Brachial plexopathy

Satish V. Khadilkar, Snehaldatta S. Khade

Department of Neurology, Grant Medical College and Sir J.J. Group of Hospitals, Mumbai, Maharashtra, India

Abstract

Brachial plexus injury can occur as a result of trauma, inflammation or malignancies, and associated complications. The current topic is concerned with various forms of brachial plexopathy, its clinical features, pathophysiology, imaging findings, and management. Idiopathic brachial neuritis (IBN), often preceded with antecedent events such as infection, commonly present with abruptonset painful asymmetric upper limb weakness with associated wasting around the shoulder girdle and arm muscles. Idiopathic hypertrophic brachial neuritis, a rare condition, is usually painless to begin with, unlike IBN. Hereditary neuralgic amyotrophy is an autosomal-dominant disorder characterized by repeated episodes of paralysis and sensory disturbances in an affected limb, which is preceded by severe pain. While the frequency of the episodes tends to decrease with age, affected individuals suffer from residual deficits. Neurogenic thoracic outlet syndrome affects the lower trunk of the brachial plexus. It is diagnosed on the basis of electrophysiology and is amenable to surgical intervention. Cancer-related brachial plexopathy may occur secondary to metastatic infiltration or radiation therapy. Traumatic brachial plexus injury is commonly encountered in neurology, orthopedic, and plastic surgery set-ups. Trauma may be a direct blow or traction or stretch injury. The prognosis depends on the extent and site of injury as well as the surgical expertise.

Key Words

Hereditary neuralgic amyotrophy, myokymic discharges, neurography, Pancoast tumor, plexopathy

For correspondence:

Dr. Satish V. Khadilkar, Room No. 110, First Floor, New Wing, Bombay Hospital, New Marine Lines, Mumbai - 400 012, Maharashtra, India. E-mail: khadilkarsatish@gmail.com

Ann Indian Acad Neurol 2013;16:12-8

Introduction

Brachial plexus problems are encountered by neurologists regularly for inpatient and outpatient consultations. A variety of disorders affect the brachial plexus and the cervical radicals inside the cervical canal. The frequently encountered problem of differentiating radiculopathies from plexopathies often proves difficult to answer. The clinical presentation of cervical radiculopathies could often be confused with brachial plexopathies, more so when multiple roots are involved. The upper trunk brachial plexopathy simulates the C5 or C6 root lesion. The natural history of the two tends to be different. Unlike brachial neuritis, it is unusual for radicular pain to subside as weakness increases. Patients with cervical radiculopathy often have persistent pain and, at times, associated neck muscle spasm. While radiculopathies tend to be sensorimotor, brachial neuritis is often a motor-dominant situation. Electrodiagnostic studies (EDS) provide further guidance to localize the site of the lesion. EDS include measurement of compound motor action potential (CMAP), sensory nerve action potential (SNAP), and needle electromyography (EMG) examination of cervical paraspinal muscles. SNAPs are present in the pre-ganglionic lesion as this lesion is proximal to the dorsal root ganglion. Needle EMG of the cervical paraspinal muscles shows fibrillation potentials in cases with recent cervical radiculopathy, but not so in cases with brachial plexus involvement. Plexopathies related to inflammation, trauma, tumors, and radiation therapy form the bulk of the clinical cases. T2WI with contrast-enhanced magnetic resonance imaging (MRI) (MR myelography) detects root avulsions, intrinsic and extrinsic masses of the brachial plexus, pseudomeningoceles, post-traumatic neuromas, hematomas, fibrosis, and inflammatory plexitis such as infectious, immune mediated, radiation induced, or idiopathic.[1] In case of root avulsion, computed tomography myelography is a standard investigation choice as a result of higher spatial resolution and better demonstration of nerve roots compared with MR myelography. Its major disadvantage is that it is invasive and very difficult to perform, especially in newborns and neonates.[2] This article will deal with the various conditions affecting the brachial plexus that are encountered by neurologists.

Access this article online	
Quick Response Code:	Website: www.annalsofian.org
	DOI: 10.4103/0972-2327.107675

Idiopathic Brachial Neuritis

Idiopathic brachial neuritis (IBN), also known as Parsonage–Turner syndrome, is a disorder of unknown etiology, with

asymmetric involvement of the brachial plexus.[3,4] It occurs in all age groups but is more common between the third and seventh decade. Men are affected more often than women. Antecedent events occurring days or weeks prior to the onset have been reported in 28-83% of the cases in various series.[5,6] Upper respiratory infection, flu-like illness, immunization, surgery and emotional stress have been the common triggers. No triggers can be found in half of the cases. The condition is commonly seen in men engaged in vigorous athletic activities such as wrestling, weight lifting, and gymnastics.

