MRI Neurography Findings in Patients with Idiopathic Brachial Plexopathy: Correlations with Clinical-neurophysiological Data in Eight Consecutive Cases

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Abstract

Objective  Idiopathic brachial plexopathy is a non-progressive disorder characterized by the sudden onset of shoulder pain associated with weakness and sometimes paraesthesia of the arm. Clinical and electrophysiological examinations are the primary diagnostic tools and allow physicians to localize the site of damage. MRI neurography is rarely performed in this setting.

Methods  We herein describe the cases of eight consecutive patients suffering from idiopathic brachial plexopathy. All patients underwent clinical visits, neurophysiological evaluations and MRI neurography.

Results  We confirmed the primary role of clinical and neurophysiological evaluations in the diagnosis of idiopathic brachial plexopathy and demonstrate the usefulness of brachial plexus MRI neurography for confirming the presence of inflammatory changes.

Conclusion  In patients with idiopathic brachial plexopathy, MR neurography is a helpful tool for excluding different aetiologies, such as compression or tumour formation, and/or confirming inflammatory changes.

Key words: idiopathic brachial plexopathy, neuralgic amyotrophy, nerve conduction studies, neurophysiology, MRI neurography


Introduction

Idiopathic brachial plexopathy is a non-progressive disorder characterized by the sudden onset of shoulder pain associated with weakness and sometimes paraesthesia of the arm (1). Since its first description (2), the condition has generally been referred to as Parsonage-Turner syndrome or acute brachial neuritis, although many other different terms have been used (3-6). Many conditions can mimic this disorder, including cervical spondylosis, rotator cuff tears, impingement syndrome, adhesive capsulitis, thoracic outlet syndrome and calcific tendinitis (1, 7). Identifying the cause of damage to the brachial plexus is crucial for selecting proper therapy (7). Clinical and electrophysiological examinations are the primary diagnostic tools for making the diagnosis of idiopathic brachial plexopathy and allow physicians to localize the site of damage (1, 7). Standard X-rays are generally used when compressive causes originating from congenital bony abnormalities (cervical ribs, C7 elongated transverse processes, abnormal first rib or clavicle) are suspected. Contrast-enhanced CT of the chest and abdomen can be used to exclude remote tumours causing paraneoplastic immunemediated syndromes. Brachial plexus MRI with neurography may be used i) when soft tissue congenital abnormalities causing compression (fibrous bands, anterior scalene syndrome) are suspected; ii) to rule out compression/infiltration by neck or pulmonary apex tumours; iii) to

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support a diagnosis of idiopathic brachial plexopathy by demonstrating the presence of inflammatory changes and, in such cases, iv) to confirm the selective involvement of roots, primary trunks or secondary cords (7-9).

We herein describe the clinical, electrophysiological and radiological findings of eight consecutive patients with idiopathic brachial plexopathy.

Materials and Methods

Patients

Eight consecutive unrelated patients were referred to our centre, the referral centre for peripheral neuropathy in the Lazio region, for an evaluation of symptoms suggestive of brachial plexopathy. We obtained a full history from all patients, including neurological symptoms, date of onset, evolution and comorbidities. All patients underwent detailed neurological examinations.

Laboratory tests

All patients underwent extensive laboratory studies to rule out possible causes of neuropathy, including measurement of the levels of fasting plasma glucose, glycosylated haemoglobin, TSH, antithyroid antibodies, serum vitamin B12 and folate, hepatic enzymes, creatinine, antinuclear antibodies (ANA), anti-extractible nuclear antigens, anti-DNA antibodies, antineutrophil cytoplasmic antibodies (ANCA), anti-Hu antibodies, anti-GM1 antibodies and circulating C3 and C4, a urinalysis, immunofixation electrophoresis and serologic tests for HBV, HCV and HIV.

Neurophysiological studies

Electromyographic examinations (EMGs) and nerve conduction studies (NCS) were performed in the upper and lower limbs in all patients. The skin temperature was approximately 34°C. Pathological values were defined according to our laboratory normative data or an amplitude of the sensory nerve action potential (SNAP) or compound muscle action potential (CMAP) of less than 50% on the contralateral side.

