

## The Spectrum of Clinical, Etiological, Electrophysiological and Radiological Findings of Non Traumatic Brachial Plexopathies from a Tertiary Care Centre

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### ABSTRACT

**Background:** Non-traumatic brachial plexitis (NTBP) is an under-recognised inflammatory and immune-mediated disorder characterised by acute or subacute upper-limb pain, weakness, and sensory disturbance in the absence of mechanical injury. Its aetiological spectrum includes diabetic radiculoplexus neuropathy, malignancy-associated plexopathy, radiation-induced injury, and connective tissue disease-related vasculitis. Published case series from Indian tertiary neurology centres remain limited. **Methods:** Eight patients diagnosed with NTBP between March 2024 and June 2025 were retrospectively analysed. Clinical features, aetiologies, nerve conduction study and electromyography findings, brachial plexus MRI, treatment, and functional outcomes were systematically recorded. **Results:** The cohort comprised four males and four females with a mean age of 50.1 years (range 16-81). Right-sided involvement predominated (87.5%). Aetiologies included diabetes mellitus (n=3, 37.5%), malignancy or its treatment (n=3, 37.5%), rheumatoid arthritis (n=1, 12.5%), and deep-vein thrombosis on anticoagulation (n=1, 12.5%)-all patients presented with upper-limb pain. NCS and EMG demonstrated post-ganglionic motor axonal lesions spanning C5-T1 in all patients, with concomitant sensory axonal involvement of C6-C7 in seven. Brachial plexus MRI was abnormal in seven of eight cases (87.5%), revealing T2/STIR hyperintensity and denervation oedema; one patient additionally showed post-gadolinium enhancement. All patients received corticosteroids and structured neurorehabilitation. Marked functional improvement was achieved in five of eight patients (62.5%), all with non-malignant aetiologies; malignancy-associated cases showed only mild improvement. **Conclusions:** NTBP presents with a consistent post-ganglionic axonal electrophysiological signature across diverse aetiologies. Early aetiological identification, immunomodulatory therapy, and neurorehabilitation are associated with favourable outcomes. Malignancy-related plexopathy carries a guarded prognosis. Dedicated brachial plexus MRI is a valuable complement to electrodiagnosis.

**Keywords:** *brachial plexitis; non-traumatic brachial plexopathy; diabetic radiculoplexus neuropathy; neuralgic amyotrophy; radiation-induced brachial plexopathy.*

### INTRODUCTION

The brachial plexus, formed by the ventral rami of C5 through T1 nerve roots, provides motor and sensory innervation to the upper limb, shoulder, and parts of the thorax. Non-traumatic brachial plexitis (NTBP) encompasses inflammatory, immune-mediated, metabolic, vasculitic, and neoplastic processes

producing focal or multifocal plexus dysfunction without direct mechanical injury. Neuralgic amyotrophy, also known as Parsonage-Turner syndrome, is the prototypical idiopathic variety, causing multifocal paresis and sensory loss in a brachial plexus distribution [1]. In a landmark 246-patient series, van Alfen and van Engelen characterised the clinical

spectrum comprehensively, documenting that pain is universal and that sensory involvement occurs in approximately 78% of cases [2]. The condition is substantially more prevalent than historically estimated; subsequent prospective cohort data documented an annual incidence of approximately 1 per 1,000 in a primary care setting, suggesting the disorder is 30-50 times more frequent than earlier reports had indicated [3].

Beyond the idiopathic form, systemic conditions predispose to NTBP across a broad spectrum. Diabetic cervical radiculoplexus neuropathy arises from immune-mediated microvasculitis causing ischaemic peripheral nerve injury [4]. Radiation-induced brachial plexopathy (RIBP) is a recognised late complication of radiotherapy to the chest wall, axilla, and neck, most commonly following breast cancer treatment [5]. Superior sulcus (Pancoast) tumours invade the inferior brachial plexus elements, producing the Pancoast-Tobias syndrome of shoulder pain, lower plexus neuropathy, and ipsilateral Horner syndrome [6]. Rheumatoid vasculitis affecting the vasa nervorum is an extra-articular manifestation of rheumatoid arthritis (RA) that can mimic idiopathic plexitis [7].

