Hand and Wrist Injuries in Golfers and Their Treatment

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KEYWORDS

• Golf injury • Swing mechanism • Trigger finger • De Quervain disease • Tendinopathy • Pisiform ligament complex syndrome • Hook of hamate fracture

KEY POINTS

- Golf injuries of the hand and wrist are common and most of these injuries are related to overuse.
- To diagnose and to provide appropriate management for golf injuries of the hand and wrist, the kinematics of the golf swing should be understood.
- Initial treatment starts with cessation of golfing to rest the wrist and includes a splint or orthotic brace, nonsteroidal antiinflammatory drug medication with corticosteroid injection, and swing modification.
- Pisiform excision is the best treatment of the most severe chronic cases of pisiform ligament complex syndrome, especially if the patient's symptoms are intolerable and nonoperative treatment measures have failed.
- Delayed diagnosis of hook of hamate fracture may lead to complications, including flexor tendon rupture of the little or ring fingers and sensory or motor deficits of the ulnar nerve. Prompt surgical resection is recommended to hasten return to sport and to prevent further complications.

INTRODUCTION

Golf has become an increasingly popular sport, attracting new players from all ages and socioeconomic groups. Irrespective of physical condition or underlying diseases, most people can enjoy the sport and the health-related benefits of walking up to 7 or 8 km per 18 holes and relaxing in a pleasant natural environment. There are an estimated 60 million golfers worldwide, playing on 32,000 golf courses. With the rapid expansion and globalization of the sport, the International Olympic Committee has included it as an Olympic sport for 2016.

The potential causes of golf injuries usually involve overuse by too much practice, poor swing mechanism, inappropriate equipment, or striking the ground or an object other than the ball, such as a tree root. McCarroll and colleagues[1] reported that, in professional golfers, wrist injuries were most common, followed by injuries to the back, left hand, left shoulder, left knee, and left thumb in reference to a right-handed golfer. In amateur golfers, the back is the most common site of injury,

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followed by the elbow, hand, wrist, and shoulder. Compared with other sports, golf is a noncontact sport, is considered a low-risk activity, and does not require much physical skill or athletic ability to participate. However, with incorrect techniques, unexpected serious injuries can happen. In most sports in which a club, bat, or racket is held, the hand and wrist directly absorb all transmitted power from the impact.

A study of injuries and overuse syndromes in amateur golfers showed that almost 40% of players had injuries in more than 2 different sites.[2] On a driving range, amateur golfers tend to practice the same swing with the same clubs over and over again. In pursuit of perfection, they believe that the constant repetition of the swing is a good way to develop a reliable golf swing. Severity of reported injuries were minor in 52% of cases, moderate in 27%, and major in 22%.[2] Overuse proved to be the most important factor resulting in golf injuries. Even though professional and low-handicap golfers have a much better swing mechanism, they tend to experience more wrist and hand problems than amateurs do, because they have spent more time hitting balls, and their impact power is much greater than with amateurs. More experienced golfers intentionally aim to hit through the ball, taking a divot of turf with the club after ball contact, producing spin on the golf ball and thereby controlling its landing. This technique results in an increased contact force when the club hits the ball and ground, and this force is transmitted to the wrist and hand. A recent study of professional golfers showed that 30% had reported various wrist problems.[3] Most injuries (67%) occurred in the leading wrist (left side of a right-handed golfer) at the most common location, the ulnar side of the wrist (35%).

This article reviews the kinematics of the golf swing and provides a diagnosis and management recommendations for golf injuries of the hand and wrist. Traumatic thrombosis of the distal ulnar artery and distal ulnar nerve neuropathy are presented in the article on nerve entrapment syndrome in cyclists.

BIOMECHANICS OF THE GOLF SWING

The ideal golf swing hits the ball a specific distance and direction following the trajectory the player wants. Therefore, the golf swing is a highly coordinated, multisegment, rotational, closed-chain activity that requires strength, explosive power, flexibility, speed, and balance.[4] Golf swing speeds can reach more than 50 m/s, producing high levels of stress in the joints. In simpler terms, the golf swing is divided into 5 stages: setup (address), backswing, downswing, impact, and follow-through.

Regarding the setup, a correct grip is the foundation of a good golf swing. An overlapping grip is currently by far the most popular. This grip enables golfers with large or strong hands to perform powerful shots. However, those with small or weak hands may find it more difficult to control the club. For right-handed golfers, the interlocking grip takes the small finger of the right hand and interlocks it with the index finger of the left hand, a grip often recommended for players with small hands, like women or children. In addition to the grip patterns, choosing the proper size of grip helps prevent tendinitis. Using a grip that is too small can cost the player some power, but, if it is too big, the player needs much greater flexion power to hit long or straight shots. Correct grip size permits the fingers in a golfers' top hand to barely touch the palm. People with hand arthritis usually benefit from using a bigger golf grip. The club should be gripped as lightly as possible in order to minimize tension in the swing.

During setup, the leading, nondominant wrist begins the golf swing in a position of ulnar deviation when addressing the ball (**Fig. 1**A). During the backswing, the club is lifted away from the

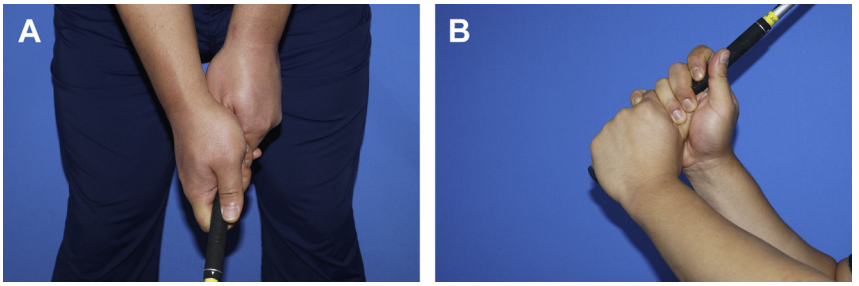

Fig. 1. (*A*) In the setup, the leading, nondominant left wrist begins the golf swing in a position of ulnar deviation when addressing the ball. (*B*) During the backswing, as the club is lifted away to the rear, the wrist moves into radial deviation until it sits maximally radially deviated at the top of the swing. The right wrist is in maximal extension at the top of the backswing.

player's core and the wrist moves into radial deviation until it sits maximally radially deviated at the top of the backswing (Fig. 1B). At this point, the club changes direction to begin the downswing and the leading, nondominant wrist returns to ulnar deviation until impact. An understanding of the opposing motion paths of ulnar to radial deviation for the leading, nondominant wrist and flexion/extension for the dominant wrist enables the mechanism of wrist and hand injuries in golf to be understood, especially when right-handed golfers use a driver. The right wrist is required to move in the planes of extension and flexion by 103° as well as radial and ulnar deviation by 45°.[5] In the left wrist, the arc is relatively small in flexion, with an extension of about 71° but slightly more radial and ulnar deviation of about 47°.[6] Through the downswing, most traumatic injuries occur at impact.

When the club hits the ball, it should strike the ball then the turf, taking a divot. However, hitting the ground before the ball can injure amateur golfers; this is known as a fat shot. By hitting fat shots, players can strike unseen tree roots, rocks, and other objects lying near the ball. The follow-through is the final phase of the swing, and it finishes in extension and radial deviation of both the leading and trailing wrists. Throughout the swing cycle, wrist motion is complex and serves as a potential source for injuries.

