Carpal-tunnel syndrome in hemodialysis

Syndrome diagnosed in 8 of 60 patients

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Clinical and electrophysiological examination for carpal-tunnel syndrome was performed in 60 consecutive hemodialysis patients. The syndrome was diagnosed in 8 patients, 6 of whom had had hemodialysis for more than 10 years. Cystic radiolucency in the carpal bone, possibly a sign of amyloid deposition, was observed more frequently in carpal-tunnel patients and amyloid-induced tenosynovitis in the carpal-tunnel had formed in 4 cases.

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Development of carpal-tunnel syndrome (CTS) in patients who have been receiving long-term hemodialysis frequently causes problem. Several etiological factors, such as vascular steal syndrome, expanded extracellular fluid, and amyloid deposition in the carpal-tunnel, have been suggested, but the precise cause remains unclear. We report clinical and electrophysiological studies of 60 hemodialysis patients.

Patients and methods

60 consecutive patients (36 men and 24 women) undergoing hemodialysis at our hospital participated in the study. The diagnosis was chronic glomerulonephritis in 57 patients, and lupus nephritis, renal tumor or kidney stones for one patient each. Their age was 55 (22–86) years. 14 patients had been hemodialyzed more than 10 years, 17 patients 5–10, and 29 patients less than 5 years. 7 patients had been diagnosed as CTS and operated on before the start of this study. The patients were asked about numbness, paresthesia or pain in the median nerve area of the hand. The patients were examined for positive Phalen's wrist flexion sign, positive Tinel's sign at the wrist, and thenar muscle atrophy. Semmes-Weinstein's monofilaments test was used for objective evaluation of disturbed sensitivity. Electrophysiological testing was done with an electromyograph (MEB 7102, Nihonkohden, Japan). Distal motor latency, distal sensory latency and motor nerve conduction velocity of the median nerve and ulnar nerve at the forearm were measured with standard techniques (Lenman and Ritchie 1970). Radio-

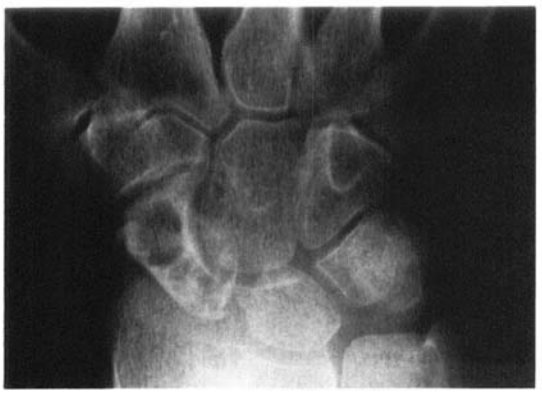

Figure 1. Cystic radiolucency in carpal scaphoid of a patient undergoing hemodialysis.

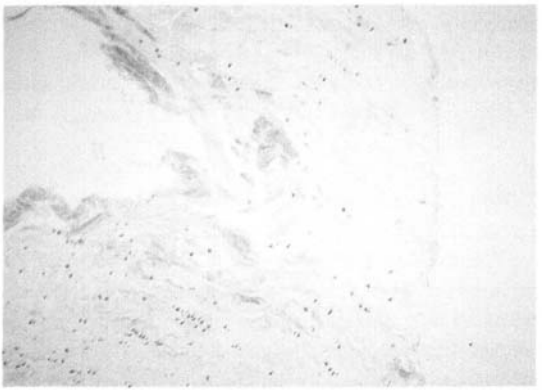

Figure 2. Amyloid deposition in transverse carpal ligament of a carpal-tunnel syndrome patient (Congo-red stain, × 100).

Table 1. 8 cases of CTS undergoing hemodialysis relation with electrodiagnosis of the median nerve

Case	Sex	Age	Blood access side	Affected side	Months on hemodialysis	Side	Preop			Postop			Postop time (months)
							DML	DSL	MCV	DML	DSL	MCV	
1	M	50	l	b	48	r	–	–	–	4.0	3.2	46	9
						l	–	–	–	4.0	3.3	49	7
2	F	74	r	b	82	r	13	NE	38	4.6	3.9	43	37
						l	7.0	3.0	40	4.0	2.9	48	12
3	M	67	r	b	192	r	–	–	–	3.9	3.1	48	22
						l	5.8	3.2	39	3.8	3.1	44	22
4	F	67	l	b	186	r	7.1	NE	48	3.7	3.0	45	39
						l	–	–	–	4.0	2.5	48	12
5	F	41	r	b	187	r	7.3	NE	38	6.6	4.3	31	36
						l	7.3	4.8	40	NO	NO	NO	
6	M	48	r	b	240	r	NE	NE	NE	4.0	2.7	52	23
						l	NE	NE	NE	4.4	2.9	53	21
7	F	52	r	b	144	r	9.9	NE	NE	4.6	3.7	44	16
						l	11	NE	38	NO	NO	NO	
8	F	50	l	l	192	l	8.2	NE	48	NO	NO	NO	

DML distal motor latency, DSL distal sensory latency, MCV motor nerve conduction velocity, – data were missing, NE not evoked, NO not operated on.

graphic examination of the hands was performed, and the level of serum ß-2 microglobulin was measured as an indicator of amyloid accumulation. Diagnostic criteria for CTS were based on clinical signs and electrophysiological results.

