Nonsystemic Vasculitic Neuropathy: A Clinicopathological Study of 22 Cases

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ABSTRACT. ***Objective.*** The involvement of the peripheral nervous system in patients with systemic vasculitis has been reported, but nonsystemic peripheral nervous system vasculitis is not so well known. We investigated the clinical, electrophysiological, and pathological features of nonsystemic vasculitic neuropathy (NSVN) in order to establish the clinical and histological manifestations and to promote the earlier diagnosis of the syndrome.

Methods. Biopsies were selected from over 700 sural nerve biopsies performed at the Section of Neuropathology, Neurological Clinic of Athens University Hospital. The diagnosis of vasculitis was based on established clinicopathological criteria. Other causes of peripheral neuropathy were excluded. Complete laboratory, clinical, electrophysiological, and pathological studies were performed in all cases.

Results. Nerve biopsies of 22 patients were diagnosed as NSVN. The pathological features were vasculitis and predominant axonal degeneration with a varying pattern of myelinated fiber loss. The vasculitic changes were found mainly in small epineural blood vessels. Mononeuritis multiplex and distal symmetrical sensorimotor neuropathy were equally frequent.

Conclusion. NSVN should be suspected in a case of unexplained polyneuropathy without evidence of systemic involvement. Clinical and neurophysiological studies are essential for the detection of nerve involvement, but the specific diagnosis of NSVN may be missed unless a biopsy is performed. (J Rheumatol 2005;32:853–8)

Key Indexing Terms:
NONSYSTEMIC VASCULITIC NEUROPATHY
NONSYSTEMIC VASCULITIS
POLYNEUROPATHY
AXONAL

The syndrome of peripheral neuropathy due to vasculitis without manifestations of disorders in other systems was first reported by Kernohan and Woltman in 1938[1]. In 1987, Dyck, *et al* described characteristic features of 20 cases with vasculitic neuropathy with no or few clinical symptoms of systemic disease or serological abnormalities, and proposed the term of nonsystemic vasculitic neuropathy (NSVN)[2]. In 1996, Davies, *et al* reported 25 patients with vasculitis confined to peripheral nerves using the term isolated peripheral nervous system vasculitis (IPNSV)[3]. There are a few reports that describe patients with vasculitis selectively affecting the peripheral nervous system[4-8]. The largest study (48 patients) was reported by Collins, *et al* in 2003[9]. As the prognosis of NSVN is good and the disease is potentially treatable, timely recognition is important for successful management of patients.

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Accepted for publication December 23, 2004.

We examined the clinical, electrophysiological, and histopathological features of 22 patients with NSVN in order to promote early diagnosis of the syndrome.

MATERIALS AND METHODS

Patient selection and clinical data. Biopsy specimens were selected from over 700 sural nerve biopsies performed at the Section of Neurology, Neurological Clinic of Athens University Hospital, during the last 20 years. Seventy-one biopsies fulfilled the pathological criteria for vasculitis[10-12]. According to these criteria a positive diagnosis requires (1) perivascular or transmural inflammatory cell infiltration in at least one endoneurial or epineurial blood vessel; and (2) signs of vessel wall necrosis (evidence of destruction and disorganization of the muscularis by fibrinoid necrosis, disruption of the endothelium, thrombosis of the lumen, or hemorrhage into the wall of the vessels). Twenty-two (30.9%) cases with no clinical or laboratory indication of other organ system involvement by vasculitis were retained for further investigation. Other causes of peripheral neuropathy such as diabetes, sarcoidosis, leprosy, malignancy, connective tissue disease, toxic exposure, nutritional deficiency, and family history of neuromuscular disorders were excluded by appropriate clinical and laboratory investigations.

Neurological interview and examination were carried out in all patients prior to nerve biopsy by at least one neurologist. Muscle strength testing was scored with the 5 point Medical Research Council scale. Deep tendon reflexes, pain, touch, and proprioception were examined and graded as normal, abnormally altered (increased or decreased), or absent. The patient's functional state was estimated using the modified Rankin scale. Complete serological tests including erythrocyte sedimentation rate, C-reactive protein, C3, C4, antinuclear antibody, anti-DNA, rheumatoid factor (RF),

cryoglobulin, and immunoelectrophoresis were carried out in all cases. Serum and urine specimens were tested for monoclonal protein. All patients had routine hematological tests and chest radiology. Nerve conduction studies and electromyography (EMG) were performed on each patient at the EMG laboratory of our institution. Motor conduction velocity studies of the median, ulnar and peroneal, and antidromal sensory conduction velocity of the median, ulnar, and sural nerves were conducted bilaterally. The first dorsal interosseus, anterior tibial, and medial gastrocnemius were sampled by means of standard concentric needle electrodes.

