Susceptibility Weighted Imaging (SWI): Contributions to Multiple Sclerosis

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Learning objectives

Usual MRI sequences used in studies of multiple sclerosis allow detection of lesions both in diagnosis and in the monitoring of disease.

Susceptibility weighted imaging (SWI) is a relatively recent sequence with high sensitivity in the detection of paramagnetic substances: hemosiderin, calcium and deoxyhemoglobin.

This feature, in the case of multiple sclerosis, can see the relationship between the plaques in white matter and venular structures and the association of lesions with different patterns of iron deposition.
Background

Multiple sclerosis is the most common neurological disease of young adult caucasian and the most common cause of neurological disability in young adults.

It is an inflammatory and demyelinating disease characterized by inflammation, demyelination plaques, axonal loss and gliosis.

MRI is nowadays a mainstay in the diagnosis and monitoring of multiple sclerosis. Diagnostic criteria proposed by McDonald et al. (revised 2010) give great importance to the findings of MRI studies with the presence of CNS demyelinating lesions scattered in space and time.

Sensitivity in the diagnosis of multiple sclerosis using classical protocols with MRI sequences (T2, FLAIR, DP, T1 after gadolinium) is very high, but there are nonspecific findings that can be misinterpreted and lead to false positives with a overestimation in the diagnosis of disease. (1)

SWI can increase the specificity of these confusing cases.
Findings and procedure details

It could increase the specificity of the cases more dubious for a diagnosis of multiple sclerosis getting greater detail some of the typical features of lesions of multiple sclerosis as: location (perivenular), distribution (periventricular) and morphology (ovoid form, perpendicular to ventricles: Dawson’s fingers).

In that sense, SWI is an additional sequence providing complementary information to that obtained with conventional sequences in terms of these characteristics.

SWI provides a detailed anatomical information of the cerebral venous system, in which the veins are hypointense structures.

Typical distribution of multiple sclerosis plaques is associated with perivenular location, which can be viewed by SWI. Venular blood supply is abundant in: periventricular deep white matter (Dawson’s fingers) and in the ependymal surface of corpus callosum, so typically, but not specifically, multiple sclerosis lesions are distributed in the interface of corpus callosum unlike in the lesions of another etiologies that affect the entire thickness of corpus callosum (fig1, fig2, fig3).

Dawson fingers, finding typical of multiple sclerosis are the morphological expression of perivenular inflammation around venules extending perpendicular to the ventricular surface. (fig3).

In cases with nonspecific lesions especially located in deep subcortical white matter, without distribution and location so typical of multiple sclerosis, SWI can delimit venules anatomically and detect lesions with perivenular distribution, such that the identification of the venule within problem lesion can help to catalog this lesion as injury multiple sclerosis (fig 3, fig 5) against other lesions in the white matter of another etiology without perivenular relationship (fig 6).

So the visibility of the central vein could be a discriminant score among the white matter lesions of multiple sclerosis of non-multiple sclerosis injuries (2). The central vein within the lesion is identified at 3T with SWI at least 45% of the lesions of multiple sclerosis compared to 8% of the incidental white matter lesions (3).

It is already known the relationship between iron deposits in gray and white matter of the brain parenchyma and the aging and neurodegenerative diseases, such as multiple sclerosis.

During the first four weeks of the inflammatory process, the activity in the lesions of multiple sclerosis is expressed with an enhancement in open ring after administration of gadolinium, as expression of the interruption of the blood brain barrier, during which
extravasation occurs red blood cells, that are kidnapped by microglia cells and whose products metabolic degradation are detected as hypointense signals in sequence SWI.

The detection of those iron deposits by SWI is possible because of is capable discriminating subtle changes in susceptibility between areas with degradation products of the blood and surrounding cerebral tissue.

Those iron deposits detected by SWI within T2 hyperintense lesions no longer enhanced after administration of gadolinium act as witnesses of an prior inflammatory episode. So SWI allows identification of hypointense signals within the lesions of multiple sclerosis (intralesional signal susceptibility) that have not been identified in white matter lesions of other etiologies and translate as extravasation of erythrocytes through the blood brain barrier. (4)

Moreover, the morphology and arrangement of the iron deposits in white matter of MS injuries detected by SWI adopt a characteristic pattern "in border" (fig11), following the edges delimiting the previous enhancement of this lesion (as a result of accumulation of the products degradation of extravasated red blood cells through interruption of the blood brain barrier during inflammatory process) opposite the patterns of iron deposit of another pathologies (fig 14, fig 15).

SWI is a sequence in T2 weighted, 3D, high resolution and a post-processing applied.

