Vasculitic peripheral neuropathy in patients with provisional cutaneous arteritis

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Abstract

Objective

Cutaneous arteritis (CA) is a skin-limited medium-vessel vasculitis. Some patients with CA also exhibit extracutaneous manifestations, such as vasculitic peripheral neuropathy (VPN). VPN is likely underdiagnosed because nerve biopsies are invasive and often impractical. This study proposed the concept of provisional CA to better identify patients with neuropathic involvement and to evaluate the prevalence and electrophysiological features of VPN.

Methods

We retrospectively analysed patients with provisional CA treated at the Severance Hospital between 2011 and 2024. Provisional CA was defined as skin-limited medium-vessel vasculitis with neuropathic symptoms in the lower limbs irrespective of a nerve biopsy. VPN was defined based on established clinical and electrophysiological criteria. Demographic, clinical, and laboratory data were collected from the electronic medical records.

Results

Thirty-six patients met the provisional CA criteria. The median age was 51.0 years, and 36.1% of the patients were male. Among these, 22 (61.1%) demonstrated electrophysiological evidence of VPN. Pure sensory neuropathy and sensorimotor involvement were observed in 59.1% and 40.9% of patients, respectively. The most frequently affected nerves were the peroneal (63.6%) and sural (54.5%) nerves. No significant differences were found between the patients with and without VPN in terms of age, sex, skin manifestations, or laboratory findings.

Conclusion

VPN was prevalent in patients with provisional CA. Clinical features alone were insufficient to predict nerve involvement. Nerve conduction studies serve as a valuable diagnostic tool when a nerve biopsy is not feasible and may facilitate the earlier detection and management of neuropathic complications in skin-limited vasculitis.

Key words

peripheral neuropathy, cutaneous arteritis, vasculitis, polyarteritis nodosa

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Introduction

Polyarteritis nodosa (PAN) is a systemic, medium-sized vessel vasculitis that can involve multiple organs, including the skin, peripheral nerves, kidneys, and gastrointestinal tract (1). Skin-limited forms of PAN, often referred to as cutaneous PAN (cPAN) or cutaneous arteritis (CA), are characterised by medium-sized arterial inflammation confined to the skin without involvement of internal organs and manifest as erythema nodosum, livedo reticularis, livedo racemosa, or ulceration (2). At the 2012 Chapel Hill Consensus Conference, CA was formally classified as a single-organ vasculitis (3).

Vasculitic peripheral neuropathy (VPN) is a common clinical manifestation of systemic vasculitis, such as PAN, resulting from ischaemic nerve injury caused by vascular occlusion (4, 5). Although CA lesions are limited to the skin, some patients present with lower-limb symptoms suggestive of sensory or motor VPN (6). Current data on the prevalence, clinical patterns, and characteristics of VPN in patients with CA are limited. VPN is generally diagnosed based on clinical and electrophysiological findings. Histological confirmation is obtained through a nerve biopsy, which has been the gold standard traditionally (7-9). However, in clinical practice, nerve biopsy is often challenging because the procedure is invasive and associated with potential complications, such as sensory deficits or neuropathic pain. Moreover, a nerve biopsy might yield nonspecific findings if the specimen does not reach the hypodermis, where medium-sized arteries reside (10-12). Furthermore, a nerve biopsy is generally only available at specialised centres, limiting its utility as a routine diagnostic tool. Consequently, VPN in patients with CA may be underdiagnosed, potentially leading to inadequate treatment and delayed symptom recovery.

In this study, we introduced the concept of provisional CA to identify patients with skin-limited medium-vessel vasculitis presenting with neuropathic symptoms more appropriately, irrespective of nerve biopsy performance, and aimed to investigate the prevalence of VPN in these patients. We also sought to describe their neuropathic and electrophysiological features and evaluate the diagnostic utility of nerve conduction studies (NCS), particularly in settings where nerve biopsy is impractical or unavailable.