Rubin's comprehensive review of brachial and lumbosacral plexopathies highlights that the non-specific clinical features and overlap with radicular and peripheral nerve disorders make a high index of suspicion and systematic evaluation essential [8]. Despite the clinical importance of NTBP, published case series from tertiary neurological centres in India remain

scarce. This study describes eight patients evaluated at our institution over fifteen months, with a focus on aetiological diversity, clinico-electrophysiological characterisation, MRI findings, and short-term functional outcomes.

Study Objective

The primary objective of this study was to describe the clinical, etiological, electrophysiological, and radiological characteristics of patients with non-traumatic brachial plexopathy presenting to a tertiary care centre. The secondary objective was to evaluate treatment response and short-term functional outcomes following corticosteroid therapy and structured neurorehabilitation.

MATERIALS AND METHODS

Study Design and Setting

It was a retrospective observational single-centre case series in the Department of Neurology of a tertiary academic hospital in India. We included patients with a clinical diagnosis of NTBP who presented between February 2024 and January 2026 and underwent electrodiagnostic testing and MRI of the brachial plexus. Patients with cervical spine trauma, prior brachial plexus surgery or cervical radiculopathy as the primary diagnosis were excluded.

Inclusion and Exclusion Criteria

The patient who has satisfied the following Inclusion and Exclusion Criteria were included in the study (Table 1 ).

| INCLUSION CRITERIA                                       | EXCLUSION CRITERIA                                                                |
|----------------------------------------------------------|-----------------------------------------------------------------------------------|
| Patients satisfying the criteria for Brachial Plexopathy | Patient with Upper limb weakness due to central causes or neuromuscular disorders |
| No history of Trauma                                     | Patient with Traumatic ethology of Brachial Plexopathy                            |
| Informed Consent                                         | Patient not willing to give informed Consent                                      |

Table 1: Inclusion and Exclusion Criteria

Clinical Assessment

Demographic data, clinical presentation, pertinent comorbidities (diabetes mellitus, malignancy, connective tissue disease), history of previous oncological therapies, muscle power rated according to the Medical Research Council (MRC) scale in proximal and distal muscle groups, deep-tendon reflexes, and cutaneous sensory defects were documented in a systematic manner. Clinical history, laboratory investigations, and imaging were used to classify aetiology.

Electrodiagnostic Studies

NCS and needle EMG were done by an experienced clinical neurophysiologist. Conducted sensory and motor NCS parameters from median, ulnar, radial, musculocutaneous and axillary nerves in the affected limb. Needle EMG was then performed in representative muscles innervated by C5-T1, including deltoid, biceps, triceps, extensor digitorum communis, first dorsal interosseous, and abductor pollicis brevis, and fibrillation potentials, positive sharp waves, and neurogenic motor unit changes.

## Brachial Plexus MRI

MRI at 1.5 or 3 Tesla scanning used T1-weighted, T2-weighted, and STIR sequences, performed in coronal and axial planes, as well as post-gadolinium fat-suppressed T1-weighted images. Qualitative analysis included assessment of changes in signal intensity in the trunks, divisions, and cords; T2/STIR hyperintensity; enlargement; gadolinium enhancement; and denervation oedema. MRI is the definitive imaging tool for brachial plexus pathology, as it offers an alternative, and complementary, anatomical localising modality to electrodiagnosis alone. 9

## Treatment and Outcome Assessment

In all cases, patients received corticosteroids (intravenous methylprednisolone 1 g/day for 3-5 days followed by oral prednisolone taper or oral prednisolone 1 mg/kg/day with taper) and structured neurorehabilitation. Considering the immune-mediated microvasculitic pathophysiology, immunotherapy is warranted. 10 Outcome was classified as improved (marked function recovery) or mild improvement (partial recovery with residual deficits).

## Statistical Analysis

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean  $\pm$  standard deviation (SD), whereas categorical variables are expressed as frequencies and percentages.

