3T Magnetic Resonance Neurography: preliminary experience

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Resumen

Propósito. Presentar las ventajas de la secuencia IDEAL (Iterative Decomposition of water and fat with Echo Asymmetry and Least-squares estimation) en la evaluación de los nervios periféricos y plexos braquial (PB) y lumbosacro (PLS) para el diagnóstico de las neuropatías por atrapamiento o compresión, las neuropatías sin atrapamiento y las condiciones subyacentes. La secuencia IDEAL proporciona 4 tipos de imágenes a partir de una sola adquisición, permitiendo la supresión uniforme de la grasa y el agua, y la obtención de imágenes en fase o fuera de fase de agua, grasa o de la combinación de éstas.

Materiales y Métodos. Estudio retrospectivo de enero de 2011 a junio de 2011. Se realizaron 11 neurografías con secuencia IDEAL en resonancia magnética 3T (HDX 3T, GE Healthcare, USA), con bobinas phased array de 8 canales en planos coronal y sagital, cortes de 1,2 - 0 mm, y plano axial 3D spoiled gradient recalled (SPGR) T1, cortes 1 - 0 mm sin y con gadolinio. El campo de visión (FOV) fue variable según el nervio o plexo a explorar.

Resultados. Se encontraron 2 schwannomas (plexo braquial, nervio ciático), 1 neuritis (inflamación por compresión del nervio mediano), 2 casos con neurofibromas múltiples (uno en plexo lumbosacro y ciático y otro en plexo braquial), 3 neuromas postraumáticos (ciático poplíteo externo -CPE-), una avulsión con pseudomeningocele (plexo braquial) y 2 casos sin alteraciones (plexo lumbosacro y ciático poplíteo externo).

Conclusión. En esta presentación preliminar, la neurografía por resonancia magnética (RM) mostró una excelente diferenciación entre el nervio y las estructuras circundantes y permitió reconocer la estructura del nervio y su señal, determinar variantes anatómicas y causas de neuropatías, así como también se pudieron evaluar los cambios de denervación en el grupo muscular involucrado.


Abstract

3T MRI neurography: preliminary experience.

Purpose. Demonstrate the advantages of the IDEAL (Iterative Decomposition of water and fat with Echo Asymmetry and Least-squares estimation) sequence in the evaluation of peripheral nerves, brachial plexus and lumbosacral plexus, for the diagnosis of compression or entrapment neuropathies, non-entrapment neuropathies, and the underlying conditions.

The IDEAL sequence provides 4 types of images from a single acquisition, allowing uniform fat or water suppression and in phase/out of phase images of water, fat or a combination of both.

Materials and Methods. This is a retrospective study, from January 2011 to June 2011. Eleven neurographies were performed on 3T MRI (HDX 3T, GE Healthcare, USA), with 8-channel phased array coils on sagittal and coronal planes, with 1.2-0 mm slices with no gap, axial 3D spoiled gradient recalled (SPGR) T1, with 1-0-mm slice thickness with and without gadolinium injection and variable field of view (FOV) according to the nerve or plexus to explore.

Results. We found 2 schwannomas (brachial plexus and sciatic nerve), 1 neuritis (compression to median nerve), 2 cases of multiple neurofibromas (lumbosacral plexus, sciatic nerve, brachial plexus), 3 traumatic neuromas (peroneal nerve) and 1 pseudomeningocele avulsion (brachial plexus), and 2 with no structural alterations (lumbosacral plexus and peroneal nerve).

Conclusion. In this preliminary experience, the use of high-resolution sequences in magnetic resonance imaging neurography studies provided excellent signal homogeneity, improving the recognition of the nerve structure and signal, the identification of anatomical variations, and causes of neuropathy, as well as the characterization of denervation changes of the affected muscle groups.


INTRODUCTION

Peripheral nerve disease or peripheral neuropathy (PN) affects all age groups, being a major cause of morbidity (1).

