Early soft-tissue complications after fractures of the distal part of the radius

SH Kozin and MB Wood

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Complications from wrist fractures are being recognized and reported with increasing frequency. The prevalence of complications following fractures of the distal parts of the radius and ulna has ranged from 20 to 31 per cent. Complications may result from the injury or as a consequence of treatment. Management and treatment of wrist fractures should maximize the outcome and minimize complications. A multitude of treatment options are available and all involve potentially adverse effects. An awareness of possible complications can lead to early recognition, allowing the surgeon to intervene to avoid or prevent permanent damage.

The soft-tissue elements of the wrist are close to the osseous structures. This proximity increases the possibility of soft-tissue injuries and complications. Despite accurate fracture-healing, damage to tendinous, neural, and vascular structures can lead to permanent disability. Therefore, primary injury or secondary damage to the soft tissues must be diagnosed and managed appropriately to avoid irreparable problems. This lecture focuses on the recognition and management of early soft-tissue complications following fractures of the distal part of the radius. The complications will be discussed separately according to the involved anatomical structure, such as the skin, tendons, nerves, fascia, or vessels (Table I).

**Skin Complications**

Most complications of the skin result from incorrect timing of the application of a cast or incorrect placement of a cast. Swelling is a natural sequela of fractures of the distal parts of the radius and ulna. Because immediate application of a constricting bandage or a circular cast cannot allow for this swelling, skin pressure necrosis and eventually a compartment syndrome may occur. This complication can be avoided through the use of a temporary splint or of sugar-tong immobilization and delayed cast application.

Cast application requires skill and a basic knowledge of cast materials. An incorrectly padded or applied cast can lead to disastrous complications (Fig. 1). Applicable pressure can be generated on the skin beneath a cast or at its sharp edges. Pressure beneath a cast may occur throughout the injured extremity or it may be isolated to a specific location. Pressure points can be avoided by placement of extra padding over the areas where pressure will be applied for the achievement or maintenance of reduction and by molding of the cast materials with the palm of the hand rather than with the fingertips. In addition, preliminary traction makes manipulation easier and decreases the pressure that is required from the cast for maintenance of reduction.

The choice of cast material can also influence the amount of pressure that is generated on the skin beneath the cast. In a recent study, skin surface pressure measurements were recorded in non-injured extremities following cast application. Much higher pressures are generated under fiberglass casts than under similar casts made of plaster. Therefore, plaster of Paris, which generates less pressure and accommodates more swelling, may be a safer material for a cast that is applied after a fracture.

**Tendon Complications**

Numerous complications can involve the flexor and extensor tendons, including the formation of adhesions, stenosing tenosynovitis, entrapment, laceration, and rupture. The most common tendon complication is formation of intertendinous and peritendinous adhesions. Intertendinous adhesions, which occur most commonly between the flexor tendons at the wrist, may be manifested either as a limitation of the independent range of motion of the finger or thumb or as poorly defined pain in the distal aspect of the forearm. Peritendinous adhesions may involve the flexor or the extensor tendon system, or both, and they can result in substantial loss of digital or wrist motion. Application of the cast with a proper technique that allows a full range of motion of the digits, and judicious limb elevation, decrease this complication. Early active finger motion and control of edema encourage resumption of normal hand activity.
and decrease adhesion formation. Once adhesions have developed, supervised physical or occupational therapy and use of a splint may be needed. Rarely, operative tenolysis may be the treatment of choice in resistant cases, but only after failure of non-operative treatment.

Stenosing tenosynovitis can complicate treatment of fractures of the distal part of the radius. Stenosing tenosynovitis occurs most often in the first dorsal compartment.75 DeQuervain tenosynovitis usually becomes apparent following cast removal. Predisposing factors include involvement of the first dorsal tendon compartment by the fracture hematoma, displaced fracture fragments, scar tissue, and residual edema about the abductor pollicis longus and extensor pollicis brevis tendons. Conservative measures, including the use of a splint, oral non-steroidal anti-inflammatory medications, and cortisone injection frequently result in resolution of this complication. Resistant cases, particularly those associated with displaced fracture fragments, necessitate division of the retinaculum overlying the first dorsal compartment.

