



ORIGINAL ARTICLE

Comparison between Gadolinium Enhanced Susceptibility Weighted Imaging and Conventional T1-Weighted Imaging in Optimizing the Recognition of Active Plaques of Multiple Sclerosis

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ABSTRACT

Background: The Gd-SWI sequence is a new important magnetic resonance imaging sequence that has been applied currently due to its ability to optimize the recognition of active plaques of MS and has a higher accurate performance than gadolinium T1WI that is currently used in clinical practice. The aim of this study is comparing the performance of the Gd-SWI sequence with the performance of other used sequences in clinical practice in detection of blood brain barrier (BBB) dysfunctions in MS plaques.

Methods: In this retrospective study, Twenty four patients were involved (16 females and 8males, their age ranging from 20- 42 years with the mean age 26.33 years), they were divided into two groups: 8 active cases and 22 inactive cases clinically and radiologically proven MS cases for MRI diagnosis and follow up.

Results: Two thirds of the studied group (66.7 %) had inactive lesions and one third (33.3 %) had active ones with (12.5 %) of both active and inactive lesions were progressive. There was higher ability of post-contrast SWI to demonstrate active plaques than post-contrast TIWI and there was highly statistically significance and good agreement (kappa=0.71) with 89.5% percent of agreement between post contrast enhanced lesions by SWI and T1 and statistically highly significant difference (p<0.001).

Conclusions: Gadolinium –SWI technique improved detection accuracy of MS- contrast-enhanced plaques better than Gd-T1 SE, including their locations and morphologies.

Keywords: Susceptibility Weighted Imaging, Multiple sclerosis, Magnetic Resonance Imaging, T1WI.



INTRODUCTION

MS is one of the world's most common neurologic disorders, and in young adults, it is the primary reason of non-traumatic neurologic disability in many places. Conventional T1WI and T2WI sequences are not sufficient in diagnosis of some cases of MS and cannot detect the activity of new plaques of MS and cannot determine if MS was progressive or not [1].

Magnetic Resonance Imaging has an important role in multiple sclerosis evaluations and recommending a necessity for an examination initially in (first six months) and (every twelve

months) a follow-up scan should be performed for detecting appearance of new T2 lesions and looking for active inflammation plaques using the gadolinium T1WI technique. MS plaque enhancement with gadolinium (Gd) is an established biomarker of multiple sclerosis plaques inflammation [2].

Because contrast enhancing lesions indicate blood brain barrier BBB (Blood brain barrier) impairment and the inflammatory response, this marker was mostly used in diagnosis and monitoring of MS lesions. Gd-enhancing lesions have also been established in recent research to have a prognostic role and clinical

correlation as a long-term independent predictor in clinically isolated syndromes [3].

SWI is a recent important sequence of MRI that is being applied clinically besides traditional T1WI and T2WI sequences used in available MRI scanners. A central vein sign inside white matter lesions have been investigated by a number of studies. SWI images have detected the “central vein sign,” which can add specificity to the MS diagnosis as a promised imaging marker of inflammatory demyelinating process. [4].

A high rate of central vein sign detection has also been shown by 1,5 tesla MRI machine. it is demonstrated a presence of non-confluent lesions three mm long with one central vein as a specific and sensitive discriminator of control individuals with other benign white matter lesions from relapsing-remitting MS patients [5, 6].

Hosseini et al also conducted a study that proved a hypointense rim around MS plaques; this additive promised marker for MS, can probably be utilized in addition to central vein as a radiological sign to discriminate other white matter lesions from MS [7].

Furthermore; information is provided by SWI about any structure having an altered susceptibility than its adjacent tissues, for example calcium, ferritin, hemosiderin and deoxygenated blood (that are exhibited by the T2* effect) [8].

In light of this, the goal of this work was to compare the Gd-SWI technique accuracy in detecting BBB dysfunctions in brain MS lesions to the other techniques performance currently utilized in clinical practice.

METHODS

This study has been conducted on 24 patients (16 females and 8males, their age ranging from 20- 42 years with the mean age 26.33), they were divided into two groups: 8 active cases and 22 inactive cases clinically and radiologically proven MS cases for MRI diagnosis and follow up, the patients were referred from the Neurology Department and Multiple Sclerosis clinic at Zagazig University Hospitals, to Radio diagnosis Department,

Magnetic Resonance Imaging (MRI unit), Hospitals of Zagazig University, over duration of 11 months starting from August 2020 to July 2021. Patient accepting the study of any sex and any age group with a defined MS diagnosis or suspicious to have MS according to 2017 revised McDonald Criteria were included in our study. Patients with very bad general condition, unwilling to complete the study, having contraindications to magnetic resonance imaging (MRI) (e.g. cochlear implants, metallic foreign bodies, cardiac pacemaker, ferromagnetic aneurysm clips), suspected unavailability throughout the study or with other neurologic disease or brain SOL were withdrawn.

