Short report

Carpal tunnel syndrome: an unusual presentation of brachial hypertrophy

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SUMMARY A patient with carpal tunnel syndrome in association with congenital hypertrophy of right upper limb is described. The median nerve also showed hypertrophy. The symptoms were relieved by decompression of the carpal tunnel.

Carpal tunnel syndrome has a wide variety of causes.¹⁻⁶ Brachial hypertrophy is a rare cause.

Case report

An 18 year old right-handed girl complained of numbness of the right thumb and index finger for six months. This used to occur after exerting the hand as in writing or lifting weights. She had noticed a gradual increase in the size of the right upper limb since childhood. No pain in the arm was noted at any time. Examination revealed a normosthenic young female. Her blood pressure was 120/80 mmHg. No angiomas or neurofibromata were detected. There was hypertrophy of the right upper extremity with no dilated veins. The upper arm and forearm girth was greater on the right than on the left, by 35 mm or 40 mm respectively. Wasting of the thenar eminence and inbending of index finger were noted (fig 1). Cutaneous sensory impairment of median nerve distribution in the hand and a positive Tinel's sign were also noted. No bruit could be heard over the neck or arm.

Routine investigation of haemogram, urinalysis, and biochemistry were normal as was a radiograph of the chest. Radiography of the right arm showed hypertrophy of the soft tissue but no bone abnormality could be seen. Brachial angiography did not show any abnormality in the

Fig 1 Right hand shows hypertrophied thumb, index and middle fingers. Wasting of the thenar eminence and inbending of the index fingers are also evident.

vascular pattern. Nerve conduction studies showed slight reduction of motor nerve conduction velocity in the forearm (table 1). Denervation potentials were recorded from the right abductor pollicis brevis on electromyography.

Table Right median nerve conduction studies

Median nerve	Before surgery	One month after Surgery
Distal latency	10.5 ms (Normal 2.63 ± 0.52)	5.8 ms
Conduction velocity	39.5 m/s (Normal 59.9 ± 5.5)	45 m/s
Sensory action potential	Not recordable	25 μ V
	(Normal $39.58 \pm 9.67 \mu$ V 2.01 ± 0.25 ms)	2.4 ms

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Surgical exploration of the carpal tunnel was undertaken. The flexor retinaculum was tight and compressing the structures in the tunnel. The median nerve was thick proximally, looked greyish, and was tightly compressed under the retinaculum. The tunnel was decompressed adequately. A single fascicular biopsy of median nerve showed perineurial collagenous proliferation, endoneurial oedema, fragmentation, and segmental demyelination of the nerve fibres (fig 2).

Fig 2 Photomicrograph showing endoneurial oedema. Haematoxylin and eosin, original magnification $\times 160$.

One month after operation there was no numbness, and no sensory deficit in the median nerve distribution could be detected. Nerve conduction studies were repeated (table 1).

Discussion

Carpal tunnel syndrome is an entrapment neuropathy of median nerve occurring at the carpal tunnel formed by the flexor retinaculum at the wrist. The median nerve is involved in a variety of conditions such as myxoedema, acromegaly, myeloma, amyloidosis, rheumatoid arthritis, and pregnancy. Rare causes are pseudogout, anomalous first lumbrical muscle, macrodystrophic lipomata, and arteriovenous fistula.^{1-3 5 6}

Our patient had right upper extremity hypertrophy and the right median nerve was thick. The occurrence of symptoms attributed to median nerve dysfunction could be related to increase in the tissue mass in the carpal tunnel and resultant pressure on the nerve and ischaemia. Ranawat *et al*¹ reported a case of macrodystrophic lipomata with carpal tunnel syndrome. The median nerve involvement was secondary to lipomatous hypertrophy of subcutaneous tissue

causing pressure on the median nerve with resultant ischaemia. The same hypothesis could be extended to our case to explain the median nerve dysfunction. The genesis of symptoms in the carpal tunnel syndrome includes possibilities of ischaemia⁷ and direct mechanical effect on nerve fibres.^{8 9} It seems that there is a dual mechanism for nerve damage.¹⁰ There is no good evidence for a direct effect on myelin¹¹ with demyelinating conduction block and conduction delay. This process appears to be relatively independent of ischaemia since fibres damaged in this way are not abnormally susceptible to ischaemia.¹⁰ The recovery is slow when the nerve is decompressed. Our patient showed features of demyelination and evidence of ischaemia of the nerve. Pain and paraesthesia are often relieved within 24 hours of carpal tunnel decompression, and this is paralleled by improvement in sensory conduction, evident within 30 minutes. Relief of ischaemia is responsible for rapid recovery.

Ringrose *et al*¹² in their review of hemihypertrophy make the comment that in normal individuals, of whom 90% are right-handed, the left cerebral hemisphere, the left half of skull, and left side of the face are larger than on the right. The right upper extremity is longer and slightly thicker than the left and, in fact, it may be as much as 10–20 mm longer than the left. Obvious asymmetry is rare. In none of their patients was there evidence of median nerve involvement in the carpal tunnel. Our case shows the unique feature of hypertrophy of the median nerve and dysfunction in relation to brachial hypertrophy.

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