Neuropathies were classified as mononeuritis multiplex (motor and/or sensory deficits in the distribution of more than one isolated peripheral nerve), distal symmetrical sensorimotor polyneuropathy (symmetrical distal stocking-glove distribution of sensory and motor deficits), or asymmetrical/overlapping neuropathy (motor or sensory deficits with substantial asymmetry between sides)[13].

Histological techniques. Biopsy of the whole sural nerve was performed under local anesthesia. Specimens were divided into 3 to 5 sections, each about 1 cm in length. One piece was fixed in 10% formaldehyde, embedded in paraffin, and cut transversely and longitudinally in sections of 7 µm thickness. The sections were stained with hematoxylin and eosin (H&E). Another piece was fixed in Flemming's solution for 24 h, dehydrated in alcohol, embedded in paraffin wax, and cut transversely and longitudinally in serial sections of 7 µm thickness. The sections were stained with Weigert Pall. A third piece was fixed in 1% glutaraldehyde, stained for 24 h in 1% osmium tetroxide, macerated in glycerol, and then teased apart under a dissecting microscope in order to isolate single nerve fibers. At least 50 fibers were sampled and assessed for pathological conditions based on the criteria of Dyck, *et al*[14]. The specimens for semithin sections and electron microscopy were fixed in a solution of 2.5% glutaraldehyde in Sorrenson buffer and embedded in epoxy resin. Semithin sections were stained with toluidine blue. Ultrathin sections were stained with uranyl acetate and lead extract and examined with a Philips EM 201 electron microscope. Another portion of the nerve was prepared for immunohistochemical staining. Immunoperoxidase procedures were used for polyclonal antibodies IgG, IgM, IgA, C3, and fibrinogen.

Pathologic changes were diagnosed and classified as predominantly axonal, demyelinating, or mixed axonal and demyelinating based on both teased fibers and resin sections, according to established criteria[15].

Morphometry. Morphometric analysis of myelinated fibers was performed using the VIDS III and Optomax V image analysis system, connected with a microscope by a color camera, and included measuring the fascical area, the number, density and mean diameter of the myelinated fibers and the fiber-size distribution histograms, the myelin sheath thickness, the mean axon diameter, and the g ratio.

RESULTS

Clinical features. Nerve biopsies of 22 patients were recognized as NSVN. Using the above pathological criteria of perivascular or transmural inflammation cell infiltration in at least one endoneurial or epineurial blood vessel and of vessel wall necrosis, all cases showed evidence of vasculitis, with no clinical or laboratory indicators of other disease.

Table 1 summarizes the clinical, electrophysiological, and serological results. The patients were 12 men and 10 women, 23 to 77 years of age. The duration of the illness before biopsy varied from 18 to 144 months, with duration of symptoms > 60 months in 12 patients.

There was no significant weight loss in any patient. Unexplained fever (37.0–37.5°C) was mentioned by one patient. Most patients presented with sensory symptoms. Pain and paresthesia were severe in 7 and mild in 12 patients. Touch sensation was decreased in all patients. Muscle weakness was mild in 12 and severe in 2 patients. Two patients reported episodes of vertigo. There were no skin lesions or signs of cranial neuropathy. No pronounced functional disability was found in any patient and the mean modified Rankin scale score was 3.18.

Based on the clinical features, there were 9 cases with mononeuritis multiplex, 9 with distal symmetrical sensorimotor neuropathy (including patients with mild asymmetries), and 4 with asymmetrical neuropathy. The electrophysiological studies were abnormal in all 22 patients. Most had mildly decreased motor and sensory conduction velocity, with significantly reduced amplitude of the motor and sensory compound action potentials, without block or dispersion. The sural nerve action potential was frequently absent in 11 patients. There was EMG evidence of chronic denervation in 9 patients and signs of active denervation in 7.

