

– Abstract –

Clinical and Electrophysiologic Review of Patients with Common Peroneal Mononeuropathy

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Objectives : This study was designed to evaluate the clinical and electrophysiologic findings of patients with common peroneal neuropathy.

Methods : Eighty-seven patients (94 lesions) with electrodiagnostically confirmed common peroneal neuropathy were analyzed via the medical records and electrodiagnostic findings. The subjects were divided into three groups according to the pathophysiologic process: conduction block; axonal loss; and the mixed. The relation of pathophysiologic process and the time from the onset to the examination was analyzed.

Results : Most common etiology of common peroneal neuropathy was fracture of the lower extremity (50%) and the second was compression around the fibular head (26.6%). The pathophysiology was predominantly axonal loss regardless of etiology: 83 axonal loss; 7 conduction block; and 4 mixed type. Conduction block pattern was significantly observed ($p < 0.05$) when electrophysiologic study was performed within 10 days post-onset.

Conclusion : Nerve conduction study must be interpreted cautiously when performed within the first 10 days after onset and follow-up examination would be needed to assess the severity and prognosis of disease.

Key Words : Common peroneal neuropathy, Axonal loss, Conduction block, Mixed type

3-6

가

가

4

가 fibular tunnel

,1,2

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(nerve entrapment)

4

가

7-9

87 7
 94 67 (77%),
 20 (23%) , 20 가 22 (25.3%)
 5 가 10 16 (18.4%)
 38 (, 8~82) (Fig. 1).
 320±646 (, 2~5475) , 87
 80 (92%)

가 (: 51 , : 43).

(axon loss), 2.

(conduction block)

(mixed axon loss/conduction block)

60% 가

3

1.

H 가

1997 1 2000 5

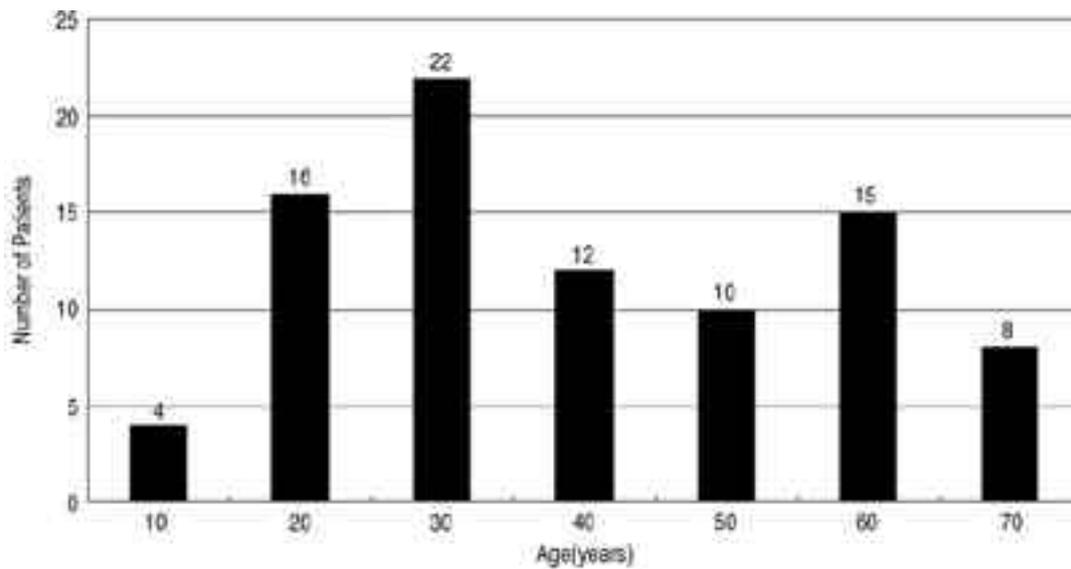


Fig. 1. Age distribution in common peroneal neuropathy.

