CARPAL TUNNEL SYNDROME IN PATIENTS ON HAEMODIALYSIS

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Eight cases of carpal tunnel syndrome are reported, all of which developed in patients on haemodialysis for chronic renal failure. In each case the arm involved had been used for a fistula. The aetiology of the syndrome in these patients is discussed; it is multifactorial, but related to the sites of arteriovenous fistulae. Decompressing the carpal tunnel provides effective and lasting relief.

Carpal tunnel syndrome is known to occur in patients having renal dialysis. The symptoms of nerve compression may be severe enough to warrant decompression in 4% to 5% of cases. The precise cause, however, has not so far been clearly established.

METHODS AND RESULTS

The case records of dialysis patients in the care of the Regional Renal Unit at St Bartholomew's and St Leonard's Hospitals were studied. Six patients who had

developed in an arm which had been used for vascular access. The delay between the formation of the fistula and the development of symptoms varied from 1 to 16 years. Symptoms were bilateral in two patients, giving a total of eight hands affected. The non-dominant hand was involved in three patients.

All the patients had undergone decompression of the carpal tunnel under general anaesthesia. In order to avoid jeopardising the fistula tourniquets had not been used. Symptoms were relieved in all patients and there

Table I. Clinical details of six dialysis patients who were operated on for carpal tunnel syndrome

Case	Sex	Renal disease	Carpal tunnel syndrome (side)	Fistula procedures (side)	Time from fistula to carpal tunnel syndrome (years)
1	Male	Glomerulonephritis	Right and Left	Right Left Left	1 10 10
2	Male	Polycystic kidneys	Left	Left	10
3	Female	Chronic pyelonephritis	Right	Right Left	16
4	Female	Polycystic kidneys	Left	Left	4
5	Male	Glomerulonephritis	Right and Left	Right Left	2 11
6	Female	Glomerulonephritis	Left	Left	3.5

required carpal tunnel decompression were found, and all were reviewed personally. Table I summarises the relevant clinical details. Nerve compression had been confirmed by EMG studies in all cases. This is particularly important for patients with chronic renal failure, in order to differentiate between carpal tunnel syndrome and uraemic peripheral neuropathy.

In this series carpal tunnel syndrome had always

has been no evidence of any recurrence after a follow-up from one year to three years.

DISCUSSION

The first report of dialysis patients developing carpal tunnel syndrome was from Warren and Otieno (1975). They reported that 23 of 36 patients with an arteriovenous fistula in the forearm had experienced symptoms like those of carpal tunnel syndrome in the same hand during dialysis. These symptoms usually resolved at the end of the dialysis, but persistent problems had necessitated carpal tunnel decompression in two patients.

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Since then there have been a number of reports of dialysis patients who have required carpal tunnel decompression, and the aetiology of the syndrome has been debated, in particular its relation to the arteriovenous fistula (Kenzora 1978; Jain, Cestero and Baum 1979; Scardapane *et al.* 1979; Halter *et al.* 1981; Martinelli *et al.* 1981). The development of the syndrome has been related to the time on dialysis, to altered haemodynamics as a result of the fistula, and to the uraemia of renal failure. The aetiology is undoubtedly multifactorial, and this is outlined in Figure 1, which emphasises the central role of the arteriovenous fistula.

the fistula is side-to-side or end-to-side does not seem to be significant (Kenzora 1978).

All the factors discussed above, acting alone or in concert, may give rise to symptoms of carpal tunnel compression. These may rarely result from local deposition of amyloid in the carpal tunnel in renal failure patients (Clanet *et al.* 1981), but no such deposition was found in this series. The cause for the original renal failure does not seem to be related to nerve symptoms and, as Figure 1 shows, an arteriovenous fistula is not an essential precursor of carpal tunnel syndrome, though its presence makes nerve compression much more likely.

Fig. 1

Diagram to show the aetiology of carpal tunnel syndrome in dialysis patients.

Peripheral neuropathy is common in chronic renal failure with its associated uraemia. As a result the peripheral nerves are unduly susceptible to minor trauma and to ischaemia (Kenzora 1978). Uraemia is also associated with an increased extracellular fluid volume which may cause raised pressure in the carpal tunnel. Both these effects of uraemia can be made worse by the arteriovenous fistula used for dialysis. During dialysis both the venous pressure and the volume of the hand are increased distal to the fistula because of the venous engorgement (Warren and Otieno 1975). High flow rates through a fistula may produce a vascular steal syndrome, causing distal ischaemia (Bussell, Abbott and Lim 1971). Ischaemia can also result from repeated procedures to gain vascular access for dialysis, which may include tying distal arterial branches and cause thrombosis of veins. Delmez *et al.* (1982) found a tendency for higher flow rates in the fistulae of the arms which developed carpal tunnel syndrome than in those which did not. Whether

The incidence of carpal tunnel syndrome in patients having dialysis is difficult to assess, and will depend on the criteria for diagnosis, be it by EMG or by clinical assessment. Scardapane *et al.* reported in 1979 that the incidence of EMG evidence of nerve entrapment rises with an increase of time on dialysis. Half of their patients who had been on dialysis for over five years had EMG evidence of carpal tunnel syndrome. Both Kenzora (1978) and Delmez *et al.* (1982) found that 4% to 5% of their patients had a carpal tunnel syndrome severe enough to require decompression. In our series six patients from a dialysis population varying from 120 to 130 needed operation, giving an incidence of about 5%. In these, as in all other reported cases, decompression has given prompt and lasting relief of pain, though some numbness may persist.

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