TYPES OF GOLF INJURIES

Golf injuries are the result of preexisting physical weaknesses that become manifest when following a specific activity. Moreover, because of overpractice, poor swing mechanism, or hitting the ground or an object, golf injuries can injure tendons, ligaments and joints, bones, vessels, and even nerves. Golf injuries are divided by timing as acute striking injury and repetitive strain injury. In the category of acute injury, there are triangular fibrocartilage complex (TFCC) injuries and carpal bone fractures, including scaphoid or hook of hamate fractures. Acute collateral ligament rupture of the digit may occur as a result of a loose grip. In amateur golfers, repetitive strain injury, such as of the trigger finger, is more common. Depending on the force transmission of the golf swing, repeated overpractice may result in tenosynovitis, like de Quervain disease, tendinitis of the flexor carpi ulnaris (FCU), extensor carpi ulnaris (ECU) dislocation, or even flexor tendon ruptures combined with hook of hamate fractures. Furthermore, peripheral nerve compression causes carpal tunnel syndrome and ulnar nerve compression at the Guyon canal.[7]

Anatomic location of the pathologic lesions can be divided into ulnar, radial, and dorsal wrist pain. The causes of ulnar wrist pain are ECU subluxation or tenosynovitis, or TFCC problems. In cases of radial wrist pain, there are de Quervain disease and intersection syndrome. Ganglia or extensor synovitis are the main causes of dorsal wrist pain from golf.

TRIGGER FINGER

With the pursuit of perfection through constant repetition of the swing, most beginner golfers feel stiffness and swelling of the digits the morning following an extended practice at a driving range. It commonly happens in golfers with a strong grip in the left hand. By practicing with the same clubs over and over again, the flexor tendons are impinged. With proximal interphalangeal (PIP) joint flexion, flexor tendons pass through a narrow A1 pulley. Particularly with a power grip, high angular loads are developed at the distal edge of the A1 pulley. It is like the effect of pulling a multifilament strand through the eye of a needle; bunching of the interwoven tendon fibers occurs,[8] resulting in reactive intratendinous swelling of the flexor tendon.[9] Moreover, the pulley shows gross hypertrophy that is whitish, cicatricial collarlike thickening. On ultrasonography, thickening and hypervascularization of the A1 pulley are the hallmarks of trigger fingers (Fig. 2A, B).

Symptoms vary greatly depending on the severity and the golfer. It usually starts as a vague pain on the palm and stiffness of the involved digits. Golfers feel catching of the digits and cannot actively extend the digit, requiring passive extension. In addition, the digits show catching with a fixed flexion contracture of the PIP joint, sometimes accompanied by pain on the dorsal aspect of PIP joint.

Trigger digit in golfers is usually spontaneously resolved in the early stage. First, the grip habit should be changed. A player's grip should not be tight, but it should be gentle. Because a worn grip causes the golfer to grasp the club tighter, clubs should be regripped regularly. The traction that a fresh grip provides lets golfers hold the club lightly. Most trigger digits can be treated with steroid injection, nonsteroidal antiinflammatory drugs (NSAIDs), and/or splinting. Corticosteroid injection has a high satisfaction rate, particularly in nondiabetic patients with involvement of a single digit, a discrete palpable nodule, and a short duration of symptoms. All patients with trigger digits should be offered 2 or 3 trials of corticosteroid injections. Two corticosteroid injections followed by surgery was the least costly

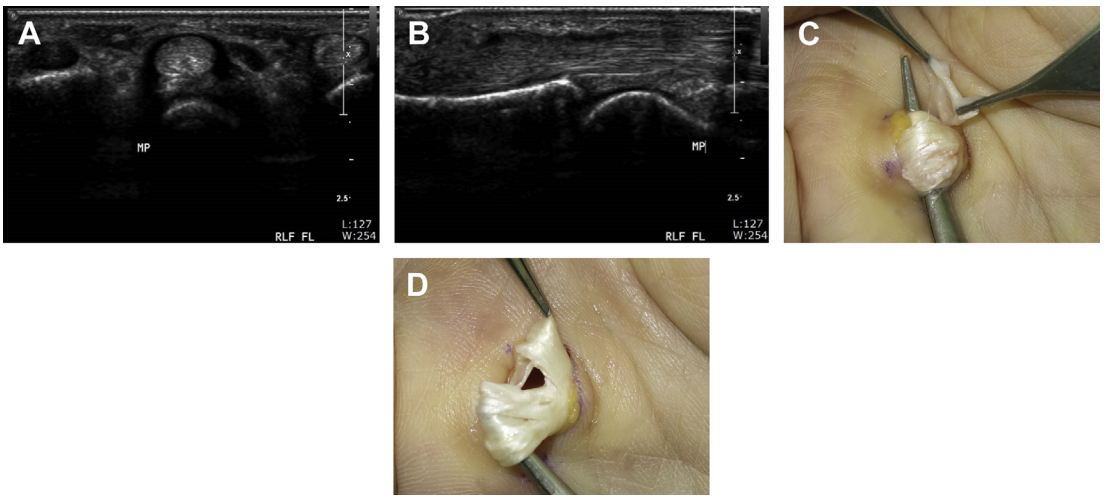

Fig. 2. (*A*, *B*) Axial and longitudinal ultrasonography images show mild hypoechoic thickening of the A1 pulley with minimal peritendinous fluid along the flexor tendon. (*C*, *D*) Beginner golfer: a 45-year-old man practiced 7 days a week at a driving range for 3 months. He hit more than 200 balls every day. On the A1 pulley release of the right long finger, the flexor tendon ruptured longitudinally and tenosynovium was hypertrophied.

algorithm and was less expensive than immediate surgical release.[10] Patients with trigger digits caused by rheumatoid arthritis or diabetes are recommended to have a second injection. When the open surgical release is scheduled, a corticosteroid injection should be avoided in the first 6 weeks before the operation. Under local anesthesia, the A1 pulley is released through a transverse, oblique, or vertical incision (Fig. 2C, D). After operation, immediate motion of the digits is advised. The incidence of postoperative complications is neither high nor serious. Pain in the incision site, fluid collection, and mild flexion contraction or stiffness of the digits resolve spontaneously. Rarely are there serious complications, including nerve transection, infection, or contracture of the PIP joint requiring secondary surgery.

DE QUERVAIN DISEASE

The first extensor compartment of the wrist, which is over the styloid process of the radius, contains the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) tendons. These tendons pass through the fibro-osseous tunnel, about 2 to 2.5 cm in length, which is covered by tough, overlying transverse fibers of the dorsal ligament. De Quervain disease is friction at the rigid retinacular sheath, with subsequent swelling or narrowing of the tunnel. It is caused by chronic inflammation of the APL and EPB, or aberrant slips of APL tendons, or presence of ganglion.

De Quervain happens more often in female golfers when they are between their 30s and 60s. Most cases are caused by poor swing mechanism. An excessive radial deviation of the left wrist in the backswing of right-handed golfers is a causal factor of de Quervain disease. Repeated thumb extension at the top of the backswing and a sudden deceleration at impact can produce or exacerbate symptoms. Beginner golfers or higher-handicapped golfers tend to release the hand early instead of retaining it in the cocked position. Early release of hand results in an off-plane swing following an outside to inside path, causing a slice of the golf ball and a significant loss of distance. In right-handed golfers, this motion allows an abrupt ulnar deviation of the left wrist, whereas the left thumb is more or less trapped in a fixed position between the right hand and the golf club.