Histopathology. Using the above pathological criteria, all cases had evidence of vasculitis (Figure 1). All biopsy specimens presented marked perivascular or transmural infiltration of inflammatory cells (14 showed both perivascular and diffuse infiltration, 6 perivascular only, and 2 diffuse only). The vasculitic changes were found mainly in the small epineurial blood vessels (88 ± 31 µm) (Figure 2). Histology revealed multifocal fiber loss due to ischemia from vasculitis (Figure 3). Subperineurial edema was observed in 5, perineurial thickening in 11, and new vessel formation in 13 cases. We found no microfasciculation forming injury neuromas. Axonal degeneration was the predominant feature in most cases, with only 3 cases showing segmental demyelination (Table 2). The immunohistochemical study was positive to antihuman polyclonal antibodies IgA, IgG, IgM, C3, and fibrinogen in 17 specimens showing depositions in infiltrated vessel walls.

Morphometry. Morphometric study was performed in all 22 cases. There was significant loss of myelinated fibers in all biopsies (2433 ± 1245) compared with a published age-matched normal control series[16]. The mean axonal diameter was especially decreased in the cases with axonal degeneration (2.87 µm ± 0.93 SD). The g ratio was markedly decreased (0.42 ± 0.11) and supported the histopathological findings of mainly axonal involvement. The histograms of fiber diameter distributions showed damage of small and large fibers in most cases.

Overall, there was no difference in the intensity of vasculitis or in the amount of damaged nerve fibers among the different types of polyneuropathy. Moreover the clinical severity of neuropathy was not related to the vasculitic changes.

Treatment. All patients received initial treatment with prednisone 60–80 mg, and azathioprine was added in 4 patients. However, followup was not sufficiently systematic to permit an accurate assessment of the response to treatment.

Table 1. Clinical, electrophysiological, and serological features of patients with NSVN.

N	Sex	Age, yrs	DS, mo	Pattern		Serological Test	DS
				Clin	NCS		
1	F	61	72	S	S	—	4
2	F	72	120	S	S	—	4
3	M	57	84	S	S	—	4
4	M	36	36	S	S	—	3
5	M	46	24	MM	MM	CRP+, ANA+ (< 1/180)	2
6	F	58	60	MM	MM	ESR 30 mm/h	2
7	M	47	36	As	S	ANA+ (< 1/180)	3
8	M	37	30	MM	As	—	3
9	F	49	48	As	As	ESR 33 mm/h	3
10	F	64	108	MM	MM	—	3
11	M	62	96	S	S	—	4
12	M	29	24	MM	MM	—	2
13	F	23	18	MM	MM	—	3
14	F	36	48	As	S	—	3
15	F	70	132	S	As	—	4
16	M	64	60	MM	MM	—	3
17	F	65	120	MM	MM	ESR 38 mm/h	3
18	M	48	36	S	S	—	3
19	M	71	60	MM	MM	—	3
20	M	54	48	As	MM	—	3
21	M	64	72	S	S	—	4
22	F	77	144	S	As	ESR 40 mm/h	4

Age: at biopsy; DS: duration of symptoms before biopsy (months); Clin: clinical pattern of neuropathy; NCS: nerve conduction studies; MM: mononeuritis multiplex; S: symmetrical sensory and motor neuropathy; As: asymmetrical sensory and motor neuropathy; DS: disability score.

DISCUSSION

Peripheral nervous system involvement is a common complication of systemic vasculitis and has been well documented[17-25]. However, there have been only a few studies of nonsystemic vasculitic neuropathy[1-6,8,9]. Twenty-two out of 700 biopsies (3.8%) performed in our laboratory were diagnosed as NSVN.

In our study mononeuritis multiplex and distal symmetrical neuropathy occurred in the same proportion of cases. Based on clinical and electrophysiological features there were 9 cases with mononeuritis multiplex, 9 with distal symmetrical neuropathy, and 4 with asymmetrical neuropathy. Although mononeuritis multiplex is thought to be the most frequent neuropathic manifestation of systemic and nonsystemic vasculitis[2,3,8,21], symmetrical polyneuropathy[2,3,10,20,26] and asymmetrical sensory or motor neuropathy[9] have also been reported in a considerable proportion of patients. The high rate of symmetrical neuropathy in our patients could possibly be due to the considerable delay between the initiation of symptoms and the clinical and neuropathological examination. At a late stage of the disease a primarily mononeuritis multiplex or asymmetric polyneuropathy could evolve toward a symmetrical picture by combination of multifocal lesions[3,20].