Owing to the small sample size and descriptive nature of the study, no inferential statistical analysis was performed.

## Ethical Considerations

This study was conducted in accordance with the Declaration of Helsinki and received institutional ethical clearance. All patient data were anonymised prior to analysis.

## Ethics Approval

The study protocol was reviewed and approved by the Institutional Ethics Committee of Vydehi Institute of Medical Sciences and Research Centre, Bengaluru. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

## RESULTS

### Demographic and Clinical Profile

Eight patients with NTBP were included. The cohort comprised four males and four females with a mean age of  $50.1 \pm 19.9$  years (range 16-81). The right brachial plexus was involved in seven patients (87.5%). All eight patients presented with upper-limb pain as the primary symptom followed by proximal weakness. Additional symptoms included numbness (n=6), difficulty gripping (n=3), and paraesthesias (n=2). Full clinical and investigative data are provided in Table 2.

| Case | Age/Sex | Etiology          | Side  | Proximal Power (MRC) | Distal Power (MRC) | Reflexes | Sensory  | NCS /EM G                                         | MRI Brachial Plexus                                          | Treatment        | Outcome  |
|------|---------|-------------------|-------|----------------------|--------------------|----------|----------|---------------------------------------------------|--------------------------------------------------------------|------------------|----------|
| 1    | 28/M    | DVT (Acitrom)     | Right | 2+                   | 5                  | Absent   | Impaired | Post-ganglionic C6-C7 sensory; C5-T1 motor axonal | Normal                                                       | Steroids + Rehab | Improved |
| 2    | 16/F    | Diabetes mellitus | Right | 2+                   | 5                  | Absent   | Normal   | Post-ganglionic C6-C7 sensory; C5-T1 motor axon   | Hyperintensity – right divisions & cords; denervation oedema | Steroids + Rehab | Improved |

|   |      |                                                |       |    |    |        |          |                                                        |                                                                                   |                  |                   |
|---|------|------------------------------------------------|-------|----|----|--------|----------|--------------------------------------------------------|-----------------------------------------------------------------------------------|------------------|-------------------|
|   |      |                                                |       |    |    |        |          | al                                                     |                                                                                   |                  |                   |
| 3 | 39/F | Diabetes mellitus                              | Right | 3+ | 5  | Absent | Normal   | Dene rvati on changes – right triceps C7–C8            | Hyperintensit y – right divisions & cords; denervation oedema                     | Steroids + Rehab | Improved          |
| 4 | 52/M | Rheumatoid arthritis                           | Right | 2+ | 5  | Absent | Impaired | Post-gang lioni c C6–C7; C5–T1 moto r axon al          | Hyperintensit y – right divisions & cords; denervation oedema                     | Steroids + Rehab | Improved          |
| 5 | 81/F | CA Breast (post-mastecto my + chemorad iation) | Right | 2+ | 5  | Absent | Normal   | Post-gang lioni c C6–C7 sens ory; C5–T1 moto r axon al | Hyperintensit y – right divisions & cords; denervation oedema                     | Steroids + Rehab | Mild improveme nt |
| 6 | 69/F | CA Endometr ium (post-TAH + chemothe rapy)     | Left  | 2+ | 5  | Absent | Impaired | Post-gang lioni c C6–C7 sens ory; C5–T1 moto r axon al | STIR hyperintensity – left trunks, divisions & cords; post-gadolinium enhancement | Steroids + Rehab | Mild improveme nt |
| 7 | 52/M | Diabetes mellitus                              | Right | 1+ | 5  | 1+     | Impaired | Post-gang lioni c C6–C7; C5–T1 moto r axon al          | Hyperintensit y – right divisions & cords; denervation oedema                     | Steroids + Rehab | Improved          |
| 8 | 64/M | CA Lung –                                      | Right | 3+ | 4+ | Absent | Impaired | Mod erate                                              | Hyperintensit y – right                                                           | Steroids + Rehab | Mild improveme nt |

|  |  |                                       |  |  |  |  |  |                                                                   |                                                |  |    |
|--|--|---------------------------------------|--|--|--|--|--|-------------------------------------------------------------------|------------------------------------------------|--|----|
|  |  | Pancoast<br>tumour<br>(right<br>apex) |  |  |  |  |  | C6-<br>C8<br>sens<br>ory;<br>C5-<br>T1<br>moto<br>r<br>axon<br>al | divisions &<br>cords;<br>denervation<br>oedema |  | nt |
|--|--|---------------------------------------|--|--|--|--|--|-------------------------------------------------------------------|------------------------------------------------|--|----|

