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Clinical characteristics and prognosis of neuralgic amyotrophy of the brachial plexus: A retrospective study in the Thai population

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Abstract

Background & Objective: Neuralgic amyotrophy, or Parsonage-Turner syndrome, is an underrecognized brachial plexopathy characterized by acute shoulder pain followed by weakness and atrophy. Limited data exist on its clinical spectrum in Thailand. The objective of this study is to describe the clinical features, electrophysiological and MRI findings, and disease course of neuralgic amyotrophy in a Thai cohort. *Methods:* We retrospectively reviewed 27 adult patients diagnosed with neuralgic amyotrophy of brachial plexus at the Neurological Institute of Thailand (2017–2024). Data included clinical presentation, nerve conduction studies, MRI findings, Medical Research Council (MRC) strength scores, and Quick Disabilities of the Arm, Shoulder and Hand (QuickDASH) functional outcomes. *Results:* The median patient age was 54 years; most were male. All presented with acute pain followed by focal weakness and atrophy, most often affecting shoulder abduction, external rotation, and elbow flexion. Triggers were identified in 25.9%, most commonly post-vaccination. Upper trunk involvement predominated. Radial nerve motor amplitudes were reduced in 51.9%, but sensory abnormalities were infrequent (18.5%). MRI showed T2 hyperintensity in 72.7% and gadolinium enhancement in 40.9%. Corticosteroids were administered to 74% of patients. Early treatment (≤30 days) was associated with significantly better QuickDASH ($p = 0.0284$) and MRC ($p = 0.0172$) scores. Most patients achieved meaningful recovery within 6 months, with peak motor improvement at second follow-up.
Conclusion: This study details clinical and MRI features of neuralgic amyotrophy in Thai patients, emphasizing early diagnosis and corticosteroid treatment to improve functional outcomes.

Keywords: Neuralgic amyotrophy, Parsonage-Turner syndrome, brachial plexitis

INTRODUCTION

Neuralgic amyotrophy, also known as Parsonage-Turner syndrome or brachial plexus neuritis, is a significant cause of brachial plexopathy in adults. It is characterized by an acute onset of severe shoulder pain, which is subsequently followed by muscle atrophy and paralysis of the affected region.[1]

Originally, the incidence of the disease was estimated at approximately 2–3 per 100,000 individuals per year.[2] However, recent studies suggest that this figure is underestimated, as under-recognition and misdiagnosis are common.[2] The clinical manifestations of neuralgic amyotrophy often overlap with other conditions, further complicating accurate diagnosis.

Over half of neuralgic amyotrophy cases are preceded by triggering events like infections, vaccinations, or pregnancy. Mechanical stress from activities such as sports or heavy labor may weaken the blood-nerve barrier, exposing nerves to immune attack.[1,2] This is supported by observations of hourglass-like nerve constrictions, likely from nerve twisting. Genetic factors, including SEPT9 mutations, have also been linked to recurrent episodes.[1,3]

Diagnosing neuralgic amyotrophy is challenging due to its variable presentation and primarily depends on clinical evaluation. Electrophysiological tests and imaging mainly help exclude other conditions, though nerve conduction studies and EMG have timing limitations. Literature reports sensory nerve conduction abnormalities in 30–45% of patients.[4,6] MRI is valuable for detecting nerve inflammation and ruling out alternatives like radiculopathy.[7,8] Regarding treatment, no specific therapy has been established for neuralgic amyotrophy. Some

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evidence supports the use of corticosteroids during the acute phase, as they may alleviate pain and facilitate more rapid improvement within the first month compared to no treatment.[1,9,10] Additionally, certain studies suggest that combining intravenous immunoglobulin with corticosteroids may further reduce the duration of symptoms.[11,12] However, most data indicate that patients beyond the acute phase (typically the first two months) are less likely to benefit from immunosuppressive therapy and are managed primarily with symptomatic treatment and rehabilitation.[4]

While the prognosis for neuralgic amyotrophy was once considered favorable for most patients, recent studies have challenged this view. For example, one study reported that fewer than 10% of patients achieve full recovery within three years, and approximately 20% remain unable to return to work.[2,5]

Although well-known, data on the epidemiology, clinical features, and natural history of neuralgic amyotrophy in Asian populations remain limited. This study aims to characterize the disease in Thai and broader Asian populations to enhance understanding and patient care.

METHODS

Patients and study design

The authors declare that all data were obtained from the Neurological Institute of Thailand. This retrospective study included 27 Thai patients diagnosed with brachial plexus neuralgic amyotrophy who attended the outpatient clinic between January 2017 and December 2024.

Inclusion criteria were age ≥18 years and a clinical diagnosis of neuralgic amyotrophy, based on acute or subacute onset of unilateral or asymmetrical bilateral upper limb weakness with pain, sensory symptoms, and muscle atrophy. Diagnosis was confirmed by characteristic findings on electrophysiologic studies and brachial plexus MRI. Patients with alternative diagnoses (e.g., cervical radiculopathy, malignancy) or incomplete records were excluded. Ethical approval was obtained (No. 68009).

