Clinical Manifestation and Treatment Responsiveness in Vasculitic Neuropathy Patients in Neurological Institute of Thailand

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Abstract

Background: Vasculitic neuropathy (VN) results from inflammation of blood vessels supplying peripheral nerves, causing ischemic nerve injury. It manifests as systemic vasculitic neuropathy (SVN) or non-systemic vasculitic neuropathy (NSVN). While SVN treatment protocols are established, data on clinical characteristics and treatment outcomes of NSVN, particularly in Thai population, remain limited.

Objective: To evaluate clinical manifestations, pathological findings, and treatment responsiveness in patients with confirmed VN at the Neurological Institute of Thailand, and identify factors associated with treatment response.

Methods: A retrospective cohort study of patients with pathologically confirmed VN diagnosed at the Neurological Institute of Thailand between June 2015, and May 2025. Clinical data, electrodiagnostic results, nerve biopsy findings, and treatment regimens were analyzed. Treatment responsiveness was assessed in patients followed over 24 months, based on improvements in pain, sensory deficits, motor weakness, relapse rates.

Results: Thirty-five patients were enrolled (65.7% female, median age 58.0 years). NSVN was present in 54.3%. Sensory loss and motor deficits were universal, pain occurred in 60%. Electrodiagnostic studies revealed axonopathy with predominant lower extremity involvement. Pathologically, 60% had definite VN. Treatment groups included corticosteroid alone (45.7%), corticosteroid plus cyclophosphamide (42.9%), and corticosteroid plus rituximab (11.4%). Overall good outcome was

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achieved in 85.7% with no significant difference between groups (p=0.575). Relapse rate was 8.6%. Pathological features did not differ significantly between treatment groups.

Conclusion: VN in Thai tertiary care patients demonstrates favorable outcomes regardless of immunosuppressive regimen. Rituximab shows promise with a favorable relapse profile. Pathological features did not correlate with severity or prognosis.

Keywords: Vasculitic neuropathy, Vasculitis, Neuropathy, Non-systemic vasculitic neuropathy, Treatment

Introduction

Vasculitic neuropathy (VN) result from inflammation of the vasa nervorum, causing ischemic nerve damage[1,2]. Patients typically present with acute to subacute dysesthesia, and motor deficits in an asymmetric distribution, often manifesting as mononeuritis multiplex. Clinical severity varies; systemic organ involvement generally correlates with higher morbidity and mortality[1]. Without treatment, mortality for primary systemic vasculitis can reach 90%[1,2].

VN is classified into systemic vasculitic neuropathy (SVN), associated with multi-organ involvement, and non-systemic vasculitic neuropathy (NSVN), isolated to peripheral nerves[1,2]. SVN treatment is based on the underlying systemic cause, primary systemic vasculitis guided by established protocols for systemic vasculitis using immunosuppressive agents. Evidence for NSVN management remains limited, based primarily on observational studies[1]. The Peripheral Nerve Society Guidelines 2010 recommend treating NSVN similarly to systemic vasculitis, with corticosteroids as first-line therapy and additional immunosuppressants for progressive or severe disease[3-7].

Studies have demonstrated superior outcomes with corticosteroids plus immunosuppressive agents compared to corticosteroids alone. Patients receiving corticosteroids plus cyclophosphamide achieved good treatment response and complete remission[5,8-10]. Rituximab has shown non-inferiority to cyclophosphamide for induction therapy in ANCA-associated vasculitis[11] and may be considered for patients with contraindications to cyclophosphamide, cryoglobulinemic vasculitis, or active glomerulonephritis[12].

For maintenance therapy, prednisolone is gradually tapered over 6-18 months in combination with other immunosuppressive agents, including azathioprine, methotrexate, mycophenolate mofetil, or rituximab. Total maintenance duration should be at least 18-24 months, as rapid tapering is associated with increased relapse rates[12].

However, treatment regimen vary in clinical practice, particularly regarding drug selection. Furthermore, data regarding clinical characteristics and treatment outcomes of VN in the Thai population remain limited.

This study aims to describe clinical manifestations and pathological findings and treatment outcomes of VN in a Thai tertiary center, and identify factors influencing outcomes and relapse rates.

