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Acute Static Volar Intercalated Segment Instability (VISI) of the Wrist: A Case Report

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Abstract

Introduction: Isolated injuries of the lunotriquetral (LTq) interosseous ligament and associated structures are less common and less well understood than other forms of dissociative carpal instability. For a VISI deformity to occur the LTq interosseous ligament must be disrupted but the progression of lunotriquetral dissociation requires further ligamentous injury, particularly the palmar LTq ligament, and for full dissociation between the lunate and triquetrum, the dorsal radiocarpal ligaments, which finally results in a static volar intercalated segmental instability (VISI) pattern.

Case report: A 52-year old left-handed male patient of black origin was presented with a rare pattern of an acute static volar intercalated instability (VISI). Complete disruption of both the intrinsic lunotriquetral ligaments and the dorsal intercarpal ligaments were identified at surgery. Direct repair and dorsal capsulodesis plus temporary pinning of the lunotriquetral joint were carried out. The patient had a good clinical outcome at 2.5 years of follow up despite the persistent static VISI deformity in the last follow up radiographs.

Conclusion: This case represents a clinical proof of both anatomical and biomechanical studies that in order for a static VISI deformity to occur (Stage III), not only must the lunotriquetral interosseous ligament and the palmar lunotriquetral ligaments be disrupted but there must also be disruption to the dorsal capsule.

Keywords: Lunotriquetral instability; Static VISI deformity; Dorsal capsulodesis

Introduction

Isolated injuries of the lunotriquetral (LTq) interosseous ligament and associated structures are less common and less well understood than other forms of dissociative carpal instability. Taleisnik et al. [1] and Reagan et al. [2] have proposed that for a VISI deformity to occur the LTq interosseous ligament must be disrupted. Viegas et al. [3,4] and Horii et al. [5] took this work further in their anatomical studies and have described how the progression of lunotriquetral dissociation requires further ligamentous injury, particularly the palmar LTq ligament, and for full dissociation between the lunate and triquetrum, the dorsal radiocarpal ligaments, which finally results in a static volar intercalated segmental instability (VISI) pattern.

We report this case of an acute static VISI deformity as the intraoperative findings demonstrated which ligaments are disrupted, thus providing in "vivo" support for the anatomical work carried out by Viegas et al. [3,4] and Horii et al. [5].

Case Presentation

A 52-year old left-handed male of black origin, was transferred to the cardiothoracic unit of our hospital with bacterial endocarditis of the mitral valve. He also had a two-week history of a painful right wrist following a fall he had sustained whilst at home. He was referred to our service where he was reviewed on the ward. Examination revealed a right wrist that was fuller than the left and was generally tender to palpation. The range of motion was limited as compared to the left wrist, with loss of 20° of extension and 10° of flexion. Supination and pronation were unrestricted. There was no neurovascular deficit. Clinical examination demonstrated a moderately positive *ballottment test* between the lunate and the triquetrum and a strong positive *derby test* after dorsal loading of the pisiform bone during the "dart-throwing" motion of the wrist. The postero-anterior X-ray of the wrist demonstrated a flexed scaphoid

with positive ring sign, step-off at the lunotriquetral joint and slight increased scapholunate gap. The lateral projection demonstrated a typical static VISI pattern with abnormal radiolunate, scapholunate and capitolunate angles (Figure 1). The patient was taken to the theatre the following day. Through a dorsal approach, between the IV-V extensor

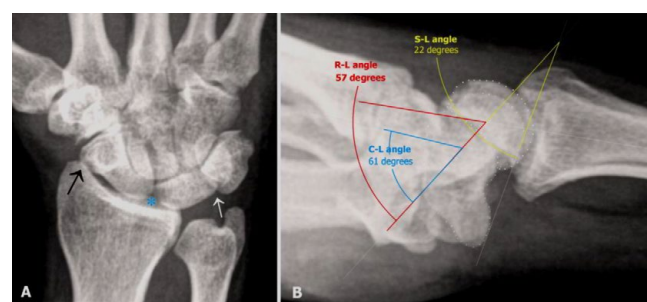


Figure 1: The postero-anterior X-ray of the wrist (A) demonstrated a flexed scaphoid (black arrow) with positive ring sign, step-off at the lunotriquetral joint (white arrow) and slight increased scapholunate gap (asterisk). The lateral projection (B) demonstrated a typical static VISI pattern with abnormal radiolunate, scapholunate and capitolunate angles.