Baseline data included demographics, comorbidities (e.g., diabetes), and possible triggers (e.g., vaccination, infection, surgery, exertion). Clinical data covered symptom laterality, time to presentation, associated symptoms, exam findings, and MRI results. Pain severity was categorized by the number of analgesics required: mild (none), moderate (one), and severe (two or more), including acetaminophen, NSAIDs, and gabapentinoids.

Electrodiagnostic testing involved nerve conduction and needle EMG. Asymmetric reductions in amplitude or abnormal EMG (e.g., denervation, reduced recruitment) were documented using contralateral comparison or normative data for bilateral cases.

Treatment data included corticosteroid use and timing from symptom onset, a key variable in the study.

Outcomes assessment

Muscle strength was evaluated using the Medical Research Council (MRC) Manual Muscle Testing Scale, applied to ten commonly examined upper limb muscles. Functional status was assessed using the QuickDASH (Quick Disabilities of the Arm, Shoulder and Hand) questionnaire, which served as the primary outcome measure of this study.

Changes in both MRC scores and QuickDASH scores were assessed longitudinally. Measurements were obtained at baseline, at 6 weeks (±2 weeks), and at the final follow-up at 12 weeks (±4 weeks). These intervals were selected to capture both early and intermediate recovery phases and allow for standardized comparison against baseline values.

The Medical Research Council (MRC) scale, widely used for its simplicity and reliability, rates muscle strength from 0 to 5. A total MRC sum score (range 0–50) was calculated from ten upper limb muscles: shoulder abduction and external rotation, elbow flexion and extension, wrist flexion and extension, finger flexion and extension, abductor pollicis brevis, and abductor digiti minimi.[13,14]

The Quick Disabilities of the Arm, Shoulder, and Hand (QuickDASH) is a validated 11-item questionnaire assessing upper limb functional impairment. It evaluates daily activity limitations, pain, and symptom burden, with each item scored from 1 (no difficulty) to 5 (unable to do). Scores range from 0 (no disability) to 100 (most severe disability).[15–17]

Subgroup analysis: Early vs. late steroid treatment

Previous studies investigating the role of corticosteroids in the acute phase of neuralgic amyotrophy have suggested that treatment within four weeks of symptom onset may positively influence clinical outcomes.[9,18] Based on this, our study categorized patients into two groups: an early treatment group, defined as those who received corticosteroids within 30 days

of symptom onset, and a late treatment group, defined as those who initiated treatment more than 30 days after symptom onset.

A prespecified subgroup analysis was performed to evaluate the impact of corticosteroid initiation timing on upper limb functional recovery, using both the QuickDASH and MRC sum scores as outcome measures. Functional and strength assessments were performed at three standardized time points for each participant: baseline, 6 weeks (±2 weeks), and 12 weeks (±4 weeks). This analysis aimed to determine whether earlier intervention with corticosteroids was associated with superior outcomes in terms of both function and muscle strength, compared to delayed treatment.

Statistical analysis

Quantitative data were reported as medians with interquartile ranges (IQR) due to non-normal distribution. Functional outcomes (QuickDASH) and muscle strength (MRC scale) were assessed at baseline, 6 weeks (±2), and 12 weeks (±4). The Friedman test was used to analyze within-subject changes over time, and Kendall's W measured consistency across time points.

To assess the effect of corticosteroid timing on recovery, linear mixed-effects models accounted for repeated measures and individual variability. Models included treatment group (early vs. late steroids), time to strength recovery, and baseline scores. Model fit was evaluated using log likelihood and Likelihood Ratio Tests. The best-fitting model balanced statistical performance and clinical relevance. A Wald test compared treatment effects between groups.

RESULTS

Demographic data and baseline characteristics

A total of 27 patient records were analyzed from a review of 42 medical charts. Among these, 15 patients initially diagnosed with neuralgic amyotrophy of the brachial plexus were later identified to have alternative etiologies for their symptoms. The most common alternative diagnosis was co-existing cervical radiculopathy. Other identified causes included active herpes zoster infection, underlying malignancy, and a history of radiation therapy.

Demographic characteristics are summarized in Table 1. Majority of patients were male, with a median age of 54 years (IQR: 45 - 62). Diabetes mellitus was present in 4 patients (14.8%). Preceding events were identified in 7 patients (25.9%), with prior vaccination—specifically COVID-19 vaccines—being the most common (11.1%), which all cases received viral vector vaccines.

All patients reported antecedent pain prior to the onset of motor weakness and presented with muscle atrophy. Left-sided brachial plexus involvement was more common, and Proximal muscle weakness more predominant than distal muscles. However, 2 patients presented with bilateral asymmetrical plexopathy. The median disease duration prior to diagnosis was 30 days (IQR: 13 - 90).