Materials and methods

Definition of provisional CA

Provisional CA was defined as patients who had not undergone nerve biopsy, presented with cutaneous lesions suggestive of medium-sized arterial inflammation (including erythema nodosum, livedo reticularis, livedo racemosa, or ulceration), exhibited sensory or motor neuropathic symptoms in the lower limbs, and showed no evidence of major organ involvement. In this study, provisional CA was applied as a clinical definition distinct from the formal diagnostic criteria for cPAN. While cPAN requires histopathologic confirmation of necrotising vasculitis in medium-sized arteries, the provisional CA group included patients without nerve or skin biopsy confirmation who exhibited clinical findings suggestive of localised medium-vessel inflammation along with neuropathic symptoms in the lower limbs. This definition of provisional CA has not been prospectively validated.

Study population and data collection

This retrospective study included patients aged 19 years or older who met the criteria for provisional CA and underwent NCS within three months before or after the diagnosis of CA at the Severance Hospital between March 2011 and February 2024. Patients with peripheral neuropathy attributable to other causes, such as diabetes mellitus, chronic alcohol use, chronic kidney disease, vitamin deficiency, or neurotoxic drug exposure, were excluded from the analysis. Demographic information, clinical data, comorbidities, and laboratory data were obtained from electronic medical records.

VPN criteria

VPN was defined according to established clinical and electrophysiological criteria, based on the recommenda-

tions of the Peripheral Nerve Society guidelines and Brighton Collaboration case definition (9). The patients were required to meet the following criteria. First, they had electrodiagnostic evidence of axonal neuropathy or clinical signs of peripheral neuropathy. Second, they had to exhibit a typical clinical presentation of VPN, which included 1) sensory or sensorimotor involvement, excluding pure motor neuropathy; 2) a multifocal or asymmetric distribution not attributable to compression or entrapment of peripheral nerves or roots; and 3) either additional clinical features or histopathological evidence of probable vasculitis on nerve biopsy. Additional clinical features include (i) predominance in the lower limbs, (ii) pain, and (iii) an acute or subacute course characterised by variable progression or episodic improvement of sensory or motor deficits (9, 13).

Electrophysiological assessment

The NCS was performed according to the standardised protocol at the Severance Hospital using a standard electromyography system. The motor and sensory nerves of the upper and lower limbs were evaluated, including distal latency, conduction velocity, and amplitude of the compound muscle and sensory nerve action potentials. Axonal neuropathy was diagnosed based on reduced amplitudes with relatively preserved conduction velocities, consistent with established electrophysiological criteria (14). All data were interpreted by experienced neurophysiologists who were blinded to the patients' clinical information.

Ethical statement

This study was approved by the Institutional Review Board (IRB) of Severance Hospital (Seoul, Korea, IRB no. 4-2020-1071) and was conducted in accordance with the Declaration of Helsinki. Given the retrospective study design and anonymised patient data, the requirement for written informed consent was waived by the IRB.

Statistical analysis

Continuous variables were presented as medians [interquartile ranges], and categorical variables were expressed as counts and percentages (%). The prevalence of VPN was calculated as a proportion, and 95% confidence intervals (CIs) were estimated using the exact binomial method. Chi-square test and Student's t-test were used for group comparisons. All statistical analyses were performed using SPSS (v. 23.0; IBM Corp., Armonk, NY, USA).

Table I. Characteristics of CA presenting clinical symptoms suggestive of VPN and having the results of NCS within 3 months around CA diagnosis.

Variables	values
Demographic data	
Age (years)	51.0 (39.5-62.8)
Male, n (%)	13 (36.1)
Female, n (%)	23 (63.9)
Cutaneous manifestations, n (%)	
Erythema nodosum	18 (50.0)
Livedo reticularis	8 (22.2)
Livedo racemosa	2 (5.6)
Ulceration	14 (38.9)
Laboratory results	
White blood cell count (/mm^3)	6.55 (5.71-7.55)
Haemoglobin (g/dL)	12.8 (12.1-13.4)
Platelet count (× 1000/mm^3)	265.0 (239.3-306.8)
ESR (mm/hr)	12.5 (6.0-27.0)
CRP (mg/L)	1.3 (0.5-2.9)
Blood urea nitrogen (mg/dL)	12.0 (10.3-15.1)
Serum creatinine (mg/dL)	0.7 (0.6-0.8)
Serum total protein (g/dL)	7.1 (6.8-7.4)
Serum albumin (g/dL)	4.4 (4.1-4.6)
Comorbidities, n (%)	
T2DM	2 (5.6)
Hypertension	5 (13.9)
Dyslipidaemia	3 (8.3)