Diagnosis is easily made by localized pain on the radial side of the wrist and is aggravated by the movement of the thumb. The Finkelstein test is the classic diagnostic test for de Quervain disease.[11] To perform the test, the examiner grasps the thumb and ulnar deviates the hand. However, it often leads to misdiagnosis. Eichhoff[12] modified the test by asking patients to flex the thumb and to clench the fist over the thumb before ulnar deviation. This process of ulnar deviation is performed by the practitioner (Fig. 3A).[12] Repetition of the patient's symptoms confirms a positive result, but it is essential to compare this with the normal contralateral side. Wrist hyperflexion and abduction of the thumb is a dynamic test that isolates the tendons within the first extensor compartment.[13] With the wrist maximally flexed, the patient is asked to abduct the thumb against

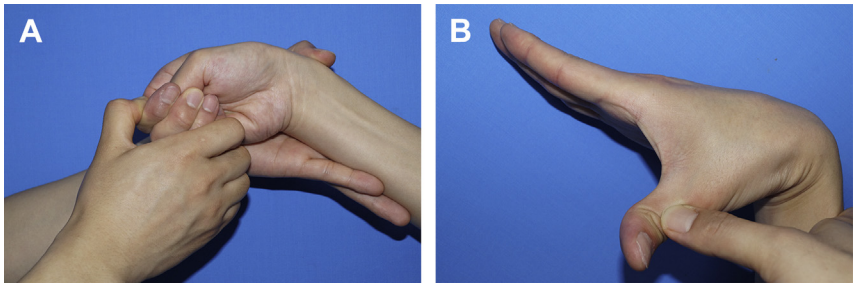

Fig. 3. (*A*) Eichhoff modification of the Finkelstein test. (*B*) The wrist hyperflexion and abduction of the thumb test is a dynamic test that isolates the tendons within the first extensor compartment.

resistance by the examiner (Fig. 3B). A positive test reproduces the patient's symptoms. With a sensitivity of 0.99 and specificity of 0.29, this test may allow clinician to more accurately diagnose de Quervain disease rather than using the Eichhoff test alone with a sensitivity of 0.89 and specificity of 0.14.[13]

Sonography is useful to diagnose de Quervain disease and to identify the presence of intracompartmental septum or ganglion as well as the number of aberrant slips of APL tendons in the first extensor compartment[14] (Fig. 4A, B).

Swing modification is the first treatment of de Quervain disease in golfers.[15] Initial treatment starts with the cessation of playing golf to rest the wrist. A thumb spica splint or orthotic brace is recommended with the wrist gently extended and the thumb abducted. NSAIDs are also helpful. The most effective conservative treatment is corticosteroid and lidocaine injection for relief of persistent symptoms. A satisfactory response to a diagnostic injection should precede the decision for surgery. Corticosteroid injection is successful in 50% to 80% of patients after 1 to 2 injections,

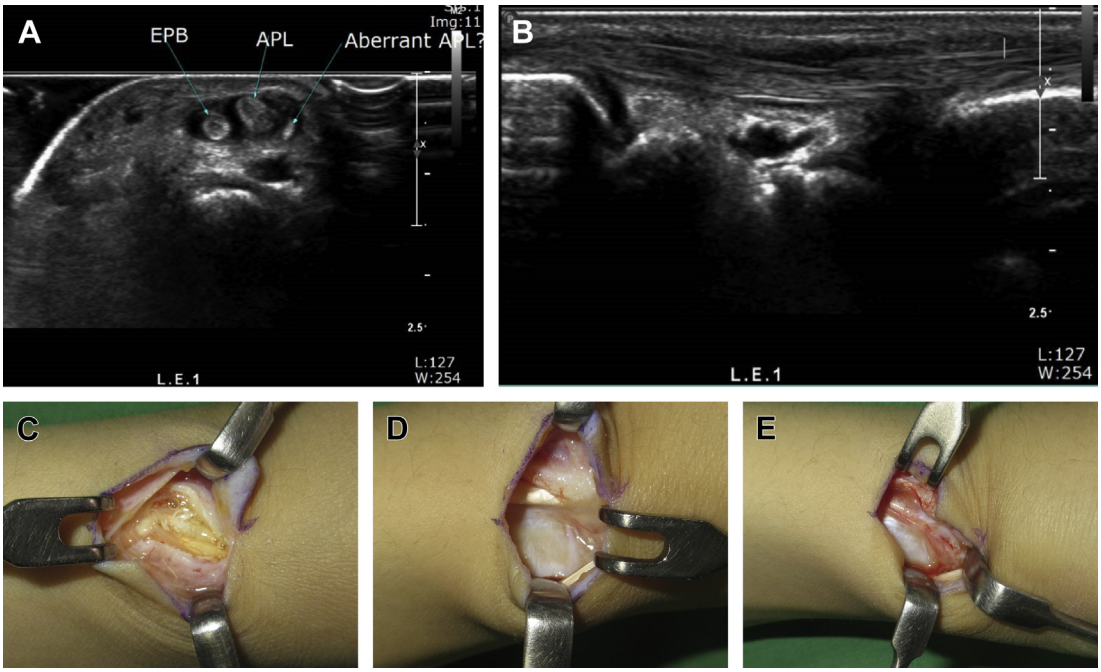

Fig. 4. A 28-year-old female golfer developed serious pain on the first extensor compartment. (*A*, *B*) On the axial and longitudinal ultrasonography views, aberrant APL tendon with fluid collection was found within the first extensor compartment. (*C*) Through the zigzag skin incision, the most dorsal part of the first extensor compartment opened revealing fluid collection and longitudinal rupture of the tendons. (*D*) Inside the first compartment, there was a septum with an aberrant tendon. (*E*) Intracompartmental septum was completely released.

and is particularly effective in acute cases.[16] Because of local complications of insoluble steroid, including localized atrophy of the subcutaneous tissues, fat necrosis, and discoloration, water-soluble dexamethasone or betamethasone mixed with 1% lidocaine is preferred.

A zigzag skin incision is made over the first dorsal compartment about 1 cm proximal to the tip of the radial styloid process. During incision of skin and dissection of the extensor retinaculum and tendons, care is taken to avoid iatrogenic injury to even small branches of the superficial sensory nerves. The hypertrophied annular ligament of the first compartment is sharply incised on its most dorsal margin to prevent tendon subluxation volarly. On traction of all exposed tendons, a thorough exploration for hidden aberrant tendons inside intracompartmental septum is necessary. Failure to identify and release a separate compartment is the main cause of recurrence of pain (**Fig. 4**C–E).

Wound massage is recommended after the stitches are removed. Short iron practice or approach shot with wedge clubs can commence about 4 to 6 weeks postoperatively.

INTERSECTION SYNDROME

Intersection syndrome is a localized inflammation of the peritendinous tissue at the intersection of the APL, the EPB, and the radial wrist extensors just proximal to the wrist's dorsal retinaculum.[17] In this condition the APL and EPB are irritated at the point where they cross over the second dorsal compartment of extensor carpi radialis longus and brevis. It should be differentiated from de Quervain disease. The tenderness point and significant soft tissue swelling with marked crepitus is about 6 to 8 cm more proximal to the Lister tubercle than de Quervain disease. These overuse-type injuries should be treated similarly to de Quervain disease. Conservative treatment includes rest, splinting, stretching and strengthening exercises, and NSAIDs. If this therapy is not effective, corticosteroid injection or surgical decompression may be required. It is frequently seen in golf swings that require repetitive wrist flexion and extension against resistance. Nonoperative treatment is usually successful. Initial measures include NSAIDs, avoidance of aggravating activities, and splint immobilization. When this fails, steroid injection is advocated into the area of the APL bursa. Following injection, immobilization should be continued for 1 to 2 weeks. In recalcitrant cases, surgical exploration and debridement of inflamed tenosynovium may be necessary. Postoperative immobilization should be limited following surgery to start gentle range-of-motion exercises. When the patient has regained range of motion and strength, golf participation can be resumed.

FLEXOR CARPI RADIALIS TENDINITIS

Flexor carpi radialis (FCR) tendinitis is usually found in the right hand of right-handed golfers. Repetitive wrist flexion against resistance causes FCR tendinitis in the right hand during the swing. The pain and tenderness are most prominent at the palmar wrist crease over the scaphoid tubercle where it is enveloped by its fibro-osseous sheath. In addition, localized swelling or ganglion may be present at that site. Resisted wrist flexion and radial deviation increase pain and are pathognomonic signs in FCR tendinitis.[18]

FCR tendon deviates about an average of 45° relative to the longitudinal axis of the forearm in the tunnel before inserting at the base of the index and long finger metacarpals.[19] This characteristic anatomic feature makes this tendon vulnerable to repetitive flexion and extension of the wrist.