In terms of motor weakness patterns, muscle groups innervated by the upper trunk of brachial plexus —specifically those responsible for shoulder abduction, shoulder external rotation, and elbow flexion—were most commonly affected, observed in 80% of patients. Muscle groups involved in elbow extension and wrist extension, typically innervated by the middle trunk and posterior cord of brachial plexus, were impaired in around 60 % of cases. Additionally, reduced motor strength in the fingers and intrinsic hand muscles was noted in around 50 % of patients (Supplementary Table 1).

Nerve conduction studies frequently revealed reduced motor nerve amplitudes, with the radial nerve most affected (51.9 %), follow by ulnar nerve (33.3 %), and median nerve (29.6 %). Sensory nerve abnormalities were found in superficial radial nerve (18.5 %) and lateral antebrachial cutaneous nerves (18.5 %). EMG study was performed in the deltoid, biceps brachii, triceps brachii, first dorsal interosseous (FDI), and abductor pollicis brevis (APB) muscles. EMG showed reduced recruitment in 50 – 100 % of muscles tested. Spontaneous activity such as fibrillation and positive sharp wave was found in 29.4 – 100 % of muscles tested (Table 2).

Most patients underwent brachial plexus MRI to confirm diagnosis or exclude alternative causes, particularly cervical radiculopathy. The most frequent MRI finding was increased T2 signal intensity, observed in 16 of 22 patients (72.7%). Contrast enhancement was seen in 40% of cases. Notably, no patients demonstrated the "hourglass" constriction pattern commonly described in neuralgic amyotrophy.

At our institution, corticosteroids were frequently administered to patients with neuralgic amyotrophy. The initial regimen typically involved intravenous pulse methylprednisolone administered over five days. This was followed

Table 1: Patients' characteristic including demographic data, associated conditions, and clinical characteristics

Demographic data	Number of patients (N=27)
Age (years, median, IQR)	54 (45 – 62)
Sex (male: female)	15: 12
Weight (kg, median, IQR)	62 (53 – 80)
Height (m, median, IQR)	1.66 (1.59 – 1.73)
Disease duration prior to diagnosis (days, median, IQR)	30 (13 – 90)
Associated conditions	
Diabetes mellitus (%)	4 (14.8)
Identifiable preceding events (%)	7 (25.9)
– Vaccination (%)	3 (11.1)
– Preceding infection (%)	1 (3.7)
– Surgery (%)	1 (3.7)
– Physical exertion (%)	2 (7.4)
Lateralization	
Left side (%)	15 (55.6)
Right side (%)	10 (37.0)
Bilateral (%)	2 (7.4)
Clinical symptoms	
Pain (%)	27 (100.0)
Mild pain	14 (51.9)
Moderate pain	5 (18.5)
Severe pain	8 (29.6)
Muscle weakness	27 (100.0)
Muscle atrophy (%)	27 (100.0)

either by weekly infusions of 1 gram of methylprednisolone or a transition to oral prednisolone at a dose of 60 mg per day. Tapering schedules varied among individuals. In addition to corticosteroid therapy, standard care included pain management—with analgesic regimens tailored to symptom severity—and ongoing rehabilitation.

In our cohort, 20 of 27 patients (74.1%) received corticosteroid therapy according to the specified regimens. Among these, 11 patients (55.0%) initiated treatment more than 30 days after symptom onset, while the remaining 9 patients received corticosteroids within the first 30 days. The median time from disease onset to initiation of corticosteroid therapy was 45 days (IQR: 22–110 days).

Among 7 patients who did not receive corticosteroid treatment, 3 patients were lost to follow-up. This unfortunately limited our ability to compare treatment efficacy between the corticosteroid and non-corticosteroid groups. Alternatively, we analyzed disease progression between early and late corticosteroid administration to assess the impact of treatment timing on clinical outcomes. Baseline characteristics of patients receiving early versus late corticosteroid treatment are presented in Table 3. There were no significant differences in demographic variables between the two groups, except that identifiable preceding events were more frequently reported in the early corticosteroid group. The severity of clinical symptoms, as measured by the QuickDASH and MRC sum score, did not differ significantly between the groups.

Functional and muscle strength outcomes

In 27 patients, functional outcomes (QuickDASH) and muscle strength (MRC sum score) were assessed at baseline, 6 weeks, and 12 weeks. The median QuickDASH score improved from 43.2 (IQR 36.4–61.4) at baseline to 34.1 at 6 weeks (IQR: 20.5–45.5) and 20.5 at 12 weeks (IQR: 9.1–38.6), indicating progressive functional recovery (Friedman χ^2=59.45, p=0.0002; Kendall's W=0.76).