Radiological studies

All patients underwent standard cervical spine X-rays and MRI in addition to contrast-enhanced CT of the chest and abdomen. Brachial plexus MR imaging was performed in all cases on a 1.5T system (Excite 2; GE Medical Systems, Milwaukee, USA) using axial and sagittal FSE T1-weighted (T1-W) sequences and coronal-oblique and axial Fast Spin Echo T2-weighted (T2-W) sequences with either the frequency-selective Fat Saturation (FS) or Short-Tau Inversion Recovery fat suppression (STIR) technique. The FS technique was first preferred in all patients in consideration of the intrinsic higher signal-to-noise ratio compared to that of STIR (10). However, since the STIR technique is relatively less susceptible to magnetic field inhomogeneity (10), STIR images were also acquired in cases in which incomplete/suboptimal fat saturation was obtained (patients #2, 7 and 8).

The T2-W acquisition planes were oriented according to the individual anatomy, basing on T1-W images. Neurographic T2-W images were obtained and evaluated after subsequent post-processing using Maximum Intensity Projection (MIP) algorithms. Post-gadolinium T1-W FS sequences were also used to help to rule out neoplastic pathology and disclose subtle inflammatory changes.

Results

Extensive laboratory tests, standard cervical X-rays and contrast-enhanced CT of the chest and abdomen were unremarkable in all patients. MRI of the cervical spine showed severe spondylosis with multiple stenosis of the lateral foramina only in case #8, while the results were completely normal in the remaining seven patients.

NCS of the lower limbs proved normal in all cases, excluding the presence of polyneuropathy.

A summary of the clinical, neurophysiological and radiological findings of the patients is shown in Table 1.

The details of the neurophysiological examinations of the patients are provided in Supplementary Tables.

Case 1

A 29-year-old woman was admitted for an evaluation of a six-month history of right hand weakness associated with paraesthesia and painful dysesthesia on the ulnar side of the arm. A clinical examination revealed weakness of the intrinsic hand muscles, with marked atrophy of the thenar muscles and slight atrophy of the abductor digiti minimi (ADM) and interossei muscles with hypoesthesia on the medial side of the right arm; the tendon reflexes were normal in all four limbs. NCS showed amplitude reduction of the right ulnar SNAP and ulnar and median CMAP compared with that observed on the opposite side. An EMG examination demonstrated reduced recruitment in the intrinsic small hand muscles with high-amplitude rapidly firing motor unit potentials without denervation signs (Supplementary Table 1, 2). All of these findings were consistent with a diagnosis of medial cord brachial plexopathy. Right brachial plexus MRI revealed thickening of the C5-C7 roots with asymmetric contrast enhancement (Fig. 1A-C).

The patient was treated with intravenous steroids (methylprednisolone: 250 mg/day intravenously for five days) resulting in marked improvement of the dysesthesia. The patient was discharged with oral steroid therapy (prednisone: 75 mg/day) that was gradually tapered over four months. After three months, a follow-up examination revealed the absence of sensory symptoms and slight improvement of the muscle strength of the ADM and first digit interosseous (strength 5 on the MRC scale). A follow-up neurophysiological examination conducted after six months revealed an increased CMAP amplitude registered from the ADM (7.9...
Table. Summary of Clinical, Neurophysiological and Radiological Findings

