Dysfunction of the Pisotriquetral Joint: Degenerative Arthritis Treated By Excision of the Pisiform

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abstract

From 1995 to 2000, 21 patients (14 women and 7 men) with a mean age of 42 were treated with excision of the pisiform for a dysfunction of the pisotriquetral joint. Follow-up ranged from 6 to 36 months (average: 30 months). The diagnoses included degenerative arthritis of the pisotriquetral joint (15 patients), degenerative arthritis associated with a ganglion (3 patients), and calcifications caused by flexor carpi ulnaris tendinopathy (3 patients). All patients had pain secondary to direct pressure on the pisiform. Side-to-side passive motion of the pisiform occasionally led to pain and crepitus. Degenerative arthritis and calcifications in the pisotriquetral joint were confirmed by a wrist radiograph (lateral view in 30° supination). In five patients, local injection with anesthetic temporarily resolved the symptoms. Excision of the pisiform resulted in complete relief of pain without functional deficit.

The pisiform is a carpal sesamoid bone lying within the fibers of the flexor carpi ulnaris tendon. The pisiform has only one articular surface—a smooth, oval, concave facet that articulates with the slightly convex triquetrum. The joint is enclosed by a loose but tough capsule that communicates in most instances with the radiocarpal joint.1

Viegas et al2 reported a communication between the proximal wrist joint and the pisotriquetral joint in 88% of the 76 wrists studied. The pisiform is bordered medially by the dorsal retinaculum and laterally by the palmar carpal ligament that forms the roof of the logo de Guyon. The ulnar nerve lies in close approximation to the radial aspect of the pisiform. The tendon of the flexor carpi ulnaris continues distally from the pisiform as the pisohamate and pisometacarpal ligaments.1 The ligamentous anatomy was categorized into three different types by Yamaguchi et al:3

- type A: pisohamate and pisometacarpal ligaments inserted on the palmar distal aspect of the pisiform,
- type B: pisohamate ligament inserted on the radial side of the pisiform, and
- type C: basic pattern of the type B anatomy with an additional ligament between the pisometacarpal ligament and the distal aspect of the hook of the hamate.

The etiologic factors that were believed to predispose, cause, or aggravate the pisotriquetral joint dysfunction were trauma (acute or chronic), instability (carpal tunnel release; hypermobile joint), ganglion, arthritis, and flexor carpi ulnaris tendinopathy.4

The degenerative changes of the pisotriquetral joint are believed to be one of the causes of ulnar-sided wrist pain. The location of degenerative changes, including chondromalacia and arthritis, was investigated in the pisotriquetral joint by Yamaguchi et al3 and classified into five types. This article reports our experience with excision of the pisiform bone to treat painful pisotriquetral joint dysfunction associated with degenerative arthritis.

MATERIALS AND METHODS

From 1995 to 2000, 21 patients (14 women and 7 men) aged 18 to 72 years (mean age: 42 years) were treated with excision of the pisiform for dysfunction of the pisotriquetral joint. Postoperative follow-up ranged from 6 to 36 months (average: 30 months). The diagnoses included degenerative arthritis in pisotriquetral

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joint (15) (Figure 1), degenerative arthritis associated with a ganglion (3), and flexor carpi ulnaris tendinopathy (3). In those with flexor carpi ulnaris tendinopathy, we observed a pisiform calcification that occurs most commonly in the flexor carpi ulnaris tendon insertion (Figure 2).

All patients initially were treated with conservative measures, which included splinting, anti-inflammatory medication, and local steroid injection. The diagnosis of dysfunction in the pisotriquetral joint was determined by a persistent pain in the joint and hypothenar eminence. The patient may report a painful clicking or locking sensation with certain motions of the wrist. Clinical examination usually reveals a well-localized tenderness to direct pressure over the pisiform. Side-to-side passive motion of the pisiform may produce pain and occasionally crepitus. Forced hyperextension of the wrist and resistance to palmar flexion and ulnar deviation, as well as to pronation and supination, may reproduce the symptoms.¹

Degenerative arthritis of the pisotriquetral joint was confirmed by a wrist radiograph (lateral view in 30° supination) and injection of the pisotriquetral joint with local anesthetic. A small injection of a local anesthetic agent into the pisotriquetral joint at the ulnar aspect of the wrist with relief of symptoms helps confirm the diagnosis. In five patients, the local injection with anesthetic temporarily resolved the symptoms. Radiographs in 30° supination may show the degenerative changes and calcifications in the pisotriquetral joint. Magnetic resonance imaging (MRI) studies confirmed the diagnosis of ganglion in three patients (Figure 3). The symptoms persisted in 21 patients, and they had a surgical treatment.