Muscle strength also increased, with median MRC sum scores rising from 40 (IQR 35–44) at baseline to 44 at 6 weeks (IQR: 38–45) and 44.5 at 12 weeks (IQR: 42–48) (Friedman χ^2=64.41, p<0.0001; Kendall's W=0.83), showing consistent

Table 2: Investigations results including nerve conduction study, electromyography and magnetic resonance imaging of brachial plexus

Nerve conduction study abnormalities		**Number of patients (N=27)**	
Median motor (%)		8 (29.6)	
Ulnar motor (%)		9 (33.3)	
Radial motor (%)		14 (51.9)	
Median sensory (%)		2 (7.4)	
Ulnar sensory (%)		1 (3.7)	
Superficial Radial sensory (%)		5 (18.5)	
Medial antebrachial cutaneous (%)		2 (7.4)	
Lateral antebrachial cutaneous (%)		5 (18.5)	
Electromyography abnormalities According to muscle group	**Numbers of test performed**	**Reduced recruitment pattern (%)**	**Active denervation features (%)**
Deltoid muscle (%)	17	10 (58.8)	7 (41.2)
Biceps brachii muscle (%)	16	9 (56.3)	7 (43.8)
Infraspinatus muscle (%)	8	7 (87.5)	4 (50.0)
Triceps brachii muscle (%)	17	11 (64.7)	6 (35.3)
Pronator teres muscle (%)	5	3 (60.0)	3 (60.0)
Extensor indicis propius muscle (%)	8	8 (100.0)	8 (100.0)
First dorsal interosseous muscle (%)	17	9 (52.9)	5 (29.4)
Abductor pollicis brevis muscle (%)	12	6 (50.0)	4 (33.3)
MRI brachial plexus abnormality		**Number of patients (N=22)**	
T2 intensity change (%)		16 (72.7)	
Gad enhancement (%)		9 (40.9)	
Hourglass appearance (%)		0 (0.0)	

Table 3: Baseline characteristic of patients who received early corticosteroid and late corticosteroid

Demographic data	**Early steroid patients (N = 9)**	**Late steroid patients (N = 11)**	**p-value**
Age (years, median, IQR)	55 (50 – 58)	50 (32 – 62)	0.447
Sex (male: female)	5: 4	6: 5	0.964
Body weight (kg, median, IQR)	65 (59 – 76)	68 (52 – 89)	0.939
Body Height (m, median, IQR)	1.66 (1.63 – 1.73)	1.65 (1.57 – 1.73)	0.676
Disease duration prior to diagnosis (days, median, IQR)	13 (10 – 30)	90 (60 – 150)	<0.001*
Identifiable preceding events (%)	4 (44.44)	0 (0.00)	0.013*
Vaccination (%)	1 (11.11)	0 (0.00)	
Preceding infection (%)	1 (11.11)	0 (0.00)	
Physical exertion (%)	2 (22.22)	0 (0.00)	
Lateralization			
Unilateral (%)	8 (88.89)	10 (90.91)	0.881
Bilateral (%)	1 (11.11)	1 (9.09)	
Baseline severity			
QuickDASH at baseline (median, IQR)	43.2 (38.6 – 63.6)	40.9 (36.4 – 52.3)	0.518
MRC sum score at baseline (median, IQR)	40 (36 – 43)	40 (33 – 43)	0.648

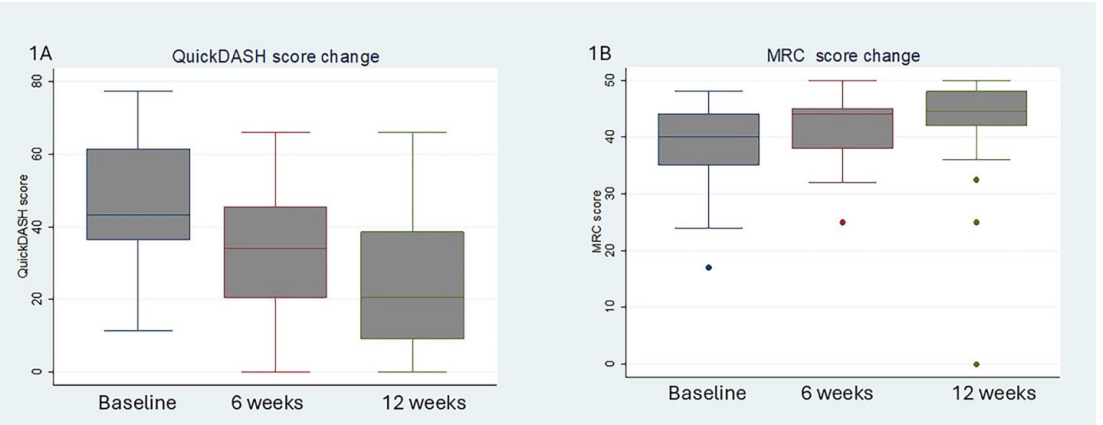

Figure 1. Recovery trends in function and muscle strength over time.
(A) QuickDASH scores at baseline, 6, and 12 weeks show progressive functional improvement (declining medians).
(B) MRC scores indicate increasing muscle strength over the same periods. Error bars = IQR. Both measures showed significant changes over time (Friedman test, $p < 0.001$) with strong within-subject concordance (Kendall's W: QuickDASH 0.76; MRC 0.83).

neuromuscular improvement alongside functional gains.