Successful relief of pain after infiltration of the tendon sheath with 1 mL of 1% lidocaine can confirm the diagnosis. However, the multiplicity of other diagnoses, including basal joint degenerative disease, scaphotrapezium-trapezoid degenerative osteoarthritis, ganglion cyst, scaphoid fractures and nonunion, and de Quervain disease, may lead to misdiagnosis. Therefore, MRI can be used to differentiate between these conditions and confirm the diagnosis.[18,20]

Nonoperative treatment is recommended initially, with a period of splint, ice, NSAIDs, wrist extensions, stretching exercises, and the avoidance of golf for 4 to 6 weeks. Corticosteroid injection into the FCR tendon sheath can be used.[15,18] In cases in which nonoperative treatment is ineffective, decompression of the FCR fibro-osseous tunnel may be required.[21,22]

FLEXOR CARPI ULNARIS TENDINOPATHY

The FCU tendon of the wrist may be injured because of microtrauma from forces produced by the golf swing just before impact. When the club hits the ground before the ball and takes a divot (a fat shot), there is a sudden overload on the flexor tendon, leading to injury.[15] FCU tendinopathy is more common in the right hand of right-handed golfers because of the range of flexion and extension during the golf swing. The

most common presentation is calcified tendinitis of the FCU.

The FCU is a large muscle and the most powerful wrist motor, but it does not have a synovial sheath. It inserts into the proximal and anterior aspect of the pisiform, a sesamoid bone located within the FCU, which articulates with the volar surface of the triquetrum. Because there is no inherent stability of the pisotriquetral joint, stability depends on the pisohamate and pisometacarpal ligaments.[23] The ulnar neurovascular bundle lies on the radial side of the FCU tendon just proximal to the wrist joint. It passes just radial to the pisiform at the Guyon canal, which may cause associated ulnar nerve symptoms.

Usually, carpal tunnel view and tangential radiographs of the volar and ulnar aspects of the wrist reveal calcium deposits distal to the ulna (Fig. 5). MRI reveals the signs of tendinopathy with increased signal on T1-weighted and T2-weighted images and can also show the calcification deposition of calcified tendinitis.

The first treatment of tendinopathy of the FCU is decreased practice intensity, or grip or swing alteration. It is most commonly treated with nonoperative options, including rest, immobilization, NSAIDs, and occasionally corticosteroid injection. At least 6 months of conservative management is recommended before adopting surgical treatment. FCU tendinitis that does not respond to nonoperative treatment may be relieved by 5-mm Z-plasty lengthening of the tendon proximal to its insertion on the pisiform.[24] Because FCU tendinopathy is degenerative tendinosis of extrasynovial tendons, surgical debridement of the pathologic tendinosis tissue is also an effective treatment of patients who fail nonsurgical management. Minor tendon slips branching from the main tendon to the adjacent soft tissue should be released. In a scraping fashion, excision of the degenerative tissue should proceed until only healthy tissue remains.[23] Excision of calcific deposits, lysis of peritendinous adhesions, and repair of the FCU tendon are necessary.[25] If the pathologic process primarily involves the pisiform, excision of the pisiform is the most commonly used surgical procedure.

PISOTRIQUETRAL ARTHRITIS

Pisotriquetral (PT) arthritis or PT instability is another potential cause of ulnar-sided wrist pain in golfers. PT arthritis is associated with local pain and tenderness that are aggravated by the grinding of the pisiform dorsally against the triquetrum. Instability may be subtle and more difficult to diagnose. A diagnostic injection of local anesthetic in combination with appropriate radiographic imaging can confirm both diagnoses.

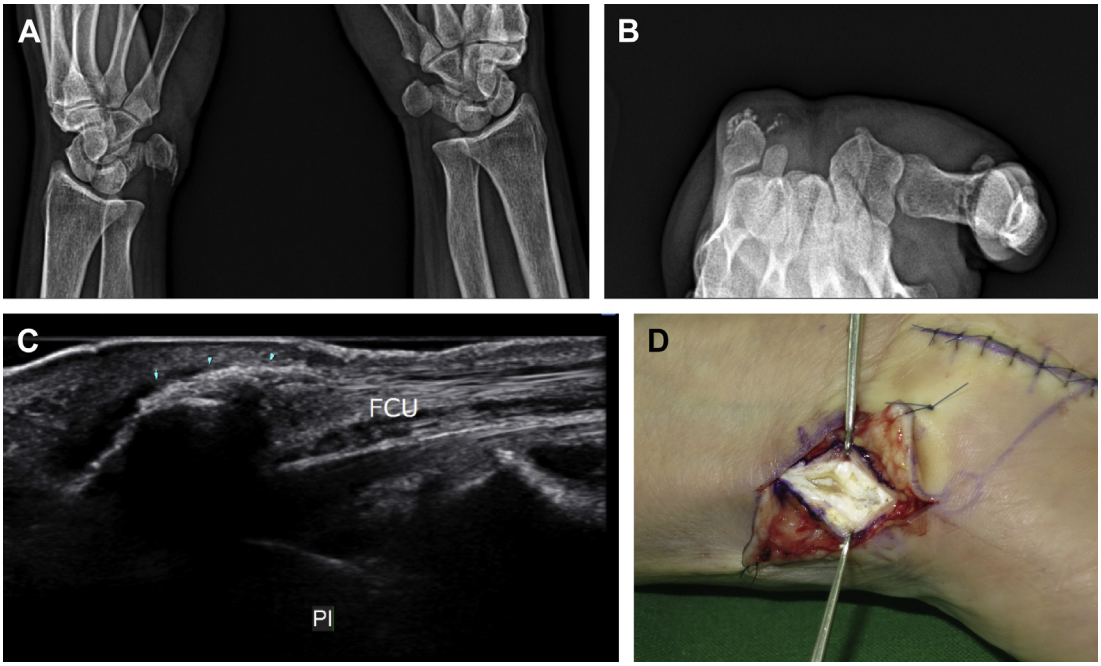

Fig. 5. (*A–C*) A 56-year-old female patient already had several corticosteroid injections because of severe ulnar-side pain. On pisiform bone view and carpal tunnel view, ultrasonography showed marked calcific deposits around the FCU and pisiform (PI). (*D*) Intraoperative view of calcification.

Pisiform ligament complex (PLC) syndrome is defined as ulnar palmar wrist pain in the vicinity of the pisiform and is caused by injury to the components of the PLC leading to PT joint instability with subsequent arthrosis. Primary osteoarthritis of the PT joint is uncommon and many arthritic disorders of this joint are posttraumatic, preceded by chronic PT joint instability. FCU tendinopathy is often associated with PT instability and PT arthritis. Narrowing of PT joint space while the wrist is in a neutral position is common in patients who have chronic instability of the PT joint and moderate arthrosis. The patient's radiograph shows marked widening of PT joint space during wrist flexion (Fig. 6).

Paley and colleagues[26] analyzed pathologic conditions of the pisiform from 216 cases identified from the literature and classified them into 4 pathologic groups: primary osteoarthritis (2.3%), secondary osteoarthritis (48.4%), other arthritides (4.7%), and FCU enthesopathy (44.6%). The most common causes were acute and chronic trauma and instability. Pisiform excision is the best treatment of the most severe chronic cases of PLC syndrome, especially if the patient's symptoms are intolerable and if nonoperative treatment measures have failed. Indications for pisiformectomy are painful nonunion of the pisiform, PTA, and FCU tendinitis.[27] A recent study on wrist function after pisiform excision showed that there is no significant difference in patients' grip and pinch strength, flexion and extension forces, ulnar and radial deviation, and flexion between the operated and nonsurgical wrists. However, wrist extension was significantly reduced in operated wrists compared with patients' nonsurgical wrists.[28]

EXTENSOR CARPI ULNARIS DISORDERS

During the golf swing, when the wrist supinates, the ulnar deviates and flexes during impact, and a painful snapping or clicking sensation can occur over the dorso-ulnar side of the wrist as the tendon shifts in and out of the shallow sulcus. In higher-handicapped golfers, a casting maneuver at the start of the golf swing risks development of this condition. Casting maneuver is an early release of the hand instead of leaving it in the retained cocked position, which allows wrist release at impact. A similar condition can be caused by the sudden ulnar load to the wrist from the club striking the ground with a fat shot or striking a stone instead of the golf ball.