<table>
<thead>
<tr>
<th>Case and gender</th>
<th>Age (years)</th>
<th>Disease duration</th>
<th>Clinical findings</th>
<th>Neurophysiological results</th>
<th>Radiological findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>#1, F</td>
<td>29</td>
<td>6 months</td>
<td>right hand weakness associated with paraesthesias and painful dysesthesias in the ulnar side of arm.</td>
<td>medial cord brachial plexopathy.</td>
<td>increased T2 signal and contrast-enhancement of the right C5, C6 and C7 roots.</td>
</tr>
<tr>
<td>#2, M</td>
<td>31</td>
<td>2 weeks</td>
<td>Bilateral weakness of deltoid, infraspinatus and muscles, more pronounced in the right side associated with pain.</td>
<td>motor involvement of C5 root or alternatively of axillary and suprascapular nerve lesion.</td>
<td>increased T2 signal and thickening of the right C6 and C8 roots, middle and inferior trunks, and middle and posterior cords. Milder T2 hyperintensity of the left C8 root and trunks.</td>
</tr>
<tr>
<td>#3, M</td>
<td>53</td>
<td>4 months</td>
<td>weakness of left deltoid, infraspinatus, supraspinatus and biceps muscles.</td>
<td>motor involvement of the left upper primary trunk.</td>
<td>swelling and increased T2 signal of the left lateral cord with abnormal enhancement after gadolinium administration.</td>
</tr>
<tr>
<td>#4, M</td>
<td>48</td>
<td>10 days</td>
<td>weakness of right infraspinatus, supraspinatus, deltoid and biceps muscles.</td>
<td>motor involvement of right primary upper trunk.</td>
<td>moderate T2 signal hyperintensity and thickening of the right C5 and C6 roots.</td>
</tr>
<tr>
<td>#5, M</td>
<td>41</td>
<td>2 months</td>
<td>mild weakness involving all muscles of right arm more pronounced in distal hand muscles.</td>
<td>diffuse involvement of right brachial plexus.</td>
<td>no specific findings.</td>
</tr>
<tr>
<td>#6, M</td>
<td>38</td>
<td>2 months</td>
<td>weakness of right triceps, extensor comuni digitorum and extensor proprius indicis muscles.</td>
<td>motor involvement of right C7 muscles.</td>
<td>thickening and T2 hyperintensity of the right C7 root and middle trunk, with subtle enhancement of the C7 root.</td>
</tr>
<tr>
<td>#7, M</td>
<td>55</td>
<td>6 months</td>
<td>mild weakness of right extensor comuni and small hand muscles.</td>
<td>motor involvement of right lower primary trunk or alternatively of right C7-T1 roots.</td>
<td>mild thickening T2 hyperintensity and contrast enhancement of the right C7 root.</td>
</tr>
<tr>
<td>#8, M</td>
<td>73</td>
<td>5 months</td>
<td>mild weakness of right extensor comuni digitorum and abductor brevis pollicis and marked weakness of first digit intersosseus.</td>
<td>motor involvement of right lower primary trunk or alternatively of right C7-T1 roots.</td>
<td>cervical spondylotic changes between C5 and D1 levels. Swelling and T2 hyperintensity of the right brachial plexus C7 root.</td>
</tr>
</tbody>
</table>

Age: current age, Disease duration: time from symptoms onset to clinical, neurophysiological and radiological investigation, M: male, F: female

mV) and a reduction in the mean latency of the F wave originating from the ulnar nerve (28.8 ms), while the other parameters were substantially unchanged. At the last follow-up visit conducted after two years, the results of the neurological examination were unchanged.

Case 2

A 31-year-old man was admitted for an evaluation of a two-week history of bilateral shoulder weakness associated with pain. A clinical examination revealed weakness of the deltoid and infraspinaus muscles that was more pronounced on the right side (Supplementary Table 3). A sensory examination showed mild hypoesthesia in the right deltoid region. The patient’s tendon reflexes were unremarkable. NCS performed two weeks after symptom onset were unremarkable. An EMG examination revealed denervation potentials (fibrillation; positive sharp waves) with reduced recruitment in both deltoid, infraspinaus and supraspinatus muscles that was more pronounced on the right side (Supplementary Table 4). These findings suggested motor involvement of the C5 root or, alternatively, axillary and suprascapular nerve lesions. Brachial plexus MRI showed the presence of bilateral abnormalities that were more pronounced on the right side (Fig. 2).