The study was conducted with institutional review based board (IRB) approval and written informed consents were taken from all patients. Our study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Patient preparation:

Participants in this study had been exposed to full history taking, general plus neurological examination, kidney function tests and MR imaging including conventional magnetic resonance imaging (T1WI, T2WI and FLAIR),and advanced techniques as(Gadolinium enhanced MR imaging, Diffusion Weighted Imaging & ADC map as well as Susceptibility Weighted Imaging (SWI).

Image acquisition:

Preparation:

Any contraindication to MRI examination were excluded by asking patients to remove any metallic subjects and also we exclude patients who have (joint prosthesis except titanium prosthesis, metallic stents, cardiac pacemaker, artificial heart valve),some instructions were given to patients about their position, examination duration, and being immobile during the examination.

Technique:

While the patient in supine position, all data was obtained by a MRI closed scanner (1.5 T),

(Philips Achieva) using encoding neurovascular coil with 16-channel sensitivity.

Sagittal volumetric FLAIR images was acquired using these parameters (acquisition time, 8 minutes and 31 seconds; slice thickness, 0.7 mm; FOV, 220 x 220 x 180 mm³; and matrix, 184 x 184; TR/TE/TI,7.000/263/ 2.300 ms).

Gd-SWI sequence was acquired with a using these parameters (acquisition time, 3minutes and 20seconds ,slice thickness, 1mm; matrix, 160 x 172x100; FOV, 200 x 211x 150 mm³; TR/TE, 52/12 ms; voxel size, 3.0 x 1.24 x 1.25 mm³; flipangle10°).

The 2D-T1WI data were acquired using these parameters:(acquisition time, 1 minute and 45 seconds; TR/TE, 614/15 ms; matrix, 244 x 168, FOV, 220 x 189 x 126 mm³; slice thickness, 5 mm, 25 slices) Gd (Gadovist [gadobutrol]; (0.1 mmol/kg) was also administered intravenously before and after. The order in which the postcontrast sequences were acquired was similar across all studies. The first obtained sequence was SWI, and then the T1WI sequence.

Diffusion Weighted MR Imaging (DWI): The imaging sequence for DWI was a multi-section single shot spin echo EPI sequence (TR/TE/NEX: 4200/140 ms/I) with diffusion sensitivities of b values = 0, 500 and 1000 s/mm². The diffusion gradients were applied sequentially in three orthogonal directions (X, Y & Z directions). Sections of 5 mm thickness, interstice gap of 1mm, FOV 240 mm and a matrix of 128x256 were used for all images. The total acquisition time was 80 sec.

Three types were obtained; orthogonal images, trace images and ADC maps. The ADC maps were calculated automatically by MRI software and included in the sequence. Measurements of ADC were made in different regions of interest (ROI) [on the lesions and in Normal Appearing White Matter (NAWM)]. The ADC values were expressed in 10⁻³mm²/sec.

Trace (D) was computed by summing ADC z x ADC y x ADC x, and on a pixel-by-pixel basis after assuming that the produced diffusion sensitization by the imaging gradients was

insignificant. To obtain the orientationally averaged ADC, trace (D) was divided by 3. The averaged water diffusion coefficient was measured at MS plaques. Measurements of ADC values were made in ROIs within the lesions.

Image reconstruction and interpretation:

Three experienced radiologists assessed all images; Gd-SWI and Gd-T1 SE were studied individually by the observers, and the data were always evaluated in that order. The interpretation of the pre-contrast FLAIR sequence was always evaluated to corroborate the lesion features. To define a lesion as enhanced on Gd- SWI, strict criteria were used; all definite enhancing lesions were included. Gd-enhancing lesions were classified as nodular, annular, punctate, or tumefactive, based on their location in periventricular, subcortical/juxta-cortical, or infra-tentorial areas. Similarly, we conducted a separate comparison of Gd-T1 SE interpretation to look for either a similar Gd enhancement in SWI lesions or a Gd-SWI acquisition that was negative for the presence of lesions enhancing in the other sequences.