Anatomically, the ECU muscle's actions vary depending on the forearm position. During supination, the ECU tendon moves dorsally closer to the extensor digiti minimi. In full supination, it is subjected to maximal traction and exits the sixth compartment at an angle of 30°, resulting in a greater contribution to true wrist extension. During pronation, the ECU tendon lies more in the palmar and ulnar positions of the ulnar head, far from the extensor digiti minimi, and exits the sixth compartment in a straight direction, resulting in a diminishing of its contribution to wrist extension. Therefore, tension on the ECU subsheath and retinaculum is increased in forearm supination with the wrist in flexion and ulnar deviation.[29]

The hallmark of the physical examination pointing to localized ECU disorder includes pain and tenderness directly over the ECU tendon and the sixth dorsal compartment where the pain is exacerbated by resisted wrist active extension with ulnar deviation. Swelling along the course of the ECU tendon is evident. In the case of ECU tendon instability, active supination, flexion, and ulnar deviation often produce visible subluxation of the tendon. In diagnosis, plain radiographs occasionally show calcification within the ECU tendon. However, in most cases, it is difficult to diagnose ECU tendinopathy and instability with only plain radiographs and clinical presentation. Therefore, ultrasonography and/or MRI are the imaging modalities of choice in the diagnosis of ECU tendinopathy and instability.[30,31] ECU tendon disorder can be broken down anatomically into 3 components that sometimes create multifactorial problems. The first is inflammation or tenosynovitis surrounding the ECU tendon; the second is tendinopathy caused by intrinsic damage or tendinosis; the third is mechanical failure resulting from bowstringing and subluxation/dislocation of the ECU tendon.

EXTENSOR CARPI ULNARIS TENOSYNOVITIS

Acute ECU tenosynovitis is defined by inflammation of the tenosynovium of the ECU without significant stenosis or an underlying bony abnormality of the sixth dorsal compartment. Ultrasonography shows compressible anechoic fluid surrounding the tendon with minimal or no vascularity on Doppler (Fig. 7). The treatment of acute ECU tenosynovitis includes the cessation of golf until symptoms subside, short-arm splinting of the wrist in a position of 30° to 40° of extension for 2 weeks, and oral NSAIDs.[15,30] If symptoms persist, a corticosteroid injection into the sixth dorsal compartment is recommended.

EXTENSOR CARPI ULNARIS TENDINOPATHY

ECU tendinopathy develops gradually. In general, it is possible to continue to play golf despite the

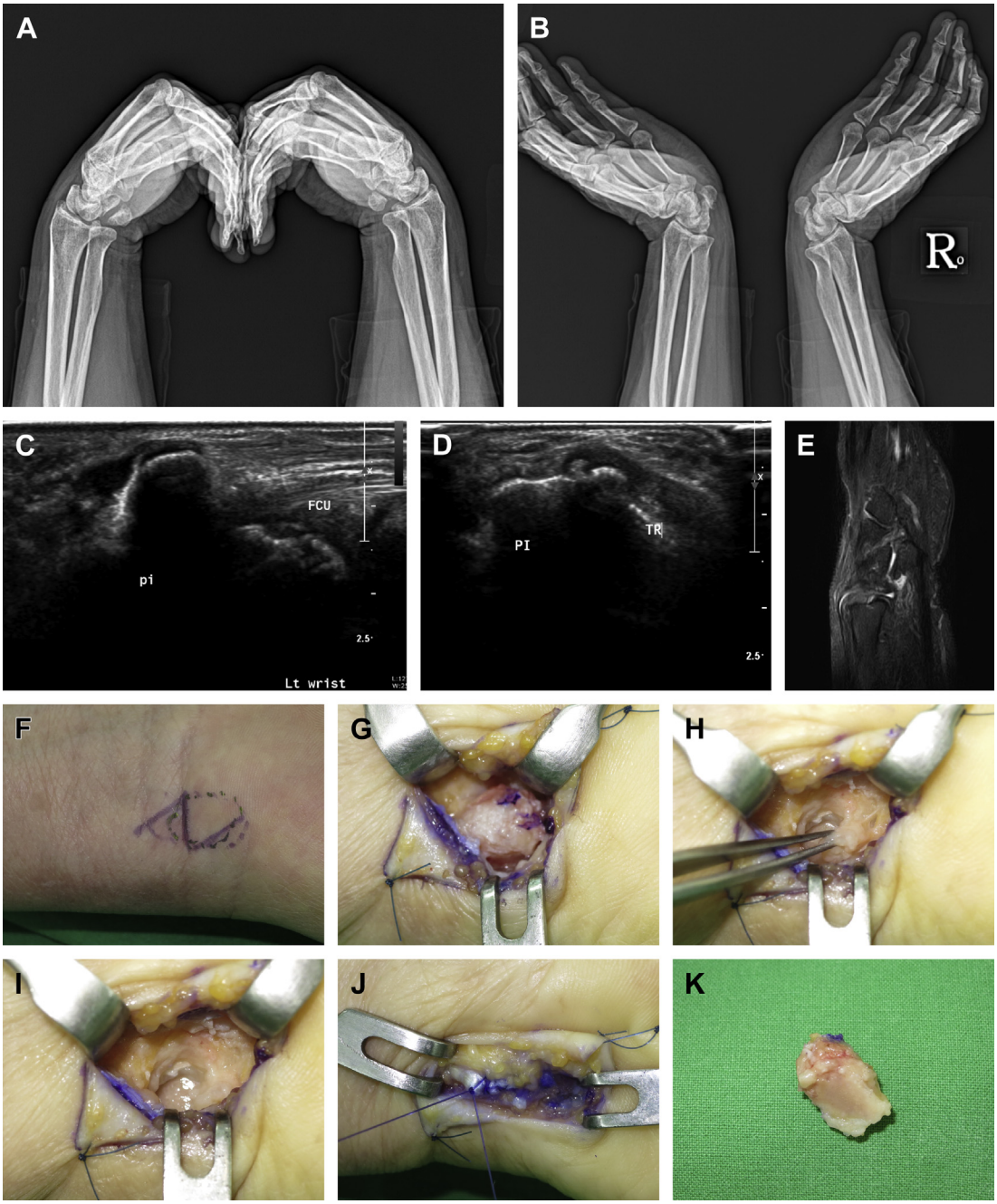

Fig. 6. (*A*, *B*) A 55-year-old female patient with severe pain in the left wrist. On semisupination oblique view of the wrist (pisiform bone view), the pisotriquetral joint of the left wrist showed marked joint space widening and sclerotic change. (*C*, *D*). Axial and longitudinal images of ultrasonography show mild FCU tendon thickening and some fluid collection around the pisiform. (*E*) Fat-suppression T2-weighted sagittal image showed FCU thickening with increased signal intensity and osteophytes and cartilage loss of the pisotriquetral joint. (*F*, *G*) Through the Z-shaped incision on the volar crease of the ulnar wrist, pisiform was subperiosteally dissected through the midline splitting of the FCU tendon. (*H*) Incidental osteophyte in the pisotriquetral joint was removed. (*I*) Cartilage of the triquetrum was denuded. (*J*) After excision of the pisiform, the flexor carpi ulnar tendon was repaired with 4-0 PDS suture in a continuous figure-of-eight method. (*K*) Excised pisiform showed almost totally denuded cartilage of the pisiform. TR, triquetrum; PI, pisiform.