The patient was treated with IVIg (2 mg/kg over five days), with resolution of pain. After one month, a follow-up examination revealed slight improvement of the patient’s strength weakness. At the last follow-up visit conducted ten months after symptom onset, the patient had fully recovered in the left arm, while mild strength impairment (grade 4 on the MRC scale) persisted in the proximal right arm muscles. The patient refused to undergo follow-up neurophysiological
Case 3

A 53-year-old man was admitted for an evaluation of a four-month history of left arm weakness. A clinical examination revealed weakness of the left deltoid, infraspinatus, supraspinatus, and biceps muscles (grade 1 on the MRC scale). The patient’s left bicipital reflex was absent. A sensory examination and other tendon reflexes were unremarkable. NCS performed four months after symptom onset revealed only a reduced CMAP of the left axillary nerve. An EMG examination showed denervation potentials (fibrillation; positive sharp waves) with reduced recruitment and high-amplitude rapidly firing motor unit potentials in the left deltoid, infraspinatus, supraspinatus, and biceps muscles (Supplementary Table 5, 6). These findings suggested motor involvement of the left upper primary trunk. Left brachial plexus MRI showed thickening and contrast enhancement of the lateral cord (Fig. 3A-C).

The patient was treated with oral steroid therapy (prednisone: 50 mg/day) that was gradually tapered over four months. A follow-up visit conducted after one year revealed only mild impairment of the proximal left arm muscles (grade 4 on the MRC scale). Follow-up NCS conducted after one year revealed a marked increase in the left axillary nerve CMAP amplitude (4.5 mV). The NCS findings of other nerves in the left upper limb were unchanged. An EMG examination of the proximal left arm muscle showed the disappearance of denervation signs and confirmed the presence of a chronic neurogenic pattern. At the last follow-up visit conducted after three years, the patient’s neurological findings were unchanged.

Case 4

A 48-year-old man was admitted for an evaluation of a 10-day history of right shoulder pain associated with progressive arm weakness. A clinical examination showed weakness of the right infraspinatus, supraspinatus, deltoid muscles (grade 5 on the MRC scale). The patient’s right bicipital reflex was absent. A sensory examination and other tendon reflexes were unremarkable. NCS performed four months after symptom onset revealed only a reduced CMAP of the right axillary nerve. An EMG examination showed denervation potentials (fibrillation; positive sharp waves) with reduced recruitment and high-amplitude rapidly firing motor unit potentials in the right deltoid, infraspinatus, supraspinatus, and biceps muscles (Supplementary Table 5, 6). These findings suggested motor involvement of the right upper primary trunk. Right brachial plexus MRI showed thickening and contrast enhancement of the lateral cord (Fig. 3A-C).

The patient was treated with oral steroid therapy (prednisone: 50 mg/day) that was gradually tapered over four months. A follow-up visit conducted after one year revealed only mild impairment of the proximal right arm muscles (grade 4 on the MRC scale). Follow-up NCS conducted after one year revealed a marked increase in the right axillary nerve CMAP amplitude (4.5 mV). The NCS findings of other nerves in the right upper limb were unchanged. An EMG examination of the proximal right arm muscle showed the disappearance of denervation signs and confirmed the presence of a chronic neurogenic pattern. At the last follow-up visit conducted after three years, the patient’s neurological findings were unchanged.
Figure 3. a. T2-W FS neurography shows swelling and an increased signal in the lateral cord of the left brachial plexus that is further illustrated in detail in (b). Diffuse abnormal enhancement following gadolinium administration at the same level (c) further demonstrates the presence of acute inflammatory changes. The small increase in the amount of articular fluid in the left shoulder had no clinical relevance.

Figure 4. T2-W FS neurography reveals moderate signal hyperintensity and thickening of the right brachial plexus C5 (solid arrow) and C6 (empty arrow) roots compared to that observed in the contralateral plexus.

and biceps muscles (grade 1 on the MRC scale). The right biceps reflex was absent. A sensory examination was unremarkable. A neurophysiological evaluation performed on admission was unremarkable. A follow-up study conducted after one month revealed amplitude reduction of the CMAP of the right axillary and musculocutaneous nerves. The sensory NCS findings were unremarkable. An EMG examination showed denervation potentials (fibrillation; positive sharp waves) with markedly reduced recruitment in the right infraspinatus, supraspinatus, deltoid and biceps muscles. All of these findings were suggestive of motor involvement of the primary upper trunk (Supplementary Table 7, 8). Right brachial plexus MRI consistently revealed thickening of the C5 and C6 roots (Fig. 4).

