High-resolution 3T Magnetic Resonance Neurography of The Lumbosacral Plexus in Several Clinical Scenarios.

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Learning objectives

1. To describe 3T magnetic resonance neurography (MRN) findings in a cohort of subjects with lumbosacral neuropathy.

2. To differentiate the normal and abnormal patterns in terms of nerve root thickening, abnormal T2WI signal and postgadolinium enhancement.

3. To describe abnormal perineural tissue signal, muscles edema, denervation atrophy, and fat infiltration in LS neuropathy.

Background

The ability of discrimination based on the high resolution provided by the techniques of MRN offers a solid morphologic aid in clinical or pre-surgical evaluation and patient management.

Current role of MRN in the evaluation of plexopathy relies on the recognition of abnormal patterns facilitating differentiation from other conditions, assessment of location and extent of pathology formerly challenging due to deep location of the nerves and variable regional muscle innervation.

Findings and procedure details

PROCEDURE DETAILS and FINDINGS

We retrospectively analyzed 62 patients from our institution and LSP branches were identified in all cases.

All images were acquired in a 3 Tesla scanner. Our MRN protocol includes 3D IDEAL T1WI, 3D IDEAL T2WI and 3D CUBE T2WI.

Between subjects with normal findings (n=21), MRN contributed to determine an alternative diagnosis in 7 cases: endometriosis (n=5) and discal herniation (n=2).

The abnormal findings were reported as diffuse nerve root thickening (n=18), focal nerve root thickening (n=14), T2WI signal hyperintensity (n=13), postgadolinium enhancement (n=5).
Findings in surrounding muscles were: edema (n=6), denervation atrophy (n=4) and fat infiltration (n=3).

Established diagnoses were: traumatic lesions (n=3), entrapment (n=7), perineural fibrosis or neuroma (n=7), benign neural sheath tumors (n=7), malignant neural sheath tumors (n=1), acute inflammatory demyelinating polyneuropathy (n=1), chronic inflammatory demyelinating polyneuropathy (n=4) and nonspecific (n = 6).

**LITERATURE REVIEW**

**A) Trauma**

Due to the protection provided by the axial skeleton, direct trauma of lumbosacral plexus (LSP) is unusual. Indirect trauma is usually secondary to spine injury, hip fracture or dislocation, pelvis fracture, iatrogenic injuries in gynecological, colorectal or inguinal hernia surgeries, other conditions such as abdominal aorta aneurism and lesions, such as hematomas or abscesses involving the psoas muscle. [1,2]

On 1943, Seddon [3] described three grades of neuronal injury depending on the severity of the lesion. The mildest type is neuropraxia, where axonal affliction occurs without loss of axonal continuity preserving supporting structures. This damage is usually temporary with full recovery. Axonotmesis is defined by loss of axonal continuity with preservation of connective tissue covering (perineurum, endoneurum and epineurum). Neurotmesis represents the most severe grade of injury, with compromise of axon continuity and perineural sheath. Prognosis depends on neuronal damage, depending on regrowth ability on the first two grades, and on the prompt surgical intervention on the in cases of neurotmesis and severe axonotmesis. Lack of recovery may develop into neuroma in continuity with neural bulb. (Figure 1)
Fig. 1: Coronal and Axial IDEAL T1WI (In-Phase), Axial IDEAL T1WI (Water). Altered signal with loss of fascicular pattern of the right sciatic nerve. Perineural fat planes were also affected in this patient with a previous history of trauma.

References: Radiology Department, FLENI, Buenos Aires, Argentina

B) Entrapment Neuropathies

LSP can be directly affected by extrinsic compression, diffuse infiltration or indirectly by systemic conditions, inflammatory processes or postoperative fibrosis. (Figure 2)

Psoas muscle pathology is one of the major causes of lumbar plexus injury, including lesions secondary to trauma, surgery, hematomas due to anticoagulation therapy, abscesses and tumoral infiltration. Pelvic tumors are usually the cause of psoas muscle tumoral infiltration, however, secondary affection from other neoplasms such as breast cancer, sarcomas, lymphomas and multiple myeloma may also be the cause.

Entrapment of LSP nerve roots may also be associated to infectious disease, sacroiliac osteoarthritis, pelvis and hip fracture as well as postoperative trauma. Colorectal and cervix cancer can directly invade the LSP. [4,5]
Fig. 2: Coronal and Axial IDEAL T1WI (In-Phase). Flattening and hypointensity in T1WI and T2WI of the right sciatic nerve. Perineural fat planes were also affected in this patient with perineural fibrosis.

References: Radiology Department, FLENI, Buenos Aires, Argentina

C) Neoplasms

1. Benign Tumors

Benign tumors of the LSP are by far more frequent than their malignant counterparts, with schwannoma and neurofibroma being the most common. Malignant schwannoma is the fifth soft tissue sarcoma in frequency. Secondary neoplasms are rare in the lumbosacral plexus.

Schwannoma usually grows eccentrically from the neural sheath. It represents 5% of benign soft tissue neoplasms and appears usually between 20 and 50 years of age.[6] (Figure 3)
Fig. 3: Coronal T2WI IDEAL (Water), Sagittal and Coronal T2WI IDEAL (Fat). Axial T2WI IDEAL (Water). Expansive lesion at the right L5-S1 neuroforamen, with distal extension through the S1 spinal root. Right psoas is hypotrophic and has increased signal in T1WI and T2WI due to denervation. Histological analysis revealed a Schwannoma.