Values are expressed as a median (25-75 percentile) or n (%).
CA: cutaneous arteritis; VPN: vasculitic peripheral neuropathy; NCS: nerve conduction study; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; T2DM: type 2 diabetes mellitus.

Results

Baseline characteristics

Baseline characteristics of the 36 patients with provisional CA are summarised in Table I. The median age was 51.0 years (interquartile range [IQR] 39.5–62.8), and 13 patients (36.1%) were male. Cutaneous manifestations included erythema nodosum in 50.0% of the patients, ulceration in 38.9%, livedo reticularis in 22.2%, and livedo racemosa in 5.6%. Laboratory findings showed a median white blood cell count of 6.55 ×10^3/mm^3 (IQR 5.71–7.55), haemoglobin 12.8 g/dL (12.1–13.4), platelet count 265 ×10^3/mm^3 (239.3–306.8), erythrocyte sedimentation rate 12.5 mm/hr (6.0–27.0), and C-reactive protein 1.3 mg/L (0.5–2.9). Renal function was normal with a median blood urea nitrogen of 12.0 mg/dL (10.3–15.1) and serum creatinine of 0.7 mg/dL (0.6–0.8). The comorbidities included hypertension (13.9%), type 2 diabetes mellitus (5.6%), and dyslipidaemia (8.3%).

VPN patterns and NCS findings

Among the 36 patients, 22 (61.1%, 95% CI 45.0–77.2%) were confirmed with VPN, showing electrodiagnostic evidence of axonal neuropathy on NCS findings (Table II). Although VPN was not confirmed by NCS in the remaining 14 patients, all 36 patients presented with clinical features typical of VPN, characterised by sensory or sensorimotor involvement, multifocal or asymmetric neuropathy, and a predominance of pain in the lower limbs. The clinical course was acute or subacute with one or more acute attacks, variable progression, or improvement in sensory or motor deficits. Pure sensory neuropathy was observed in 13 patients (59.1%), mixed sensorimotor neuropathy was present in nine patients (40.9%), and no cases of pure motor neuropathy were identified. The most commonly affect-

Table II. VPN pattern and NCS findings in CA patients.

Variables	values
Among 36 patients with NCS results	
Prevalence of VPN, n (%)	22 (61.1, 95% CI 45.0–77.2)
Clinical features suggestive of VPN, n (%)	
I. Evidence of PN	
a. electrodiagnostic evidence of an axonal neuropathy	22 (61.1, 95% CI 45.0–77.2)
OR	
b. clinical examination signs of PN*	N/A
AND	
II. Clinical presentation typical for VPN	
a. Sensory-motor or sensory (not pure motor)	36 (100)
AND	
b. Multifocal or asymmetric pattern at any time, AND this is not attributable to compression or entrapment of peripheral nerves or roots	36 (100)
AND	
c. EITHER	
i. Further clinical features:	
(1) Lower limb predominant	36 (100)
AND	
(2) Painful	36 (100)
AND	
(3) One or more acute attacks, or variable speed of progression, or improvement of motor of sensory deficit	36 (100)
OR	
ii. Biopsy or nerve shows histopathologically probable vasculitis (N=2)	1 of 2 (50.0)
Among 22 CA patients with VPN on NCS	
NCV findings	
Types of VPN, n (%)	
Mixed	9 (40.9)
Pure sensory	13 (59.1)
Pure motor	0 (0)
Sites affected by VPN, n (%)	
Median nerve	4 (18.1)
Ulnar nerve	5 (22.7)
Peroneal nerve	14 (63.6)
Posterior tibial nerve	4 (18.1)
Saphenous nerve	2 (9.1)
Sural nerve	12 (54.5)
Plantar nerve	0 (0)

