

An Unusual case of Scaphoid Fracture with Both Fragments Avascular Necrosis with Delayed Carpal Tunnel Syndrome

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Abstract

A case of carpal tunnel syndrome (CTS) occurring after 22 years of scaphoid fracture is presented. The patient reported two months after the onset of symptoms. The cause of CTS due to fracture of scaphoid was evident after history, physical examination, investigations and excluding other known causes of CTS. Roentogram and MRI findings revealed avascular necrosis of both the proximal and distal poles, which is an unusual finding. The patient experienced improvement in symptoms after conservative treatment and local injection of steroid.

Key words: Scaphoid fracture, Avascular Necrosis, Carpal tunnel syndrome, Rehabilitation

Introduction

Fractures of scaphoid are most common amongst wrist fractures, most prevalent in active energetic adolescents. Scaphoid fractures are produced by wrist hyperextension greater than 90 degree combined with radial deviation. Compression and hyperextension forces have been shown to result in scaphoid waist fractures.¹ These injuries are prone to complications like non-union, malunion and late degenerative changes. The displacement of fracture, avascular necrosis can cause non-union. Symptomatic non-unions have a high probability of degenerative change^{2,3,4} and even asymptomatic non-unions are likely to develop degenerative changes and eventual symptoms.⁵ Avascular necrosis is common in the proximal fragment because of its peculiar vascular supply and distal fragment avascular necrosis is rare and so far not we have not come across any report in the literature.

Carpal tunnel syndrome (CTS) is the commonest entrapment neuropathy. The dorsal subluxation of carpal bones can cause narrowing of the cross-sectional area of the carpal tunnel thus leading to CTS. Median neuropathy can occur immediately at the time of scaphoid

fracture⁶, secondary to fracture reduction technique or late associated with immobilization and as a chronic complication related to malunion of the fracture and compromise of the carpal tunnel.⁷

Few cases of delayed CTS have been reported in literature. In 2003 Goyal et al reported a case of delayed CTS after 22 years due to malunited Colle's fracture.⁸ Only one case of delayed CTS because of old displaced scaphoid fracture has been reported in literature.¹² Here a case is reported which developed CTS after 22 years of scaphoid fracture. Roentograms and CT showed both proximal and distal poles' avascular necrosis which is an unusual finding. As per our knowledge this is the first case to be reported of such type.

Case Report

A 43 years old, non-alcoholic, salesman by occupation presented to our out patients department with the chief complaints of pain, numbness and tingling on the radial side of left hand in the distribution of median nerve for about two months. The symptoms were more at night and in cold weather and exacerbated after driving two-wheeler. After massaging the hand he used to get relief from symptoms. The patient gave a history of trauma 22 years back when the patient fell on both of his hands while playing kabbadi (a type of contact sport). The

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patient had pain in left wrist; he was then treated conservatively elsewhere by immobilization with plaster of Paris cast, duration of which is not known correctly. The pain subsided in few months time. No radiographs were taken at the time of injury. The patient remained asymptomatic till two months back when the symptoms started developing and gradually increased in severity. The patient denied any significant medical or surgical history. Review of systems was negative for diabetes mellitus, gout, any infection or hypothyroidism; there was no other evident cause for CTS like rheumatoid arthritis, Colle's fracture. Also there was no history suggestive of similar symptoms in the past.

Physical examination revealed mild wasting of left thenar muscles and a palpable soft tissue mass with central bony prominence on the radial side of the dorsal aspect of wrist. No abnormal sweat pattern in median nerve distribution was observed. There was terminal restriction of dorsiflexion at wrist joint due to swelling on the dorsal aspect. Tinel's sign and Phalen's test were positive and there was no sensory deficit. Roentograms revealed non-union of scaphoid fracture with sclerosis of both proximal and distal fracture fragments suggesting avascular necrosis of both fragments (Fig-1). Later Axial NCCT was done which revealed fracture of scaphoid bone with



Fig-1: PA radiograph of left hand showing non-union of scaphoid bone fracture with sclerosis of both the fracture fragments suggesting avascular necrosis of both fragments with collapse of proximal pole.

sclerosis of both the fracture fragments with areas of lucencies inside suggesting post traumatic avascular necrosis of scaphoid (Fig-2). MR image findings suggested fracture scaphoid with displacement of fracture

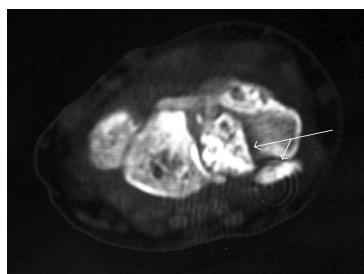


Fig-2: Axial NCCT image of wrist showing fracture of scaphoid bone with sclerosis of both the fracture fragments with areas of lucencies inside suggesting post traumatic avascular necrosis of scaphoid.



Fig-3: Coronal T1 weighted MR image of wrist showing fracture of left scaphoid bone with loss of normal marrow signal intensity in both fragments. In addition foci of low signal intensity areas are also seen in trapezoid and hamate suggesting degenerative changes.

fragment just below the extensor tendons on dorsal aspect of left radius with severe degenerative changes of capitate and hamate bones.