Statistical analysis:

Microsoft Excel software was used to code, enter, and analyze data obtained during the history, clinical examination, laboratory investigations, and outcome measures. Statistical Package for the Social Sciences program was used for analyzing data. The following tests were employed depending on the type of data.

Unless otherwise indicated, mean (M) ± Std. Deviation (SD) expressed all quantitative data. Checking differences in continuous measurements is done by using Independent samples t test.

For comparing qualitative variables, the Fisher's exact test (or Chi-square test, if appropriate) was utilized. The best cutoff point for ADC value was determined using ROC curve, which was based on the following principle: specificity plus sensitivity has the highest number.

All tests were two tailed and parameters were considered significant with $p < 0.05$ and statistically highly significant difference ($p < 0.001$).

RESULTS

Twenty four patients were involved in the study, the average studied group age was (26.3 ± 8.3) ranged from 20 to 51 years with two thirds (66.7%) of the studied group was females. Two thirds of the studied group (66.7 %) had inactive lesions and one third (33.3 %) had active ones with (12.5 %) of both active and inactive lesions were progressive.

Distribution of MS lesions:

Most of lesions were periventricular (48.4 %) followed by cortical & subcortical (40.3 %) then brain stem, cerebellar, callosal-septal and U fibers (4.9 %, 2.6 %, 2.1 % and 1.6%) respectively (Table 1).

Comparing pre and post contrast non-enhanced lesions by SWI:

There was statistically significance higher ability of post-contrast SWI to demonstrate lesions with positive central vein than pre-contrast SWI and in contrast there was statistically significance decrease on number of homogenous hypointense lesions with post contrast than pre-contrast while regarding scattered hypointense dots and peripheral hypointense rim, they were more obvious by post contrast than pre contrast but not different enough to be significant and also isointense lesions not seen were more among pre-contrast than post-contrast (Table 2).

Concordance between T1 and SWI regarding post contrast enhanced lesions among the studied group:

There was highly statistically significant good agreement ($\text{kappa}=0.71$) with 89.5% percent of agreement between post contrast enhanced lesions by SWI and T1 (Table 3).

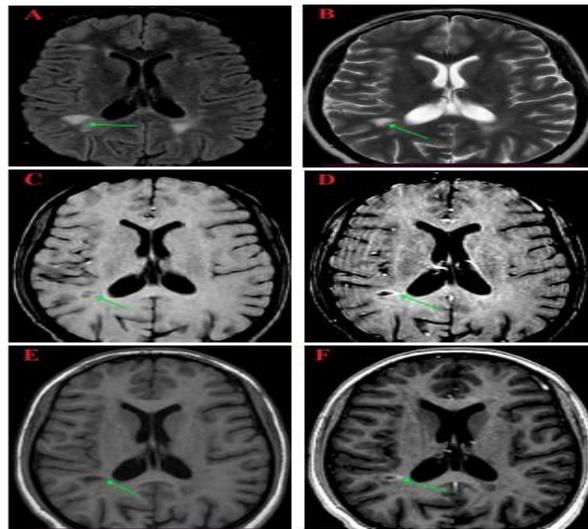


Fig. (1): A 22-year-old female patient, presented with right hemi-paresis, headache and generalized weakness of one-week duration. (The initial MRI) A & B): axial FLAIR and Axial T2WI: Show right periventricular and subcortical white matter hyperintense plaques, C): Axial pre contrast SWI: white matter hypointense plaques, D): Axial post contrast SWI: strong ring pattern of enhancement of one plaque which demonstrate BBB dysfunction in the plaque that is characterized more obvious by the GD-SWI sequence, E & F): Axial T1WI (pre and post contrast study): The pre contrast T1WI (D) shows periventricular and subcortical white matter hypointense plaques, the post contrast T1WI (E) shows faint ring pattern of enhancement of one plaque (arrow).