The patient was discharged without therapy. A follow-up visit conducted after six months revealed marked improvement of the patient’s strength impairment (grade 4 on the MRC scale in the deltoid and grade 3 in the biceps). At the last follow-up visit conducted after one year, only mild strength impairment of the biceps (grade 4 on the MRC scale) was detected. A follow-up neurophysiological evaluation conducted after six months showed increased CMAP amplitudes of the axillary (3.6 mV) and musculocutaneous (2.6 mV) nerves. An EMG examination confirmed a chronic neurogenic pattern with high-amplitude rapidly firing motor unit potentials in the same muscles.

Case 5

A 41-year-old man was admitted for an evaluation of a two-month history of right neck pain associated with progressive arm weakness. A clinical examination showed mild weakness involving all muscles of the right arm that was more pronounced in the distal hand muscles. The patient’s tendon reflexes and sensory examination findings were unremarkable. NCS performed two months after symptom onset revealed slight amplitude reduction of all right CMAPs and SNAPs compared with those observed on the opposite side. An EMG examination showed mild reduction of recruitment in all tested muscles (Supplementary Table 9, 10). Sporadic fibrillation potentials were also detected in the right small hand muscles. All of these findings suggested diffuse in-
volvement of the brachial plexus. MRI of the brachial plexuses showed no specific findings in either plexus (Fig. 5).

The patient was treated with oral steroid therapy (prednisone: 75 mg/day) that was gradually tapered over five months. After six months, he revealed complete resolution of his symptoms. The results of follow-up NCS were unchanged. An EMG examination confirmed a chronic neurogenic pattern in all right arm muscles without denervation signs. At the last follow-up visit conducted after one year, the neurological examination was unremarkable.

**Case 6**

A 38-year-old man was admitted for an evaluation of a two-month history of neck pain associated with mild right arm weakness. A clinical examination revealed weakness of the right triceps, extensor communis digitorum and extensor proprius indicis muscles (grade 4 on the MRC scale). A sensory examination was unremarkable, while the right triceps reflex was absent. NCS proved normal. An EMG examination showed denervation potentials (fibrillation; positive sharp waves) with reduced recruitment in the right triceps, extensor communis digitorum and extensor proprius indicis muscles (Supplementary Tables 11, 12). These findings suggested motor involvement of the right C7 muscles. Right brachial plexus MRI confirmed an increased T2 signal with contrast enhancement of the C7 root and middle primary trunk (Fig. 6A, B).

The patient was treated with intravenous steroids (methylprednisolone: 250 mg/day intravenously for three days) followed by oral therapy (prednisone: 75 mg/day) that was
F i g u r e 7. Mild thickening and hyperintensity of the right C7 root is depicted on frontal (a) and right-oblique (b) MR STIR neurography. The corresponding right-oblique post-gadolinium T1-W image reveals subtle enhancement of the nerve root (c).

gradually tapered over six months. After six months, the patient’s clinical symptoms completely resolved. At the last follow-up visit conducted after one year, the neurological examination was unremarkable.

**Case 7**

A 55-year-old man was admitted for an evaluation of weakness in the right hand that began six months earlier associated with arm and neck pain. A clinical examination revealed only mild weakness of the extensor communis digitorum (grade 4 on the MRC scale) and small hand muscles (grade 3 on the MRC scale). NCS revealed only a prolonged mean F wave latency of the right ulnar nerve. An EMG examination showed denervation potentials (fibrillation; positive sharp waves) with reduced recruitment in the right extensor communis digitorum, extensor proprius indicis, first digitii interosseus and abductor brevis pollicis muscles (Supplementary Table 13, 14). These findings suggested motor involvement of the right lower primary trunk or, alternatively, the right C7-T1 roots. Right brachial plexus MRI confirmed an increased T2 signal with contrast enhancement of the C7 root (Fig. 7a, b). The patient was treated with intravenous steroids (methylprednisolone: 250 mg/day intravenously for three days) followed by oral therapy (prednisone: 75 mg/day) that was gradually tapered over six months. After six months, the patient’s sensory symptoms disappeared; only mild motor strength impairment persisted in the small hand muscles (grade 4 on the MRC scale). At the last follow-up visit conducted after one year, the patient had recovered completely.