Tendon entrapment in the fracture site is rare. However, this diagnosis should be considered if the fracture cannot be reduced despite adequate anesthesia and proper technique. The flexor or extensor tendons may be entrapped in the fracture site, preventing accurate reduction (Fig. 2).78,26,57,84. Failure to recognize this complication results in residual fracture malalignment and tendon dysfunction. Following cast removal, tenodesis of the incarcerated tendons is easily detectable on examination. For example, entrapment of the digital flexor tendons causes exaggerated finger flexion during wrist dorsiflexion. In addition, full digital extension is possible only with the wrist in palmar flexion. Early recognition of osseous tendon entrapment combined with open reduction and freeing of the tendon prevents permanent disability. Late diagnosis leads to the need for operative extrication of the entrapped tendons and tenolysis.

Entrapment of the extensor tendons can also occur at the sites of fractures of the distal part of the radius with distal radio-ulnar joint involvement (Galeazzi fracture types). Recent reports of this complication have suggested that it is more common than had been previously recognized.42,45. The affected tendon is usually the extensor carpi ulnaris, but individual cases of entrap-ment of the extensor digiti minimi also have been reported. Radiographs demonstrate a displaced fracture of the distal part of the radius and a widened distal radio-ulnar joint. The ulnar styloid may sustain an avulsion fracture and displace into the distal radio-ulnar joint with the extensor carpi ulnaris tendon. Physical examination of the distal part of the ulna reveals a vacant extensor carpi ulnaris sulcus (empty sulcus sign). The distal radio-ulnar joint is irreducible even after internal fixation of the radial fracture, and dorsal exploration delineates the interposed extensor carpi ulnaris tendon, with or without the avulsed ulnar styloid.

At the time of the injury, the extensor carpi ulnaris may displace in an ulnar direction around the ulnar head or directly radially into the distal radio-ulnar joint (Fig. 3). Both mechanisms block reduction of the ulnar head. Open reduction of the distal radio-ulnar joint, suture repair of the extensor carpi ulnaris fibro-osseous canal, and internal fixation of the ulnar styloid fracture are necessary. Unless the distal radio-ulnar joint is reduced and the extensor carpi ulnaris tendon sheath is repaired, secondary instability and incongruity will result.

Tendon discontinuity can occur following fractures of the distal parts of the radius and ulna. Displaced bone fragments may cause acute flexor or extensor tendon laceration. Careful physical examination identifies the specific functional loss. Exploration and primary repair of acute lacerations is indicated. Attritional ruptures can occur later as gradual tendon-fraying over an exostosis or a malunited fragment causes disruption.2,12,37,47,74. Accurate fracture reduction minimizes the exposed os-
Entrapment of the flexor tendons may occur after fractures of the distal part of the radius. (Reproduced with permission from the Mayo Foundation.)

Entrapment of the extensor carpi ulnaris in the distal radio-ulnar joint. (Reproduced with permission from the Mayo Foundation.)

Nerve Injuries

The median, ulnar, and radial nerves cross the wrist to innervate the hand. Fractures of the distal part of the radius may be complicated by injuries to these structures. Early reports of fractures of the distal part of the
Rupture of the extensor pollicis longus tendon after a fracture of the distal part of the radius. 

**Fig. 4**

Rupture of the extensor pollicis longus tendon after a fracture of the distal part of the radius.

Neurological complications most frequently involve the median nerve. Its close proximity to the distal part of the radius and its confinement within the carpal tunnel predispose the median nerve to injury. Anatomical studies have demonstrated the nerve to be located three millimeters from the distal part of the radius. Following simulated Colles fracture, the nerve was seen to lie within two millimeters of the distal fragment of the radius and to be angulated over the proximal fragment. The reported prevalence of median neuropathy after Colles fractures has ranged from 0.2 to 79 per cent. Abbott and Saunders classified median-nerve injuries accompanying fractures of the distal part of the radius into four groups: (1) primary injuries, apparent immediately at the time of the injury; (2) secondary injuries, following unstable or partial reduction and malunion; (3) late or delayed injuries, occurring months or years after fracture-healing; and (4) injuries following forced manipulation and immobilization in pronounced palmar flexion and ulnar deviation.