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Figure 2: Intraoperative photo which shows the disruption of the dorsal capsule (A) and the grade III dissociation of the LTq joint (B).

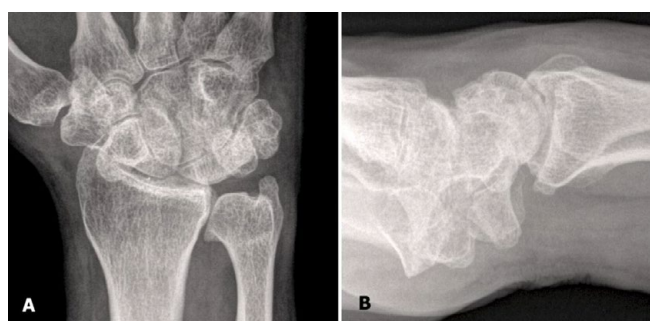


Figure 3: Follow up radiographs demonstrated flexion of both the scaphoid and lunate and proximal migration of the triquetrum; in the lateral projection there was a persistent static VISI deformity.

compartments, a complete rupture of the dorsal radiocarpal ligaments was identified together with a grade III disruption of the intrinsic LTq ligaments (Figure 2A and 2B). A 2.0 KW was used to stabilize the lunotriquetral joint and a direct repair of the interosseous ligament was performed with multiple sutures followed by dorsal capsulodesis. A below elbow back-slab was applied for a period of 6 weeks. The KW was removed after 6 weeks in the clinic, but the patient didn't attend any of his scheduled follow up appointments since then. He presented 2.5 years after the operation in the outpatient office for a different orthopaedic problem. Regarding his right wrist he confessed that he had had occasional pain and moderate difficulties with his work as a plaster. Posteroanterior check X-ray revealed the typical carpal bone displacement that occurs with the chronic static VISI deformity: flexion of both the scaphoid and lunate and proximal migration of the triquetrum; in the lateral projection there was a persistent static VISI deformity (Figure 3). However, physical examination showed good range of motion with 70° of dorsiflexion, 60° of palmar flexion, 20° of ulnar deviation and 30° of radial deviation. The Disabilities of the Arm, Shoulder and Hand (DASH) score was 33 and his grip strength 62% compared to the uninjured left wrist. As his present medical history involved an ischemic heart disease and a myelodysplastic syndrome requiring chemotherapy and multiple admissions in the hospital he refused any kind of surgical intervention regarding his right wrist.

Discussion

Volar Intercalated Segmental Instability (VISI) is defined by the International Wrist Investigators' Workshop [6] as a general class of symptomatic carpal instability characterized by pathologic volar flexion of the lunate, with or without a similar posture of the other proximal

carpal row bones, and asynchronous movement of the proximal and/or distal carpal rows. The carpal instability dissociative (CID) pattern of VISI represents the more advanced stage of lunotriquetral dissociation (LTD) which shows a static deformity with both scaphoid and lunate flexed, and the triquetrum neutral or extended. With further progression by involvement of the extrinsic or capsular ligaments, the deformity becomes severe and eventually fixed, as in our case, and becomes a CIC (carpal instability complex) type lesion at this stage.