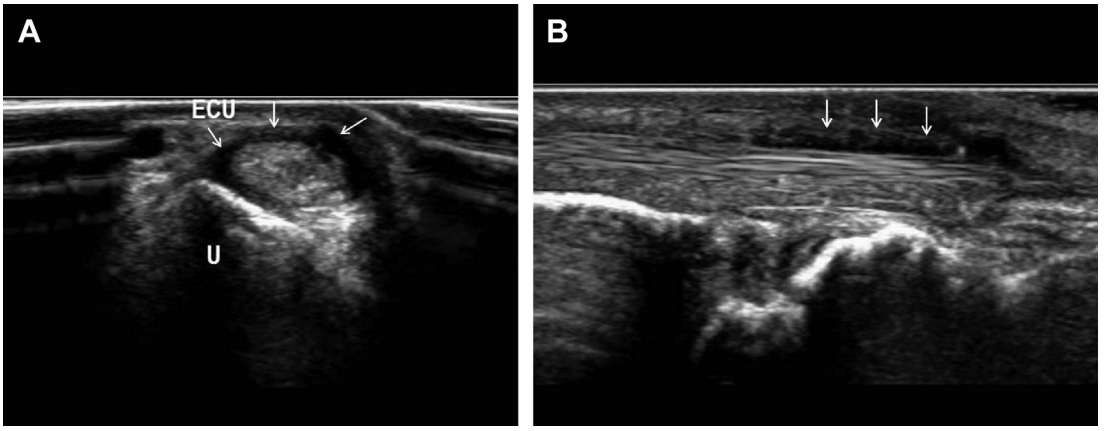

Fig. 7. Ultrasonography of the right wrist ECU tenosynovitis. Longitudinal (*A*) and axial (*B*) views showed anechoic fluid in the tendon sheath (*white arrows*). U, ulna.

pain or failure of a patient with tenosynovitis to respond to appropriate treatment. As the disease progresses, tendon thickening becomes more pronounced, resulting in stenosing tenosynovitis. The tendon can become unstable and dislocated from attenuation or tearing of its subsheath.[30,32] The ECU tendon can be ruptured partially by gliding over the ulnar ridge of the groove or a bony spur. MRI shows moderate increased signal intensity at the area of tendinopathy and tendon thickening. In the case of partial tendon tears, MRI reveals clefts or splits within the tendon substance (**Fig. 8**). Initial treatment should be conservative. If symptoms are not relieved by conservative measures, it can also be treated by corticosteroid injection into the sheath. In patients with recalcitrant symptoms, sixth dorsal compartment release should be considered.[33] After the division of retinaculum and subsheath, the tendon should be inspected for tearing of the sheath; spur and prominent ridges should be repaired or trimmed.

EXTENSOR CARPI ULNARIS TENDON INSTABILITY

The ECU tendon is stabilized by a unique fibro-osseous sheath (subsheath) deep to the extensor retinaculum. This deep subsheath maintains the tendon's normal position.[30,34] Therefore, instability of the ECU can result following disruption of the subsheath even if the extensor retinaculum was

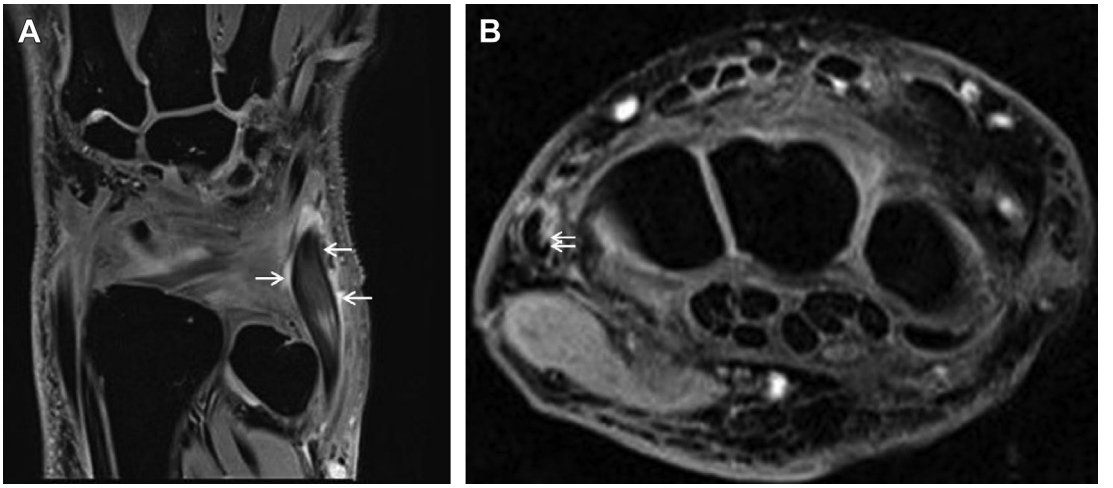

Fig. 8. (*A*) Coronal section of fat-saturated T2-weighted magnetic resonance images of ECU tendinopathy show moderately increased signal intensity at the area of tendinopathy and tendon thickening (*white arrows*). (*B*) Axial section shows a linear area of high signal intensity within the ECU tendon (*white arrows*) representing a partial tear.

intact. The exact mechanism is not clear, but generally this condition is seen with forceful supination with wrist flexion and ulnar deviation.[33,35] During impact, a painful snapping sensation can occur over the ulnar aspect of the wrist. Confirmed diagnosis can be made with dynamic ultrasonography and MRI. Treatment of the acute ECU tendon instability consists of 4 to 6 weeks of long-arm splinting or casting with the wrist in extension, radial deviation, and forearm in pronation followed by an additional 4 weeks of removable splint.[36] In chronic ECU tendon instability, surgical reconstruction of the ECU subsheath may be indicated.[37]

HOOK OF HAMATE FRACTURE

Most patients do not realize that a hamate fracture can happen from a golf swing error. Hook of hamate fractures occur almost exclusively in the leading hand. Anatomically, the prominent hook of hamate is easily broken when the golfer strikes the ground abruptly. After repetitive practice of golf swing or a sudden painful event such as hitting the ground or stone, players may feel a vague pain and focal tenderness on the hypothenar eminence. Tenderness to palpation is felt over the hamate hook approximately 2 cm distal and radial to the pisiform. Patients complain of pain aggravated by active grasping. Sometimes, patients complain of numbness of the ring and small fingers. However, standard radiographs of the hand and wrist fail to show a definite fracture line. Even hand surgeons tend to misdiagnose this as a repetitive strain injury or nerve-related problem. Over time, finger flexion of the small and/or ring fingers becomes affected, resulting in complete rupture of the flexor tendons.

The mechanism of fracture is the force directly transmitted through the butt of the golf club to the hook of hamate. Prevention of this injury is by selecting golf clubs of appropriate length. Correct fitting and a proper club grip should allow the butt of the club to extend beyond the hypothenar border of the hand. If the clubs are short, the club end is directly against the hamate.

Diagnosis of a hook of hamate fracture mainly depends on the history of overpractice or painful memories of club strike and physical examination. On the radiograph, the carpal tunnel view is the best tool to check the profile of the hook of hamate (**Fig. 9**). Computed tomography scan can show definite fracture line with high sensitivity and accuracy.[38] In suspicious cases of combined flexor tendon ruptures, ultrasonography provides real-time dynamic images of a moving flexor tendon, unlike static images. Ultrasonography may be a viable diagnostic tool in preoperative identification of the proximal tendon stump. High-resolution 3-T MRI can diagnose complete or partial tendon tears, helps determine the location of the tears and the degree of tendon retraction, and excludes any associated fractures. On T2-weighted fat-suppressed and proton density–weighted fat-suppressed sequences, flexor tendon tears are seen as fluid signal at the site of the tear. Electromyography and nerve conduction study are required in cases of paresthesia or numbness of the ring and small fingers to confirm the ulnar nerve neuropathy.