The pathophysiology of IBN is not fully elucidated but is believed to be an immune-mediated disorder. The temporal relationship of antecedent events with the onset of brachial neuritis initially suggested the possibility of an autoimmune basis. Subsequent studies were carried out in search of immune pathogenesis. A study conducted by Pierre *et al.*, demonstrated the presence of oligoclonal bands in the cerebrospinal fluid and raised serum IgG titers against herpes simplex and varicella zoster virus. Thus, reactivation of virus was thought to be involved in the pathogenesis of the disease.[7] There has been some evidence of altered lymphocyte subsets, with a decrease in CD3 values and an increase in the CD4:CD8 ratio in the brachial plexus nerves, further substantiating the role of the immune process.[8] The demonstration of antiganglioside antibodies in sera and multifocal mononuclear infiltrates in brachial plexus biopsies of patients with brachial neuritis also supports an immune basis.[9,10]

IBN starts abruptly with intense pain at sites such as the shoulder, trapezius ridge, scapular area, upper arm, forearm, or hand. Pain may be sharp, stabbing, throbbing, or aching in nature, which lasts from few hours to weeks. A minority of the patients of IBN do not experience the initial painful stage.[11] As the pain abates, shoulder girdle and arm weakness develops. Upper plexus muscles, including deltoid, supra and infraspinatus, serratus anterior, and biceps are commonly affected. Weakness usually progresses over few days. The forearm and hand muscles are less commonly involved. Wasting of the weak muscles follows rapidly and can be very striking in some patients. The wasting can be misleading, prompting the thoughts of a disease of much longer duration. Muscle stretch reflexes are impaired or absent in weakened muscles. Most plexopathies are incomplete as one or more muscles in the same root distribution are spared. Motor deficit is usually predominant as compared with sensory loss. At times, discrete lesions of individual peripheral nerves are seen in cases with IBN. These could be the axillary nerve, the suprascapular nerve and the anterior interroceous nerve.[12-14] Diaphragmatic involvement is seen in few cases.[15,16] Recurrent isolated alternating phrenic neuropathy may also be a part of the brachial neuritis spectrum.[17] One-third of the cases have bilateral plexopathy. Majority of the patients have a slow but steady recovery of motor function over the following 6-18 months. The degree of recovery may vary from area to area.[3] IBN is typically monophasic and recurrence is rare. Electrophysiological studies help in the confirmation of diagnosis, extent of lesion, and prognostication. MRI findings in brachial plexitis range from normal to mild thickening of the plexus and hyperintensity on T2WI with or without enhancement. Fat deposition and denervation signal-intensity changes appear in the muscles of the shoulder girdle and chest in the subacute and chronic phases of brachial plexitis.[18,19]

Case 1

A 50-year-old male presented with a complaint of acute-onset breathlessness in a lying position since the past 1 week, which was preceded by a herpes zoster infection causing facial palsy. The patient did not have any weakness in any of the limbs. On examination, he had paradoxical breathing, which was confirmed on fluoroscopy. The patient's routine blood investigations were within normal limits. Conventional nerve conduction studies were within normal limits. Phrenic nerve stimulation showed no response bilaterally that was suggestive of diaphragmatic failure. Considering it to be a presentation of brachial neuritis spectrum disorder, the patient was put on a trial of steroids. On follow-up at 2 months, he showed significant improvement in the symptoms of orthopnea.[20]

Unilateral or bilateral phrenic nerve involvement in neuralgic amyotrophy has been described, but isolated phrenic nerve involvement in brachial neuritis is unusual.

Treatment

Patients often require analgesic drugs for relief of pain. The pain can be severe in some patients, requiring a combination of agents and, at times, requiring opiod derivatives.

Corticosteroids have been used regularly in the management of IBN, but limited data are available to support its use. A study by van Eijk *et al*,[21] indicates that oral prednisolone may be an effective pain treatment for brachial neuritis. These investigators evaluated the effects of prednisolone treatment in terms of pain relief and strength recovery when administered in the acute phase. This is a retrospective case series of 50 treated patients compared with 203 untreated cases. The study found that the median time required for initial pain relief was 12.5 days in the treated group compared with 20.5 days in the untreated patients. The study showed that 18% of the prednisolone patients recovered strength within the first month of treatment, with only 6.3% of the control group patients showing recovery. Moreover, 12% of the patients in the prednisolone group attained a full recovery within 1 year, while only 1% of the untreated group fully recovered within that period. The authors recommended that oral prednisolone be used during the acute phase of brachial neuritis; but, they also advised that a prospective, randomized trial be conducted to verify their results.