The standard classification of peripheral neuropathies divides them into two large groups: those caused by entrapment or compression (cPN) and those in which there is no entrapment (ncPN) (1, 2).

The etiologies of peripheral neuropathy are diverse and include anatomical or extrinsic compression, traumatic injury (penetrating injury or injury from

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stretch and/or friction), inflammatory-infectious processes, tumor invasion, metabolic, ischemic or genetic causes and physical-chemical damage (thermal or radiation injuries) (1).

The study of PNs has traditionally been within the field of neurophysiology with the use of electromyography (EMG), providing functional and qualitative-quantitative data on the conduction properties of the affected nerve (velocity, latency, etc.), serving as a supplement to clinical examination in the delimitation of the affected territory, and thus allowing the identification of the injured nerve location (3).

The advent of the magnetic resonance (MRI) neurography in 1992 (4) made a major contribution to the non-invasive diagnostic study of peripheral neuropathies. Magnetic resonance neurography complements the EMG evaluations providing further anatomical data and allowing characterization of the distribution of muscle involvement, based on signal changes and the trophism of tissue (denervation edema, fatty replacement, atrophy). Thus, it is possible to estimate the time of injury evolution and in many cases to clarify the underlying pathology.

As a result of the growing use of MRI in peripheral neuropathies, this technique has become a widely used diagnostic method.

The standard imaging protocol for the evaluation of PN includes T1- and T2-weighted spin-echo images and T1-weighted images with intravenous gadolinium injection, using variable section thickness. The three anatomical planes are used, as well as the oblique plane with reduced slice thickness to increase resolution at specific locations. In these studies, T1- and T2-weighted images are often complemented with fat saturation images, usually weighted on T2 (T2 Fat Sat, STIR) which, due to the abundant fat in the perineural tissue, provide improved sensitivity for the detection of signal abnormalities and higher specificity for the chronological characterization of such abnormalities in the studied structures (5).

With the appearance of new high-resolution neurography imaging (3D T2 SPACE - Siemens Healthcare, IDEAL - GE Healthcare, T2 CUBE - GE Healthcare, etc) for MRI assessment of PNs, further diagnostic data are available and, often, exploration becomes simpler.

The aim of this study is to report our preliminary experience in the use of high-resolution neurography sequences for the study of PN in high field MRI (3T), to discuss the advantages and disadvantages of the method and illustrate its applications in the evaluation of PN, considering some basic concepts of this pathology.

**MATERIALS AND METHODS**

A retrospective study was performed from January to June 2011. Eleven patients with a clinical and/or electromyographic diagnosis of peripheral neuropathy and an age range between 3 and 61 years (mean age: 25.9 years) were examined (Table 1).

Eleven neurographies were performed on high field MRI (HDX 3 Tesla, GE Healthcare, USA) using high resolution neurographic sequences called IDEAL by the manufacturer (Iterative Decomposition of water and fat with Echo Asymmetry and Least-squares estimation - GE Healthcare, USA) on coronal and sagittal planes with 1.2-0-mm slices (Table 2). Axial T1-weighted 3D SPGR (spoiled gradient recalled) images were performed.

**Fig 1:** Thermal injury (secondary to laser treatment for varicose veins) of the common peroneal nerve. STIR axial image. (a) Increased nerve signal (arrow) and hyperintensity of perineural tissue; (b) IDEAL T2 Fat Sat images allow for the detection of signal abnormalities in the affected nerve and perineural tissues with improved definition of nerve architecture (please note the fascicular pattern of the nerve).
also acquired with 1-0-mm slice thickness, before and after gadolinium injection. Eight-channel phased array coils appropriate for the anatomical region of interest were used. The field of view (FOV) used varied according to the explored nerve or plexus. Clinical data (history, duration of the condition, physical examination, etc) were taken into account, as well as data provided by the EMG when it was performed prior to the MRI exam.

