Compression of median nerve at elbow

Compression of the median nerve at the wrist causing carpal tunnel syndrome is an entrapment neuropathy known to every final year medical student. What is not so well known, however, is that compression of the median nerve may occur in the region of the elbow and simulate the clinical features of carpal tunnel syndrome.

Case report

A 46-year-old man was admitted to St Helier Hospital, Carshalton, for treatment of primary hypercholesterolaemia. He was observed to have considerable wasting of the muscles of the left thenar eminence and probable wasting of the left forearm. On direct questioning he admitted that he had had some weakness of the left hand for several years but denied sensory symptoms. Neurological examination showed gross weakness of the left thumb with wasting of abductor pollicis brevis, flexor pollicis brevis, and possibly flexor carpi radialis. Perception of sharp and blunt in the pulp of the left index finger was diminished, but sensation in the hand was otherwise normal. There were no localised areas of tenderness in the arm or hand. Investigations included normal radiographs of the chest, elbows, and cervical spine. No supracondylar spurs were present. Electromyography suggested compression of the median nerve at the level of the elbow and it was decided to explore this region.

With a tourniquet on the upper arm a 10-cm Z-shaped incision was made over the anterior aspect of the elbow and the bicipital aponeurosis divided. The median nerve was identified and traced along its course. A shiny white ligamentous band 3 mm in diameter was found lying parallel with, but separate from, the upper border of pronator teres and passing from the medial epicondyle of the humerus to just below the tuberosity of the radius. This band passed across both the median nerve and the brachial artery and tightly constricted the nerve (see figure). The band was divided and excised to release the nerve. Histological examination of the band showed normal ligamentous tissue.

Diagram showing ligament passing across elbow joint from medial epicondyle to radius and constricting median nerve.

Discussion

The pronator syndrome is caused by the entrapment of the main trunk of the median nerve in the region of the two heads of pronator teres before it dips beneath the tendinous bridge of flexor digitorum sublimis.¹ It may easily be misdiagnosed as carpal tunnel syndrome or thought to be caused by cervical spondylitis; occasionally it has been mistaken for a toxic neuritis. The sensory impairment may be identical to that in carpal tunnel syndrome, but weakness of the long and short flexor muscles of the fingers often predominates as the median nerve supplies the lateral part of flexor digitorum profundus and the whole of flexor digitorum sublimis. Tenderness over the pronator teres at the site of entrapment is sometimes a diagnostic pointer. Classically, the compression is caused by hypertrophy of the pronator teres muscle itself or by a tendinous band within the ulnar head of pronator teres. Rarely, the tendinous bridge of flexor digitorum sublimis may be the cause. There may, however, be considerable variation in the amount of fibrous tissue associated with the two heads of both flexor digitorum

sublimis and pronator teres, as well as variation in the origin of pronator teres.² Although we can find no published record of the ligament we describe in this paper, it seems unlikely that it is a unique case, and we believe the diagnosis in our patient was that of pronator syndrome.

The main trunk of the median nerve may also be compressed by the supracondylar ligament of Struthers, which lies above the hinge of the elbow joint and passes from a spur on the anteromedial aspect of the humeral shaft to the medial epicondyle.³ Rarely, the anterior interosseous branch of the median nerve may be separately compressed by a fibrous band of variable origin and insertion associated with either flexor digitorum sublimis or pronator teres, causing weakness of the thumb and index finger.⁴

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Severe hypertension produced by interaction of phenylpropanolamine with methyldopa and oxprenolol

The sympathomimetic amine phenylpropanolamine, which is present in many proprietary "cold cures," antagonises the hypotensive effect of bethanidine and guanethidine and causes hypertension in patients on monoamine oxidase inhibitors.¹ I would like to report a severe hypertensive reaction to this drug in a patient with renal hypertension treated with methyldopa and oxprenolol.

Case report

The patient was a 31-year-old mining engineer. After an attack of acute glomerulonephritis in 1958, when he was 14, his urine was noted to contain red cells, albumin, and casts on several occasions. He remained well, however, until May 1975, when he went to his general practitioner complaining of headaches and was found to have a blood pressure of 200/120 mm Hg and a blood urea level of 22 mmol/l (133 mg/100 ml.) His hypertension was easily controlled on methyldopa 250 mg twice daily and oxprenolol 160 mg three times a day with repeated clinic readings of 140/80 mm Hg, but his renal function continued to deteriorate and by September 1975 his blood urea level was 30.4 mmol/l (183 mg/100 ml). On 15 December 1975 he visited his general practitioner, who recorded his blood pressure as 120/70 mm Hg and prescribed Triogesic tablets (phenylpropanolamine 12.5 mg and paracetamol 500 mg) in a dose of two tablets three times a day as symptomatic treatment for a cold. When seen at the clinic two days later the patient complained of decreasing energy, occasional headaches, and increasing nausea over the preceding few weeks and paraesthesiae in his hands and feet since the onset of his cold. In addition to the Triogesic he had continued on his normal antihypertensive treatment. At this time his blood pressure was 200/150 mm Hg and pulse rate 90 beats/min. There were no objective signs of peripheral neuropathy, no hypertensive retinopathy, and no signs of heart failure. His blood urea level had risen to 42 mmol/l (253 mg/100 ml). The Triogesic was stopped but he continued on his usual doses of methyldopa and oxprenolol, and by the next day his blood pressure was 140/110 mm Hg and his paraesthesiae had disappeared. He continued to vomit, however, and was therefore started on peritoneal dialysis the next

day. Since then his blood pressure remained steady at 140/90 mm Hg on oxprenolol 80 mg three times a day alone.