Materials and Methods

Study Design: This retrospective study was conducted at the Neurological Institute of Thailand, including VN patients diagnosed and treated between June 1, 2015, and May 31, 2025. All patients were performed nerve biopsy and

diagnosed according to the Peripheral Nerve Society Guideline 2010[3] and Thai guideline for immunomodulating therapy in immune mediated neuromuscular disease[12]. Diagnosis requires clinical assessment, electrodiagnostic studies demonstrating asymmetric axonal neuropathy, and nerve biopsy.

Clinically probable VN required

1) Nerve or muscle biopsy consistent with VN or meeting criteria for systemic vasculitis from other organs.

2) Clinical features including sensory-motor or sensory deficit, asymmetric or multifocal distribution, lower limb and distal predominance, presence of pain, or recurrent episodes.

3) Electrodiagnostic findings of sensory-motor or sensory axonal neuropathy.

4) Absence of red flags.

Inclusion criteria:

1. Adults aged ≥18 years-old

2. Diagnosed with Vasculitic neuropathy according to the Thai guideline for immunomo dulating therapy in immune mediated neuromuscular disorders[12], the Peripheral Nerve Society Guideline 2010[3]

3. All patients were performed nerve biopsy proven vasculitic neuropathy

Exclusion criteria:

1. Incomplete medical records

2. Patients with alternative diagnosed mimic vasculitis neuropathy

Study Protocol

Data Collection:

- Baseline characteristics: age, gender, disease duration, underlying disease, and clinical manifestation including sensory symptoms, muscle strength assessed by Medical Research Council (MRC) grading, and other symptoms.

  Medical Research Council (MRC) Grading:
  - Grade 0: No contraction.
  - Grade 1: Trace contraction, no movement.
  - Grade 2: Movement with gravity eliminated.
  - Grade 3: Movement against gravity but not resistance.
  - Grade 4: Movement against some resistance.
  - Grade 5: Normal strength.

  MRC sum score; assessment of muscle action of each limb (0-60 points) evaluating shoulder abduction, elbow flexion, wrist extension, hip flexion, knee extension, and ankle dorsiflexion.
- Laboratory data included serum ESR, CRP, β2-microglobulin, ANA, cryoglobulins, Anti Ro, Anti La, C-ANCA, P-ANCA, Rheumatoid factor, Anti HIV, Anti HCV, HbsAg, electrodiasnostic studies, and nerve biopsy results were recorded.
- Classification of vasculitic neuropathy: Systemic vasculitic neuropathy (SVN), and Non-systemic vasculitic neuropathy (NSVN).

  Systemic vasculitic neuropathy (SVN) required at least one abnormal systemic marker (ESR, CRP, ANCA, complement, RF, β2-microglobulin) plus evidence of extraneural organ involvement or associated conditions (connective tissue disease, infection, malignancy, cryoglobulins).

  Nonsystemic vasculitic neuropathy (NSVN) was diagnosed when patients met clinically probable or pathologically definite criteria but without evidence of systemic involvement.

- Treatment regimens included corticosteroids (CS) alone, CS combined with Cyclophos phamide (CS+CY), and CS combined with Rituximab (CS+RTX).
- Outcomes were assessed at 6 months and at last follow-up, evaluating MRC score changes, pain and sensory loss improvement, and refractory or relapse events.

Statistical Analysis

Data was analyzed using SPSS for Windows version 17.0. Continuous variables were expressed as median with interquartile range (IQR). Categorical variables were expressed as frequencies and percentages. Group comparisons used Chi-square test or Fisher's exact test for categorical data and Kruskal-Wallis test or Mann-Whitney U test for continuous data. Statistical significance was set at $p < 0.05$.

This study was approved by the Institutional Review Board of Neurological Institute of Thailand (study number: 68066) on September 11th 2025.

Results

Baseline Characteristics

A total of 35 patients with vasculitic neuropathy were included (Table 1). There was female predominance (65.7%) with median age at onset of 58.0 years (IQR 45.5-65.0). Median disease duration from symptom onset to diagnosis was 8.0 weeks (IQR 4.5-22.0). NSVN (54.3%) was slightly more common than SVN (45.7%). SVN etiologies included microscopic polyangiitis (MPA) 14.3%, eosinophilic granulomatosis with polyangiitis (EGPA) 8.6%, systemic lupus erythematosus (SLE) 8.6%, systemic sclerosis (SSc) 2.9%, rheumatoid arthritis (RA) 2.9%, cryoglobulins 11.4%, HBV infection 2.9%, and HIV infection 2.9%.