RESULTS

In 2 of the 11 patients studied (Table 1) no structural causes of PN were identified on high-resolution neurographies. Of those cases in which pathology was documented by MRI, only 3 had a history of trauma (2 traffic accidents and 1 secondary to laser treatment for varicose veins in the lower limb) (Fig. 1). We also found: 2 schwannomas (brachial plexus -BP- and sciatic nerve) (Fig. 2); an inflammatory process (neuritis due to compression to median nerve) (Fig. 3); 2 cases of multiple neurofibromas (one involving the lumbo-sacral plexus -LSP- and the sciatic nerve, and the other in the BP) (Fig. 4), of which only one had a history of neurofibromatosis at the time of examination; 1 post-traumatic neuroma (external popliteal sciatic nerve-EPSP-) and 1 avulsion with associated pseudomeningocoele (BP) (Fig. 5).

In cases of cPN with trauma history, signal changes in the affected nerve and surrounding tissues were mainly observed, with hyperintense signal on

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Clinical history</th>
<th>EMG</th>
<th>Neurography findings</th>
<th>Diagnosis</th>
</tr>
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<tbody>
<tr>
<td>Patient 1</td>
<td>F</td>
<td>14 ys-o Chronic low back pain radiating to left lower limb</td>
<td>-</td>
<td>Complex regional neuropathic pain</td>
<td></td>
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<tr>
<td>Patient 2</td>
<td>F</td>
<td>13 ys-o Pain and functional impairment in the territory of the right common peroneal nerve</td>
<td>Myelinic neuropathy with axonal component of the common peroneal nerve</td>
<td>-</td>
<td>Regional neuropathic pain</td>
</tr>
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<td>Patient 3</td>
<td>F</td>
<td>38 ys-o Carpal Tunnel Syndrome</td>
<td>Edema of the median nerve in carpal tunnel</td>
<td>Carpal Tunnel Syndrome</td>
<td></td>
</tr>
<tr>
<td>Patient 4</td>
<td>M</td>
<td>35 ys-o Progressive functional impairment of the foot and associated atrophy</td>
<td>Axonal neurogenic injury and denervation in the L4-L5 territory.</td>
<td>Increased signal - thickening of the common peroneal nerve</td>
<td>Entrapment neuritis of the common peroneal nerve</td>
</tr>
<tr>
<td>Patient 5</td>
<td>M</td>
<td>15 ys-o Trauma after fall from motorcycle</td>
<td>Hypersignal of the right popliteal nerve and muscle atrophy</td>
<td>Traumatic paralysis of the common peroneal nerve</td>
<td></td>
</tr>
<tr>
<td>Patient 6</td>
<td>M</td>
<td>35 ys-o Fall from motorcycle with fracture dislocation of the left sternoclavicular joint</td>
<td>Pseudomeningocoele after left C7-D1 avulsion and secondary to lower trunk brachial plexus avulsion</td>
<td>Post-traumatic avulsion of left T1 root with pseudomeningocoele</td>
<td></td>
</tr>
<tr>
<td>Patient 7</td>
<td>F</td>
<td>61 ys-o Right common peroneal nerve injury secondary to laser treatment for varicose veins</td>
<td>Peroneal nerve injury</td>
<td>Peroneal nerve neuroma</td>
<td></td>
</tr>
<tr>
<td>Patient 8</td>
<td>F</td>
<td>46 ys-o Pain in the neck and right arm</td>
<td>Right supraclavicular Schwannoma</td>
<td>Brachial plexus Schwannoma</td>
<td></td>
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<tr>
<td>Patient 9</td>
<td>M</td>
<td>7 ys-o Non-traumatic acute onset right common peroneal nerve palsy</td>
<td>Image in common peroneal nerve - right peroneal nerve</td>
<td>Neuroma</td>
<td></td>
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<tr>
<td>Patient 10</td>
<td>M</td>
<td>3 ys-o Neurofibromatosis type I and giant neurofibroma in right lower limb</td>
<td>Expansive lesions in lumbo-sacral plexus, gluteal region and lower limbs</td>
<td>Neurofibromatosis type I</td>
<td></td>
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<tr>
<td>Patient 11</td>
<td>M</td>
<td>31 ys-o Operated neck Schwannoma</td>
<td>Bilateral foraminal neurofibromas C4-C5, C5-C6 and C6-C7</td>
<td>Multiple Schwannomatosis</td>
<td></td>
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T2-weighted pulse sequences. A case of loss of nerve continuity and associated pseudomeningocele was also observed.