Despite the anatomical proximity of the median nerve to the fracture site, primary direct injury to this nerve is uncommon. However, direct contusion, nerve transection, and entrapment have been reported. Fracture fragments can impale the median nerve.
and cause neurapraxia or axonotmesis (Fig. 6). Spicules of bone can sever the nerve and result in a neurotmesis injury, with profound median-nerve deficit. These direct neurological insults are associated with high-velocity injuries and severe fracture displacement. Scrutiny of radiographs made at the time of the injury may aid in the diagnosis. A volarly displaced fracture fragment can cause the injury and should be treated with reduction to alleviate persistent pressure on the nerve. Closed reduction is often inadequate and operative intervention is then necessary to alleviate this acute compression.

Nerve entrapment within the fracture site can also occur, and operative extrication of the nerve must be done to prevent permanent sequelae.

Secondary median neuropathy is more common than the primary injury is, and it is related to carpal tunnel compression. The unyielding carpal tunnel accommodates a fixed volume. An increase in the content causes median-nerve compression and carpal tunnel syndrome. Edema, hematoma, and displaced bone fragments can increase the carpal canal contents following fractures of the distal part of the radius. Measurement of carpal tunnel pressures demonstrates elevated values after fractures of the distal part of the radius. The prevention of persistent median neuropathy depends on prompt treatment. Management is directed at achievement of fracture reduction and alleviation of median-nerve compression. Gentle fracture reduction with the patient under adequate anesthesia minimizes further osseous and soft-tissue damage. Excessive force should be avoided during reduction to prevent iatrogenic injury.

Accurate reduction will eliminate osseous encroachment on the carpal canal and prevent direct osseous compression of the median nerve. Immobilization devices that allow for swelling and for the treatment of excessive swelling both are critical elements of the treatment algorithm. Most median-nerve deficits resolve spontaneously after the initiation of these therapeutic measures. Progressive deficits mandate exploration and carpal tunnel release. Factors contributing to the nerve deficit should be delineated and fracture stabilization should be performed to eliminate the need for a postoperative cast.

A late or tardy median neuropathy may occur months or years after a fracture of the distal part of the radius. The presenting signs and symptoms are usually those of carpal tunnel syndrome. As with acute median neuropathy, contributing factors that compromise carpal canal volume should be identified. Radiographic examination may reveal median-nerve compression because of osseous malalignment or because of excessive volar callus. Persistent volar displacement of fracture fragments or volar callus may necessitate removal of the fragments or callus and ligament division to alleviate the compression. A malunion with severe dorsal angulation may necessitate correctional osteotomy for complete relief of median neuropathy. Non-osseous elements that encourage median neuropathy include residual swelling and paraneural scarring. Non-operative measures may alleviate this non-osseous carpal-tunnel syndrome, but operative decompression is frequently necessary.

The position in which the limb should be immobilized for reduction and treatment of a fracture of the distal part of the radius remains controversial. However, excessive palmar flexion coupled with ulnar deviation (the Cotton-Loder position) should be avoided. Measurements of carpal tunnel pressure are markedly elevated with maximum wrist flexion. This increased pressure further contributes to median-nerve compression. In addition, the proposed advantages of pronounced wrist flexion — that is, assistance in fracture reduction and resistance of dorsal tilting — appear unwarranted. The dorsal radiocarpal ligament complex is relatively lax, except at maximum flexion. The position of flexion necessary to attain adequate dorsal ligament tension would induce pain and interfere with circulation. Therefore, the ligamentotaxis capability of the dorsal ligaments to maintain reduction is limited.