Results

Among the 60 patients examined, 15 hands of 8 patients (3 men and 5 women) were diagnosed as CTS including 12 hands of those 7 patients who had been operated on before this study (Table 1). The incidence of CTS was correlated to the period of hemodialysis: 1/29 patients with less than 5 years on dialysis, 1/17 with 5–10 years, 6/14 with more than 10 years. The incidence showed no correlation to sex or blood access site. Of the 15 hands with CTS, preoperative electrophysiological data were available in 11 hands and showed severe compromise of the median nerve function at the carpal-tunnel. All operated patients were satisfied with the result after a mean follow-up of 21 (7–39) months. All but one (Case 5) showed improvement at the electrophysiological examination.

6 patients with CTS had cystic radiolucency in their carpal bones (Figure 1), a significantly high incidence compared with that for the patients without CTS; (8/48 patients whose radiographs were available). Serum ß-2 microglobulin of the CTS patients was increased to 27 *SD 5.0* mg/L (normal value < 1.0 mg/L) which did not differ from that of the patients without CTS (27 *12* mg/L). This increase in the level of serum ß-2 microglobulin correlated with increasing periods on hemodialysis. In 8/12 operated hands, a biopsy from the transverse carpal ligament was analyzed, and amyloid deposition was recognized in 4 (Figure 2).

For all 120 hands, nerve conduction studies showed generally deteriorated nerve conduction velocity in both forearms (49 *6.2* m/s for the median nerve and 48 *5.8* m/s for the ulnar nerve). To determine whether the median nerve function at the forearm influences the degree of compromise of the median nerve at the carpal-tunnel, we examined the relation between the distal motor latency and motor nerve conduction velocity of the median and ulnar nerves in the forearm. The 12 operated hands were excluded from this study in order to eliminate the influence of the operation. In the remaining 108 non-operated hands, the median distal motor latency was extended to 4.6 *1.2* ms, and the distal sensory latency to 3.2 *0.6* ms, whereas the ulnar distal motor latency and distal sensory latency were 3.4 *0.6* ms and 2.9 *0.4* ms, respectively. There was a correlation between motor nerve conduction velocity and distal motor latency of the median nerve in which the retardation of the motor nerve conduction velocity was related to the delay in distal motor latency, but this was not the case for the ulnar nerve (Figure 3). There were no differences between the electrophysiological data for the blood access and the non-blood access sites.

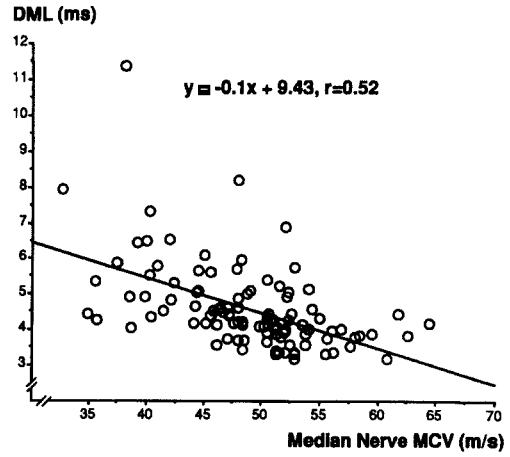

Figure 3. Relationship between motor nerve conduction velocities and distal motor latencies of 108 median nerves of 55 patients undergoing hemodialysis. DML: distal motor latency. MCV: motor nerve conduction velocity.

Discussion

A high incidence of CTS developing in patients undergoing long-term hemodialysis has been reported (Warren and Otieno 1975, Kenzora 1978, Delmez et al. 1982, Bradish 1985, Minami and Ogino 1987, Naito et al. 1987, Gilbert et al. 1988). Although the pathomechanism in these cases is still controversial and has not been clearly explained, various hypotheses have been advocated for the etiology of CTS related to hemodialysis. One explanation is related to the altered hemodynamics caused by the arteriovenous fistula. Warren and Otieno (1975) attributed the cause to edema induced by increased venous pressure at the blood access site. Harding and Le Fanu (1977) suggested that ischemia resulting from the vascular steal syndrome contributed to the symptoms. Halter et al. (1981), however, insisted that the presence of forearm blood access is not crucial to the development of CTS and showed that the highest incidence of CTS was found in dialysis patients with peripheral neuropathy, as confirmed by a nerve conduction study. After Assenat et al. (1980) reported amyloid deposition in the transverse carpal ligament, amyloid deposition in the carpal-tunnel has come to be recognized as a possible causative factor in hemodialysis patients (Gejyo et al. 1986a).