Diabetes mellitus was present in 11.4%. The most common presentation was asymmetrical sensorimotor polyneuropathy (71.4%), followed by multiple mononeuropathy (17.1%), and distal symmetric polyneuropathy (11.4%). All patients had sensory and motor deficits. Sensory loss was reported in 100%, while pain was reported in 60.0%. Median baseline MRC sum score was 52.0 (IQR 47.5–55.5). Constitutional symptoms (fever, weight loss, myalgia, or anorexia) were presented in 54.3%.

Laboratory and Electrodiagnostic Findings

Laboratory investigations revealed elevated ESR in 66.7% and elevated CRP in 64.0% of tested patients. Positive autoantibodies included rhematoid factor (64.7%), anti-Ro (12.5%), P-ANCA (30.0%), and cryoglobulins (36.4%) of tested patients. Hypereosinophilia was recorded in 20.0%, and anemia in 40.0%.

Nerve conduction studies demonstrated an axonopathy pattern in all patients. Lower extremity nerves were predominantly affected, with peroneal nerve involvement in 91.4%, sural nerve in 85.7%, and tibial nerve in 82.9%. Upper extremity involvement included median nerve in 74.3%, ulnar nerve in 57.1%, and radial nerve in 14.3%.

Table 1: The demographic and clinical characteristic

Variable	Total (n=35)	Treatment regimens			p-value
		CS alone (n=16)	CS + CY (n=15)	CS + RTX (n=4)	
Sex, n (%)					0.042
Male	12 (34.3%)	9 (56.3%)	2 (13.3%)	1 (25.0%)	
Female	23 (65.7%)	7 (43.8%)	13 (86.7%)	3 (75.0%)	
Age at onset, years; median (IQR 25, 75)	58.0 (45.5, 65)	58.0 (37.3, 67.5)	58.0 (48.0, 61.0)	65.0 (29.8, 71.0)	0.767
Disease duration, weeks; median (IQR 25, 75)	8.0 (4.5, 22.0)	12.0 (6.0, 27.0)	12.0 (3.0, 24.0)	3.0 (1.3, 7.0)	0.080
Constitutional symptoms, n (%)	19 (54.3%)	7 (43.8%)	10 (66.7%)	2 (50.0%)	0.433
Fever	8 (22.9%)	3 (18.8%)	3 (20.0%)	2 (50.0%)	0.390
Anorexia	4 (11.4%)	1 (6.3%)	2 (13.3%)	1 (25.0%)	0.322
Weight loss	11 (31.4%)	4 (25.0%)	6 (40.0%)	1 (25.0%)	0.769
Myalgia	5 (14.3%)	1 (6.3%)	4 (26.7%)	0 (0.0%)	0.218
Classification, n (%)					0.214
- Systemic VN	16 (45.7%)	5 (31.2%)	8 (53.3%)	3 (75.0%)	
MPA	5 (14.3%)	1 (6.3%)	3 (20.0%)	1 (25.0%)	
EGPA	3 (8.6%)	1 (6.3%)	1 (6.7%)	1 (25.0%)	
CTD	5 (14.3%)	0 (0.0%)	4 (26.7%)	1 (25.0%)	
Cryoglobulins	4 (11.4%)	2 (12.5%)	2 (13.3%)	0 (0.0%)	
HBV infection	1 (2.9%)	1 (6.3%)	0 (0.0%)	0 (0.0%)	
HIV infection	1 (2.9%)	1 (6.3%)	0 (0.0%)	0 (0.0%)	
- Non-systemic VN	19 (54.3%)	11 (68.8%)	7 (46.7%)	1 (25.0%)	
Clinical presentation, n (%)					
Sensory symptoms	35 (100.0%)	16 (100.0%)	15 (100.0%)	4 (100.0%)	-
Pain	21 (60.0%)	10 (62.5%)	8 (53.3%)	3 (75.0%)	0.798
Paresthesia	18 (51.4%)	9 (56.3%)	7 (46.7%)	2 (50.0%)	0.649
Sensory loss	35 (100.0%)	16 (100.0%)	15 (100.0%)	4 (100.0%)	-
Motor symptoms	35 (100.0%)	16 (100.0%)	15 (100.0%)	4 (100.0%)	-
Pattern of distribution					
Asymmetric polyneuropathy	25 (71.4%)	10 (62.5%)	12 (80.0%)	3 (75.0%)	
Multiple mononeuropathy	6 (17.1%)	3 (18.8%)	2 (13.3%)	1 (25.0%)	
Distal symmetric polyneuropathy	4 (11.4%)	3 (18.8%)	1 (6.7%)	0 (0.0%)	

