
CLINICAL ARTICLE

The incidence of asymptomatic Kienböck's disease

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Abstract

The aim of the study was to determine the incidence of asymptomatic Kienböck's disease in patients who attended the Dr George Mukhari Hospital (formerly: Ga-Rankuwa Hospital), and to determine the relevance of ulnar variance on the disease.

This was a retrospective study. In a 12-month period we reviewed postero-anterior X-rays of 1 287 patients seen at our radiology department, with complaints unrelated to the upper limb including the wrist and hand.

We identified 23 cases (1.87%) of asymptomatic Kienböck's disease. The majority (63%) were male with an average age of 49 years, and 37% were female with an average age of 46.5 years. All were unilateral and all were in the dominant hand. Thirteen cases (57%) had an ulna neutral wrist and the remaining ten (43%) had an ulnar negative variance. The vast majority (83%) were unemployed.

From the results of our study we could deduct with reasonable confidence that at least a 1.87% incidence of asymptomatic stage II, III and IV Kienböck's disease is present in the black African population. Analysis of the data shed no further light on the aetiology. The relevance of ulnar variance as an aetiological factor is seriously questioned.

Introduction

Kienböck's disease is a condition of uncertain aetiology that ultimately results in osteonecrosis of the carpal lunate. Presently, it is thought that the aetiology is probably multifactorial.

In 1843, Peste¹ first described the collapse of the carpal lunate in anatomic specimens. At that time he believed it was the result of a fracture and hence had a traumatic cause. In 1910, Dr Robert Kienböck,² a professor of radiology in Vienna wrote his classic article describing "Traumatic malacia of the lunate and its consequences:

degeneration and compression fractures". He also believed that the lesion was the result of trauma. Repeated sprains or contusions were thought to lead to ligamentous and vascular injury. Fifteen years later, Goldsmith³ reported the first cases of Kienböck's disease in the American literature and credited Speed⁴ with ascribing to it the name "Kienböck's disease". Goldsmith stated that because of the mildness of the symptoms and the frequent absence of a traumatic history, Kienböck's disease was frequently overlooked without the use of radiographs.

Two years later, in 1928, 18 years after the description of Kienböck's disease, Hulten⁵ made the association between ulnar variance and lunatomalacia. He examined 23 cases of Kienböck's disease and found that in 17 (74%) there was an ulnar negative variance, and in six (26%) the ulnar was equal in length to the radius.

Kostler⁶ however disagreed with the importance of an ulnar minus variant in the production of Kienböck's disease.

Gelberman and co-authors⁷ also found a statistically significant association between negative ulnar variance and Kienböck's disease but were however very careful to point out that it should not be considered a primary aetiological association but at best a predisposing factor.

Lee,⁸ in cadaver experiments, found that the lunate had three predictable vascular patterns: (1) a single volar or dorsal vessel; (2) several vessels, volar and dorsal, without a central anastomosis; and (3) several vessels, volar and dorsal, with a central anastomosis. He theorised that patients with the first two patterns were at greater risk of developing Kienböck's disease. Gelberman *et al*⁹ studied the extra- and intra-osseous blood supply in fresh specimens. He suggested that it is the intra-osseous disruption, caused by repeated trauma that resulted in avascular necrosis.

In the literature there is very little if anything written about the incidence in the general population with radiographic changes in keeping with Kienböck's disease but who seek no medical attention as a result of symptoms. In fact, only Taniguchi¹⁰ has written a report in which Kienböck's disease was diagnosed on wrist X-rays of 14 patients who had the radiographs taken for other hand and wrist problems.

The aim of our study was to determine the incidence of such a group of patients in our population.

Methods

In a 12-month period, all patients who presented to the radiology department of Dr George Mukhari Hospital with complaints unrelated to the upper limb, had, in addition to their requested radiographs, a posterior-anterior (PA) view taken of both their wrists on a single X-ray plate. All patients were specifically questioned about any previous injuries to their wrists and hands as well as any pain or discomfort of their wrists at the time of presentation. All patients therefore included in this survey had symptomless wrists and no previous injury to their wrists. Pathologies for which they presented to the hospital ranged from mandibular fractures and pulmonary tuberculosis to lumbar degenerative disease.

With the help of the radiographers, a form was completed that included the following information: age, gender, hand dominance, occupation and presenting complaint.

These radiographs were then collected and analysed by a single individual (senior orthopaedic registrar) to ascertain whether or not there were any with radiographic evidence of stage II-IV Kienböck's disease. These X-ray findings were also scrutinised by the senior author.

Results

A total of 1 287 PA X-rays of the wrist were analysed which included 734 males (74%), and 553 females (43%). Of all of these, 23 cases of Kienböck's disease were identified which comprised 1.87% of the patients. Fourteen of them were male (63%) with an average age of 49 years (28 years–77 years) and the rest (9) were female (37%) with an average age of 46.5 years (34 years–68 years). All of them were in the dominant hand. Only nine had an ulnar negative variance (43%), and the rest had an ulna neutral variance (57%). None had an ulna positive variance. In terms of occupation, only three were employed (17%) and the vast majority (83%) were unemployed. Of the 23 cases identified, seven had stage II disease (30%), 11 had stage III disease (48%) (*Figure 1*), and the remaining five had stage IV disease (22%).

Discussion

Our study indicates that 1.87% of persons in the black African population have asymptomatic stage II, III, and IV Kienböck's disease. This incidence must be higher (possibly over 2%) because stage I Kienböck's disease were not included. Of course, to determine stage I, cases need to be examined with MRI, which was not done.

Ulnar negative variance per se cannot be a contributing factor



Figure 1: Example of asymptomatic Kienböck's disease stage III A of the left wrist

One may argue that our sample group may not represent 'the general African population'; however, it would be practically impossible to find a representative group 'out there' and submit them to X-rays as well as MR imaging.

This study further indicates that not all cases of Kienböck's disease are painful or symptomatic, and therefore do not seek medical treatment. Taniguchi *et al* found radiographic worsening in 14 out of 20 patients (70%) whom they had followed for a period of 35 years. However, of those, only three (20%) had disabling symptoms. Long-term follow-up of our patients with re-examination would help in prognosticating the natural history of Kienböck's disease.

Our initial aim with this study was also to question the generally held belief that Kienböck's disease is at least in part due to ulnar variance. Our findings, also based on 23 cases, has had an ulnar negative variance of only 43% and a neutral variance if 57%, i.e. the majority. These statistics are meaningful and confirm that ulnar negative variance per se cannot be a contributing factor. This is in keeping with the study by Nakamura *et al*¹¹ where in 41 wrists with Kienböck's disease, they found no positive correlation with ulnar variance.

Since none of the identified cases had previous injury to their upper limbs, including the hand and wrists, trauma can also be excluded as a contributing factor to the development of Kienböck's disease.

Conclusion

Asymptomatic Kienböck's disease occurs in about 2% of the general African population. Trauma and ulnar variance do not seem to play a role in the development of this disease. The aetiology therefore still remains uncertain.

This article was submitted to an ethical committee for approval. The content of this article is the sole work of the authors.

No benefits of any form have been derived from any commercial party related directly or indirectly to the subject of this article.

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