The pathophysiology of all the vasculitic syndromes, including NSVN, remains unknown, and the mechanism of vessel damage in these diseases is poorly understood. It is possible that several mechanisms may operate in the pathogenesis of vasculitis of peripheral nerves.

The inflammatory process in NSVN tends to affect smaller epineurial arterioles than in systemic vasculitis, but the cellular composition of the inflammatory infiltrates appears to be similar[2,4,27-29]. Moreover there is a predilection for the epineurial blood vessels, which are more severely affected than the endoneurial ones[2,6,27,28]. These findings are confirmed in our study, in which there was predominant involvement of smaller epineurial vessels, the endoneurial vessels rarely being damaged.

The neuropathy is caused by nerve infarction due to occlusion of vasa nervorum. The result of this infarction is a loss of sensory and motor axons. Analysis of the histopathological findings in our study revealed a varying degree of loss of myelinated fibers of all diameters in all cases. Active axonal degeneration is usually found in NSVN[2,3,5,6,8]. The dominant finding in sural nerve biopsies in our study was axonal degeneration, but 3 cases showed predominant demyelination. Segmental demyelination as an atypical feature of vasculitic neuropathy has been reported before[26,30]. Moreover there have been reports of isolated cases with conduction block in systemic and nonsystemic vasculitis[31-33], presumably not due to demyelination but to focal axonal conduction failure related to infarctive axonal injury[32]. These "pseudo conduction blocks," which appear in the course of ongoing Wallerian degeneration, give way to

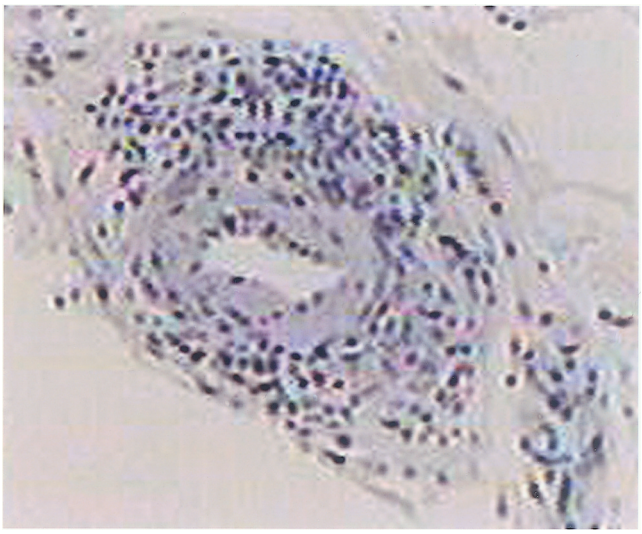

Figure 1. Pathologic appearance of a sural nerve from a patient with NSVN. The wall of small arteriole is involved and infiltrated with inflammatory cells (transverse paraffin section, H&E stain; original magnification ×40).

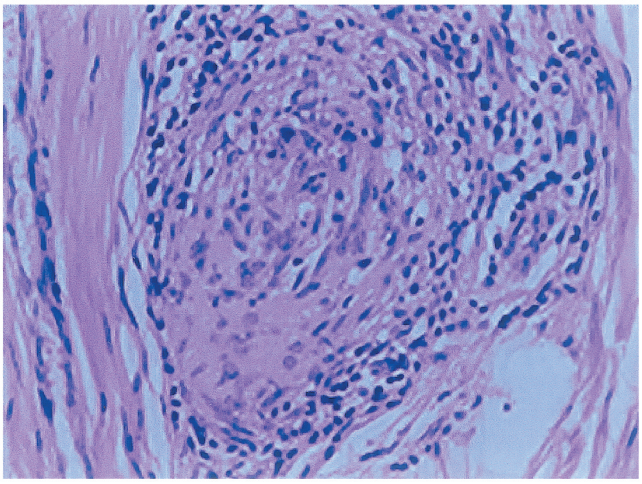

Figure 2. Longitudinal paraffin section of sural nerve, showing epineurial arteriole of a patient with NSVN (H&E stain; original magnification ×40).