A Cochrane review points out the paucity of randomized controlled trials for steroids in IBN in establishing the efficacy of treatment with corticosteroids or other immune-modulating therapies.[22]

No controlled trials of IV immunoglobulin (IVIG) have been carried out in brachial neuritis. Anecdotal reports of the beneficial role of IVIG in the management of brachial neuritis are available in the literature (Class IV).[23-25]

Immobilization of the extremity during the short painful stage is recommended. As recovery begins, physical therapy for patients with brachial neuritis should be focused on the maintenance of full range of motion in the shoulder and other affected joints once severe pain subsides.[26] Strengthening of

the rotator cuff muscles and scapular stabilization may be necessary in severely affected patients. Assistive devices and orthotics may be used, depending on the particular disabilities present.

In brachial neuritis, nerve grafting or tendon transfers may be considered for the few patients who do not achieve good recovery by 1-2 years. Surgery is usually aimed at improving shoulder abduction.

Idiopathic Hypertrophic Brachial Neuritis

This disease is uncommon and tends to affect the brachial plexus gradually. The history goes on for months to even years, and there is slowly progressive weakness and wasting of the segments affected by the disease. Motor disability is overwhelming and sensory findings, when present, are mild. Unlike the acute form, this condition is painless from the beginning.

It differs from IBN in its painless course, although some patients may have significant pain. In this disorder, EMG/NCS will show demyelinating features not seen in IBN. Histopathological studies reveal features of a localized form of other peripheral demyelinating disorders, such as chronic inflammatory demyelinating polyneuropathy or multifocal motor neuropathy. Enlargement of the brachial plexus may be seen on MRI.[26]

Case 2

A 42-year-old male presented with complaints of progressive numbness of the right hand since 2 years, with similar complaints of the left hand since 6 months. He noticed progressive weakness of the right upper limb since 3 months. During the course of the illness, he noted significant wasting of the intrinsic muscles of the right hand. On examination, predominant proximal weakness (grade 1/5) was observed. Pin prick and temperature sensation were reduced in the right hand, whereas joint position sense and vibration were normal. Deep tendon reflexes were absent in both upper limb. A burn mark was observed over the right arm. He did not have thickened nerves. With the presence of flail arm right more than left and sensorimotor affection, brachial plexus or spinal cord was thought to be the probable site of the lesion. The CSF report showed nil cells, mildly raised proteins, and normal sugar. Electrodiagnostic studies showed asymmetric right more than left, multifocal motor more than sensory and demyelinating more than axonal, with evidence of minimal demyelination in the lower limbs. MRI right brachial plexus showed hypertrophied plexus while MRI cervical spine was normal. Therefore, a final diagnosis of multifocal acquired demyelinating sensory and motor neuropathy (MADSAM) was made [Figure 1].

Idiopathic hypertrophic brachial neuropathy as a presentation of MADSAM has been reported in the literature.[27]

Hereditary Neuralgic Amyotrophy

Hereditary neuralgic amyotrophy (HNA) is an autosomal-dominant disorder characterized by repeated episodes of paralysis and sensory disturbances in an affected limb preceded by severe pain. HNA is genetically linked to chromosome 17q25, where mutations in the septin-9 (SEPT9) gene have been found.[28] HNA is a rare disorder and its worldwide prevalence is unknown.

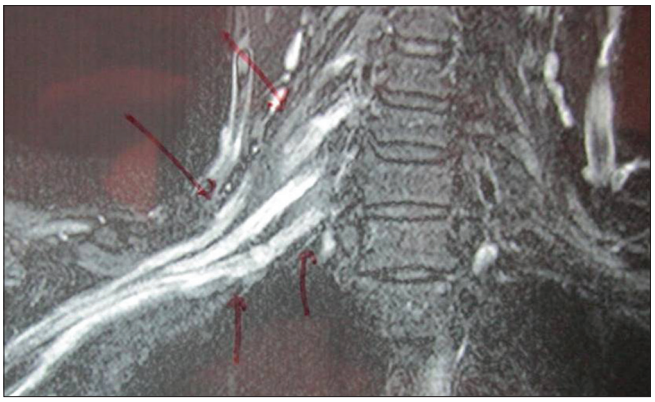

Figure 1: Magnetic resonance imaging brachial plexus showing hypertrophy of the right plexus with contrast enhancement

The onset of HNA is at birth or later in childhood, with good prognosis for recovery following each attack. However, persons with HNA may have permanent residual neurological deficit following repeated attacks. Like the idiopathic variety, in HNA, episodes are triggered by infections, immunizations, surgery, the puerperium, and stress. While the frequency of the episodes tends to decrease with age, affected individuals are often left with residual problems, such as chronic pain and impaired movement, which accumulate over time. In some HNA pedigrees, there are characteristic dysmorphic facial features, including hypotelorism, epicanthal folds, long narrow face, facial asymmetry, cleft palate and neck and forearm skin folds or creases, syndactily or webbing of toes and primitive pinna with folded helix. It differs from the idiopathic form in that there are no gender differences, recurrence is common, cranial nerves are more commonly affected and that it may be associated with dysmorphic features mentioned above.[29]

Electrophysiological studies show normal or mildly reduced motor nerve conduction velocities distal to the affected brachial plexus. Pathological studies have found mild focal axonal degeneration in nerves examined distal to the plexus abnormality.