SWI is a particularly sensitive in detecting of paramagnetic substances, than in the brain are represented mainly by iron, bound to heme, deoxyhemoglobin and products derived from the destruction of the same. So all those tissues in which those components are involved may be detected with higher sensitivity by this sequence. Compared to T2* sequence presenting better discrimination between tissues paramagnetic and no paramagnetic.

Estimated time to obtain all brain coverage is 3 to 5 minutes, which are used in the signal acquisition and remove incidental phase variations due to non homogeneous static magnetic fields, a process that is automatically achieved by the system providing 3D images. The phase image (previously filtered with a mask) is multiplied to the magnitude image to improve visualization of microvessels. Finally, minIP reconstruction is performed with selected thickness (1-2 cm) generating images to contrast all the hypointense signals produced by the different tissue susceptibility to find hemorrhage, calcium, iron deposits ... 

As limitations of the sequence, we can mention that despite having very good sensitivity, has enough noise that can limit the anatomic location, the quality of the image is relatively poor in areas that are close to the skull base and although provides a detailed identification of the cerebral venous system, it is not good for the superior sagittal sinus venosus.
At present, its main applications include studies of vascular malformations (cavernomas), chronic vasculopathy and future applications are investigated in neoplastic disease and in multiple sclerosis.
**Fig. 1:** FLAIR sagittal: Involvement of the ependymal surface of the corpus callosum showing hyperintense signal in male patient aged 40 diagnosed with multiple sclerosis.

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Fig. 2: FLAIR sagittal: Involvement of the ependymal surface of the corpus callosum showing hyperintense signal in the same patient who is referred in fig.1.

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**Fig. 3:** SWI sagittal: Perivenular distribution of the lesion which is referred in fig.1. Correlating lesion of the corpus callosum ependymal surface in FLAIR sagittal with SWI sagittal showing perivenular distribution of the lesion: venulas as hypointense structures linear perpendicular to the ventricular surface.

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Fig. 4: T2 coronal: Hyperintense injury in periventricular white matter. 43 year old woman with diagnosis MS.

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Fig. 5: SWI coronal: Detail of structure venular within white matter lesion periventricular white as hyperintense T2 signal of the patient who is referred in Fig. 4.

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**Fig. 6:** SWI axial: Locator value of SWI, identifying venular structures in a patient with multiple perivascular Virchow-Robin spaces shown as hypointense signals with linear morphology, which are hyperintense in T2 of Fig7.

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Fig. 7: T2 axial: Perivascular spaces which are referred in Fig. 6 (red arrow). Hyperintense injury in deep subcortical white matter of the left hemisphere (blue arrow) in a patient with known chronic ischemic lesions, in this case, is not distributed as perivenular (No structure as venule is identified within this lesion, probably of ischemic etiology, in SWI in fig.6)

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**Fig. 8:** FLAIR axial: Hyperintense juxtacortical left anterior frontal injury in male patient aged 44 diagnosed of multiple sclerosis.

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**Fig. 9:** Detail of the left anterior frontal lesion yuxtacortical which is referred in Fig. 8.

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Fig. 10: FLAIR coronal: Cortical atrophy predominantly in the left hemisphere in the patient who is referred to in fig8 and fig9.

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Fig. 11: SWI axial: Characteristic pattern of iron deposit of multiple sclerosis, showing hypointense signals, are grouped around the edge, perfectly defined, of the lesion described in fig 8 and fig 9.

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**Fig. 12:** T2 axial: Several lesions in deep subcortical white matter of both hemispheres.

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**Fig. 13:** FLAIR axial: Involvement of periventricular white matter and brain atrophy associated in the patient who is referred in Fig12.

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Fig. 14: SWI axial: Findings described in fig 12 and fig 13 are nonspecific and may be shared by several diseases, including MS. SWI in fig 14 and fig 15 shows a pattern of iron deposit, which in this case is not characteristic of multiple sclerosis, with the presence of multiple and small hypointense signals of anarchic distribution suggestive of vasculitis.

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Fig. 15: SWI axial: Cranial plane to referred in fig14 showing lot of punctate iron deposits in patient with diagnosis of vasculitis.

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Conclusion

SWI is an additional sequence, that together with usual MRI sequences, helps to characterize the white matter lesions detected by the conventional protocol, in multiple sclerosis diagnosis, providing an additional valuable information for the diagnosis of CNS pathology, particularly for debugging the differential diagnosis of lesions of multiple sclerosis respect to other lesions of white matter nonspecific, especially in subcortical distribution.

Offers interesting insights for understanding the pathophysiology of this disease, and the basis for future research.

It could be useful as predictive early marker of inability to identify abnormal iron deposits, which are the first step in the development of neurodegenerative diseases, such as multiple sclerosis.
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