Ulnar neuropathy is less common than is median-nerve injury after fractures of the distal part of the radius. This decreased susceptibility to injury is due to
Onset of Dupuytren disease following a fracture of the distal part of the radius.

the anatomical differences between the nerves. At the level of the wrist, the ulnar nerve is located within three millimeters of the ulna and the median nerve is located within three millimeters of the radius. However, the ulnar nerve courses superficial to the transverse carpal ligament and enters Guyon's canal, which is located distal to the site of the fracture and is more yielding than the carpal tunnel is. This arrangement allows a greater excursion of the ulnar nerve at the fracture site and a decreased prevalence of pressure-induced neuropathy.

Ulnar nerve lesions may present with a sensory, motor, or combined neurological deficit, depending on the site of compression. A careful physical examination helps in the localization and quantification of the nerve injury. Classification of ulnar nerve injuries is similar to that used for median neuropathies. Primary, secondary, or late ulnar-nerve injuries can occur after fractures of the distal part of the radius. Primary trauma to the ulnar nerve is associated with certain fracture characteristics. High-energy injuries with severe angulation or displacement and a coexistent ulnar fracture increase the probability of ulnar nerve injury. In addition, marked disruption of the medial complex of the distal part of the radius increases the potential for direct nerve damage. These injuries are usually neurapraxic in nature and resolve following fracture reduction. A persistent nerve deficit or a progression to a complete nerve block should be treated with open reduction and nerve decompression.

Secondary ulnar-nerve lesions occur after the initial trauma. Compression within Guyon's canal from persistent hematoma, fracture malalignment, and localized swelling leads to neuropathy. Fracture reduction and measures that control edema usually alleviate this compression. Persistent symptoms should be treated with release of Guyon's canal to prevent permanent neurological damage. Improper cast immobilization has been implicated as a cause of secondary ulnar-nerve compression. Removal and reapplication of the cast may improve neurological findings.

Irritation or injury to the sensory branches of the nerves about the wrist may complicate treatment. The sensory branches of the radial, median, ulnar, or musculocutaneous nerves may be involved. The sensory branches of the radial nerve are the most likely to be injured, and damage can cause severe pain and disability. Neuroma formation is especially difficult to treat, and all inciting causes should be avoided. This extremely sensitive nerve may be damaged by compression, irritation, laceration, or pin penetration.

A leading cause of radial sensory neuritis is a compressive or malpositioned cast or fracture brace. The immobilization device should not compress the extremity but should fit securely to prevent motion and friction. Nerve irritation can occur at the radial styloid area, the dorsum of the thumb, or in the index ray. If radial sensory neuritis results, non-operative treatment and desensitization are recommended. Laceration or pin penetration of the radial sensory nerve may occur at the time of operative treatment.

Awareness of the anatomy and potential aberrant locations of the nerve prevents direct nerve injury. The radial sensory nerve emerges from beneath the brachioradialis and courses along the dorsoradial aspect of the forearm. This location is a common site for placement of external fixation pins. Careful open exposure of the radius with preservation and protection of the radial sensory nerve reduces the risk of iatrogenic injury to this nerve. Percutaneous pin fixation can impale the nerve and create a severe injury. A similar risk may exist for...
terminal branches of the musculocutaneous nerve (lateral antebrachial cutaneous nerve).

Fascial Complications

The fascia of the hand and forearm provides support and function to the upper extremity. The fascia divides the hand into various compartments and coalesces into the palmar aponeurosis. The forearm fascia forms boundaries for the flexor and extensor muscle compartments. The fascial structures are composed of dynamic elements, capable of activity and transformation. Complications of fractures of the distal part of the radius that can involve the hand or forearm fascia include Dupuytren disease and compartment syndrome.

Dupuytren disease results from the transformation of fascial tissue into pathological nodules and cords. The nodules are usually the initial presentation of the disease, while the cords cause flexion deformities of the involved digits. The disease has a heterogeneous involvement and a variable natural history. Known risk factors include northern European descent, a positive family history, and epilepsy. The exact role of trauma as a predisposing factor remains controversial. A single episode of injury to the extremity can lead to, or exacerbate pre-existing, Dupuytren disease. Whether the response of abnormal fascial formation is triggered by the initial trauma or by secondary swelling is unclear. The inciting event may be a fracture of the distal part of the radius, a burn, or a penetrating injury (Fig. 7). The development or increased activity of Dupuytren disease has been reported in 0 to 11 per cent of patients who had a fracture of the distal part of the radius.