Hereditary neuropathy with pressure palsies can present phenotypically like acute brachial plexopathy.[30] But, unlike the classical phenotype, recurrence is unusual.

Neurogenic Thoracic Outlet Syndrome

Thoracic outlet syndrome is characterized by pain, paresthesias, and weakness in the upper extremity, which may be exacerbated by elevation of the arms or by exaggerated movements of the head and neck. It is commonly seen in women. Often, bilateral cervical rib or enlarged down-curving C7 transverse processes, fibrous band across the cervical rib and scalene tubercle of the first cervical rib are noted in these cases. Compression can occur between the anterior and middle scalene muscles, beneath the

clavicle in the costoclavicular space, or beneath the tendon of the pectoralis minor.[31]

Classic symptoms of neurogenic thoracic outlet syndrome (nTOS) include pain, paresthesias or weakness in the upper extremity. Paresthesias most commonly affect the ring and small fingers. Symptom severity tends to increase after certain activities, and worsens at the end of the day or during sleep.[32] Advanced cases of nTOS are characterized by objective signs of weakness of the hand, loss of dexterity of the fingers, and atrophy of the affected muscles. X-rays of the chest should be performed to rule out the possibility of an infiltrative process or space-occupying mass (e.g., Pancoast tumor) compressing the brachial plexus. MRI, especially sagittal T1WI through neurovascular bundles as well as MR angiography and MR venogram of the subclavian vessels in both neutral and abduction positions, aid in depiction of neurovascular compression, stenosis, thrombosis, and aneurysms.[33]

The provocative tests, which may be corroborative evidence of nTOS, are based on creating maximal tension on the anatomic sites of constriction. There are various provocative tests like Adson test, Wright test, Roos test, and others. The Adson test involves extension of the neck with rotation of the head toward the involved extremity, which is held in an extended position at the side. This maneuver constricts the interscalene triangle. It is considered positive on detection of change in the radial pulse when the patient inhales deeply and holds his/her breath. During the Wright test, the patient places the arm in full abduction and external rotation, leading to constriction of the costoclavicular space. The test is considered abnormal if typical symptoms are elicited along with detection of a change in pulse. In the elevated arm stress test (Roos test), the patient keeps the affected arm in full abduction and external rotation and then opens and closes the fist slowly over 3 min. This test causes constriction of the costoclavicular space. It is considered positive on elicitation of typical symptoms and the patient's inability to sustain this manoeuvre for the full 3 min. It should be noted that a high false-positive rate is seen and that it cannot replace confirmatory EDS – the most definitive test.[32,34]

Electrodiagnostic features for the diagnosis of nTOS require the following parameters: [35,36]

- *Sensory findings*: Absent or reduced amplitude (<12 μV) of the ulnar antidromic sensory nerve action potential (SNAP) or absent or reduced amplitude (<10 uV) of the medial antebrachial cutaneous nerve (MABC) antidromic SNAP, with normal amplitude of the MABC SNAP in the contralateral (unaffected) extremity.
- *Motor findings:* One or more of the following should be present : (1) Absent or reduced amplitude (<5 mV) of the median nerve compound motor action potential (CMAP). (2) Absent or prolonged minimum latency (>33 msec) of the ulnar F-wave (with or without abnormalities of the median F-wave), and with normal F-waves in the contralateral (unaffected) upper extremity. (3) Needle electromyography (EMG) which shows denervation (e.g. fibrillation potentials, positive sharp waves) in atleast one muscle supplied by each of two different nerves from the lower trunk of the brachial plexus, with normal EMG of the cervical paraspinal muscles and at least one muscle supplied by a nerve from the middle or upper trunk of the brachial plexus.

  In addition a) Exclusion of other focal neuropathies or polyneuropathy as a cause for the abnormalities described above and b) the normal amplitude (≥ 15 μV) of the median nerve antidromic SNAP and normal conduction velocity (≥ 50 m/s) of the ulnar motor nerve across the elbow is required.