Values are expressed as n (%).
*Because of the retrospective study design, neurological examination findings corresponding to this item could not be collected from all patients.
VPN: vasculitic peripheral neuropathy; NCS: nerve conduction studies; CA: cutaneous arteritis; PN: peripheral neuropathy; CI: confidence interval.

ed nerves were the peroneal (63.6%), sural (54.5%), ulnar (22.7%), median (18.1%), and posterior tibial nerves (18.1%). The saphenous nerve was less frequently involved (9.1%), whereas none of the patients demonstrated plantar nerve involvement. Two patients underwent nerve biopsy, one of whom (50.0%) showed probable vasculitis.

Comparison of provisional CA patients with and without VPN

We compared the demographic characteristics, cutaneous manifestations, and laboratory findings between patients with and without VPN on NCS (Table III). No significant differences were observed in age, sex distribution, skin lesion type, or baseline laboratory parameters between the two groups.

Discussion

In this study, we proposed the concept of provisional CA to facilitate the identification of VPN in patients with skin-limited vasculitis when a nerve biopsy is challenging. Using this definition, 61.1% of patients showed electrophysiological evidence of VPN on NCS, a prevalence higher than previously reported ranges of 2.4–33.3% in CA (3, 15). This relatively high prevalence may be explained by our modified diagnostic criteria for provisional CA based on typical CA skin lesions and clinical neuropathic symptoms. Unlike previous studies that relied on selective nerve biopsies (16, 17), this approach enabled the detection of subclinical or milder manifestations of VPN that would have remained undetected, indicating a potential underestimation of peripheral nerve involvement in clinical practice. The neuropathic characteristics observed in our cohort were predominantly axonal neuropathy, a pattern of sensory or sensorimotor involvement, multifocal or asymmetric distribution, and predominance in the lower limbs, particularly the peroneal and sural nerves. These features are consistent with the typical presentation of VPN in systemic PAN, even in skin-limited vasculitis (1, 18).

Although a nerve biopsy is considered the gold standard for diagnosing VPN, its clinical utility is often limited by several factors, including its invasiveness, technical complexity, potential sampling errors owing to the patchy distribution of nerve involvement, and uncertain sensitivity (10, 19). Although biopsy can provide valuable histopathological information, particularly in patients with atypical features or a progressive clinical course, these limitations frequently make biopsy impractical in routine clinical settings (4). Furthermore, Goel et al. demonstrated that nerve biopsy results do not always correspond with clinical findings in patients with VPN (20). In such cases, electrodiagnostic studies can serve as complementary diagnostic approaches. In both large arteriolar and microvasculitic neuropathies, NCS typically reveals acute to subacute axonal loss affecting the sensory and motor fibres, often in a patchy and multifocal pattern (4, 21, 22). Among these findings, asymmetrical or non-length-dependent patterns of axonal damage are considered particularly supportive of a VPN diagnosis (23). The high prevalence of VPN in our cohort of patients with provisional CA further highlights the need for early recognition of neuropathic

Table III. Characteristics of provisional CA patients with and without VPN.

Variables	With VPN (n=22)	Without VPN (n=14)	*p*-value
Age	51.5 (42.8–66.3)	46.0 (35.5–56.0)	0.224
Male	9 (40.9)	4 (28.6)	0.501
Erythema nodosum	11 (50.0)	7 (50.0)	1.0
Livedo reticularis	6 (27.3)	2 (14.3)	0.441
Livedo racemose	1 (4.5)	1 (7.1)	1.0
Ulceration	11 (50.0)	3 (21.4)	0.160
Hypertension	3 (13.6)	2 (14.3)	1.0
Diabetes mellitus	1 (4.5)	1 (7.1)	1.0
Hyperlipidaemia	2 (9.1)	1 (7.1)	1.0
HBV	3 (13.6)	0 (0.0)	0.267
WBC, x10^3/mm^3	6.5 (5.6–7.6)	6.6 (5.9–7.7)	0.864
Hb, g/dL	13.1 (12.3–13.5)	12.3 (12.1–13.1)	0.105
Plt, x10^3/mm^3	254.5 (223.3–283.5)	298.5 (267.3–336)	0.144
ESR, mm/hr	14.0 (5.8–28.3)	11.5 (5.0–27.3)	0.639
CRP, mg/L	1.2 (0.6–3.3)	1.3 (0.4–2.1)	0.976
Fasting glucose, mg/dL	98.5 (93.0–114.3)	93.5 (83.5–106)	0.079
BUN, mg/dL	12.0 (10.1–16.6)	12.0 (9.9–15.1)	0.663
Cr, mg/dL	0.7 (0.6–0.8)	0.7 (0.6–0.8)	0.054
Total protein, g/dL	7.1 (6.8–7.4)	7.1 (6.7–7.4)	0.472
Albumin, g/dL	4.4 (4.3–4.5)	4.4 (4.0–4.7)	0.813