Non-traumatic cPN demonstrated a frank association between signal changes and adjacent anatomical structures in the upper limb (carpal tunnel) and lower limb (EPSN - fibular head), as well as tumor compression in cases of neurofibromatosis and schwannomas.

Those pathological cases with no compression involvement proved to be morphologically healthy, but with signal changes.

DISCUSSION

Peripheral neuropathies result from an isolated or a group of damaged peripheral nerves with or without involvement of a nerve plexus. They affect all age groups but, depending on which nerve is involved, there may be some sex-specific prevalence (for example, there is a relationship between the female gender and the carpal tunnel syndrome). In addition, PNs are a major cause of morbidity with a significant economic and occupational impact. Thus, in the general population, only entrapment neuropathies (the most frequent) generate approximately 100,000 surgical procedures per year in USA and Europe.

Peripheral neuropathies may be classified into two large groups: those caused by entrapment or compression (cPN) and those in which there is no entrapment (ncPN).

Regarding etiology, in the case of cPN, nerves damage is caused by physical forces from both anatomical and external structures. Such forces, as described by H. Seddon in 1943, cause different grades of nerve damage, called neuropraxia, axonotmesis and neurotmesis, each one with different functional prognoses related to the nerve capacity of regeneration.

Neuropraxia is the lesser degree of severity in case of peripheral nerve injury, as there is nerve injury but no discontinuity of the sheath and axon. It is generally a temporary disorder with regeneration ad integrum.

Axonotmesis is a more severe nerve injury with disruption of the axon but preservation of the encapsulating connective tissue of the nerve (endoneurium, perineurium and epineurium), with capacity for regeneration being variable.

<table>
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<tr>
<th>Table 2: T1- and T2-weighted IDEAL sequence acquisition parameters.</th>
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<tbody>
<tr>
<td>FOV: 16 - 35 cm</td>
</tr>
<tr>
<td>Slice thickness / spacing between slices: 1 - 1.2 mm / 0 - 0.2 mm</td>
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<tr>
<td>Frequency: 320</td>
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<tr>
<td>NEX: 3</td>
</tr>
<tr>
<td>TE: 90 ms (T2) / 9.1 ms (T1)</td>
</tr>
<tr>
<td>TR: 7160 ms (T2) / 575 ms (T1)</td>
</tr>
<tr>
<td>Echo Train: 20 (T2) / 3 (T1)</td>
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<tr>
<td>Matrix: 320 x 256</td>
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Fig. 2. Schwannoma at the right supraclavicular triangle: (a) and (b) IDEAL images, T1-weighted (a) and T2-weighted (b) coronal planes: a nodular lesion can be seen (short arrows) with a hypointense signal on T1 and a hyperintense signal on T2 in relation to structures of the right brachial plexus (long arrows in a); (c) Multiplanar axial and (d) coronal oblique reconstructions of IDEAL T2 Fat Sat images (short arrows). Reconstructions allow to open out the brachial plexus (long arrows in d) and to show the relationship between the tumor and the surrounding nerves.
Finally, the most severe form of entrapment injury of the peripheral nerve is neurotmesis, in which both the axon and perineural sheaths are damaged. The injured nerve loses its capacity for regeneration and this may result in aberrant regeneration processes (as in case of neuromas) (7).

As mentioned above, these three degrees of nerve injuries imply different functional prognoses. Surgical repair is performed in cases of neurotmesis and severe axonotmesis (8).