Compartment syndrome following fracture of the distal part of the radius is rare; the prevalence is less than 1 per cent. Elevated pressure within the closed osseofascial compartments can occur in either the hand or the forearm. Circulation to the enclosed muscles and nerves is impaired and ischemia, with myonecrosis and neuropathy, occurs. Fasciotomy and decompression interrupt the vicious cycle of interstitial edema and persistent ischemia. Immediate clinical recognition of impending compartment syndrome is necessary to prevent irremediable damage. Clinical features include pain that is out of proportion to the injury, paraesthesias, and a tense compartment. Passive stretching of the involved muscles elicits severe pain. Because peripheral circulation and distal pulses are rarely obliterated, they are unreliable indicators of compartment syndrome. Measurements of intracompartmental pressures are a useful adjunct to diagnosis in equivocal cases.

Several factors contribute to the development of compartment syndrome following fractures of the distal part of the radius. Most reported compartment syndromes have occurred after high-velocity accidents with associated severe bone and soft-tissue injury. Direct vascular trauma or compromise is usually not the inciting event. Hematoma, edema, and fracture displacement are potential etiological factors. Techniques of immobilization can also contribute to increased intracompartmental pressures. Use of a compressive dressing on the extremity has been demonstrated to increase compartment pressures to as much as twenty-five millimeters of mercury. Excessive wrist or elbow flexion diminishes venous return and aggravates compartment syndrome.

Compartment syndromes in the forearm after wrist fractures usually involve the volar compartment. Physi-

**Fig. 8**

A fasciotomy of the volar compartment done to alleviate increased intracompartmental pressures.
early recognition of this complication is based on physical examination. Increased pain, loss of digital motion, and continued swelling suggest impending compartment syndrome (Fig. 9)⁶⁹. If non-operative measures fail, operative release of the involved compartments is necessary⁶⁹. The dorsal and volar interossei and the adductor compartments are released through two longitudinal incisions over the second and fourth metacarpals. The thenar and hypothenar compartments must be released through separate longitudinal incisions.

Following fractures of the distal part of the radius, the interossei are prone to intrinsic edema⁶⁹. Measures for the control of edema are crucial to relieve this trapped fluid and allow finger motion. A gradual reduction in digital range of motion may suggest a progressive subacute compartment syndrome of the interosseous muscles. Intrinsic tightness becomes evident on physical examination as motion of the proximal interphalangeal joint becomes dependent on the position of the metacarpophalangeal joint. More proximal interphalangeal motion is possible with metacarpophalangeal flexion than with metacarpophalangeal extension⁶⁹. Failure to recognize this progressive intrinsic contracture compromises the final outcome.

**Vascular Complications**

Acute vascular complications after fractures of the distal part of the radius are extremely rare⁶⁹. Entrapment of the vascular structures has been reported as a reason why a fracture cannot be reduced⁶. Recognition and removal of the entrapped vessels is necessary to prevent further damage. Disruption of the radial or ulnar arteries can occur in association with high-velocity wrist injuries. A complete assessment of the circulatory status of the limb reveals the diagnosis. Treatment of vascular compromise is often complex and can require consideration of multiple factors, including soft-tissue viability, the patency of the remaining vessels, and associated injuries. After careful consideration of the patient and the extremity, management of these injuries should be individualized.

**Conclusions**

Soft-tissue injuries can complicate fractures of the distal part of the radius. Damage to the skin, fascia, tendons, and neurovascular structures is possible. Knowledge of the potential complications assists in early diagnosis. Prompt recognition as well as immediate intervention minimizes functional loss, diminishes disability, and improves the over-all outcome of the injured extremity.

**References**