**Case 8**

A 73-year-old man was admitted for an evaluation of progressive right hand weakness lasting for five months. A clinical examination revealed only mild weakness of the extensor communis digitorum and abductor brevis pollicis muscles (grade 4 on the MRC scale) and marked weakness of the first digitii interosseus muscles (grade 2 on the MRC scale). A sensory examination was unremarkable. NCS revealed mild bilateral carpal tunnel syndrome, compression of the right ulnar nerve in the elbow and prolonged mean F wave latencies originating from all upper limbs nerves. An EMG examination showed denervation potentials (fibrillation; positive sharp waves) with reduced recruitment in the right extensor communis digitorum, first digitii interosseus and abductor brevis pollicis muscles (Supplementary Table 15, 16). These findings suggested motor involvement of the right lower primary trunk or, alternatively, the right C7-T1 roots. MRI of the cervical spine showed severe spondylosis with multiple areas of bilateral stenosis of the lateral foramina (Fig. 8a). However, right brachial plexus MRI neurography also revealed swelling and an increased T2 signal in the C7 root (Fig. 8b). The patient was treated with intravenous steroids (methylprednisolone: 250 mg/day intravenously for three days) followed by oral therapy (prednisone: 75 mg/day) that was gradually tapered over six months. At the last follow-up visit conducted after six months, mild weakness (grade 4 on the MRC scale) in the small right hand muscles persisted.

**Discussion**

The brachial plexus (BP), which supplies most of the upper extremities and shoulder, is the most complex structure in the peripheral nervous system. Considering its large size, superficial location and position, the BP can be damaged by different mechanisms, including trauma, inflammation or disorders involving the neighbouring structures (pulmonary, vascular or skeletal) (1, 7).

The precise cause of idiopathic brachial plexopathy is unknown, although a viral aetiology (3, 11), autoimmune mechanisms (12), trauma, physical exercise and surgery (13) have all been proposed.

Idiopathic brachial plexopathy has a reported incidence of 2-3/100,000/year (14, 15). The age range of affected patients is extremely wide, with most patients presenting in the third to seventh decades of life (1). Men are predominantly affected, and bilateral involvement is observed in up to one-
Assessment of brachial plexopathy is invaluable for determining clinical management. Performing an electrodiagnostic assessment of the corresponding lateral foramina with mild swelling and hyperintensity of the right brachial plexus C7 root, as demonstrated on STIR neurography (arrows in b), suggestive of inflammatory changes.

Our small series confirmed the epidemiological data, showing a male predominance (M/F: 7/1) and one case (12.5%) with bilateral involvement. The mean age of onset in our series was 46 years (range: 29-73).

Clinically, impairment of the proximal shoulder muscles is more often observed (approximately two-thirds of cases), while small hand muscles are less frequently affected (1). Almost all patients experience typical, very severe, relentless, neuropathic pain during their attacks, while sensory symptoms, such as hypoesthesia or paraesthesia with a radicular or peripheral nerve distribution, are less frequently noted (1).

In our series, three of the eight (37.5%) patients presented with selective involvement of the proximal arm muscles. Of the remaining five patients, three exhibited small hand weakness (37.5%), one exhibited predominant involvement of the C7 muscles and one exhibited diffuse impairment of all right arm muscles. Seven patients experienced pain at symptom onset, while only two patients (case #1 and #2) demonstrated sensory impairment.

We treated eight patients (seven patients treated with steroids and one patient treated with IVIg), with improvement of symptoms observed in all cases. The good response of idiopathic brachial plexopathy to immunosuppressive or immunomodulatory treatment has been reported in the literature (1, 16).

Clinical visits and electrophysiological examinations are crucial for making the diagnosis of plexopathy (1, 7). Conducting a detailed clinical evaluation is vital for determining the localization and severity of the lesions, both of which have diagnostic and prognostic implications that contribute to clinical management. Performing an electrodiagnostic assessment of brachial plexopathy is invaluable for determining the lesion site. In general, extensive NCS and EMG evaluations are required in addition to contralateral comparison studies (7).