Treatment

No randomized controlled trials have been conducted to measure the efficacy of conservative treatments for nTOS.[37] Examples of conservative treatment include modification of activities that exacerbate symptoms, education, postural exercises, physical therapy and anti-inflammatory drug therapy.

An observational study of 50 patients showed that strengthening and stretching exercises reduced pain among 80% of the patients after 3 months and among 94% of patients after 6 months, and a 2007 systematic review of the available literature concluded that conservative treatment appears to be effective in reducing symptoms, improving function and facilitating return to work.[38] If there is no response to conservative treatment within 6 weeks, or if time loss extends longer than 2 weeks, specialist consultation should be obtained. The "disputed thoracic outlet syndrome" terminology has been applied to the cases in which there are little or no clinical findings of TOS. Hence, the judicious and accurate use of EDS has a significant role in the diagnosis of nTOS, as surgical outcome in such cases with disputed TOS has often been detrimental.[39]

Cancer-Related Brachial Plexopathy

Brachial plexus involvement is a well-known complication of cancer. Brachial plexopathy in such cases could be either due to metastatic spread or secondary to radiation therapy for the cancer.

Metastatic Brachial Plexopathy

Incidence of metastatic neoplasm of the brachial plexus increases with age; thus, the condition is more common in the elderly patients. Lung and breast cancers most commonly metastasize to brachial plexus. Neoplasms reach the plexus by direct extension or, more commonly, by metastasis through the lymphatics from the axilla. Other less common associated tumors are lymphoma, sarcoma and melanoma.

The salient feature of metastatic plexopathy is the pain, which is often severe, located in the shoulder girdle radiating to the inner aspect of the upper limb.[40] Peripheral pain mechanisms proposed include reduction in nociceptor threshold by prostaglandins and other noxious chemical substances and persistent nociceptor stimulation, compression or infiltration of the nerves of the plexus by a tumor. There is a preferential involvement of the lower trunk as lateral axillary lymph nodes draining the lung and breast regions are in proximity to the lower trunk. The Pancoast syndrome is usually caused by carcinoma at the lung apex, encroaching on the lower trunk of the brachial plexus. The associated Horner syndrome is noted in about half of the cases.[41] MR neurography may

help in excluding tumor in patients presenting with brachial plexopathy.[42] MRI can identify the mass adjacent to the plexus and detect whether the epidural space is encroached. Fluorodeoxyglucose-positron emission tomography (PET) aids in confirming metastases in patients with indeterminate MRI findings, and is useful for depicting metastases in the other part of the body as well.[1]

Treatment is often difficult and thus the patient receives palliative care. Treatment of metastatic plexopathy is based upon two pillars, radiotherapy and chemotherapy of the underlying tumor. Results are often disappointing. In patients with Pancoast tumor, the common approach is preop radiotherapy followed by extended surgical resection, with a 5-year survival rate of 20-35%.[43]

Radiation-Induced Brachial Plexopathy

Radiation therapy to the chest, neck or axillary region for the underlying tumor may result in brachial plexopathy. Factors like radiation dose, technique and concomitant chemotherapy play a vital role in the brachial plexus injury.[44] Radiation dose < 6000 cGy less likely leads to plexopathy. The interval from the last dose of radiation to the first symptom of plexus disorder is usually a mean of 6 years. Breast carcinoma is most commonly associated with radiation plexopathy (40-75%), which is followed by lung carcinoma and lymphoma.[45]

Limb paresthesia, swelling, and motor weakness are common presenting complaints. Pain is not a consistent feature of such plexopathies. Unlike metastatic injury, radiation-induced plexopathy has a predilection for the upper trunk and not for the lower trunk, probably secondary to the protective effect of the clavicle and relatively shorter course of the lower trunk through the radiation port. Endoneural and perineural fibrosis, occlusion of microvasculature and direct injury to the myelin sheaths and axons are the proposed mechanisms for radiation-induced plexopathy. MRI may show thickening and diffuse enlargement of the brachial plexus without the focal mass, but does not always differentiate metastatic and radiation injuries.[46] Radiation fibrosis in the chronic form appears as hypointense on T1WI and T2WI.[47] Nerve conduction studies in the early stages may show features of demyelinating conduction blocks. Unlike metastatic plexopathy, EMG studies in radiation injury show spontaneous activity in the form of myokymic discharges.[48]

It has dismal prognosis, with the patient requiring palliative care depending on the distressing symptoms. Lymphatic bypass surgery to relieve lymphedema may rarely be required. The patient is advised to continue rehabilitative measures.