A zigzag incision is made on the anterior aspect of the pisiform bone, and a subperiosteal dissection of the pisiform is performed. The insertion of the flexor carpi ulnaris is preserved as are the pisiform metacarpal ligaments. The entire pisiform is removed. Since the pisiform metacarpal ligaments are preserved and the insertion of the flexor carpi ulnaris tendon is also preserved, the function of the flexor carpi ulnaris is essentially left intact.⁵ Associated pathologic findings in the pisotriquetral joint area included calcification in the flexor carpi ulnaris in three patients and a ganglion cyst in another three cases. Usually, the increased size of the ganglion necessitates surgical intervention. The wrist is immobilized with a dorsal plaster splint for about 10 days.

Macroscopic aspects of the pisiform bone revealed degenerative changes in all patients, which ranged from mild chondromalacia to advanced osteoarthritis. The changes were worse on the pisiform than on the triquetrum. At the end of the study, 20 patients were pain free and one had pain on strenuous activity. No patient complained of constant pain. Wrist range of motion was similar to that of the contralateral hand and no loss of range of motion was observed.

**DISCUSSION**

Dysfunction of the pisotriquetral joint complex may result from injury, either acute or chronic, to any of the supporting retinacular structures. In some patients this leads to pisotriquetral instability, followed by degenerative changes.⁶ Pisotriquetral joint dysfunction with or without a loose body is an uncommon diagnosis. Pisotriquetral joint dysfunction most commonly presents as either chondromalacia, osteoarthritis, or flexor carpi ulnaris tendinopa-
Dysfunction of the pisotriquetral joint | GÓMEZ ET AL

Figure 2: PA (A) and lateral (B) radiographs of a patient with flexor carpi ulnaris tendinopathy. Note the calcification present in the flexor carpi ulnaris tendon insertion.

Figure 3: MRI showing a ganglion in the pisotriquetral joint area (A, B).

However, it is necessary to consider other entities affecting the ulnar wrist, such as triangular fibrocartilage complex tears, ulnar impingement, lunotriquetral dysfunction, distal radioulnar joint subluxation, hook-of-hamate fracture/non-union and ulnar artery thrombosis.

The exact function of the pisiform is not known, but aside from being a focal point of soft tissue attachment on the medial wrist, it acts as a lever, much like the patella, and increases wrist flexion force. The pisiform derives its stability from soft-tissue attachments. The soft-tissue attachments to the pisiform include the flexor carpi ulnaris, extensor retinaculum, transverse carpal ligament, abductor digiti minimi, ulnar collateral ligament, triangular fibrocartilage complex, pisohamate ligament, pisometacarpal ligament, pisotriquetral joint capsule, and the presence of a ligamentous band running from the pisiform to the hook of the hamate as described by Hayes et al. The normal forces of the flexor carpi ulnaris tend to pull the pisiform proximally and mediially. The flexor carpi ulnaris tendon covers the proximal and anterior aspect of the pisiform. The function of this musculotendinous unit is dependent on the pisiform to act as a mediator, transferring the load to the pisometacarpal and pisohamate ligaments to flex the wrist. This tendon and the pisometacarpal and pisohamate ligaments appear to be the main stabilizers of the pisiform.

The multitude of pathologic diagnoses were summarized by Paley et al into four categories:

- primary osteoarthritis, no etiologic factors;
- secondary osteoarthritis, associated with a previous acute trauma, chronic repetitive trauma, instability (postdislocation, iatrogenic, joint laxity or subluxation), or other pathologic conditions (ganglion, osteochondritis dissecans, or synovial chondromatosis);
- other arthropathies (rheumatoid arthritis, gout, or psoriatic arthritis); and
- flexor carpi ulnaris tendinopathy.

In our study, the majority of the cases were attributable to primary osteoarthritis. The literature showed that degenerative changes in the pisotriquetral joint are associated with a previous acute trauma, chronic repetitive trauma, or instability. Other arthropathies are rarely a cause of pisotriquetral joint dysfunction. While rheumatoid arthritis generally involves this joint early on, involvement is rarely isolated to the pisotriquetral joint and rarely necessitates treatment. Gout and psoriatic arthritis have been reported to affect this joint. We did not observe any patient with pisotriquetral joint dysfunction and rheumatoid arthritis, gout, or psoriatic arthritis.