Table 2: Patient Demographics, Clinical Features, Investigations, and Outcomes

MRC = Medical Research Council; NCS = nerve conduction study; EMG = electromyography; STIR = short-tau inversion recovery; CA = carcinoma; DVT = deep-vein thrombosis; TAH = total abdominal hysterectomy.

Aetiological Profile

Diabetes mellitus was then the most frequent aetiology (Cases 2, 3, and 7; 37.5%). Malignancy or its treatment was responsible for three other cases (37.5%): breast carcinoma on a background of mastectomy, chemotherapy, radiotherapy (Case 5); endometrial carcinoma on a background of total abdominal hysterectomy, chemotherapy (Case 6); and carcinoma of the lung on a background of right apical Pancoast tumour (Case 8). One patient had rheumatoid arthritis (Case 4) and one had deep-vein thrombosis treated with acenocoumarol (Case 1).

Neurological Examination

With respect to the cohort, proximal MRC power varied from 1 to 3+. In seven patients, power at MRC 5 was preserved distally at MRC 5; Case 8 (Pancoast tumour) sustained distal weakness to MRC 4+, which was consistent with inferior plexus involvement. Seven patients had absent deep-tendon reflexes, except Case 7,

who had significantly obliterated (1+) reflexes. Five patients (Cases 1, 4, 6, 7 and 8) had a sensory impairment.

Electrodiagnostic Findings

Post-ganglionic axonal lesion to C5-T1 motor fibres was the most common conduction defect, with sensory axonal involvement of C6-C7 in seven patients. Selective denervation limited to right triceps (C7-C8) in case 3, suggesting more focal involvement of cord or trunk. Case 8: Moderate axonal changes involving sensory nerves C6-C8 and motor nerves C5-T1 were found in the same patterns consistent with inferior plexopathy from Pancoast tumor involvement of the plexus.

Brachial Plexus MRI

Seven of 8 patients (87.5%) had abnormal MRI. T2/STIR hyperintensity (hyperintensity) of the divisions and cords with features of denervation oedema was the most common finding in seven patients. Post-gadolinium enhancement along the left brachial plexus trunks, divisions, and cords was also seen in case 6 (Figure 1). A normal MRI was present in Case 1 (DVT on anticoagulation).

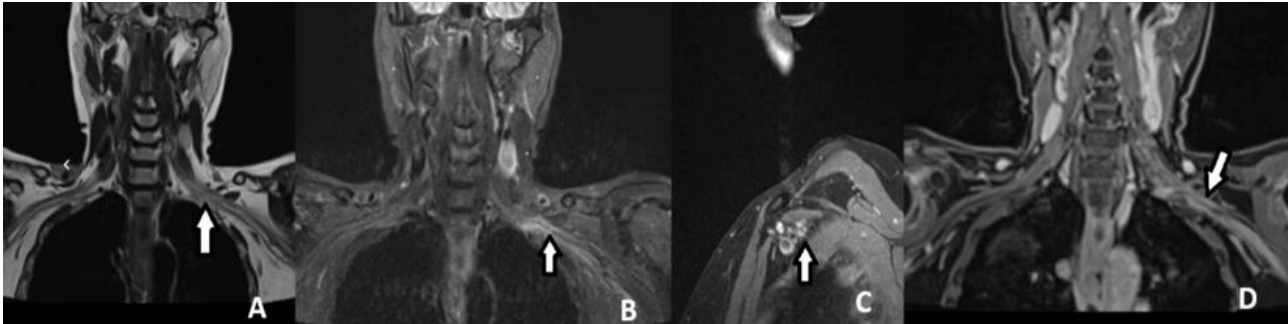


Figure 1: MRI of the patient with Carcinomatous Left Brachial Plexopathy - A : T2 images showing hyperintensity in Left Brachial Plexus; B : STIR images showing hyperintensity in Left Brachial Plexus at the level of the trunks, divisions and cords; C : Axial images and D : Sagittal images showing T1 Post Contrast enhancement of Left Brachial Plexus.