Comment

There seems little doubt that this hypertensive episode was caused by phenylpropanolamine. Although the combination of methyl dopa and intravenous propranolol has occasionally produced severe hypertension² there was no evidence of any major fluctuation in the patient's blood pressure until phenylpropanolamine was introduced. Methyl dopa is known to increase the pressor effects of sympathomimetic amines³ and it has been postulated that beta-blockade may have a similar action by allowing an unopposed alpha-constrictor response to adrenergic stimulation.⁴ The combination of methyl dopa and oxprenolol may therefore have caused a particular severe hypertensive reaction. The patient's renal failure might have exacerbated the effect since metabolites of methyl dopa with possible pressor effects are increased in uraemia.⁵

In view of the widespread use of methyl dopa and beta-blockers in combination it is surprising that an interaction with this amine has not been reported before. On this occasion the hypertension did not result in any permanent damage, but the consequences might be serious in patients with ischaemic heart disease.

The manufacturers of Triogesic do give hypertension as a contraindication to its use but no mention is made of beta-blockers that might be given for other reasons. In addition phenylpropanolamine is a constituent of many cold cures obtainable without prescription, and although warnings are printed on the packet these may well be disregarded.

It seems desirable that wider publicity should be given to the potentially serious effects of interaction of phenylpropanolamine with beta-blockers, methyl dopa, and other drugs, which might well outweigh its very limited role as a nasal decongestant.

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Detection of urinary amyloid in familial Mediterranean fever

Derosena *et al*¹ and Neale² reported finding amyloid fibrils by electron microscopy in the urinary sediment of two patients with renal amyloidosis. It was suggested that this technique could be used to diagnose renal amyloidosis when renal biopsy cannot be performed. We have used this technique to diagnose renal amyloidosis in a patient with familial Mediterranean fever, who subsequently showed a good response to colchicine.

Case report

A 72-year-old man was admitted to this hospital with a history of recurrent fever of undetermined origin for 35 years. Urine had a specific gravity of 1.011, contained protein 4+, and was negative for glucose. Microscopical examination of the urinary sediment showed hyaline, granular, waxy, and fatty casts. Doubly refractive fat bodies were visible with polarised light. The urinary protein was 4.2 g/24 hours, and the total serum protein 61 g/l (albumin 25 g/l). Pedal oedema and Muehrcke's lines were present. Packed

cell volume (PCV) was 0.26 (26%), blood urea nitrogen 23.2 mmol/l (65 mg/100 ml), and serum creatinine 398 μ mol/l (4.5 mg/100 ml). An intravenous pyelogram showed poor excretion and decreased size of both kidneys. Rectal biopsy was positive for amyloid, but renal biopsy was contraindicated because of the possibility of bleeding and the decreased renal function and small kidneys.

Urine was processed according to the method of Derosena *et al*,¹ and sections of the sediment were examined by electron microscopy. This showed fibrillar material with the characteristics of amyloid (see fig). The patient's mother was of Sephardic Jewish descent, and the working diagnosis of familial Mediterranean fever was made. The patient was maintained on colchicine 0.6 mg by mouth twice daily and remained asymptomatic and free of oedema for four months. At the time of writing, the urinary protein was 2 g/24 hours, serum albumin 37 g/l, serum creatinine 327 μ mol/l (3.7 mg/100 ml), and PCV 0.31 (31%).

Electronmicrograph of urinary sediment showing fibrillar material having size and periodicity of amyloid fibrils. (Uranyl acetate and lead citrate stain. $\times 32\,500$.)

Comment

Familial Mediterranean fever occurs most commonly in Sephardic Jews, Armenians, and Arabs, but a few cases have been reported in Ashkenazic Jews.³ It is characterised by intermittent acute episodes of fever and polyserositis, usually peritonitis, and its most serious sequela is renal amyloidosis.³ Some patients may have fever as the only manifestation of the disorder³ and are therefore subjected to repeated investigations for fever of unknown origin. This was true in the present case, and it was only after the appearance of the nephrotic syndrome and amyloidosis that the diagnosis was established.

Colchicine prevents acute exacerbations of familial Mediterranean fever, and response to the drug has been recommended as the most reliable diagnostic test for the disorder.⁴ Since renal amyloidosis is notoriously refractory to treatment,⁵ the apparent response in this patient is noteworthy. Renal biopsy may be hazardous in patients with renal amyloidosis because of an increased bleeding tendency. This is particularly true in the presence of renal insufficiency. The detection of urinary amyloid fibrils by electron microscopy is a practical and non-invasive alternative diagnostic method for patients suspected of having renal amyloidosis. Evaluation of its reliability and sensitivity will require further study.

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