The localization of degenerative changes in the pisiform and triquetrum were classified into five types by Yamaguchi et al: type 1, central; type 2, peripheral; type 3, fan-shaped; type 4, combination of types 1, 2, or 3; and type 5, total. The incidence of degenerative changes in the pisotriquetral joint increases with age, and the degenerative changes in this joint occur most frequently in the distal, distal-radial, and radial aspect of the pisiform and triquetrum. In our study we observed degenerative changes in all excised pisiform bones, which ranged from mild chondromalacia to advanced osteoarthritis. The pathological changes are like those of osteoarthritis in other joints: destruction of articular cartilage, cleft, or annular osteophyte formation at the articular margins.

Vasiliou et al. in a kinematic study of the pisotriquetral joint, described radiographically the normal motion occurring at the pisotriquetral joint. Under normal circumstances, the width of the pisotriquetral joint, on the lateral radiograph with the forearm in 30° supination, is 1-4 mm. Pisiform kinematics were studied in vivo by Moojen et al. With ulnar deviation of the wrist, the
Brief Report

What is already known on this topic

- Dysfunction of the pisotriquetral joint is an uncommon diagnosis.
- It is necessary to consider other entities affecting the ulnar wrist, such as triangular fibrocartilage complex tears, ulnar impingement, lunotriquetral dysfunction, distal radioulnar joint subluxation, hook-of-hamate fracture/nonunion, and ulnar artery thrombosis.
- Pisotriquetral joint dysfunction most commonly presents as either chondromalacia, osteoarthritis, or flexor carpi ulnaris tendinopathy.

What this article adds

- We treated, with excision of the pisiform, the painful pisotriquetral joint dysfunction associated with degenerative arthritis and flexor carpi ulnaris tendinopathy.
- Excision of the pisiform resulted in complete relief of pain without functional deficit.

pisiform deviates ulnarily; with radial deviation, the pisiform extends and deviates radially. During wrist flexion the pisiform flexes; with wrist extension, the pisiform extends and deviates radially. Under normal circumstances multiple forces act on the pisiform, causing considerable motion between the pisiform and the triquetrum.

Painful diseases of the pisiform and the pisotriquetral joint are not rare. Early degenerative changes, mostly found at the radial-distal border of the pisiform joint surface, are mainly caused by forceful ulnar deviation and extension of the wrist. Compressing soft-tissue constraints of the pisiform by injury increases motion and secondary arthritis at the pisotriquetral joint.

The osteoarthritic changes of the pisotriquetral joint may be caused by a tendinous rupture or ulnar neuropathy. Takeda et al. reported a case of rupture of the flexor digitorum profundus tendon to the little finger secondary to osteoarthritis of the pisotriquetral joint. Mechanical attrition against the radial side of the pisiform was thought to be responsible for the rupture. Hayes et al. described a fibrous band superficial to the pisohamate ligament passing from the pisiform to the hamate. Ulnar nerve compression, called "pisohamate hiatus syndrome," may be caused by this band. However, ulnar neuropathy may also be caused by pisotriquetral joint instability. The ulnar nerve lies on the lateral side of the pisiform. Increased motion of the pisiform may cause nerve irritation or compression by distorting the Guyon's canal anatomy. The incidence of ulnar neuropathy associated with pisotriquetral joint dysfunction has been reported to be 33%. In our study, we observed no such complication.

Excision of the pisiform bone is indicated when medical treatment is unsuccessful and the symptoms continue to be disabling. Removal of the pisiform bone does not seem to interfere with the normal function of the hand. Arner and Hagberg used isometric and isokinetic testing to compare pisiformectomy with contralateral limb control. They found that pisiformectomy decreased wrist flexion strength without functional deficit or loss of range of motion. The soft-tissue confluence over the pisiform allows for subperiosteal pisiform excision and repair of the tissues without disturbing the flexor carpi ulnaris insertion. This should prevent significant decrease in wrist flexion force following pisiform excision. In our study, all patients reported complete relief of pain without loss of range of motion. Steinmann et al. reported a group of 8 patients with pisotriquetral joint dysfunction with associated loose bodies. Excision of pisotriquetral loose bodies appears to provide satisfactory relief of symptoms in patients without evidence of joint degeneration. In patients with associated chondromalacia or osteoarthritis of the pisotriquetral joint, pisiformectomy has been shown to provide symptomatic relief of ulnar-sided wrist pain.

REFERENCES