Subgroup analysis based on timing of corticosteroid administration

To assess the impact of corticosteroid timing on recovery outcomes, linear mixed-effects models were fitted for both QuickDASH and MRC motor sum scores. For the QuickDASH outcome, the model incorporating random intercepts and slopes with an unstructured covariance matrix and baseline adjustment demonstrated the best fit (log-likelihood = –203.71; LRT: $\chi^2(3) = 29.45$, $p < 0.001$). Both early and late steroid groups showed improvement, but the early group (≤30 days) exhibited a significantly greater reduction in scores (–4.84 vs. –2.44; Wald $\chi^2 = 4.80$, $p = 0.0284$; Figure 2A and Table 2 supplementary).

A similar approach applied to MRC motor scores identified a model with random intercepts and slopes and unstructured covariance as optimal (log-likelihood = –162.36; LRT: $\chi^2 = 65.04$, $p < 0.001$). Early treatment was associated with a larger mean strength gain (+1.38 vs. +0.60), and the group difference was statistically significant (Wald $\chi^2 = 5.68$, $p = 0.0172$; Figure 2B and Table 3 supplementary).

These findings consistently support that initiating corticosteroid within 30 days of symptom

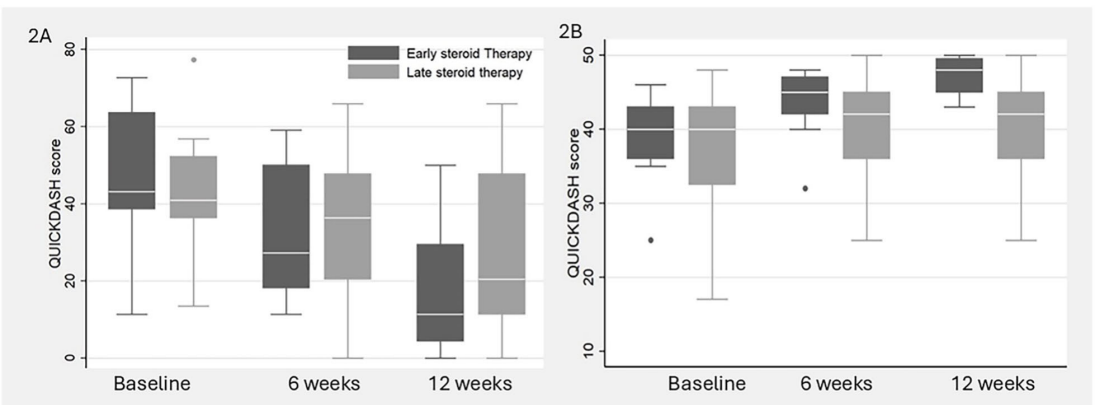

Figure 2. Impact of corticosteroid timing on recovery.
(A) Mean QuickDASH score changes at baseline, 6, and 12 weeks for early (≤30 days) vs. late (>30 days) treatment groups, with greater improvement in the early group.
(B) Mean MRC sum scores similarly favored early treatment. Error bars = standard error of means. Group differences were statistically significant (QuickDASH p=0.0284; MRC p=0.0172).

onset confers significantly greater functional ability and muscle strength improvement.

DISCUSSION

The epidemiological characteristics of the patient cohort were consistent with previous studies, with a median age of approximately 50 years and a higher prevalence in males compared to females. However, in contrast to prior reports, involvement of the left side was more common in our cohort.[5,9]

Antecedent events are often linked to neuralgic amyotrophy, supporting an immune-mediated cause. While prior studies report triggers in 40–50% of cases—including infections, vaccinations, surgery, or peripartum periods—only 25.9% of our patients had such events, mostly COVID-19 vaccinations.[2,5]

In our study, all patients with neuralgic amyotrophy reported pain in the affected arm. Motor weakness primarily involved muscles innervated by the upper trunk of the brachial plexus including those responsible for shoulder abduction, external rotation, and elbow flexion. It was affected in over 80% of cases. These findings align with prior studies, which reported pain in 96% of patients and upper trunk involvement in 71%, with frequent involvement of the infraspinatus and serratus anterior muscles. Although less common, distal hand weakness can also occur. In our cohort, about half of the patients showed weakness in finger and intrinsic hand muscles at presentation—higher than the 26–30% reported in earlier studies.[5]

Neuralgic amyotrophy is typically an axonal neuropathy characterized by reduced motor and sensory nerve conduction amplitudes. EMG often shows active denervation and reduced recruitment. However, routine nerve conduction studies may be normal due to predominant proximal nerve involvement not captured by standard tests.[1,3] Consistent with prior reports, only a few patients in our study had nerve conduction abnormalities. Motor abnormalities were most common in the radial nerve (extensor indicis proprius), while sensory changes were less frequent, mainly affecting the superficial radial and lateral antebrachial cutaneous nerves—the latter being the most commonly involved sensory nerve in previous studies.[1,2,6] These findings highlight the need for careful interpretation of nerve conduction studies in suspected neuralgic amyotrophy, as normal studies do not exclude the diagnosis.