Our result of 13 percent CTS incidence agrees with Gilbert's (1988) report of approximately 10 percent. This incidence of CTS is even higher for patients on hemodialysis for longer periods. A CTS incidence as high as 30 percent has been reported in patients who had been hemodialyzed for more than 10 years (Halter et al. 1981). This correlation between the duration of hemodialysis and development of CTS suggests that some accumulating factor is related to the incidence of CTS. Our electrophysiological study revealed generally slowed nerve conduction velocity of the median and ulnar nerves of the forearm in hemodialyzed patients, suggesting that nearly half of them suffered from peripheral neuropathy according to Melvin et al.'s (1966) criteria for peripheral neuropathy. The same study suggested a high incidence of uremic neuropathy in hemodialyzed patients and a close relationship with the development of CTS.

One of our patients operated on with severely delayed nerve conduction velocity showed only a minimal improvement in the median distal motor latency after carpal-tunnel release. Compromise of the median nerve in the carpal-tunnel in this patient might have occurred subclinically as a result of severe peripheral uremic neuropathy, even after adequate decompression. There was no difference between the values for serum ß-2 microglobulin of CTS patients and those of non-CTS patients in our series, which is in accordance with the past report by Gejyo et al. (1986b). They suggested that some factors other than the level of serum ß-2 microglobulin, such as calcium or sulfated glucose of aluminum, might play an important role in the mechanism where amyloid is deposited in human tissues, though amyloid deposits in hemodialyzed patients consist mainly of ß-2 microglobulin itself. In half of the specimens derived from the transverse carpal ligaments of our operated patients, amyloid deposits were identified. In view of all these results, we conclude that in many patients receiving long-term hemodialysis, median nerves are more susceptible, due to the underlying uremic neuropathy, and are easily damaged by amyloid-induced tenosynovitis in the carpal-tunnel.

References

Assenat H, Calemard E, Charra B, Laurent G, Terrat J C, Vanel T. Hemodialyse: syndrome du canal carpien et substance amyloide. Nouv Presse Med 1980; 9 (24): 1715.

Bradish C F. Carpal tunnel syndrome in patients on haemodialysis. J Bone Joint Surg (Br) 1985; 67 (1): 130–2.

Delmez J A, Holtmann B, Sicard G A, Goldberg A P, Harter H R. Peripheral nerve entrapment syndromes in chronic hemodialysis patients. Nephron 1982; 30 (2): 118–23.

Gejyo F, Homma N, Suzuki Y, Arakawa M. Serum levels of beta 2 microglobulin as a new form of amyloid protein in patients undergoing long-term hemodialysis (letter). N Engl J Med 1986a; 314 (9): 585–6.

Gejyo F, Odani S, Yamada T, Honma N, Saito H, Suzuki Y, Nakagawa Y, Kobayashi H, Maruyama Y, Hirasawa Y, et al. Beta 2 microglobulin: a new form of amyloid protein associated with chronic hemodialysis. Kidney Int 1986b; 30 (3): 385–90.

Gilbert M S, Robinson A, Baez A, Gupta S, Glabman S, Haimov M. Carpal tunnel syndrome in patients who are receiving long-term renal hemodialysis. J Bone Joint Surg (Am) 1988; 70 (8): 1145–53.

Halter S K, DeLisa J A, Stolov W C, Scardapane D, Sherrard D J. Carpal tunnel syndrome in chronic renal dialysis patients. Arch Phys Med Rehabil 1981; 62 (5): 197–201.

Harding A E, Le Fanu J. Carpal tunnel syndrome related to antebrachial Cimino Brescia fistula. J Neurol Neurosurg Psychiatry 1977; 40 (5): 511–3.

Kenzora J E. Dialysis carpal tunnel syndrome. Orthopedics 1978; 1 (3): 195–203.

Lenman J A R, Ritchie A E. Clinical electromyography. Pitman Med & Scient Publ Co, Kent 1970.

Melvin J L, Harris D H, Johnson E W. Sensory and motor conduction velocities in the ulnar and median nerves. Arch Phys Med Rehabil 1966; 47 (8): 511–9.

Minami A, Ogino T. Carpal tunnel syndrome in patients undergoing hemodialysis. J Hand Surg (Am) 1987; 12 (1): 93–7.

Naito M, Ogata K, Goya T. Carpal tunnel syndrome in chronic renal dialysis patients: clinical evaluation of 62 hands and results of operative treatment. J Hand Surg (Br) 1987; 12 (3): 366–74.

Warren D J, Otieno L S. Carpal tunnel syndrome in patients on intermittent haemodialysis. Postgrad Med J 1975; 51 (597): 450–2.