In patients in whom damage is caused by anatomical compression of the nerve (i.e.: nerve entrapment syndromes), pain is determined both by the pressure exerted on the nerve (a conflict between the container and its content) and the duration of such pressure.

In this type of mechanism, the pressure exerted affects nerve irrigation, first impairing venous blood flow and higher pressures are required to impair arterial blood flow in nerve structures. In case of compression pressures above 80 mmHg, ischemic damage...
becomes irreversible. In addition, if compression becomes chronic (>6 months), the aforementioned ischemic damage is associated with reactive fibrosis.

Non-entrapment peripheral neuropathies include those caused by inflammatory and/or infectious conditions, metabolic and ischemic disorders and physiological changes (thermal or ionizing radiation injuries). These processes may affect both the cytoarchitecture of the axon and the constitution of the myelin sheath at molecular level (dysmyelinating disorders) and structural level (demyelinating disorders), resulting in nerve conduction abnormalities of PNs.

Tumor involvement in PNs may be related to tumors of the nerve itself (i.e.: schwannomas) or from tumors of adjacent structures that provoke nerve compression and/or infiltration.

The study of PNs has traditionally been within the field of neurophysiology, with EMG being the standard diagnostic test. This test evaluates and records the spontaneous or induced electrical activity of a muscle related to its innervating nerve, providing data on the conduction properties of such nerve. Even if electromyographic testing may be performed with surface electrodes, it is more often performed percutaneously, with needle electrodes, for a higher diagnostic efficacy.

With the advent of neurographic sequences in 1992 (6), the diagnostic efficacy of MRI for PN has improved, providing further anatomical details and making possible to trace the course of the studied nerve. Thus, MRI neurography contributes to reveal signs of anatomic conflict in cPN and to rule out such conflicts in ncPN, providing a better assessment of the morphology and signal of the nerve of interest. Due to these advantages, and its noninvasive nature, MRI neurography has become a currently validated method for the study of PN and, often, the first diagnostic test, making possible to complement the functional and qualitative-quantitative data provided by EMG with anatomical data.

As a first step in the evaluation of PN, the normal appearance of the nerve should be taken into account to identify changes in morphology that may be associated with signal abnormalities; the presence of anatomical variants should also be detected.

Consequently, normal peripheral nerves have a fascicular appearance, uniform caliber and well-defined borders, with a straight course without sharp angulations (characteristics that are absent in a large number of peripheral neuropathies). Normal peripheral nerves signal in MRI is isointense to skeletal muscle, with T2 hyperintensity and signal abnormalities of perineural structures with loss of their interface. For detecting these abnormalities and due to the fat content of the nerve, the use of fat saturation imaging (T2 Fat Sat, STIR, SPIR, etc) becomes highly useful, providing increased sensitivity.

Depending on the etiology of nerve injury (e.g., infections or tumors), findings derived from the administration of paramagnetic contrast medium should be considered, which become evident on T1-weighted sequences (to which fat saturation pulses may be added).

Changes in the perineural tissue signal are often associated with changes in the abnormal nerve. This tissue loses its signal homogeneity and there is perineural fat stratification, poor fat saturation in T2 pulses with fat saturation and linear tracts of low signal intensity corresponding to fibrosis.

It is important to highlight the presence of signal changes in muscles innervated by the affected nerve(s).

At an early stage, and due to an early denervation edema, muscle structures show a variable hypointensity on T1 and hyperintensity on T2, with no decrease in signal intensity on T2-weighted fat saturation pulse sequences. These signal changes are not usually associated with significant changes in muscle volume.

Approximately one month after nerve injury, if the harmful agent persists, signs of fatty replacement will be seen in the affected muscle. In this setting, signal intensity gradually increases on T1-weighted images and decreases in fat saturation sequences. In this subacute stage, a slight decrease in muscle volume may be observed; therefore, it may be useful to compare it with the contralateral muscle.