The electrophysiological findings of the patient are shown in Table- 1. With the above findings patient was diagnosed as left delayed CTS due to old scaphoid fracture. The patient refused any surgical intervention; he was



Fig-4: Coronal T1 weighted MR image of wrist showing foci of low signal intensities in capitate bone suggesting degenerative changes.

prescribed NSAIDS (Valdecoxib) and carpal tunnel splint. He was advised to avoid excessive movements at wrist joint and for proper posture of limb like to keep arm elevated during sleep to reduce edema around carpal



Fig-5: Axial T2 weighted MR image of wrist showing displaced fracture fragment of scaphoid on the dorsal aspect beneath the extensor tendons.

tunnel. A local injection of triamcinolone in left carpal tunnel was given. He reported 75% reduction of symptoms after two weeks. The patient did not show any exacerbation of symptoms in the ensuing three months.

Discussion

The exact mechanism by which this patient developed CTS is not clear. We have ruled out all other possible causes for CTS. The likely mechanism for development of CTS may be due to a degenerative change after scaphoid fracture. Generally any physical insult like trauma or fracture can lead to early degenerative changes, which are associated with fibrosis and synovitis⁸, which may lead to compression, ischemia of nerve and pain. Coonay et al found that volar fracture fragments, excessive callus formation and localized swelling were responsible for most cases with CTS in Colle’s fracture⁹.

A few cases with the development of CTS after scaphoid fracture has been reported in literature. Olerud et al reported a case of acute CTS, which developed within three hours of fracture scaphoid and fifth metacarpal bone due to compression by a haematoma, which responded dramatically after surgical decompression of the carpal tunnel.⁶ Lee DJ et al reported a case of delayed CTS due to old displaced fracture of scaphoid, the patient got relief after the excision of the displaced fragment.⁷ In 1992 Monsivais et al, reported a case of persistent CTS which did not respond to standard surgical release, later it was found that the median nerve was compressed against the flexed distal pole of scaphoid. The patient was treated with internal fixation of scaphoid.¹⁰

Neurophysiological studies are still one of the most valuable techniques to determine the extent of nerve compression or injury. However, in cases where the nerve conduction studies or symptoms are ambiguous¹¹, ultrasound, CT scan and MRI may help to establish the diagnosis by demonstrating compression of the median nerve. Our patient’s MRI, CT and roentograms revealed non-union with avascular necrosis and displacement of both the fracture fragments along with arthritic changes. The avascular necrosis of the distal fragment was not found reported in literature. The significant number of scaphoid fracture complications development can be attributed to its peculiar vascular anatomy, especially of the proximal pole, which is vulnerable to post traumatic ischemia and avascular necrosis because of its precarious blood supply. Gelberman and Menon¹² found that the scaphoid bone receives its vascularization mainly from the radial artery with the dorsal and volar branches entering through the distal portion of the bone. The vasculature enters the dorsal area of the bone through numerous small foramina along the spiral groove and

dorsal ridge. These feeding vessels arise from the dorsal scaphoid branch of the radial artery and the dorsal radial carpal arch. These sources account for approximately 80% of the total blood supply to the scaphoid. The distal area of the bone is supplied with 20% of the total blood supply to the scaphoid by palmar vessels that enter through the tubercle and distal pole. In this patient at the time of trauma there was possibly complete disruption of vascular supply causing avascular necrosis of both proximal and distal poles of scaphoid. Green et al reported that total avascular necrosis was present in only 5-10% of scaphoid fractures and almost certainly in proximal pole fractures.¹³ The development of CTS in this can be due to the avascular necrosis of the both fragments, leading to severe arthritis causing swelling and irritation of the median nerve. The swelling on the dorsal aspect of the wrist was due to displaced bony fragment and secondary osteoarthritis of carpal bones. The swelling partially reduced after the local steroid injection and the symptoms also reduced markedly. The other possible mechanism for development of CTS can be due to the distal fragment avascular necrosis causing compression on the nerve or the carpal tunnel either directly or through the secondary osteoarthritis changes of other carpal bones. We were not able to establish the compression of the tunnel or the nerve through the MRI findings.

Table 1: Electrophysiological parameters of the patient

<i>Motor Nerve</i>	<i>Distal latency (msec)</i>	<i>CMAP amplitude (µV)</i>	<i>Velocity (m/sec)</i>
Median Right	3.30	11.8	88.5
Median Left	5.20	6.7	47.3
Ulnar Right	3.00	2.7	47.4
Ulnar Left	2.95	1.1	46.6
<i>Sensory Nerve</i>	<i>Distal latency (msec)</i>	<i>SNAP amplitude (µV)</i>	<i>Velocity (m/sec)</i>
Median Right	4.50	27.9	41.7
Median Left	6.15	27.9	27.8
Ulnar Right	1.50	34.0	62.0
Ulnar Left	4.45	28.7	33.3

Normal values of our electrophysiology laboratory
 Median motor distal latency <3.2msec, median sensory distal latency <3.2msec,
 CMAP= compound motor action potential, SNAP= sensory nerve action potential

To conclude a detailed history especially of trauma and radiographic findings help to know the definite etiology and thus accurate management of CTS. Delayed CTS with avascular necrosis of both scaphoid fracture fragments is not documented in the literature. Old fracture

of scaphoid with avascular necrosis of both fragments may be a cause for CTS and should be considered in cases presenting as late CTS.

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