Traumatic Brachial Plexopathy

Trauma is one of the most common causes of brachial plexopathy. These injuries usually result from a motorcycle accident or a high-speed motor vehicle accident, fall from a significant height secondary to traction or from a direct blow. It may occur with penetrating injuries and gunshot wounds. It could result from iatrogenic injury, especially as a complication of nerve block administration. In traction-type brachial plexus injuries, the head and neck are stretched away violently from the shoulder.[49] Upper plexus injuries are commonly seen if the arm is at the side because the first rib acts as a fulcrum to direct the traction forces preferentially in line with the upper plexus. The lower plexus lesion predominates when the arm is abducted and raised overhead violently because the coracoid acts as a fulcrum in a similar fashion. The pre-ganglionic site of injury is usually associated with nerve root avulsion, with rootlets torn from the spinal cord, and thus carries a poor prognosis. Supraclavicular injuries are more common and more severe and have a worse prognosis than infraclavicular injuries.[50]

Injured patients need to be thoroughly evaluated, determining for head, neck, and shoulder injuries. In open injuries, there could be damage to the great blood vessels and lungs, in which case urgent life-saving operative intervention would be necessary. Clavicular integrity should be assessed in such cases. Along with motor and sensory deficit at the shoulder and or upper limb, the presence of signs of Horner's syndrome suggest complete lower trunk plexopathy as the sympathetic ganglion for T1 is in close proximity to the brachial plexus. In a stretching injury, MRI findings of asymmetric thickening, T2 hyperintensity, and diffuse contrast enhancement of the injured plexus are observed.[51] The MRI in pre-ganglionic injuries may show root avulsion, pseudomeningocele (a tear in the meningeal sheath around the nerve roots with extravasation of the CSF in the neighboring tissue), enhancement of the root exit zone, signal-intensity changes in the spinal cord at the level of root avulsion and/or paraspinal muscles and avulsion of the spinal cord.[51,52] In post-ganglionic lesions, enhancing nodular thickening (neuroma) and hematoma in the vicinity of the plexus are common imaging findings.[51] Most researchers are of the view that in an acute situation, surgical interventions like nerve resection or grafting would be difficult to carry out due to the difficulty in assessment of nerve continuity. Once the general condition of the patient is stabilized, a careful neurological assessment is carried out. Prognosis is better when the plexus elements are in continuity and the nerve fibers have neuropraxic injury with minimal axonotmesis. The main limiting factor is the distance between regenerating axon sprouts and end organs; thus, upper plexus proximal muscles recover more likely than hand muscles supplied by the lower trunk. Nerve transfers can be performed to accelerate recovery from pre-ganglionic injuries. Such procedures, performed ideally within 6 months, reduce time to re-innervation by reducing the distance to the site of the nerve injury.[53] Intra-operative motor-evoked potentials help in assessing the functional status of the anterior motor roots and motor fibers. Primary nerve reconstruction, joint fusion and tendon transfers combined result in a decent recovery of functions in many patients.[54]

References

1. Bowen BC, Seidenwurm DJ, Expert Panel on Neurologic Imaging. Plexopathy. AJNR Am J Neuroradiol 2008;29:400-2.
2. Yoshikawa T, Hayashi N, Yamamoto S, Tajiri Y, Yoshioka N, Masumoto T, *et al*. Brachial plexus injury: Clinical manifestations, conventional imaging findings, and the latest imaging techniques.