References: Radiology Department, FLENI, Buenos Aires, Argentina

There are two neurofibroma sub-types, the solitary and the plexiform. Solitary neurofibroma is the most common, with affection of peripheral nerves rather than the LSP itself. It represents the 5% of soft tissue neoplasms and usually affects young adults in their 20’s and 30’s. [7] (Figure 4)
**Fig. 4:** Coronal T2WI IDEAL (Water). Hyperintense fusiform lesion adjacent to the postganglionic segment of right L5 nerve root. Histological analysis revealed a solitary neurofibroma.

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The plexiform neurofibroma is almost pathognomonic of NF1. It tends to appear during childhood and has a rate of malignization from 8 to 12%. These tumors expand and distort long segments of the nerves and in MR images they show high signal intensity on T2 WI with a "bunch of grapes" pattern. The post-contrast enhancement is similar to the one seen in solitary neurofibromas. [8] (Figure 5)

Schwannomas and neurofibromas share some imaging characteristics in MRN. Both lesions have well defined fusiform morphology rarely exceed 5 cm in diameter. [8,9]
Fig. 5: Coronal and Axial STIR. Axial contrast enhanced axial T1WI. Multiple expansive lesions with high signal intensity on T2WI extending through the lumbosacral plexus, inguinal region and thigh in a patient with history of NF1.

References: Radiology Department, FLENI, Buenos Aires, Argentina

2. Malignant Tumors

Malignant Schwannoma

Malignant schwannoma, neurogenic sarcoma and neurofibrosarcoma are tumors that arise de novo or from a neurofibroma. They account the 5 to 10% of soft tissue sarcomas. [8] 25 to 50% of malignant peripheral nerve sheath tumours (MPNST) are discovered in patients with NF1, arising half of them de novo and half of them from a preexistent neurofibroma.[9] They typically develop in main nerve trunks, in the proximal LSP and in the main nerves of the inferior limbs. They present as gross tumors that affect the whole nerve with proximal and distal extension. Histological analysis typically shows areas of mixoid degeneration, hemorrhage and necrosis.[10] (Figures 6 and 7)
Fig. 6: Coronal T2WI and contrast enhanced axial T1WI FAT SAT. Enlargement of L5-S1 right neuroforamen due to intraforaminal expansive lesion which shows heterogeneously hyperintense signal intensity on T2WI and homogeneous enhancement post gadolinium. Pathological specimen revealed a MPNST.

References: Radiology Department, FLENI, Buenos Aires, Argentina
Fig. 7: Coronal T2WI IDEAL (Water) shows a recurrent MPNST with neural infiltration and thickening.

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Secondary Tumors

Secondary tumors are extremely rare. Primary affection of the LSP denotes an extraganglionar manifestation of non-Hodgkin lymphoma in high-grade B cell lymphomas. They show diffuse thickening of a neural segments with high signal intensity on T2WI and isointensity on T1WI. Post-contrast enhancement is variable. [11,12]

Melanoma can also compromise LSP. Breast and lung metastasis are infrequent.

Though MRI cannot differentiate primary from secondary tumours, knowing patient clinical history is key to reach the diagnosis. Presence of lymphadenopathies, bone lesions, multiplicity of lesions and infiltrating characteristics aim towards metastatic etiology. [13]

D) Polyneuropathy

1. Diabetic Plexopathy

Diabetic polyradiculoneuropathy is the most studied diabetic neuropathy. This condition, also known as diabetic amyotrophy, usually has a subacute course with pain, weakness and weight loss. LSP affection is progressive, bilateral and multifocal.[14,15]

MRN most remarkable abnormality is hyperintense signal of affected nerves in T2WI. Hypertrophy of nerve roots and postcontrast enhancement can also be present. Dependent muscles of a nerve/trunk may show structural changes and hyperintensity on T2WI as a manifestation of subacute denervation. Chronic denervation shows fatty infiltration and muscle atrophy.[16] (Figure 8)
Fig. 8: Coronal T2WI IDEAL (Water). Slight thickening and fascicular pattern of both lumbosacral trunks in a patient with type II diabetes.

References: Radiology Department, FLENI, Buenos Aires, Argentina

2. Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)
CIDP is one of the most frequent forms of LSP polyneuropathy. The diagnosis is based on clinical examination and electrophysiology studies in the majority of cases. Patients usually have sensitive and motor affection in both upper and lower limbs. Less frequently, cranial nerves could be affected. The evolution of the disease lasts between 4 to 8 weeks. [17]

MRN shows hypertrophy and thickening of roots and peripheral nerves in most patients. Thickening of the cauda equina roots can exceptionally be present with obliteration of the lumbar spinal canal.[18,19] (Figure 9)

**Fig. 9**: Coronal and Axial T2WI IDEAL (Water), Coronal T1WI IDEAL (Water) and Coronal DWI. Hyperintensity and thickening of post-ganglionar spinal roots of the lumbosacral plexus from L2 up to S1 in a patient with CIDP.

**References**: Radiolgy Department, FLENI, Buenos Aires, Argentina

3. **Unknown Origin Neuropathy/Plexopathy**

In cases in which clinical evaluation can´t reveal the etiology of the neuropathy, MRN can identify focal or diffuse abnormalities of the LSP that disappear on posterior studies. This is thought to be caused by idiopathic inflammatory or post-viral conditions.[20] (Figure 10)
Fig. 10: Coronal T2WI IDEAL (Water), shows thickening and loss of normal fascicular pattern of the left lumbosacral trunk in a patient with unknown origin plexopathy. 

References: Radiology Department, FLENI, Buenos Aires, Argentina

Images for this section:

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**Fig. 10:** Coronal T2WI IDEAL (Water), shows thickening and loss of normal fascicular pattern of the left lumbosacral trunk in a patient with unknown origin plexopathy.
Conclusion

3T MRN of the LSP was able to differentiate normal from abnormal cases, representing an important tool in the differential diagnosis of lumbosacral plexopathy.

Personal information

References