Previous studies have reported varying MRI findings in patients with neuralgic amyotrophy. In one study utilizing high-resolution MRI in 27 patients, over 80 % demonstrated normal imaging results.[19] However, many studies have reported differing findings. Recent research suggests that patients with clinically diagnosed neuralgic amyotrophy often exhibit signal changes in parts of the brachial plexus, with T2 signal alterations being the most common.[2,7,20] The cervical roots and trunks were the regions most frequently involved, and many patients demonstrated involvement of multiple areas. According to the data, the C5 root and upper trunk were most commonly affected.[20] Moreover, MRI has been utilized in additional ways to support diagnosis. Changes in muscle signal intensity have been studied, with findings such as muscle edema and atrophic changes in the shoulder girdle muscles aiding in diagnosis.[21] Unsurprisingly, the supraspinatus and infraspinatus muscles — innervated by the upper trunk — were most commonly affected. In our study, we focused exclusively on brachial plexus findings, which predominantly demonstrated abnormalities. T2 signal changes with some degree of contrast enhancement were observed, consistent with findings reported in the literature.[1,2]

Regarding disease management, there is currently no high-quality evidence supporting the use of immunosuppressive therapy in the treatment of patients with neuralgic amyotrophy.[22] Some studies have suggested that the use of corticosteroids may have a positive effect on pain relief and could potentially aid in the recovery of motor function.[9,10,23] Immunoglobulin therapy has also been studied in small groups of patients.[11,12] In contrast, some previous studies have reported differing outcomes. A Cochrane review identified only three observational studies involving more than ten patients who received corticosteroid treatment.[22] Of these, one study suggested that systemic corticosteroids may not significantly alter the disease course in terms of motor weakness, although they may provide partial relief of pain symptoms. Furthermore, a study on lumbosacral radiculoplexus neuropathy, a condition believed to share a similar microvasculitic pathogenesis, reported negative effects associated with corticosteroid use.[24] These inconsistencies may be partly explained by variations in the timing of treatment initiation, underscoring the potential importance of early intervention. Several studies have proposed that the efficacy of immunosuppressive therapy in neuralgic amyotrophy depends on how early it is administered, with some suggesting that corticosteroids started beyond 14 to 30 days

after symptom onset may offer limited or no benefit.[3,9,18] Consequently, a 4-week cutoff has been used in some studies to define early intervention. At our institution, corticosteroids are routinely administered to nearly all patients with neuralgic amyotrophy. In this context, we aimed to explore whether the timing of corticosteroid initiation—early versus late—might influence clinical outcomes.

Our study shows that early corticosteroid treatment (within 30 days of symptom onset) significantly improves functional recovery in patients with upper limb neuralgic amyotrophy, as measured by QuickDASH and MRC motor scores. These results support the inflammatory basis of the disease, suggesting that prompt immunosuppression may reduce nerve damage and enhance recovery.

This study has limitations inherent to its retrospective design, including inconsistent documentation and inability to establish causality. Confounding factors such as disease severity, comorbidities, and healthcare access, may have influenced both treatment timing and outcomes. Selection bias is possible, as early corticosteroid initiation was likely based on clinical presentation. Due to the small number and poor follow-up of untreated patients, treatment comparisons were limited. Additionally, the five-month follow-up may not reflect long-term recovery or relapse rates.

In conclusion, this study is one of the few to present detailed data on the clinical features and progression of brachial plexus neuralgic amyotrophy in the Thai population. The low reported incidence may reflect underdiagnosis. Electrophysiological and MRI findings remain underreported in Thailand, likely due to limited access to specialized expertise. Our findings highlight the clinical variability and diagnostic challenges of this condition and may serve as a foundation for future research to improve recognition and management.

DISCLOSURE

Ethics: This study was approved by The Ethics Committee of Prasat Neurological Institute approved this study (approval number 68009).