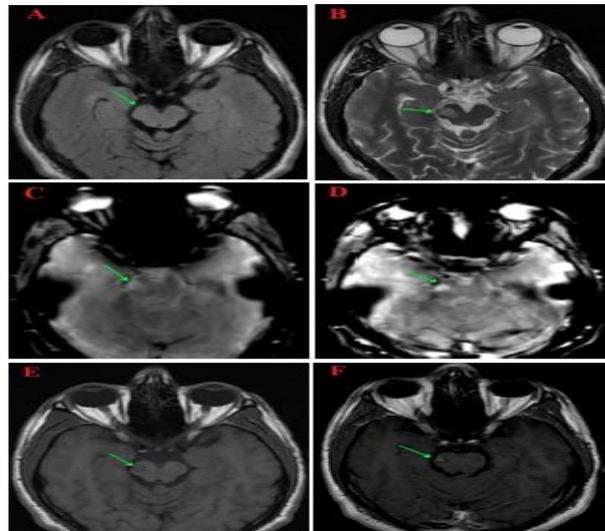


Fig. (2): A 22-year-old male patient, presented with, lumbar weakness and visual troubles of two weeks duration. (The initial MRI) A & B): Axial FLAIR and axial T2WI: Show right midbrain hyperintense plaque on T2WI and isointense plaque on FLAIR, C): Axial pre contrast Susceptibility Weighted Images (SWI): Show right midbrain hyperintense plaque, D): Axial post contrast Susceptibility Weighted Images (SWI): shows strong homogenous nodular pattern of enhancement of plaque which demonstrate BBB dysfunction in the plaques that is characterized more obvious by the GD-SWI sequence more than post contrast T1WI, E & F): Axial T1WI (pre and post contrast study): The pre contrast T1WI (E) shows right midbrain hypointense plaques, the post contrast T1WI (F) shows faint nodular pattern of enhancement of plaque (arrow).

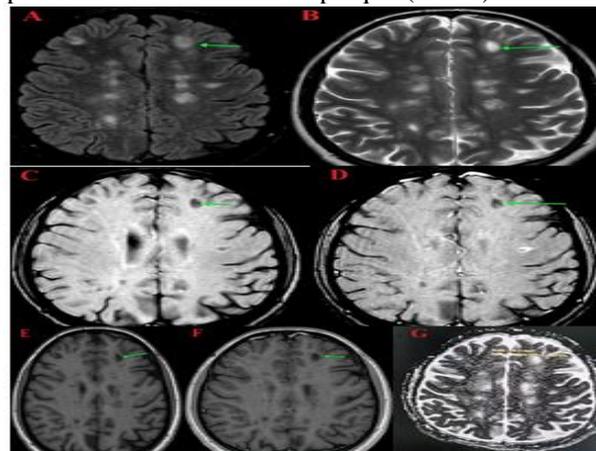


Fig. (3): A 22-year-old female patient, presented with right hemi-paresis, headache and generalized weakness of one week duration. (The initial MRI) A & B): Axial FLAIR and axial T2WI: Show large left sub cortical white matter hyperintense plaques, C): Axial SWI: characteristic incomplete rim of hypointense dots in the periphery of the plaque (arrows) which demonstrate iron deposition in periphery of lesion, D): Axial post contrast SWI: two plaques the first show complete ring pattern of contrast enhancement of one plaque and the second one show ring-shaped distribution of hypointense susceptibility dots, E & F): Axial T1WI (pre and post contrast study): shows subcortical white matter hypointense plaques, G): ADC map: The plaques are hyperintense on ADC map (G). The ADC value of MS plaque of (NEL) on the left side (ROI1) is elevated = $1.550 \times 10^{-3} \text{ mm}^2/\text{second}$. *** The clinical and the radiological findings are consistent with primary progressive case of multiple sclerosis. The current MRI revealed progressive course of the MS disease in the form of elevation of the ADC value of the old plaques and presence of peripheral hypointense rim in GD-SWI sequence.

Table 1: Distribution of lesions among the studied group

Distribution	Number of lesions (range/ one patient)	%
Cortical & sub cortical	154 (1 – 35)	40.3 %
Periventricular	185 (1-31)	48.4%
Brain stem	19 (0 – 4)	4.9 %
U Fibers	6 (0 – 1)	1.6 %
Callososeptal	8 (0 – 1)	2.1 %
Cerebellar	10 (0 – 3)	2.6 %
Total	382	100 %

Table 2: Comparing pre and post contrast non-enhanced lesions by SWI

Lesions	Pre-contrast No (range/one patient)	Post-contrast No (range/one patient)	X ²	P value
Lesions with positive central vein	109 (0-33)	132 (0-33)	5.4	0.02*
Scattered hypointense dots	3 (0-3)	4 (0-4)	0.2	0.6
Peripheral hypointense rim	4 (0-1)	5 (0-1)	0.17	0.6
Homogenous hypointense lesions	152 (0-17)	112 (0-12)	6.5	0.01*
Isointense lesion not seen	99 (0-17)	95 (0-17)	0.01	0.9
Total	367	348		

SWI: Susceptibility Weighted Imaging

Table 3: Concordance between T1 and SWI regarding post contrast enhanced lesions among the studied group

SWI	T1		Kappa agreement	P value
	Homogenous enhancing lesions	Ring shaped enhancing lesions		
Homogenous enhancing lesions (No=14)	8	3	0.71	0.001*
Ring shaped enhancing No=9	0	9		
Total =23	8	12	-	-

DISCUSSION

Non-traumatic neurologic disability is mainly caused by MS in young adults in many nations.