Appendix 1. Criteria for different pathophysiologic processes affecting the peroneal nerve

I. conduction block (presumably secondary to focal demyelination)

; the amplitude of the compound muscle action potential (CMAP), stimulating proximally, was less than 50% of the distal amplitude, recording from the extensor digitorum brevis (EDB) and/or tibialis anterior (TA)

II. axonal loss

- 1) the amplitude of the peroneal CMAP, while stimulating distally, recording either EDB or TA, was unelicitable, low compared with normal values for age, or relatively low (less than 50%) compared with the corresponding contralateral response
- 2) the amplitude of the superficial peroneal sensory nerve action potential (SNAP) was unelicitable, low compared with normal values for age, or relatively low (less than 50%) compared with the corresponding contralateral response

III. mixed conduction block and axonal loss

; the nerve conduction study results fulfilled both criteria

		(Biceps femoris short head)		
	5			
			1.	
Katirji-Wilbourn ³ (1988)				
(conduction block),		(axonal loss)		47 (50%)
(mixed type)			가	25
(Appendix 1). ²			(26.6%)	
50%			10 (10.6%)	
			1	
			가 5	(Fig. 2).
			47	16 (34%) 가
		50%		(13 ,
			27.7%).	8 (17%) ,
			2	1 ,
			가	
			47	39 (Fig. 3).
			7	2
(complete lesion)				, 5 (71.2%)
		(incomplete lesion)	가	
			10	
	1 , 11	30	2 , 31	3
		10		
			10	11
			39 (41.5%) 가	가
			가	7 (7.4%)
			2.	
3.				18 (19.1%),
				76
SPSS 9.0 for window			(80.9%)	

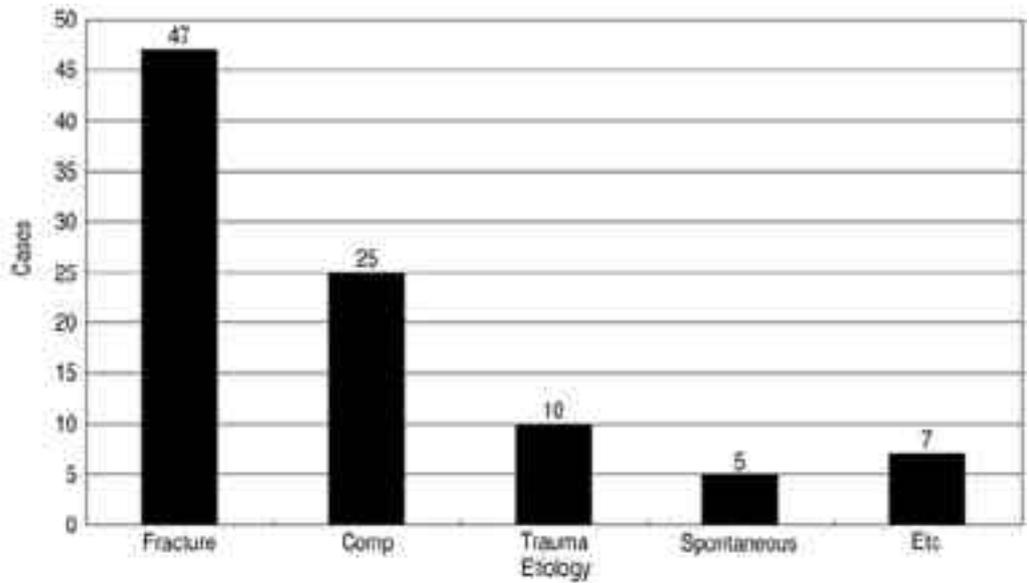


Fig. 2. Etiology of common peroneal neuropathy. Comp, compression; Spontaneous, neither trauma or compression; Etc, hip dislocation (1), vasculitis (1), ankle sprain (1), glass nerve injury (4).

