Acute carpal tunnel syndrome secondary to thrombosis of a persistent median artery

Case report

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A rare case of thrombosis of a persistent median artery as a cause of acute carpal tunnel syndrome is reported. The sudden onset of pain, local tenderness of the palm, and decreased sensation in the median nerve distribution were the symptoms. The operative findings and subsequent progress are described.

KEY WORDS - acute carpal tunnel syndrome - median artery thrombosis

Carpal tunnel syndrome is produced by compression of the median nerve within the carpal tunnel. The carpal tunnel, bounded by the transverse carpal ligament anteriorly and an arch formed by the carpel bones, conducts the median nerve and the long flexor tendons from the anterior compartment of the forearm into the central compartment of the palm. The median nerve passes directly beneath the volar transverse carpal ligament and lies superficial to the nine flexor tendons of the digits. Any condition that crowds the structures within the carpal tunnel may result in median nerve compression. Symptoms may be pain, paresthesia or sensory loss in the median nerve distribution, and progressive atrophy of the thenar muscles.

Marie and Foix, in 1913, observed bilateral thenar atrophy and a neuroma of the median nerve proximal to the transverse carpal ligament in a patient at autopsy. They were the first to recommend decompression of the median nerve by sectioning the transverse carpal ligament. In 1946 Cannon and Love reported 38 cases of tardy median palsy in which nine were treated by sectioning the transverse carpal ligament. Brain, et al., in 1947 described six cases treated surgically that presented as spontaneous compression of both nerves in the carpal tunnel, and established this as a clinical syndrome.

In spite of the many cases of carpal tunnel syndrome reported, the development of acute carpal tunnel syndrome is rare. Phalen, in a series of 654 patients with symptoms of median nerve com-
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sion in the hand, did not encounter an acute carpal tunnel syndrome.

The following is a case of acute carpal tunnel syndrome caused by thrombosis of a persistent median artery.

Case Report

This 43-year-old neuropathologist (second author) was admitted to the University of Kansas Medical Center in February, 1972, with a history of severe pain and decreased sensation in the median nerve distribution of the left hand. Two weeks prior to admission he was awakened in the middle of the night by an aching pain in the palm of the left hand along the base of the thenar eminence. This pain persisted, was intermittent, and became worse at night, particularly 2 hours after he had gone to sleep. He could partially relieve the pain by hanging his hand down and massaging the base of the thumb. He noticed at these times a tenderness over the flexion crease of the wrist. The patient had suffered a small but deeply incised wound over the distal interphalangeal joint of the left thumb 3 weeks prior to the onset of pain. The wound of the thumb had completely healed but the scar was still somewhat tender.

Examination. The left hand showed slight atrophy of the thenar eminence. The soft tissues of the palm were not swollen, and the skin was not reddened. There was a healed laceration over the distal interphalangeal joint of the thumb. Neurological examination showed hypesthesia in the median nerve distribution. There was no weakness of the hand. Focal tenderness was present at the midpoint of the crease at the base of the thenar eminence. Nerve conduction studies of the right and left median nerves were all normal. X-ray films of the left wrist and hand were normal.

Operation. An exploration of the left carpal tunnel was carried out on February 21, 1972, under local anesthesia. The routine hand incision that closely paralleled the crease at the base of the thenar eminence did not give adequate proximal nerve exposure, and the incision was therefore extended in a standard zigzag fashion designed to cross the flexor wrist crease. The thrombosed section of an artery 4 mm in diameter accompanying the median nerve through the carpal tunnel lay on the volar aspect of the nerve, integrally related to the perineurium. Distally the artery joined the mid-aspect of the superficial arterial arch. The thrombosis stopped at a patent arterial arch. The thrombosed section of the artery 5.2 cm long was excised (Fig. 1).

Fig. 1. Operative photographs. Left: The thrombosed section of the left median artery is exposed. Right: The median artery is being removed from its attachment to the median nerve.
Pathological Examination. The excised artery appeared completely obliterated by a red-gray thrombus. Microscopically the thrombus showed early organization with a few fibroblasts growing into the clot from the intima (Fig. 2 left). No inflammation was seen in the intima and the inner half of the media, but the outer layers of the media and the adventitia showed rather marked subacute inflammatory changes consisting of plasma cells, lymphocytes, a few neutrophil granulocytes, and occasional eosinophils. There was also a proliferation of newly formed capillaries in the adventitia (Fig. 2 right). Necrosis and granulomatous changes were not present. Bacterial and fungal stains were negative. The changes were consistent with subacute nonspecific periarterial inflammation with secondary thrombosis of the median artery.

Postoperative Course. Recovery following surgery was uneventful, and the patient was discharged to be followed as an outpatient. In view of the inflammatory changes in the artery, polycillin treatment was given for 1 week. He was relieved of discomfort, and normal sensation returned. In the 6 months following surgery there has been no recurrence of symptoms.

Discussion

Acute carpal tunnel syndrome is rare and for it to be caused by a thrombosed persistent median artery is even more unusual. One case of median artery thrombosis as a cause of acute carpal tunnel syndrome has been reported by Burnham, and Bralliar has related his experience with this entity. The persistence of the median artery is in itself uncommon. McCormack, et al., in their detailed study of anatomical variations of arteries in the forearm and hand found it to be present in 7.5%. They found the artery usually reinforcing the superficial volar arterial arch. In our case the median artery connected to the midpoint of the volar arterial arch. Obviously the mere presence of an anatomically variant artery does not by itself necessarily predispose thrombosis. In Burnham's case the possible etiology of the thrombotic process is not discussed.

In our case there is a strong possibility that the deep cut of the thumb (which was inflicted with a sharp blade while peeling a pineapple) carried infection with a low virulence organism into the deep fascial planes. Since the so-called radial bursa connects the deep tissue spaces of the thumb

![Fig. 2. Left: Cross section of the artery showing complete occlusion by mostly laminated thrombus. Black spots are scattered red blood cells. H & E, X 20. Right: Higher power view of artery showing thrombus with red blood cells trapped between fibrin strands (left), smooth muscle of the media with scattered red blood cells (center), and adventitia with lymphocytic and plasmacellular infiltration and newly formed capillaries (right). H & E, X 120.](image-url)
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with those of the palm (Lampe\textsuperscript{9}), a ready-made route existed for the spread of infection which appeared to be of sufficiently low virulence to preclude external signs of inflammation (swelling and redness that were present in Burnham's case). The microscopic alterations of the adventitia, however, showed unequivocal proof of inflammation in the arterial wall and its immediate surroundings.

Arterial insufficiency of the hand was not a problem in this case, probably because the thrombosis was confined to the median artery and left the volar arterial arch patent.

References

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