In our cohort, the neurophysiological evaluations revealed pure motor involvement in six cases, involving the proximal arm muscles in three cases (#2, #3 and #4), the right small hand muscles in two cases (#7 and #8) and the C7 muscles in the remaining case (#6). In the other two cases (#1 and #5), a reduction of the SNAP amplitude was detected. In all cases, the clinical and neurophysiological findings were concordant, allowing us to determine the site of the lesion.

However, no test is specific for the diagnosis of Parsonage-Turner syndrome. Electromyography, nerve conduction studies and MRI must be interpreted in light of the patient’s clinical history. The correlations between imaging results and clinical and neurophysiological findings may further increase the overall specificity and sensitivity (15, 16). Moreover, conducting an imaging workup is useful for ruling out other pathologies that can mimic the symptoms, including primitive and secondary tumours, paraneoplastic immunemediated syndromes, bony and soft tissue thoracic outlet syndromes and joint shoulder disease. MRI is the technique of choice in patients with shoulder pain and weakness (17, 18). In our small series, MRI was able to exclude compressive aetiologies in the plexus and support the clinical and neurophysiological findings of idiopathic plexopathy by demonstrating the occurrence of discrete acute inflammatory changes in the brachial plexus structures in seven of the eight patients (#1, 2, 3, 4, 6, 7 and 8). However in one case (#8), nerve conduction studies were misleading, showing bilateral carpal tunnel syndrome, right ulnar nerve compression in the elbow and bilateral prolonged median latencies of F waves originating from the ulnar and median nerves. Furthermore, in this case, cervical spine MRI was also ab-

**Figure 8.** Neuroradiological findings characterized by the concurrence of severe cervical spondylotic changes between the C5 and D1 levels (a coronal T1-W image - arrowheads in a) causing stenosis of the corresponding lateral foramina with mild swelling and hyperintensity of the right brachial plexus C7 root, as demonstrated on STIR neurography (arrows in b), suggestive of inflammatory changes.
normal, showing severe spondylosis with multiple areas of bilateral stenosis of the lateral foramina (thereby explaining the increased F wave latencies). In this case, MRI neurography proved to be an essential tool, providing clues to inflammatory changes that could account for the patient’s acute clinical picture.

On the other hand, a clear correlation between clinical-neurophysiological and MRI data with respect to the site of damage was established in only four cases (#4, 6, 7 and 8), while in one case (#5), there was a mismatch between the imaging findings and clinical and electrophysiological evidence of definitive right lateralized plexopathy.

The MR neurography findings observed in patients with idiopathic plexopathy can be similar to those observed in patients with less common inflammatory neuropathies, such as chronic inflammatory demyelinating polyneuropathy (CIDP) (19) and multifocal motor neuronopathy (MMN) (20). However, T2 signal abnormalities of the brachial plexuses in patients with CIDP tend to be bilateral (19). On the other hand, MMN may be hardly distinguishable based on MR findings alone. Conducting clinical examinations, which generally show slowly progressive, asymmetric distal limb weakness without sensory loss, and neurophysiological investigations, which generally detect motor conduction blocks, is crucial for making a proper diagnosis (20, 21).

Finally, MRI neurography may provide additional information if compared with the findings of neurophysiological examinations in the acute phase of idiopathic brachial plexopathy. In fact, denervation signs may not be detectable for two or three weeks after symptom onset, while radiological alterations appear earlier (see case #2 and #4) (19). Unfortunately, we have not performed follow-up radiological studies in our patients to assess whether the radiological alterations persist after the acute phase of inflammation and the administration of therapy.

We speculate that MRI is sensitive to subclinical and “subneurophysiological” involvement of the plexus roots, although, in most cases, this modality may also be able to more specifically confirm the primary site of involvement and the nature of the lesions.

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**Conclusion**

In conclusion, we confirmed the primary role of clinical and neurophysiological evaluations in the diagnosis of idiopathic brachial plexopathy and the usefulness of integrating these data with the findings of imaging workups. In this setting, we recommend the use of MRI as an important modality that should be routinely performed because it allows physicians to exclude compressive aetiologies and, at the same time, confirm the presence of selective inflammatory changes in most cases.

The authors state that they have no Conflict of Interest (COI).

Marco Luigetti and Emanuele Pravatà contributed equally to this work.

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