Radiographics 2006;26:S133-43.
3. Misamore GW, Lehman DE. Parsonage–Turner syndrome (acute brachial neuritis). J Bone Joint Surg Am 1996;78:1405-8.
4. Parsonage MJ, Turner JW. Neuralgic amyotrophy, the shoulder girdle syndrome. Lancet 1948;1:973-8.
5. Kelkar P, Parry GJ. Brachial plexus disorders. In: Noseworthy JH, editor. Neurologic Therapeutics: Principles and Practice. London: Martin Dunitz Publishers; 2003:2065-7.
6. Spillane JD. Localized neuritis of the shoulder girdle. Lancet 1943;2:532-5.
7. Pierre PA, Laterre CE, Van den Bergh PY. Neuralgic amyotrophy with involvement of cranial nerves IX, X, XI and XII. Muscle Nerve 1990;13:704-7.
8. Sierra A, Prat J, Bas J, Romeu A, Montero J, Matos JA, *et al*. Blood lymphocytes are sensitized to branchial plexus nerves in patients with neuralgic amyotrophy. Acta Neurol Scand 1991;83:183-6.
9. Suarez GA, Giannini C, Bosch EP, Barohn RJ, Wodak J, Ebeling P, *et al*. Immune brachial plexus neuropathy: Suggestive evidence for an inflammatory-immune pathogenesis. Neurology 1996;46:559-61.
10. van Eijk JJ, van Alfen N, Tio-Gillen AP, Maas M, Herbrink P, Portier RP, *et al*. Screening for antecedent *Campylobacter jejuni* infections and anti-ganglioside antibodies in idiopathic neuralgic amyotrophy. J Peripher Nerv Syst 2011;16:153-6.
11. Martínez-Salio A, Porta-Etessam J, Berbel A, Alonso A, Gutiérrez-Rivas E, Trueba J. Amyotrophic neuralgia: Review of 37 cases. Rev Neurol 1998;27:823-6.
12. Gaitzsch G, Chamay A. Paralytic brachial neuritis or Parsonage-Turner syndrome anterior interosseous nerve involvement. Report of three cases. Ann Chir Main 1986;5:288-94.
13. Nacır B, Genç H, Çakıt BD, Karagöz A, Erdem HR. Brachial neuritis presenting with isolated long thoracic nerve involvement. Turk J Phys Med Rehab 2009;55:83-6.
14. Cruz-Martínez A, Barrio M, Arpa J. Neuralgic amyotrophy: Variable expression in 40 patients. J Peripher Nerv Syst 2002;7:198-204.
15. Lahrmann H, Grisold W, Authier FJ, Zifko UA. Neuralgic amyotrophy with phrenic nerve involvement. Muscle Nerve 1999;22:437-42.
16. Kumar N, Folger WN, Bolton CF. Dyspnea as the predominant manifestation of bilateral phrenic neuropathy. Mayo Clin Proc 2004;79:1563-5.
17. Gregory RP, Loh L, Newsom-Davis J. Recurrent isolated alternating phrenic nerve palsies: A variant of brachial neuritis? Thorax1990;45:420-1
18. Gaskin CM, Helms CA. Parsonage–Turner syndrome: MR imaging findings and clinical information of 27 patients. Radiology 2006;240:501-7.
19. Scalf RE, Wenger DE, Frick MA, Mandrekar JN, Adkins MC. MRI findings of 26 patients with Parsonage-Turner syndrome. AJR Am J Roentgenol 2007;189:W39-44.
20. Khadilkar S, Deshmukh S, Gupta N. Neuromuscular disorders in the critical care unit. In: Banerji D, Pauranik A, editors. Progress in Clinical Neurosciences. Vol. 26. Delhi: Byword Books; 2012:45.
21. van Eijk JJ, van Alfen N, Berrevoets M, van der Wilt GJ, Pillen S, van Engelen BG. Evaluation of prednisolone treatment in the acute phase of neuralgic amyotrophy: An observational study. J Neurol Neurosurg Psychiatry 2009;80:1120-4.
22. van Alfen N, van Engelen BG, Hughes RA. Treatment for idiopathic and hereditary neuralgic amyotrophy (brachial neuritis). Cochrane Database Syst Rev 2009 Jul 8;:CD006976
23. Johnson NE, Petraglia AL, Huang JH, Logigian EL. Rapid resolution of severe neuralgic amyotrophy after treatment with corticosteroids and intravenous immunoglobulin. Muscle Nerve 2011;44:304-5.
24. Nakajima M, Fujioka S, Ohno H, Iwamoto K. Partial but rapid recovery from paralysis after immunomodulation during early stage of neuralgic amyotrophy. Eur Neurol 2006;55:227-9.
25. Tsao BE, Avery R, Shields RW. Neuralgic amyotrophy precipitated by Epstein–Barr virus. Neurology 2004;62:1234-5.
26. McCarty EC, Tsairis P, Warren RF. Brachial neuritis. Clin Orthop Relat Res 1999;:37-43.
27. Simó M, Casasnovas C, Martínez-Yélamos S, Martínez-Matos JA. Neurological picture. Multifocal acquired demyelinating sensory and motor neuropathy presenting as idiopathic hypertrophic brachial neuropathy. J Neurol Neurosurg Psychiatry 2009;80:674-5.
