**Piso-Hamate hiatus syndrome**

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A 49-year-old woman presented with progressive weakness of the right hand of two months duration. Her clinical history was unremarkable except for a carpal tunnel release at the right wrist. Clinical examination showed wasting of the right hand with disability of spreading the fingers. Sensory conduction study of the right ulnar nerve was normal. Motor needle conduction study showed loss of contraction of the ulnar innervated hand muscles indicating neuropathy of the right ulnar nerve.

A comparative ultrasound study of both wrists (Fig. A) showed a cyst of 7 mm medial to the right hamate bone. The deep motor branch of the ulnar nerve (caliper) was running between the cyst and the hamate bone. The nerve was slightly thickened and hypoechoic compared to the left wrist (arrow). Further examination at the level of the metacarpal bones (Fig. B) showed loss of bulging of the dorsal contour of the interosseous muscles at the right side compared to the left side (arrows), reflecting volume loss due to muscle atrophy. Fat-suppressed T2-weighted MR image of the wrist at the level of the hamate bone (Fig. C) confirmed the presence of the cyst causing compression of the deep motor branch of the right ulnar nerve which appeared slightly thickened and hyperintense (arrow). Asymmetry of the dorsal contour of the interosseous muscles was best demonstrated on T1-weighted MR images the level of the metacarpal row (Fig. D, arrows).

Based on imaging findings the diagnosis Piso-Hamate Hiatus Syndrome was made. Patient underwent surgery with ulnar nerve release and resection of the synovial cyst originating from the intercarpal joint. Nerve conduction study performed two months after surgery showed residual denervation activity of the first dorsal interosseous muscle. Patient made a full recovery three and a half months after surgery without neurological symptoms of the right hand.

Comment

The ulnar nerve enters the volar compartment of the forearm after exiting the cubital fossa. It passes anteriorly to the flexor digitorum profundus under the flexor carpi ulnaris where it is accompanied by the ulnar artery and veins. At the ulnar side of the carpus, the nerve enters Guyon's canal and splits distally into a superficial sensory branch and a deep motor branch supplying the intrinsic hand muscles. The proximal medial side of Guyon's canal is formed by the pisiform bone and the distal lateral side by the hook of the hamate bone. The floor is formed by the transverse carpal ligament and the hamate and triquetrum bones, whereas the pisohamate ligament represents the roof. The latter ligament spans between the hook of the hamate and the pisiform bone.

Furthermore, the flexor brevis digiti quinti muscle has two separate attachments at the hook of hamate and pisiform bone respectively. These attachments are bridged by a firm concave musculotendinous arch. Between this arch and the opposite pisohamate ligament, a narrow oblique opening exists, the pisohamate hiatus. The deep motor branch of the ulnar nerve and the accompanying artery passes through this opening and leaves the canal of Guyon to enter the deep palmar space.

Guyon's canal is a potential site for neuropathy by a variety of causes including accessory muscles, cysts, occupational neuritis, soft tissue tumors, hematoma and callus surrounding a fracture of the hook of the hamate bone and arteriovenous malformation. Clinical symptoms depend on the site of the underlying lesion and have been divided into three types. Type 1 syndrome is caused by a lesion proximal to the Guyon canal and results in weakness of all the ulnar innervated muscles in the hand as well as a sensory deficit to the palmar surfaces of the hypothenar eminence and of the little finger and the ulnar side of the ring finger. Type 2 syndrome is caused by a lesion of the deep motor branch of the ulnar nerve at any site distal to the bifurcation. The sensation to the hand is spared but there is motor weakness of the muscles which are innervated by the deep motor branch of the ulnar nerve. Type 3 syndrome is caused by a lesion in or distal to the Guyon's canal involving only the sensory branch of the ulnar nerve resulting in sensory deficit of the volar surface of the hypothenar eminence and the ring finger. There is no associated muscle weakness.

Ultrasound is a reliable technique for assessment of the site of ulnar nerve compression and evaluation of the underlying etiology as well as the consequences of muscle denervation. Ultrasound is able to evaluate the size and the echotexture of the affected muscles. However, compared to MRI, ultrasound is less capable to distinguish early muscle denervation, in which changes are mainly related to extracellular edema, from late muscle denervation in which muscle atrophy and fat replacement occur gradually. MRI is also superior to ultrasound in quantification the extent of muscle atrophy, denervation edema and fatty infiltration. Treatment of the pisohamate hiatus syndrome depends on the cause. If the syndrome is caused by chronic mechanical trauma, the patient can be treated by conservative means such as immobilization or local injection of corticosteroids. When conservative treatment fails or in case of an underlying mass, surgical decompression may be mandatory. Recovery may take several months to years and is prolonged in older patients. With severe compression recovery may be incomplete.