Delayed diagnosis of this injury may lead to complications, including flexor tendon rupture of the little or ring fingers and sensory or motor deficits of the ulnar nerve. Thus, prompt surgical treatment is recommended to hasten the return to sport and to prevent further complications. Successful cast immobilization within 7 days of injury is required. The average period of immobilization is 11 weeks.[39] The hook of hamate has a tenuous blood supply. Pull of the pisohamate ligament along with the origins of the flexor digiti minimi brevis and opponens digiti minimi displaces the fracture fragment, causing nonunion. Failure rates after conservative management ranged from 80% to 100% in previous studies because of nonunion and associated complications.[40–42] Excision remains the operation of choice for most surgeons.[43] In high-level amateur athletes especially, excision of hook of hamate fractures is an effective procedure.

Incision for decompression of the Guyon canal provides a good approach for excision of ununited hook of hamate fractures.[44] Some of the complications reported, such as injury to the motor branch of the ulnar nerve, superficial palmar arch injury, or median nerve paresthesia secondary to retraction, are complications of surgical exposure rather than of the excision itself. The open carpal tunnel approach is another option. Its familiarity, ease of performance, excellent visualization, and low morbidity make it a successful technique for open excision of symptomatic ununited hook of hamate fractures. A slightly curved linear incision is taken just ulnar to the midline in line with the ring finger and over the carpal tunnel with a slight proximal extension above the transverse wrist crease. After the release of the transverse carpal ligament in carpal tunnel syndrome, the ulnar wall of the carpal tunnel is exposed to be palpated in the nonunion area. Dissection starting from the radial aspect of the ulnar wall of the carpal tunnel proceeds in careful subperiosteal excision. A rectangular floor-based periosteal flap can be used to cover the raw bony surface of the hamate. Almost

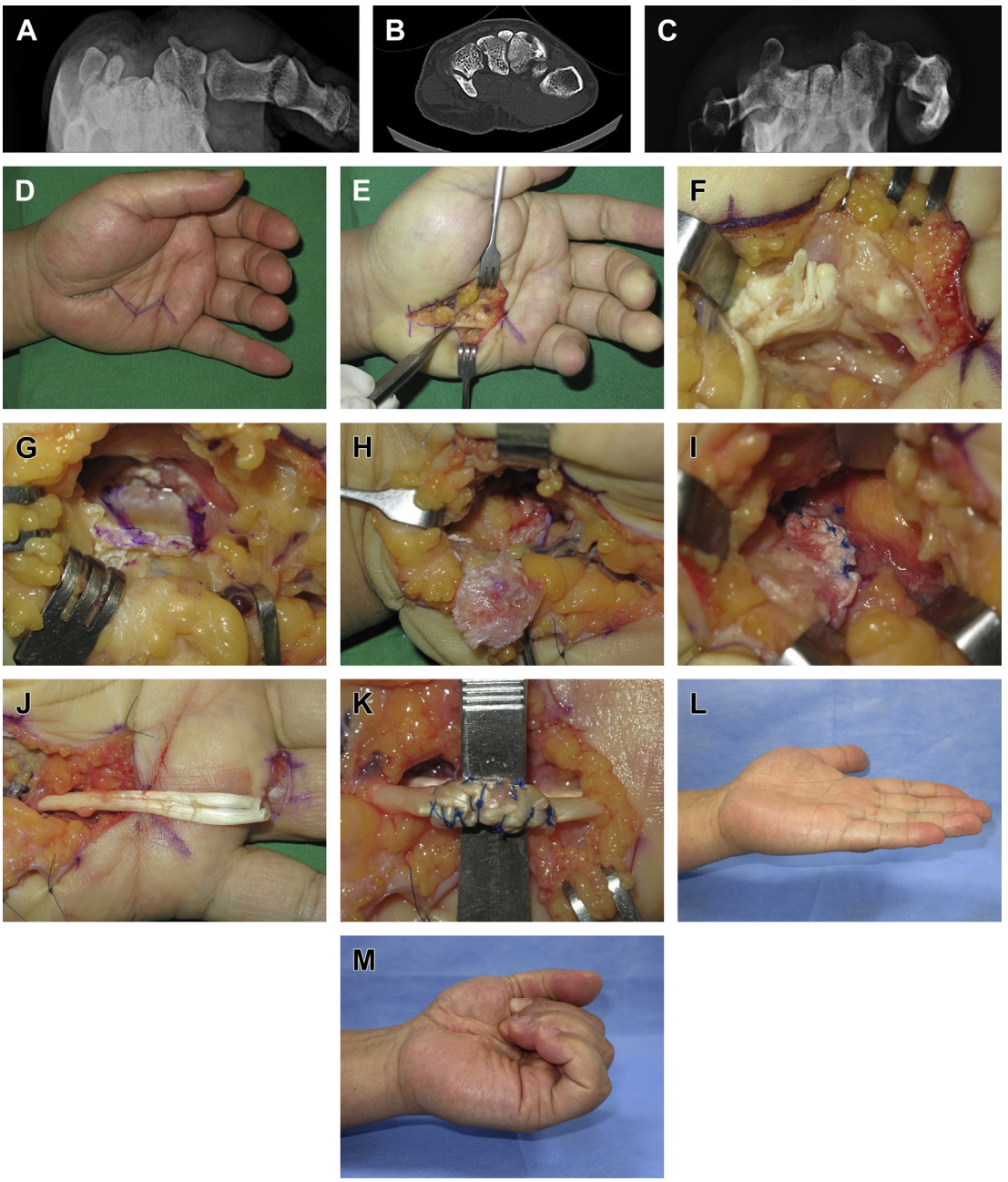

Fig. 9. Beginner golfer: a 56-year-old male patient had chronic pain on the left palm and limitation of flexion on the ring and small fingers after intensive golf practice for 3 months. (*A*, *B*) On the radiograph of the carpal tunnel view and computed tomography, the base portion of the hook of hamate was fractured. (*C*) Postoperative carpal tunnel view after resection of the fracture segment. (*D*) The preoperative view showed destroyed cascade of the ring and small finger and preoperative design for resection of the fractured segment of the hook of hamate, and fourth flexor digitorum superficialis (FDS) tendon transfer for the reconstruction of the fifth flexor digitorum profundus (FDP) together. (*E*) Complete ruptured fifth FDP at zone III. (*F*) The fourth FDP was also longitudinally torn. (*G*) Fractured hook of hamate was identified and periosteal flap based from the ulnar wall of carpal tunnel was designed with gentian violet. (*H*, *I*) The fracture segment was excised and then the raw surface was resurfaced with periosteal flap. (*J*, *K*) The fourth FDS was harvested from the small transverse incision on the metacarpophalangeal volar crease. Pulvertaft interweaving suture was performed between the proximal end of the fifth FDP and distal end of the fourth FDS. (*L*, *M*) 36 months postoperation.

all cases show longitudinal tearing of the fourth or fifth flexor tendons. Debridement or sometimes tendon repair is necessary.

In case of associated flexor tendon ruptures, incision should be extended to identify and expose the proximal and distal end of ruptured tendons. The preferred treatment of these ruptures is interposition tendon grafting. A tendon gap at the palm or wrist level can be reconstructed by the transfer of an adjacent intact flexor digitorum superficialis tendon passed deep to the neurovascular bundle or median nerve and sewn to the distal segment of the injured tendon. Flexor digitorum superficialis tendon transfer from the ring to the little finger has proved to be another good option, but has been considered less favorable because it may compromise ring finger function and overall grip strength.[45] Because adjusting tension of the transfer has always been difficult, tendon transfer is best performed using wide-awake surgery. With the patient being awake, the digits and the hand can move actively to determine correct tension of the transfer.[46,47] Intraoperatively, unsedated patients can observe active motion of their digits in a pain-free state. Patients also develop a much stronger desire for rehabilitation.