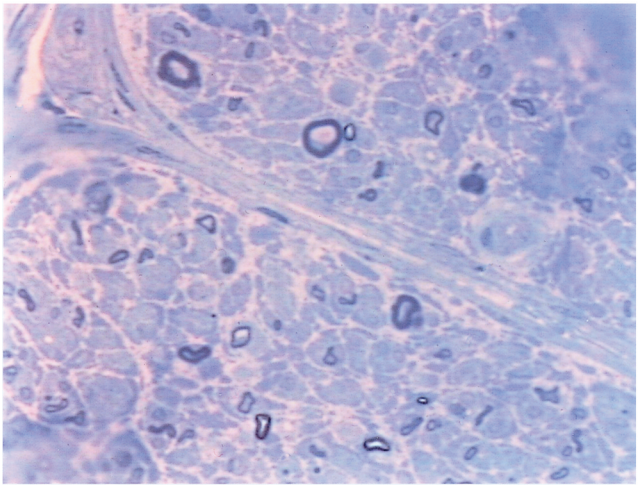

Figure 3. Semithin transverse epoxy section showing the prominent fiber loss (original magnification ×40).

unequivocal electrophysiological findings of axonal damage in subsequent examinations[9,34]. In our cases with demyelination there was no evidence of conduction block in the electrophysiological studies.

Until now there has been no fully satisfactory explanation for the presence of an organ-specific vasculitis confined to the peripheral nervous system. Some investigators suggest that isolated vasculitis of the peripheral nerves represents an early and mild form of systemic vasculitis[4,35]. A study by Said, *et al* demonstrated that in patients with clinically isolated peripheral neuropathy the vasculitic process could often spread in skeletal muscle, indicating that the vasculitis in these patients was not organ-specific[35]. In contrast, there are reports suggesting that selective involvement of nerve is not due to lack of severity, and that localized vasculitis of nerve represents a distinct clinicopathological entity with immunological mechanisms directed against antigens shared by nerve[2,3,29]. This point is supported by our findings. Indeed, in our patients, despite the long duration of illness, which would be sufficient for the systemic features to appear, the disease was confined in the peripheral nerves.

The usefulness of sural nerve biopsy in the detection of neuropathy from systemic or nonsystemic vasculitis is uncertain[2,3,7,10]. Dyck, *et al* reported that sural nerve biopsy was diagnostic in 58% of patients with systemic, but in only 25% of those with nonsystemic vasculitis[2]. Collins, *et al* showed that the estimated sensitivity of the combined superficial peroneal nerve and peroneus brevis muscle biopsy for vasculitic neuropathy was about 60%[7,9]. Davies, *et al* believe that nerve biopsy is critical to the diagnosis of NSVN[3]. They suggest that if prominent acute axonal degeneration is seen without mononuclear cuffs around vessels, further sections should be cut through the paraffin blocks of nerve tissue.

NSVN seems to have a favorable prognosis in contrast to that of systemic vasculitis neuropathy, and we believe that nerve biopsy is essential for appropriate diagnosis and treatment.

Our study confirms that (1) NSVN should be suspected when there is unexplained polyneuropathy without evidence of systemic involvement; (2) the diagnosis of NSVN is difficult on the basis of clinical and electrophysiological data only; (3) there is need of further classification and description of the characteristic pathological features; and (4) in many such cases nerve biopsy can establish the diagnosis and should be routinely performed, especially when noninvasive methods have been ineffective and when there is evidence of nerve damage.

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Table 2. Pathology in sural nerve biopsies with NSVN.

N	NFC			IC	IgA	IgG	IgM	C3	Fb
	Ad	D	B						
1		+		++			+	+	
2		+		+					
3			+	++	+		+		
4		+		++				+	+
5	+			+++		+		+	+
6	+			+++			+	+	
7	+			++	+		+	+	
8	+			+			+		
9	+			++	+	+		+	+
10	+			+++		+		+	+
11	+			+++	+		+	+	
12	+			+++		+		+	+
13	+			++		+			+
14	+			++		+		+	
15			+	+++		+	+	+	+
16	+			+		+			+
17	+			+++	+	+		+	+
18	+			++		+		+	
19			+	+++				+	+
20	+			++		+		+	+
21			+	++	+	+		+	
22			+	+++				+	+

NFC: nerve fiber changes; AD: axonal degeneration; D: demyelination; B: both (demyelination and axonal degeneration); IC: infiltration by inflammatory cells, C3, Fb: fibroinogen.

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