers or men active in sports activities like athletics that continually load the wrist [2,3]. These patients' common presentations include stress related dorsal wrist pain, reduced wrist flexion-extension, and impaired grip strength. Manual workers tend to have more repetitive trauma to the carpal bones leading to degenerative changes and subsequent loss of lunate blood supply. It can be theorized that athletes using their hands may also have an increase in repetitive trauma to the wrist leading to an increased risk of developing Kienbock's disease. There is a predilection in males compared to females with a male to female ratio of up to 9:1 [4]. It affects the dominant and non-dominant hand equally and is usually a unilateral condition, but bilateral involvement may occur [2,3,5]. A history of a traumatic event is common and may have occurred prior to signs and symptoms of wrist pain and dysfunction [3]. Clinically, dorsal wrist swelling and tenderness are commonly apparent over the radiocarpal joint. Kienbock's disease is also associated with negative ulnar variance. It is postulated that the lunate bone in patients with Kienbock's is prone to high stresses causing repeated micro trauma and



Figure 3: X-ray showing avascular necrosis of lunate with pellet embedded inside it

eventually leading to necrosis. Subsequently, altered kinematics cause abnormal rotation of the scaphoid, which in turn, further increase the lunate loading and necrosis. Radiographs and magnetic resonance imaging (MRI) are the optimal diagnostic modalities for Kienbock's disease. Depending on the radiological progression [3,4], the disease has four stages:

Stage I: Radiographic findings are normal, but the bone scan findings are positive for the disease. MRI shows a decreased signal on T1- and T2-weighted images.

Stage II: Sclerotic changes and fractures are visible on radiographs; however, carpal integrity is intact.

Stage III (A and B): This stage occurs when the lunate collapses and the capitate migrates proximally. In Stage IIIA, no fixed carpal derangement is noted. In Stage IIIB, decreased carpal height, ulnar migration of the triquetrum, scapholunate dissociation, and flexion of the scaphoid are seen.

Stage IV: This stage is associated with additional carpal degeneration and generalized arthritis. Frequently, the patient will report tenderness with a bony palpation of the lunate. The final result of Kienbock's disease is lunate sclerosis, fragmentation, and collapse secondary to AVN [3].

Currently, there are many etiological theories of Kienbock's disease. According to the mechanical theory, AVN of the lunate bone is a consequence of a slow progressive collapse of the lunate under excessive bony stresses and excessive loading. Excessive load causes small repeated micro fractures of the lunate trabecula leading to subsequent bony collapse [3]. One of the most common mechanical etiological factors is an ulnar minus variant. A short ulna cannot share axial loads with the radius thus increasing force transmission through the lunate. This creates a "nutcracker effect" with increased compressive forces through the lunate from the radius and the capitate leading to repeated micro fractures [3,6-8].

The vascular supply theory states that the etiology of Kienbock's disease may be from a limited arterial supply to the dorsal or palmar aspect of the lunate. The proximal pole of the lunate is likely to become osteonecrotic due to its limited anastomosis of arterial supplying and the presence of only terminal arterial branches supply the proximal pole. Furthermore, impairment in the venous outflow can lead to an increase in intraosseous pressure in the lunate causing avascular changes and osteonecrosis [2,3,6,7]. Due to the limited blood supply of the lunate, especially the proximal pole, it is the main carpal bone vulnerable to post-traumatic AVN [9]. It has been theorized that vascular disturbances to the lunate, either congenital or acquired, may result in insufficient bony nutrition leading to an increased risk of developing AVN.

The traumatic theory of Kienbock's disease states that ligamentous rupture of ligaments attaching to the lunate may disrupt nutrient arteries supplying the internal aspect of the lunate causing avascular changes. Small micro fractures in the lunate may also lead to sclerosis, fragmentation, and collapse of the bone [2,3,6,7].

In our case, the vascular and traumatic theories may both have played a role. Interruption of the vascular supply of the lunate by the pellet and the foreign body reaction to the iron body plus vascular trauma at the time of injury compounded by the cavitation effect of the missile could be implicated in the causation of the necrosis. Furthermore, another possibility is mechanical overloading of the bone post-trauma causing ligamentous laxity.

The management of Kienbock's disease is highly dependent on the stage of the disease based on the Lichtman classification system, symptomatology, and functional deficits [3,6]. The methods for treatment of Kienbock's disease ranges from conservative care to operative care and focuses on the alleviation of subjective pain, improving function, and limiting the progression of the disease [3,6,10]. The most commonly used conservative therapies include immobilization with splinting or casting and activity modification [3,6-9]. When unsuccessful, various surgical options are available which include lunate excision with or without silicon replacement arthroplasty, shortening osteotomy of the radius to compensate for the negative ulnar variance, ulnar lengthening, intercarpal fusion, and revascularization. Radial shortening [5] is most commonly used, resulting in decreased pain and improved function in up to 90% of patients. Recent evidence, however, has questioned the trend of ulnar minus wrists leading to Kienbock's disease [11]. In spite of surgical correction, collapse and degeneration of the lunate may continue.

CONCLUSION

Kienbock's disease should be considered a differential diagnosis in patients with insidious, progressive wrist pain, especially in males between 20 and 40 years of age and those with a history of repetitive wrist trauma. Our case presented with dorsal and ventral pain that was insidious over 15 months and progressive in nature. There was pain on deep palpation of the lunate and surrounding ligaments. Since the blast injury was the only precipitating factor and considering Kienbock's disease may have a vascular or traumatic origin, we hypothesize that intralunate vascular anastomosis may have been disrupted by the initial trauma or subsequent foreign body reaction leading to bony ischemia, necrosis, and ultimately collapse of the proximal pole. We conclude that patients with foreign body trauma to the wrist should be regularly followed clinicoradiologically to preclude the development of carpal AVN and take early corrective measures in cases where it is seen on follow-up.

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