Treatment and Outcomes

All eight patients were given the same pharmacological treatment of corticotherapy and then underwent neurorehabilitation (methodology is highly structured). There were five patients with a post-operative functional

improvement which were statistically significant (62.5%); all had non-malignant aetiologies. For the three patients (Cases 5, 6, and 8) with malignancy-associated plexopathy, only mild improvement was observed. Table 3 contains a complete summary of these results.

| Parameter                | Finding                                     |
|--------------------------|---------------------------------------------|
| Total cases              | 8                                           |
| Mean age (years)         | 50.1 ± 19.9 (range 16–81)                   |
| Sex (M: F)               | 4:4                                         |
| Right-sided involvement  | 7/8 (87.5%)                                 |
| Left-sided involvement   | 1/8 (12.5%)                                 |
| Diabetes mellitus        | 3/8 (37.5%)                                 |
| Malignancy-associated    | 3/8 (37.5%)                                 |
| Rheumatoid arthritis     | 1/8 (12.5%)                                 |
| DVT / anticoagulation    | 1/8 (12.5%)                                 |
| Corticosteroid treatment | 8/8 (100%)                                  |
| Neurorehabilitation      | 8/8 (100%)                                  |
| Marked improvement       | 5/8 (62.5%) – all non-malignant aetiologies |
| Mild improvement         | 3/8 (37.5%) – all malignancy-related cases  |

**Table 3: Summary of Demographic and Outcome Data**  
Data presented as n (%) or mean ± standard deviation.

## DISCUSSION

This case series illustrates the diverse aetiological landscape of non-traumatic brachial plexitis while demonstrating a remarkably consistent electrophysiological signature across all cases. Our findings are discussed below in comparison with prior published literature.

### Aetiological Profile and Comparison with Published Series

Neuralgic amyotrophy is the commonest described aetiology in NTBP cohort studies undertaken in the Western population. The disorder's classic characteristics of acute severe pain followed by patchy proximal weakness with a preference for the upper and middle trunk were described in the 246-patient largest dedicated clinical series by van Alfen and van Engelen. Neuralgic amyotrophy was the most common cause of non-traumatic plexopathy, comprising 48% of cases in the series by previous studies, with neoplastic and radiation-related aetiologies combined approximating 24%, an overall distribution somewhat similar to the estimate of between 20-40% for malignant aetiologies across

published series by Rubin [8,9]. Unlike more purely idiopathic series, our cohort exhibited a systemic identifiable aetiology in all patients, reflecting a tertiary neurology referral bias toward metabolic and oncological comorbidities.

The most common identifiable aetiology in our series (37.5%) was diabetes mellitus, similar to the work by Massie et al., who described 85 patients with diabetic cervical radiculoplexus neuropathy at the Mayo Clinic [4]. In this seminal cohort pain was the predominant initial symptom (53/85), weakness was almost universal (84/85), and electrophysiology showed multisegmental axonal involvement a feature closely replicated in our three diabetic cases. The rationale for corticosteroid therapy is therefore directly based on the pathological substrate of immune-mediated microvasculitis causing ischaemic nerve injury, established by Dyck and Windebank [9,10].