A number of different pathomechanics have been described for the static VISI deformity to establish. Linscheid et al. [7] in their classic article believed that traumatic or congenital laxity of the palmar radiocarpal ligament was the key for the development of the "palmar-flexed intercalated segment instability". Garth et al. [8] reported that laxity of the capitotriquetral ligament results in failure of the triquetrum-hamate joint to produce a dorsiflexion moment, and the unbalanced volar flexion moment generated by the scaphoid produces VISI. Trumble et al. [9] suggested that release of lunotriquetral ligament in addition to the volar arcuate ligament was necessary to cause significant rotation of the lunate. According to the "progressive perilunar instability" theory by Mayfield et al. [10] injury to the lunotriquetral ligament occurs in stage III, following rupture of the scapholunate ligament (stage I) and lunocapitate ligament (stage II). Viegas et al. [3] suggested that isolated lunotriquetral ligament rupture may result from a reverse pattern of injury originated on the ulnar side of the wrist. In their anatomical and biomechanical study the authors found that the dorsal radiocarpal ligament must be attenuated or disrupted in order for a static VISI to develop; in less advanced ligament sectioning, when only the palmar and dorsal lunotriquetral ligaments were sectioned, increased mobility of the lunotriquetral joint was detected (dynamic VISI), but not complete disorganization of the carpus. Horii et al. [5] also confirmed these findings in their kinematic study: the essential lesion in producing static VISI was division of the dorsal radio-triquetral and dorsal scapho-triquetral ligaments in association with the lunotriquetral ligaments and interosseous membrane sectioning. In an ulterior study, Viegas et al. [4] investigated the anatomy and mechanical properties of the dorsal radiocarpal (DRC) and dorsal intercarpal (DIC) ligaments. They suggested that the lateral V configuration of the DRC and DIC ligament construct allows dorsal stability of the proximal pole of the scaphoid while still allowing a 3-fold increase in the distance between the DRC ligament radial attachment and the DIC ligament scaphoid attachment. The authors concluded that these findings have to be taken into account when a repair or reconstruction of the DISI (Dorsal Intercalated Segmental Instability) and VISI deformities is attempted. Finally, a modification of the Osterman and Seidman's [11] classification of lunotriquetral instabilities have been proposed by Green et al. [12] into six types, in which an acute static VISI is classified as type 3.

Regarding the mechanism of injury little information is known, but a fall on the outstretched hand with the wrist in radial deviation and midcarpal pronation has been proposed [2,13]. Our patient wasn't able to recall the mechanism of injury.

Radiological appearance of the static VISI deformity in the PA view is characterized by disruption of the normal convex arc of the proximal row, a shallow-appearing outline of the triquetrum, a moonlike appearance of the lunate and a possible ring sign of the flexed scaphoid. In the lateral view the lunate is tilted palmarly as is the scaphoid to provide a decreased scapholunate angle and an abnormally increased radiolunate and capitolunate angles. Reagan et al. [2] state that sometimes a decreased lunotriquetral angle (normally, 14°) can be identified, although the interpretation of the triquetrum is difficult

in the lateral view. The follow up radiograph of our patient revealed a persistent static VISI deformity, with marked flexion of the scaphoid and proximal migration of the triquetrum.

Treatment options in the static type of VISI are controversial. Ligament repair or reconstruction and partial or more extensive arthrodesis is the options but there are not forfeited complications. When the injury is acute, as in our case, direct repair of the ligaments and temporary stabilization of the lunotriquetral joint is an option, although it was proven that this kind of repair was probably insufficient to restore such a complex disorganization of the carpus. Shin et al. [14] reviewed 57 patients with isolated lunotriquetral injuries and reported a 40.7% complication rate after ligament repair. In the same study the probability of remaining free from complications at five years was 68.6% for the reconstruction group (8 patients), 13.5% for repair (27 patients) and less than 1% (22 patients) for arthrodesis. In this last group of arthrodesis, 40.9% patients developed nonunion and 22.7% ulnocarpal impaction. The authors stated that ligament reconstruction or repair seems superior to arthrodesis in terms of pain relief, strength and movement.

Conclusion

This rare case represents a clinical proof of the anatomical and biomechanical studies of Viegas et al. [3,4] and Horii et al. [5] that in order for a static VISI deformity to occur (Stage III), not only must the lunotriquetral interosseous ligament and the palmar lunotriquetral ligaments be disrupted but there must also be disruption to the dorsal capsule.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Authors' Contributions

RA analyzed and interpreted the patient data, clinical symptoms and signs as well as radiological evaluation and intraoperative imaging. AP and JC performed literature review and were the major contributors

in writing the manuscript. All authors read and approved the final manuscript.

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