Pathological Findings

Sural nerve biopsy was performed in all cases (Table 2), revealed definite VN in 60.0%, probable in 25.7%, possible in 5.7%, and inflammatory neuropathy (not reach criteria diagnosis) in 8.6%. Epineurial perivascular inflammatory collection was small in 54.3%, moderate in 34.3%, and large in 31.4%. Small vessel involvement in epineurium was present in 51.4%, and large arteriole involvement in 45.7%. Vessel wall disruption was present in 54.3%. Severe nerve fiber density loss occured in 60.0% with generalized (62.9%) or multifocal pattern (37.1%). Active axonal degeneration was present (61.8%), and late stage (8.8%). Immunohistochemistry

demonstrated CD3 positivity in 97.0%, CD20 positivity in 93.9%, CD3 positivity more predominant than CD20 positivity in 39.4%, CD3 and CD20 positivity same proportion in 54.8%, and polyclonal pattern (both CD3 and CD20 positivity) in 93.9%. Pathological features did not differ significantly between treatment groups (p>0.05).

Table 2 : Sural nerve biopsy

Pathological findings	Total (n=35)	Treatment regimens			p-value
		CS alone (n=16)	CS + CY (n=15)	CS + RTX (n=4)	
Diagnostic certainty n/N (%)					0.499
Definite VN	21/35 (60.0%)	9/16 (56.3%)	9/15 (60.0%)	3/4 (75.0%)	
Probable VN	9/35 (25.7%)	4/16 (25.0%)	5/15 (33.3%)	0/4 (0.0%)	
Possible VN	2/35 (5.7%)	2/16 (12.5%)	0/15 (0.0%)	0/4 (0.0%)	
Inflammatory neuropathy	3/35 (8.6%)	1/16 (6.2%)	1/15 (6.7%)	1/4 (25.0%)	
Size of perivascular inflammatory collection in epineurium, n (%)					
Small	19 (54.3%)	9 (56.3%)	10 (66.7%)	0 (0.0%)	0.064
Moderate	12 (34.3%)	6 (37.5%)	4 (26.7%)	2 (50.0%)	0.686
Large	11 (31.4%)	7 (43.8%)	3 (20.0%)	1 (25.0%)	0.355
Vessel size involvement in epineurium, n (%)					
Large arteriole	16 (45.7%)	7 (43.8%)	7 (46.7%)	2 (50.0%)	1.000
Small vessel	19 (51.4%)	9 (56.3%)	8 (53.3%)	1 (25.0%)	0.644
Large and small vessel	3 (8.6%)	2 (12.5%)	1 (6.7%)	0 (0.0%)	-
Distribution of inflammation in endoneurium, n/N (%)					
Collection	3/34 (8.8%)	2/16 (12.5%)	1/14 (7.1%)	0/4 (0.0%)	1.000
Scatter	25/34 (73.5%)	11/16 (68.8%)	12/14 (85.7%)	2/4 (50.0%)	0.250
Disruption of vessel wall (Seperation of smooth muscle actin stain), n/N (%)	19/35 (54.3%)	9/16 (56.3%)	7/15 (46.7%)	3/4 (75.0%)	0.638
Neovascularization	18/35 (51.4%)	8/16 (50.0%)	7/15 (46.7%)	3/4 (75.0%)	0.717
Hemosiderin deposits	7/35 (20.0%)	0/16 (0.0%)	4/15 (26.7%)	3/4 (75.0%)	0.001
Fibrinoid necrosis	4/35 (11.4%)	1/16 (6.3%)	2/15 (13.3%)	1/4 (25.0%)	0.322
Severity of nerve fiber density loss, n/N (%)					
Mild	4/35 (11.4%)	0/16 (0.0%)	4/15 (26.7%)	0/4 (0.0%)	0.107
Moderate	10/35 (28.6%)	6/16 (37.5%)	2/15 (13.3%)	2/4 (50.0%)	
Severe	21/35 (60.0%)	10/16 (62.5%)	9/15 (60.0%)	2/4 (50.0%)	
Pattern of fiber loss, n/N (%)					
Multifocal	13/35 (37.1%)	4/16 (25.0%)	6/15 (40.0%)	3/4 (75.0%)	0.180
Generalize	22/35 (62.9%)	12/16 (75.0%)	9/15 (60.0%)	1/4 (25.0%)	0.180
Axonal degeneration, n/N (%)					
Early (active)	21/34 (61.8%)	10/16 (62.5%)	8/14 (57.1%)	3/4 (75.0%)	1.000
Late	3/34 (8.8%)	2/16 (12.5%)	1/14 (7.1%)	0/4 (0.0%)	1.000
Immunohistochemistry stains, n/N (%)					
CD3 positive	32/33 (97.0%)	15/15 (100.0%)	13/14 (92.9%)	4/4 (100.0%)	0.545
CD20 positive	31/33 (93.9%)	14/15 (93.3%)	13/14 (92.9%)	4/4 (100.0%)	1.000