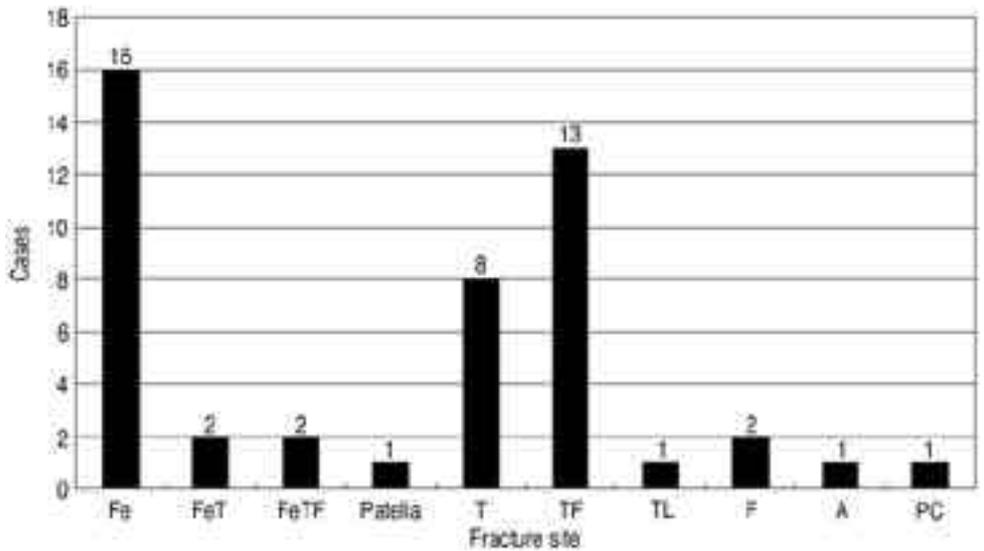


Fig. 3. Fractures in common peroneal neuropathy (Fe, femur; FeT, femur+tibia; FeTF, femur+tibia+fibula; T, tibia; TF, tibia+fibula; TL, tibia+lateral collateral ligament; F, fibula; A, ankle; PC, pelvis+calcaneus).

%) 7† (30.1%)
 4
 13
 6 ,
 6 ,
 1
 1, 2
 가 (ventral ramus) (pos-
 83 (88.3) 1
 , 25 , 3
 7 67 (97.1%)
 (5 , 71.4%). (p<0.001, Table 2).
 (Table 1).
 (p>0.05).
 4, 5

Table 1. Pathophysiologic Processes and Etiology of Common Peroneal Neuropathy

Etiology	Pathophysiologic process			Total
	Conduction block	Axonal loss	Mixed lesion	
Fracture	1	46	0	47
Compression	3	19	3	25
Trauma	1	9	0	10
Spontaneous	2	2	1	5
Etc	0	7	0	7
Total	7	83	4	94

($p > 0.05$ compared pathophysiologic processes with etiology of common peroneal neuropathy) Spontaneous, neither trauma or compression; Etc, hip dislocation (1), vasculitis (1), ankle sprain (1), glass nerve injury (4)

Table 2. Pathophysiologic Processes and Time Duration from onset to Examination

Time duration	Pathophysiologic process			Total
	Conduction block	Axonal loss	Mixed lesion	
I	6	6	1	13
II	1	10	1	12
III	0	67	2	69
Total	7	83	4	94

($p < 0.001$ compared pathophysiologic processes with time duration from onset to examination)

I, 10 days post-onset; II, 11 days, 30 days; III, 31 days post-onset

terior divisions)

가
 가 10 cm
 가
 가 (superficial head) .^{4,10} Berry fibular tunnel
 tendinosus arch , fibular tunnel .⁴ 0.5 cm (ankle inversion)
^{4,9} Sunderland 가
^{4,11,12} 20 1
² , 7 ,
 11 가 .⁴ , fibular tunnel
⁴ , 4 cm 가
 . Nobel
 2
 (leg crossing) (nerve sheath) .¹⁰
 10 가 가
 . Nobel

가
 Watson Jones 13 6 46.2% 10 85.7%
 , , , , 가 31 2.9%
 , , , , 13 7~10
 14 Wallerian 가
 가 16 (34%) 10
 8 (17%) (27.7%)
 가
 Katirji 80 79% 30% 가 가
 16.5% 2 가 가
 가 가
 가 가
 34% 가
 Katirji , , , 83 , 7 , 4 10
 가 가
 가 55% 가 , 20%,
 22.5% Wilbourn
 60% 가
 25%
 5 10 11

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가 83% 7.4% 1. Clawson DK, Seddon HJ: The late consequences of sciatic nerve injury. J Bone Joint Surg[Br] 1960; 42: 213-225

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