### Malignancy-Associated Plexopathy

Malignancy or its treatment comprised 37.5% of our cases, broadly in keeping with published series in which

neoplastic and post-radiation aetiologies represent 20-40% of non-traumatic plexopathies [8,11,12]. Radiation-induced brachial plexopathy (RIBP) is an established late sequela of radiotherapy when given for breast cancer, as in case 5 [5]. Distinction of RIBP from neoplastic plexus infiltration is a key diagnostic dilemma; MRI with gadolinium is the primary investigation for this differential diagnosis. Radiation fibrosis appears as diffuse plexus thickening with enhancement (but low T2 signal) and with no discrete mass, whereas neoplastic infiltration contains irregular enhancing lesions with high T2 signal (or cord discontinuity) as shown by contrast-medium-enhanced MRI, Wittenberg, and Adkins [9]. Sureka et al similarly highlighted that MRI signal composition and lack of a focal mass are the main distinguishing characteristics [13].

Pancoast tumours directly invading the C8 and T1 nerve roots and lower trunk. Case 8 exhibited the anticipated distribution of weakness being distal and moderate axonal alteration in inferior plexus. They also highlighted that Pancoast tumours may present with musculoskeletal shoulder pain as an initial presenting symptom, and that their actual recognition requires a careful clinical index of suspicion with adjunct imaging of the chest [6].

### **Rheumatoid Vasculitis and DVT-Associated Plexopathy**

Rheumatoid vasculitis is an extra-articular complication of seropositive RA due to immune complex deposition in the vasa nervorum, causing nerve ischaemia [7]. Makol et al confirmed that peripheral neuropathy is one of the most common neurological manifestations of rheumatoid vasculitis, and that perhaps management as they experienced in their case series may necessitate escalated immunosuppression beyond corticosteroids alone [7]. The plexopathy associated with DVT (Case 1) likely represents ischaemic plexus injury from venous outflow obstruction and secondary microvascular insufficiency. The lack of abnormality on MRI in this case, combined with profound electrodiagnostic abnormality, may reflect early-phase disease. Wittenberg and Adkins have previously reported cases of electrophysiologically confirmed plexopathy with normal initial MRI [9].

### **Electrophysiological Signature**

The electrophysiological studies of all eight patients indicated a post-ganglion axonal pattern (consistent with plexitis), rather than a corresponding preganglionic cervical radiculopathy in which sensory nerve action potentials are intact. The marked C5-T1 motor axis with C6-C7 sensory predilection corresponds to upper/middle trunk predilection as reported in previously published

electrodiagnostic series [1,2,14]. Van Eijk et al, in their recent review of the literature on neuralgic amyotrophy, have proposed that patchy multifocal denervation on needle electromyography indicating simultaneous fascicular involvement is the key feature that discriminates plexitis from radicular or peripheral nerve lesions [14]. This electrodiagnostic framework led to plexus-level localisation in each of the eight patients examined, in line with the brachial plexopathies defined by Ferrante [13].

### **MRI Contribution**

The 87.5% rate of MRI abnormality in our series agrees with the data in the literature. In the study of Abid et al, Wittenberg and Adkins, MRI gives diagnostically useful information in most cases of nontraumatic plexopathies, and T2/STIR hyperintensity was the most sensitive sign. Authors from a South Asian tertiary centre directly comparable to the setting of this study have previously confirmed the role of MRI in identifying signal intensity changes, perineural pathology, and enhancement patterns across inflammatory, infiltrative, and compressive plexopathies [9,15]. An extensive review by Rubin emphasizes that confirmation of a plexopathy and accurate determination of its aetiology necessarily require the correlation of contributions from neuroimaging, electrodiagnosis and serologic evaluation [8].

### **Treatment Outcomes and Prognostic Factors**

The proportion (62.5%) of marked improvement available in the non-malignant cases is in keeping with the outcomes from the Mayo Clinic diabetic cervical radiculoplexus neuropathy cohort [4]. Thaisethawatkul and Dyck evaluated immunity in lumbosacral radiculoplexus neuropathies and reported that about 1/3 benefitted from corticosteroids and intravenous immunoglobulin; thus, although objective improvement is modest, corticosteroids promote relief of pain and augmented motor recovery [10]. The worse outcomes seen in malignancy-associated cases represent irreversible radiation fibrosis and/or tumour infiltration, as IIB has reported in his published RIBP series [5]. Neurorehabilitation was part of care in all cases, in line with guidance which states that physiotherapy and occupational therapy are essential to prevent secondary complications [10,14].