Table 3 : Outcomes

Response	Total	Treatment regimens			p-value
		CS alone (n=16)	CS + CY (n=15)	CS + RTX (n=4)	
Composite MRC score, point; median (IQR 25,75)					
At baseline	52.0 (47.5, 55.5)	52.5 (48.8, 57.0)	52.0 (47.0, 55.0)	48.5 (44.8, 55.3)	0.596
Changed at 6 months	3.0 (3.0, 6.0)	5.0 (2.2, 8.5)	3.0 (2.7, 5.2)	4.0 (2.0, -)	0.559
Changed at last follow up	6.0 (3.5, 8.5)	6.0 (2.0, 8.0)	5.0 (4.5, 10.5)	6.0 (3.0, -)	0.760
Pain at 6 mo, n/N (%)					
Improve	17/21 (81.0%)	8/8 (100.0%)	6/10 (60.0%)	3/3(100%)	0.384
Pain at last follow up, n/N (%)					
Improve	19/19 (100%)	7/7 (100.0%)	9/9 (100.0%)	3/3 (100.0%)	-
Sensory loss at 6 mo, n/N (%)					
Improve	17/21 (81.0%)	6/8 (75.0%)	9/10 (90.0%)	2/3 (66.7%)	0.449
Sensory loss at last follow up, n/N (%)					
Improve	19/19 (100%)	7/7 (100.0%)	9/9 (100.0%)	3/3 (100.0%)	-

Table 4 : Relapse and Refractory events

Response, n (%)	Total	Treatment regimens			p-value
		CS alone (n=16)	CS + CY (n=15)	CS + RTX (n=4)	
Response to treatment	30 (85.7%)	14 (87.5%)	12 (80.0%)	4 (100.0%)	0.575
Refractory	2 (5.7%)	0 (0.0%)	2 (13.3%)	0 (0.0%)	0.243
Relapse	3 (8.6%)	2 (12.5%)	1 (6.7%)	0 (0.0%)	0.684

Treatment and Outcomes

Patients received CS alone (n=16), CS+CY (n=15), or CS+RTX (n=4), based on clinical judgment and disease severity. Most NSVN patients received CS alone due to lower disease severity. Baseline characteristics were similar across treatment groups except for sex distribution, with more females in CS+CY and CS+RTX groups (p=0.042). Patients receiving rituximab had shorter duration before treatment initiation (median 3.0 vs 12.0 weeks) and higher proportion of SVN (75.0% vs 31.2-53.3%).