28. Klein CJ, Dyck PJ, Friedenberg SM, Burns TM, Windebank AJ, Dyck PJ. Inflammation and neuropathic attacks in hereditary brachial plexus neuropathy. J Neurol Neurosurg Psychiatry 2002;73:45-50.
29. Chance PF, Windebank AJ. Hereditary neuralgic amyotrophy. Curr Opin Neurol 1996;9:343-7.
30. Bulusu S, McMillan HJ. A report of hereditary neuropathy with liability to pressure palsy (HNPP) presenting with brachial plexopathy: The value of complete electrodiagnostic testing. Am J Electroneurodiagnostic Technol 2011;51:183-90.
31. Watson LA, Pizzari T, Balster S. Thoracic outlet syndrome part 1: Clinical manifestations, differentiation and treatment pathways. Man Ther 2009;14:586-95.
32. Brantigan CO, Roos DB. Diagnosing thoracic outlet syndrome. Hand Clin 2004;20:27-36.
33. Demondion X, Herbinet P, Van Sint Jan S, Boutry N, Chantelot C, Cotten A. Imaging assessment of thoracic outlet syndrome. Radiographics 2006;26:1735-50.
34. Nord KM, Kapoor P, Fisher J, Thomas G, Sundaram A, Scott K, *et al*. False positive rate of thoracic outlet syndrome diagnostic maneuvers. Electromyogr Clin Neurophysiol 2008;48:67-74.
35. Tolson TD. "EMG" for thoracic outlet syndrome. Hand Clin 2004;20:37-42.
36. Rousseff R, Tzvetanov P, Valkov I. Utility (or futility?) of electrodiagnosis in thoracic outlet syndrome. Electromyogr Clin Neurophysiol 2005;45:131-3.
37. Hanif S, Tassadaq N, Rathore MF, Rashid P, Ahmed N, Niazi F. Role of therapeutic exercises in neurogenic thoracic outlet syndrome. J Ayub Med Coll Abbottabad 2007;19:85-8.
38. Vanti C, Natalini L, Romeo A, Tosarelli D, Pillastrini P. Conservative treatment of thoracic outlet syndrome. A review of the literature. Eura Medicophys 2007;43:55-70.
39. Cherington M, Happer I, Machanic B, Parry L. Surgery for thoracic outlet syndrome may be hazardous to your health. Muscle Nerve 1986;9:632-4.
40. Kori SH, Foley KM, Posner JB. Brachial plexus lesions in patients with cancer: 100 cases. Neurology 1981;31:45-50.
41. Pancoast HK. Superior pulmonary sulcus tumor: Tumor characterized by pain, Horner's syndrome, destruction of bone and atrophy of hand muscles. J Am Med Assoc (JAMA) 1932;99:1391-6.
42. Du R, Auguste KI, Chin CT, Engstrom JW, Weinstein PR. Magnetic resonance neurography for the evaluation of peripheral nerve, brachial plexus, and nerve root disorders. J Neurosurg 2010;112:362-71.
43. Arcasoy SM, Jett JR. Superior pulmonary sulcus tumors and Pancoast's syndrome. N Engl J Med 1997;337:1370-6.
44. Johansson S, Svensson H, Denekamp J. Timescale of evolution of late radiation injury after postoperative radiotherapy of breast cancer patients. Int J Radiat Oncol Biol Phys 2000;48:745-50.
45. Dropcho EJ. Neurotoxicity of radiation therapy. Neurol Clin 2010;28:217-34.
46. Wouter van Es H, Engelen AM, Witkamp TD, Ramos LM, Feldberg MA. Radiation-induced brachial plexopathy: MR imaging. Skeletal Radiol 1997;26:284-8.
47. Todd M, Shah GV, Mukherji SK. MR imaging of brachial plexus. Top Magn Reson Imaging 2004;15:113-25.
48. Shimazaki H, Nakano I. Radiation myelopathy and plexopathy. Brain Nerve 2008;60:115-21.
49. Rovak JM, Tung TH. Traumatic brachial plexus injuries. Mo Med 2006;103:632-6.
50. Midha R. Epidemiology of brachial plexus injuries in a multitrauma population. Neurosurgery 1997;40:1182-8.
51. Yoshikawa T, Hayashi N, Yamamoto S, Tajiri Y, Yoshioka N,

Masumoto T, *et al*. Brachial plexus injury: Clinical manifestations, conventional imaging findings, and the latest imaging techniques. Radiographics 2006;26:S133-43.
52. Carvalho GA, Nikkhah G, Matthies C, Penkert G, Samii M. Diagnosis of root avulsions in traumatic brachial plexus injuries: Value of computerized tomography myelography and magnetic resonance imaging. J Neurosurg 1997;86:69-76.
53. Rohde RS, Wolfe SW. Nerve transfers for adult traumatic brachial plexus palsy (brachial plexus nerve transfer). HSS J 2007;3:77-82.
54. Giuffre JL, Kakar S, Bishop AT, Spinner RJ, Shin AY. Current concepts of the treatment of adult brachial plexus injuries. J Hand Surg Am 2010;35:678-88.

How to cite this article: Khadilkar SV, Khade SS. Brachial plexopathy. Ann Indian Acad Neurol 2013;16:12-8.
Received: 29-09-12, **Revised:** 09-11-12, **Accepted:** 24-10-12

Source of Support: Nil, **Conflict of Interest:** Nil