### **Strengths of the Study**

- This study provides one of the few Indian tertiary-care case series describing the clinical, etiological, electrophysiological, and MRI spectrum of non-traumatic brachial plexopathies.
- All patients underwent a uniform diagnostic evaluation, including detailed neurological examination, nerve conduction studies,

electromyography, and dedicated brachial plexus MRI.

- The study demonstrates the correlation between clinical presentation, electrophysiological findings, and MRI abnormalities across different etiologies.
- Standardized treatment with corticosteroids and structured neurorehabilitation allowed consistent assessment of short-term clinical outcomes.

### Clinical Implications

Early recognition of non-traumatic brachial plexopathy, supported by electrodiagnostic studies and dedicated brachial plexus MRI, facilitates accurate localization, timely etiological diagnosis, and appropriate treatment. Prompt initiation of corticosteroid therapy and structured neurorehabilitation may improve functional recovery, particularly in inflammatory and metabolic causes.

### Patient Consent

Written informed consent was obtained from all participants or their legally authorized representatives for participation in the study and publication of anonymized clinical information and radiological images.

### LIMITATIONS

Limitations include the small sample size, retrospective design, variable follow-up duration, absence of biopsy-based pathological confirmation, and lack of validated functional outcome instruments such as the DASH questionnaire or MRC sum scores. Prospective multicentre Indian cohort studies with standardised follow-up intervals are warranted.

### Future Directions

Future prospective multicentre studies involving larger patient populations, longer follow-up durations, standardized functional outcome measures, and advanced imaging techniques are required to better understand disease mechanisms, optimize treatment strategies, and improve prognostic prediction in patients with non-traumatic brachial plexopathy.

### CONCLUSIONS

Non-traumatic brachial plexitis encompasses a clinically diverse but electrophysiologically consistent syndrome. In this single-centre Indian case series, diabetes mellitus and malignancy-related aetiologies were the most common etiologies followed rheumatoid vasculitis and DVT-associated plexopathy comprising the remainder. Post-ganglionic axonal NCS/EMG findings and T2/STIR brachial plexus MRI hyperintensity were the dominant diagnostic features across all aetiologies. Corticosteroids combined with structured

neurorehabilitation produced marked improvement in significant number of patients with non-malignant causes, consistent with outcomes reported in diabetic and idiopathic plexitis cohorts in published literature. Malignancy-associated plexopathy carried a guarded prognosis, reflecting irreversible underlying mechanisms. Systematic aetiological evaluation, electrodiagnostic characterisation, and dedicated brachial plexus MRI remain the pillars of diagnosis and management in this important condition.

### Ethics Statement and Conflict of Interest Disclosures

**Human subjects:** Informed consent for treatment and open access publication was obtained or waived by all participants in this study.

**Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue.

**Conflicts of interest:** In compliance with the [ICMJE uniform disclosure form](#), all authors declare the following:

**Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work.

**Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

**Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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### Data Availability Statement

The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

### Author Contributions

**Rashmi Devaraj:** Conceptualization, study design, patient management, supervision, interpretation of data, manuscript review, and final approval of the manuscript.

**Aishwarya Thallapally:** Data collection, literature review, clinical data analysis, manuscript drafting, and revision.

**Shreyashi Ganguly:** Data interpretation, manuscript editing, literature review, and critical revision of the manuscript.

**Mohan P C:** Study supervision, interpretation of findings, critical revision of the manuscript for important intellectual content, and final approval of the manuscript.

### Abbreviations

NTBP – Non-traumatic brachial plexopathy  
MRI – Magnetic Resonance Imaging  
NCS – Nerve Conduction Study  
EMG – Electromyography  
MRC – Medical Research Council  
STIR – Short Tau Inversion Recovery  
DVT – Deep Vein Thrombosis  
RA – Rheumatoid Arthritis  
TAH – Total Abdominal Hysterectomy  
SD – Standard Deviation

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