At 6-month follow-up, median MRC sum score improvement was 5.0 (CS alone), 3.0 (CS+CY), and 4.0 (CS+RTX) (p=0.559). Pain improvement occurred in 100% of CS alone and CS+RTX groups compared to 60.0% in CS+CY group (p=0.384). Sensory improvement was similar across groups (66.7-90.0%, p=0.449). At last follow-up, all groups achieved 100% improvement in pain and sensory symptoms, with no significant MRC differences (p=0.760).

Overall treatment response was achieved in 85.7%. Refractory disease (5.7%) occurred only in CS+CY. Relapse rate was 8.6%, two patients in CS alone (at 3 and 36 months) and one in CS+CY (at 30 months). The CS+RTX group had no relapses or refractory cases. Good outcome rates did not differ significantly between treatment groups.

In subgroup analysis, VN classification did not affect good outcome rates across treatment regimens (all p=1.000). However, within the CS alone group, NSVN showed significantly better MRC recovery than SVN at 6 months (60 vs 57, p=0.031) and last follow-up (60 vs 59, p=0.036).

Adverse events occurred only in CS+CY group (3 pateints), pneumonia, sepsis with urinary tract infection, herpes zoster infection.

Predictors of Outcomes

Univariate analysis was performed to identify factors associated with treatment outcome. The clinical, laboratory testings, nerve conduction study and pathological findings did not correlate with treatment outcome. Notably, disease severity (MRC sum score) did not correlate with pathological severity.

Discussion

This study demonstrates the clinical sprectrum and treatment outcomes in 35 VN patients at Neurological Institute of Thailand, with an overall good outcome rate of 85.7%. Demographics, with female predominance and median age in the sixth decade, are similar to previous studies[4,5]. NSVN was slightly more common than SVN (54.3% vs 45.7%). However, most SVN patients were classified by secondary from systemic disease and laboratory abnormalities without significant extraneural organ involvement. Previous cohort reported 6-37% of NSVN patients developing systemic manifestations over 5-6 years[5,13], which was not observed in our study, possibly due to small sample size and loss to follow-up.

All patients presented with sensorimotor deficits, predominantly as asymmetrical sensorimotor polyneuropathy (74.3%). While pain is classically a hallmark of VN, only 60% of our patients reported, comparable to previous NSVN studies (66-80%)[4,13,14]. This might be under-reported by paresthesia symptoms (51.4%) or NSVN more common in our study.

Electrodiagnostic findings showed axonopathy pattern with lower extremity predominance, and asymmetric distribution is typical for vasculitic neuropathy. The peroneal nerve was most frequently affected (91.4%), followed by sural and tibial nerves, are similar to previous studies[4,5,13,14].

Treatment response was comparable across the three regimens, although selection bias was present. The CS alone group was predominantly comprised NSVN patients, who demonstrated better MRC recovery despite similar good outcome rates compared to SVN (91% vs 80%, p=1.000), possibly due to higher baseline MRC in NSVN (55 vs 52). The CS+RTX group, although small, demonstrated no refractory disease or relapse, potentially superior to CS alone and CS+CY group, however, this group was biased toward ANCA-positive patients for whom rituximab is recommended. Evidence for rituximab efficacy in ANCA-negative vasculitic neuropathy remains limited. Regarding safety, both CS+RTX and CS alone group showed favorable profiles with no serious infection observed in either group.

Nerve biopsy remains essential for establishing the diagnosis of vasculitic neuropathy. However, pathological findings did not correlate with clinical severity or prognosis, possibly due to short duration to treatment in our study.

This study has several limitations. First, the retrospective design and small sample size, particularly in the rituximab group. Second, non-randomized treatment selection, leading

potential selection bias. Third, the treatment regimens in our study categorized by induction therapy, but maintenance therapy may have also affected or predicted long term outcomes. Fourth, missing data in some laboratory variables or in loss follow up patients may have affected the analysis.

Conclusion

Vasculitic neuropathy in Thai tertiary care setting demonstrates favorable treatment outcomes with an overall good outcome rate of 85.7%, regardless of the immunosuppressive regimen used, Rituximab shows promise as an effective therapy with no relapse observed. Pathological features did not correlate with clinical severity or prognosis. These findings support individualized treatment approaches based on disease severity. Larger prospective studies are needed to further stratify risk factors and optimize treatment protocols.

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