In chronic stages (> 12 months), progressive replacement of muscle fibers becomes evident, and therefore the muscle becomes markedly hyperintense on T1- and T2-weighted images and hypointense on fat saturation images. Even if muscle has a tendency to atrophy with a decrease in volume, in rare cases, an enlargement may be observed due to conspicuous fatty infiltration.

Regarding the study of PN by MRI, traditional image acquisition protocols used standard T1- and T2-weighted images, as well as fat saturation sequences, usually weighted on T2 (T2 Fat Sat, STIR, etc.), with administration of intravenous paramagnetic contrast depending on clinical suspicion.

In our practice, as from January 2011, we have gradually added to 3T MRI neurographies (HDX 3T, GE Healthcare, USA), high-resolution multiple contrast sequences. This recently developed sequence, called...
IDEAL (Iterative decomposition of water and fat with echo asymmetry and least-squares estimation - GE Healthcare, USA) \(^{(20)}\) by the manufacturer, allows to obtain with a single acquisition T1- or T2-weighted images and combine them with fat-only or water-only images, or both, as well as with in-phase or out-of-phase images.

These imaging sequences are derived from the principles first described by Dixon, based on decomposing fat proton signals according to their precession frequency (a widely used principle for in-phase or out-of-phase imaging) \(^{(20)}\).

This type of imaging allows for an adequate exploration and its advantages include optimization of the signal-to-noise ratio and a more uniform fat saturation than that achieved with traditional fat saturation sequences (T2 Fat Sat, STIR, etc), reduced presence of artifacts (because of less field inhomogeneity), and an adequate visualization of the structures being imaged, mainly in challenging anatomies (plexus, nerve canals, etc.). Furthermore, it is important to highlight the possibility offered by these sequences and 3D T1 SPGR and T2 CUBE images for multiplanar and/or curved reformatted images using the workstation.

Probably, the main advantage of this type of imaging is the optimization of the signal-to-noise ratio, as it allows for improved signal homogeneity. This development becomes even more evident in fat saturation sequences, where there is a significant decrease in the incidence of artifacts.

Regarding the different saturation options of IDEAL sequences, we would like to highlight the usefulness of water-saturation and out-of-phase images. Both sequences provide a better delineation of the nerve structure and surrounding tissue.

Anyway, at least some standard imaging sequences should be used, particularly because of the different contrasts between both types of images. Then, we think it would be wise to have a training time, during which both standard and neurographic sequences should be used \(^{(20,21)}\).

In addition, clinical and EMG data should be available to optimize acquisition parameters and improve imaging accordingly. This would avoid to explore vast regions, which affects the length of the examination and, secondarily, its quality.

In our patients, high-resolution neurographies demonstrated pathology in most cases and ruled it out in patients with normal imaging findings in whom structural pathology was suspected based on clinical and / or EMG data.

Regarding imaging findings, the morphology or signal changes of the different nerves that we observed in our group of patients, were consistent with those reported in the literature \(^{(1,2,3,4,5,6,7,8,9,10,11)}\).

For the characterization of associated muscle abnormalities, traditional imaging sequences with a wide FOV are a useful supplement to neurographic sequences for the study of muscle groups innervated by the nerve of interest.

A limitation of the study was the small sample size and the lack of a study comparing standard imaging sequences with high-resolution neurographic imaging. We think that further studies with larger sample sizes are needed to provide further data on this described technique.

CONCLUSION

It is important to be aware of the norm characteristics of peripheral nerves on MRI and their appearance in pathological conditions, considering the changes in signal and volume of muscle structures as a result of their innervating nerve injury.

Although our study did not compare the benefit of these imaging sequences with those of traditional imaging, we may affirm that in this group of patients, the use of high resolution imaging for MRI neurography allowed to detect abnormalities in most cases and to rule them out in 2 cases with clinical suspicion of associated structural pathology. Hence, high resolution neurographic sequences allowed for an adequate visualization of the nerve and the perineural tissue, optimizing their evaluation, mainly by improving signal homogeneity.

References


The authors declare no conflicts of interest.