Values are presented as median (interquartile range) for continuous variables and n (%) for categorical variables.
WBC: white blood cell count; Hb: haemoglobin; Plt: platelet count; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; BUN: blood urea nitrogen; Cr: creatinine.

symptoms. Notably, more than half of the patients showed electrophysiological evidence of axonal neuropathy; however, no significant differences in demographic characteristics, cutaneous manifestations, or laboratory findings were observed between patients with and without VPN on NCS. This indicates that clinical features alone are insufficient to predict nerve involvement. These findings underscore the pivotal role of NCS in evaluating peripheral nerve involvement in CA. NCS provides an objective and sensitive tool for detecting early or subclinical neuropathy, allows accurate characterisation of neuropathic patterns, and can help differentiate vasculitic neuropathy from other causes of peripheral nerve dysfunction, such as metabolic or compressive aetiologies (24, 25). Becker *et al*. showed that electrodiagnostic studies could identify cases that may have been missed by histology alone (26). Moreover, electrophysiological abnormalities may serve as surrogate markers of active vascular inflammation, guiding the timely initiation or escalation of immunosuppressive therapy (24).

Although CA generally has a more benign clinical course than systemic PAN, the presence of skin ulcers or sensory neuropathies at diagnosis has been suggested as a predictor of poor prognosis (27, 28). Nerve involvement can lead to pain, sensory deficits, motor impairment, and a decreased quality of life (29, 30). Therefore, patients with CA who present with these features may require more intensive treatment, including immunosuppressive therapy (2, 27). The early detection of VPN is clinically important because timely and appropriate interventions can help prevent irreversible neurological damage and improve long-term functional outcomes (8, 31). In this context, the identification of VPN through NCS may serve not only as an objective diagnostic tool but also as an early indicator of potential systemic disease extension, warranting closer monitoring and comprehensive management (13, 32). Therefore, when nerve biopsy is not feasible, early and proactive use of NCS in patients with CA may improve the prompt recognition of neuropathic involvement, enabling individualised management strategies, and potentially preventing irreversible neurological sequelae.

This study has several strengths. First, NCS was performed in all patients with provisional CA, enabling a more reliable estimation of the prevalence of VPN. Second, we propose a practical provisional definition for CA with suspected neuropathic involvement, which may be useful in clinical settings where nerve biopsy is of limited practicality and could enable earlier recognition and appropriate clinical management of neuropathic involvement in skin-limited vasculitis. This study also has several limitations. First, this retrospective single-centre study with a relatively small sample size may limit the generalisability of our findings. Second, only a small number of patients underwent nerve biopsy, which prevented direct histopathological confirmation in most cases. Third, patients were preselected based on the presence of neuropathic symptoms, which could introduce referral and selection bias, potentially leading to an overestimation of the prevalence of VPN. Despite these limitations, our study suggests that VPN should not be considered uncommon in CA. Furthermore, our findings indicate the need for early neurological evaluation and appropriate management.

In conclusion, NCS provides a reliable method for detecting VPN when a nerve biopsy is challenging. Clinicians should actively assess neuropathic symptoms in patients with skin-limited vasculitis. Future prospective studies are needed to correlate the NCS findings with treatment response and long-term prognosis.

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