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Conflict of interest: none

REFERENCES

1. Feinberg JH, Radecki J. Parsonage-turner syndrome. *HSSJ* 2010;6(2):199-205. doi: 10.1007/s11420-010-9176-x
2. Rubin DI. Brachial and lumbosacral plexopathies: A review. *Clin Neurophysiol Pract* 2020;5:173-93. doi: 10.1016/j.cnp.2020.07.005.
3. IJspeert J, Janssen RMJ, van Alfen N. Neuralgic amyotrophy. *Curr Opin Neurol* 2021;34(5):605-12. doi: 10.1097/WCO.0000000000000968
4. Kim TU, Chang MC. Neuralgic amyotrophy: an underrecognized entity. *J Int Med Res* 2021;49(4):3000605211006542. doi: *10.1177/03000605211006542*.
5. van Alfen N, van Engelen BGM. The clinical spectrum of neuralgic amyotrophy in 246 cases. *Brain* 2006;129(Pt 2):438-50. doi: 10.1093/brain/awh722
6. van Alfen N, Huisman WJ, Overeem S, van Engelen BGM, Zwarts MJ. Sensory nerve conduction studies in neuralgic amyotrophy. *Am J Phys Med Rehabil* 2009;88(11):941-6. doi: *10.1097/PHM.0b013e3181a5b980*.
7. Park MS, Kim DH, Sung DH. Magnetic resonance neurographic findings in classic idiopathic neuralgic amyotrophy in subacute stage: a report of four cases. *Ann Rehabil Med* 2014;38(2):286-91. doi: 10.5535/arm.2014.38.2.286
8. Kim DH, Kim J, Sung DH. Hourglass-like constriction neuropathy of the suprascapular nerve detected by high-resolution magnetic resonance neurography: report of three patients. *Skeletal Radiol* 2019;48(9):1451-6. doi: 10.1007/s00256-019-03174-4
9. van Eijk JJJ, van Alfen N, Berrevoets M, van der Wilt GJ, Pillen S, van Engelen BGM. Evaluation of prednisolone treatment in the acute phase of neuralgic amyotrophy: an observational study. *J Neurol Neurosurg Psychiatry* 2009;80(10):1120-4. doi: 10.1136/jnnp.2008.163386
10. Butt MA, Agadi K, Butt MM, Butt MM. Successful Management of Parsonage–Turner syndrome with steroids in the post-acute weakness phase: A case report. *EMJ Neurol* 2021;81-7. doi: 10.33590/emjneurol/21-00068
11. Naito K, Fukushima K, Suzuki S, *et al*. Intravenous immunoglobulin (IVIg) with methylprednisolone pulse therapy for motor impairment of neuralgic amyotrophy: clinical observations in 10 cases. *Intern Med* 2012;51(12):1493-500. doi: 10.2169/internalmedicine.51.7049
12. Sedlacek CM, Leone M, Foster AD, Hinkelman A. Off-label use of intravenous immunoglobulin with methylprednisolone to treat Parsonage-Turner syndrome in a United States marine. *Case Rep Med* 2021;2021:6663755. doi: 10.1155/2021/6663755
13. James MA. Use of the Medical Research Council muscle strength grading system in the upper extremity. *J Hand Surg Am* 2007;32(2):154-6. doi: 10.1016/j.jhsa.2006.11.008
14. Turan Z, Topaloglu M, Ozyemisci Taskiran O. Medical Research Council-sumscore: a tool for

evaluating muscle weakness in patients with post-intensive care syndrome. *Crit Care* 2020;24(1):562. doi: 10.1186/s13054-020-03282-x
15. Hudak PL, Amadio PC, Bombardier C. Development of an upper extremity outcome measure: the DASH (disabilities of the arm, shoulder and hand) [corrected]. The Upper Extremity Collaborative Group (UECG). *Am J Ind Med* 1996;29(6):602-8. doi: 10.1002/(SICI)1097-0274(199606)29:6<602::AID-AJIM4>3.0.CO;2-L
16. Moradi A, Menendez ME, Kachooei AR, Isakov A, Ring D. Update of the Quick DASH Questionnaire to account for modern technology. *Hand* (NY) 2016;11(4):403-9. doi: 10.1177/1558944715628006
17. Eren Y, Yavasoglu NG, Comoglu SS. The relationship between QDASH scale and clinical, electrophysiological findings in carpal tunnel syndrome. *Adv Clin Exp Med* 2018;27(1):71-5. doi: 10.17219/acem/67947
18. Holle JF, Limmroth V, Windisch W, Zimmerman M. Neuralgic amyotrophy. *Dtsch Arztebl Int* 2024;121(15):483-9. doi: 10.3238/arztebl.m2024.0077
19. Sneag DB, Rancy SK, Wolfe SW, *et al*. Brachial plexitis or neuritis? MRI features of lesion distribution in Parsonage–Turner syndrome. *Muscle Nerve* 2018;58(3):359-66. doi: 10.1002/mus.26108
20. Upadhyaya V, Upadhyaya DN, Bansal R, Pandey T, Pandey AK. MR neurography in Parsonage-Turner syndrome. *Indian J Radiol Imaging* 2019;29(3):264-70. doi: 10.4103/ijri.IJRI_269_19
21. Scalf RE, Wenger DE, Frick MA, Mandrekar JN, Adkins MC. MRI Findings of 26 patients with Parsonage-Turner syndrome. *AJR Am J Roentgeno*l 2007;189(1):W39-44. doi: 10.2214/AJR.06.1136
22. van Alfen N, van Engelen BGM, Hughes RAC. Treatment for idiopathic and hereditary neuralgic amyotrophy (brachial neuritis). *Cochrane Database Syst Rev* 2009;2009(3):CD006976. doi: 10.1002/14651858.CD006976.pub2
23. Lee GW, Kwon YH. The purport of steroid treatment in the weakness phase of Parsonage-Turner syndrome. *J Korean Orthop Assoc* 2012;47(4):311. doi: 10.4055/jkoa.2012.47.4.311
24. Pinto MV, Ng PS, Howe BM, *et al.* Lumbosacral radiculoplexus neuropathy: Neurologic outcomes and survival in a population-based study. *Neurology* 2021; 96(16):e2098-e2108. doi: 10.1212/WNL.0000000000011799