TRIANGULAR FIBROCARTILAGE COMPLEX SPRAIN/TEARS

TFCC is the term most commonly used to describe the interconnected soft tissues that span and support the distal radioulnar joint (DRUJ) and ulnocarpal articulations. The role of the TFCC is to stabilize the bones in the wrist. It acts as a shock absorber and enables smooth movement. However, cartilages and ligaments of the complex are prone to degeneration and wear-and-tear injuries, which can lead to ulnar-side pain, weakness of grip strength, and instability of the DRUJ. During the golf swing, rotational injuries may occur when hitting the ball out of deep rough. When the club head gets trapped and twisted in long grass, it produces a sudden strain on the structures that stabilize wrist rotation, thus resulting in acute tears of the TFCC. The wrists are locked when a golfer holds a club, but, once the club swings, the wrist movement may cause pain in the TFCC region. A poor golf shot on the little-finger side of the wrist may also sprain or tear the TFCC.

Magnetic resonance arthrography of the wrist is a valuable tool in diagnostic evaluation to detect full-thickness tears of the TFCC. Arthroscopy is sensitive in identifying traumatic TFCC tears or degeneration of the central portion of the disk, chondromalacia, and ulnocarpal ligament injuries. Arthroscopy is more sensitive and more accurate than noninvasive imaging modalities.[48]

Most acute, isolated TFCC tears do not require prompt aggressive treatment. The treatment modality usually depends on the presence of persistent joint pain from mechanical irritation or synovitis caused by the tear or DRUJ instability. Conservative treatment consists of rest, immobilizing the wrist with a splint, ice application, and taking NSAIDs. Most golf-induced wrist injuries are caused by overuse and are successfully treated without surgery, although the golfer may need to give up the game for an extended period of time. If initial treatments fail, cortisone injections may provide relief. If a sudden injury causes a wrist sprain or a tear of the TFCC, the wrist should be immobilized for 3 to 4 weeks. After that, depending on the injury, the golfer can slowly return to play. If a golfer experiences persistent pain and instability, wearing a wrist brace may help. For the treatment of degenerative TFCC lesions, debridement of the joint, reduction of load across the ulnocarpal joint, DRUJ stability, DRUJ articular congruity, and presence of developmental or acquired skeletal deformities should all be considered. In severe cases, surgery is usually necessary to reconstruct the TFCC. Symptomatic and complete tears with grossly unstable or even chronic injury can be repaired by arthroscopic-assisted techniques, including transosseous or direct capsular suture repair.

Following TFCC reconstruction, the wrist and forearm should be immobilized for 4 to 6 weeks. By 6 weeks, the golfer is usually able to initiate up and down palm motions and wrist flexion and extension. The main goals of physical therapy are to achieve full range of motion in the forearm and the wrist, and for the patient to be able to return to playing pain-free golf.

CARPAL TUNNEL SYNDROME

Although playing golf is not a direct cause of carpal tunnel syndrome, the repetitive swing or strong grip can contribute. Playing a few rounds of golf every month is not a major factor in the development of carpal tunnel syndrome, but serious amateurs, beginners, or professional golfers spend countless hours in practice and play. In this case, inflammation and swelling of flexor tendons and tenosynovium causes crowding and increased pressure on the median nerve (**Fig. 10**). The increased pressure on the nerve causes it to malfunction, resulting in the symptoms of carpal tunnel syndrome. Flexor tenosynovitis and carpal tunnel syndrome result from repetitive grasping and wrist motions. Repetitive digital flexion in individuals

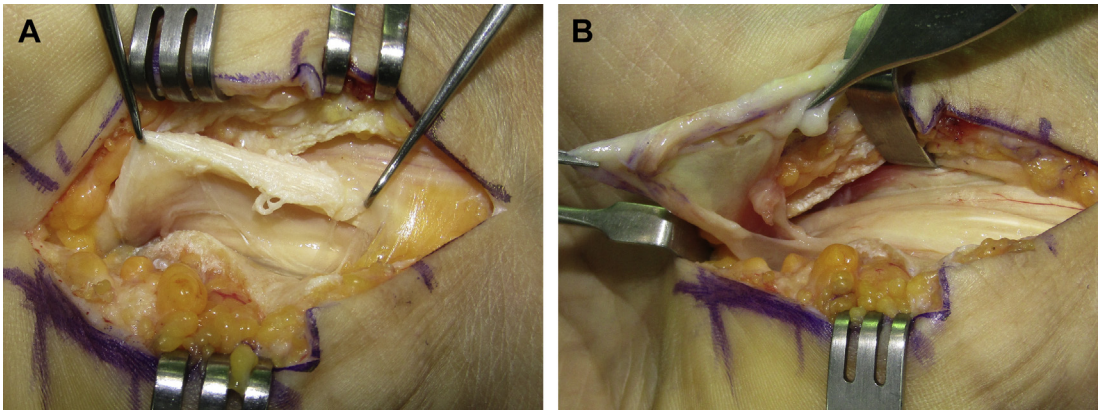

Fig. 10. With a golfing handicap of 10, a 45-year-old male patient developed numbness of the right thumb and index, long, and ring fingers after aggressive practice and 3 to 4 rounds of golf a week. (*A*, *B*) After confirmed diagnosis of carpal tunnel syndrome with the nerve conduction test, open carpal tunnel release was performed with long incision. Severe synovial hypertrophy was found around all flexor tendons and there was longitudinal tearing of the fourth and fifth FDS tendon in the carpal tunnel.

unaccustomed to such activity can induce significant tenosynovitis of the digital flexors.[49]

Rest, wrist splinting at night, NSAIDs, and steroid injections are frequently used as initial treatments for carpal tunnel syndrome. For beginner golfers, easing up on grip pressure during the address phase of the swing and the replacement of worn golf grips may reduce stress on the wrists and hands. Wearing a glove on each hand should also provide extra cushioning. Decreasing the frequency of rounds of golf played and the number of balls hit at the range each week may help injured nerves to recover.

Surgery is the treatment of choice when nonsurgical treatments fail or when abductor pollicis brevis and/or sensory denervation are problematic. Release of the transverse carpal ligament may be performed using an open or endoscopic technique. Open carpal tunnel release is the most common method of decompression and can be performed under local anesthesia or through wide-awake anesthesia. Practicing approach shots with wedge clubs or short iron clubs may resume as little as 4 weeks after operation. At 10 to 12 weeks after surgery, the use of all clubs, including a long iron and driver, is possible without restriction.

SUMMARY

Golf injuries of the hand and wrist are preventable through proper understanding of swing mechanics, avoidance of repetitive and excessive golf practice and play, and ensuring that the hands are appropriately placed on the club to minimize impact injuries. A thorough understanding of the swing phases and mechanisms of injury in golf facilitates accurate diagnosis, treatment, and future prevention of injuries. Initial treatment starts with the cessation of practice to rest the wrist, a splint or orthotic brace, and NSAIDs with corticosteroid injection. Swing modification is recommended for trigger finger, de Quervain disease, and tendinopathy. Pisiform excision is the best treatment of the most severe chronic cases of PLC syndrome, especially if the patient's symptoms are intolerable and nonoperative treatment measures have failed. Delayed diagnosis of hook of hamate fracture may lead to complications, including flexor tendon rupture of the little or ring fingers and sensory or motor deficits of the ulnar nerve. Prompt surgical resection is recommended to hasten return to sport and to prevent further complications.

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