Supplementary Table 1: Characteristics of muscle weakness of involved extremity at the first examination according to muscle action

Pattern of weakness	Number of patients (N = 27)
Shoulder abduction	
– MRC motor power score 5 (%)	6 (22.2)
– MRC motor power score 3-4 (%)	13 (48.2)
– MRC motor power score 0-2 (%)	8 (29.6)
Shoulder external rotation	
– MRC motor power score 5 (%)	5 (18.5)
– MRC motor power score 3-4 (%)	17 (63.0)
– MRC motor power score 0-2 (%)	5 (18.5)
Elbow flexion	
– MRC motor power score 5 (%)	6 (22.2)
– MRC motor power score 3-4 (%)	17 (63.0)
– MRC motor power score 0-2 (%)	4 (14.8)
Elbow extension	
– MRC motor power score 5 (%)	11 (40.7)
– MRC motor power score 3-4 (%)	14 (51.9)
– MRC motor power score 0-2 (%)	2 (7.4)
Wrist flexion	
– MRC motor power score 5 (%)	1 (3.7)
– MRC motor power score 3-4 (%)	14 (51.9)
– MRC motor power score 0-2 (%)	12 (44.4)
Wrist extension	
– MRC motor power score 5 (%)	11 (40.8)
– MRC motor power score 3-4 (%)	12 (44.4)
– MRC motor power score 0-2 (%)	4 (14.8)
Finger flexion	
– MRC motor power score 5 (%)	16 (59.3)
– MRC motor power score 3-4 (%)	10 (37.0)
– MRC motor power score 0-2 (%)	1 (3.7)
Finger extension	
– MRC motor power score 5 (%)	12 (44.5)
– MRC motor power score 3-4 (%)	8 (29.6)
– MRC motor power score 0-2 (%)	7 (25.9)
Abductor pollicis brevis (APB)	
– MRC motor power score 5 (%)	14 (51.9)
– MRC motor power score 3-4 (%)	11 (40.7)
– MRC motor power score 0-2 (%)	2 (7.4)
Abductor digiti minimi (ADM)	
– MRC motor power score 5 (%)	13 (48.2)
– MRC motor power score 3-4 (%)	12 (44.4)
– MRC motor power score 0-2 (%)	2 (7.4)

Supplementary Table 2: QUICKDASH mixed model summary table

Model	Description	Log Likelihood	Early steroid Coef. (p-value)	Late steroid Coef. (p-value)	LRT vs Linear Model	Wald Test (Early vs Late)
Model 1	No group structure; fixed effects only	-258.38	-4.84 (0.001)	-2.44 (0.055)	N/A	χ^2=1.59, p=0.207
Model 2	Random intercept (group = A)	-237.27	-4.84 (<0.001)	-2.44 (<0.001)	χ^2=42.22, p<0.001	χ^2=7.31, p=0.0068
Model 3	Random intercept and slope (unstructured, group = A)	-235.68	-4.84 (<0.001)	-2.44 (0.000)	χ^2=45.42, p<0.001	χ^2=5.29, p=0.0215
Model 4	Random intercept and slope + baseline adjusted (group = A)	-203.71	-4.84 (<0.001)	-2.44 (0.001)	χ^2=29.45, p<0.001	χ^2=4.80, p=0.0284

Supplementary Table 3: MRC mixed model summary table

Model	Description	Log Likelihood	Early steroid (p-value)	Late steroid Coef. (p-value)	LRT vs Linear Model	Wald Test (Early vs Late)
Model 1	No group structure; fixed effects only	-194.88	1.38 (0.005)	0.60 (0.176)	N/A	χ^2=1.40, p=0.236
Model 2	Random intercept	-168.32	1.38 (<0.001)	0.60 (0.001)	χ^2=53.11, p<0.001	χ^2=8.65, p=0.0033
Model 3	Random intercept and slope	-162.36	1.38 (<0.001)	0.60 (0.006)	χ^2=65.04, p<0.001	χ^2=5.68, p=0.0172
Model 4	Random intercept and slope + baseline adjusted	-155.8	1.38 (<0.001)	0.60 (0.006)	χ^2=32.47, p<0.001	χ^2=5.68, p=0.0172