At different phases of disease progression, it is primarily defined by the separate processes of inflammation, demyelination,

neurodegeneration, re-myelination, and axonal repair in distinct multifocal combinations [1, 9]. Despite the fact that activity and progression are nearly similar, their analysis is done separately in clinical practice. Lesion activity in MR imaging and/or Clinical relapses are used to define disease activity, which is linked to tissue damage episodes and inflammation [11]. Increased neurologic dysfunction is connected to progression, which (according to present knowledge) reflects neurodegenerative processes [12]. As per the current criteria [10]. Detecting the BBB dysfunction represented by Gadolinium enhancement and MRI determining lesions load (increase in the pre-existing lesions volume or the new lesions number) are used to perform active disease estimation that help in therapeutic evaluation analysis [2].

SWI is a recent MRI technology that is used currently in clinical practice. It has been documented that the post-Gd SWI is more sensitive technique in detection of a blood brain barrier dysfunction with MS inflammatory activity (Figure 1) and our study shows that pre- and post-Gd SWI show a higher diagnostic accuracy than T1 SE Sequence and also demonstrate the ideal role of the post-Gd SWI technique in evaluating inflammatory activity of MS plaques (Figure 2). This was in agreement with Do Amaral et al. who reported that Gadolinium-SWI technique improved detection accuracy of MS- CE lesions better than Gd-T1 SE, including their locations and morphologies [13].

Our study has confirmed the ideal role of SWI in detection of central vein inside white matter plaques that could be used as ideal specific biomarker in the diagnosis of MS. This was agreeing with Tallantyre, et al. who confirmed the sensitive role of SWI sequence in diagnosis of MS by detection of central vein sign [4].

Our study has also investigated the role of SWI in imaging of hypointense rim around MS plaques that considered a recent specific marker for MS that help us in the diagnosis of MS. This was in agreement with Hosseini et al. reported that around MS lesions there may be hypointense rim; this additive promised marker

for MS, can probably be utilized in addition to central venous sign as a radiological sign in differentiation of MS from other white matter lesions [7]. Our study has also investigated the role of post GD –SWI in detection of central vein sign better than pre GD SWI. It improved small veins visualization that was already visible in the SWI before contrast. This was agreeing with the study conducted by Maggi et al.; who reported that when using SWI, GD improves the veins visibility inside MS white matter lesions, and this is remarkably noticeable in the contrast enhancing lesion on the same SWI technique [14].

Our study assesses the role of SWI in Characterization of MS plaques Contrast-Enhancing and Non-contrast-enhancing. A 1.5 T MRI was used for evaluating 24 patients of MS, 381 lesions were analyzed: 23/381 (6%) were contrast-enhancing lesions and 358/381 (94%) were non-enhancing. Two patterns were seen in CE lesions: ring-shaped enhancement in 9/23; nodular enhancement in 14/23. In 132/381(35%) post GD-SWI lesions, were associated with central veins. In 119/381 (31%) pre GD-SWI lesions, were associated with central veins, whereas 5/358 (1.4%) showed peripheral hypointense dots/rims, lesions with scattered hypointense dots represented 4/358 (1.1%), and in 112/358 (31%), no SWI hypointensity was detected. additionally, 95/358 (27%) lesions were invisible on SWI and isointense. This agreed with Eisele et al. study using Susceptibility-Weighted Imaging in Characterization of Contrast-Enhancing and Non-contrast-enhancing Multiple Sclerosis Lesions [15]. Our study included 24 patients, 8 active and 16 inactive cases proven as MS patients clinically and radiologically, fulfilled the inclusion criteria. 8 males and 16 females; the ages were from 20-42 years with mean age 26.33 years. This was in agreement with Harbo et al. who reported that MS is more common in females than males as most of the autoimmune disorders and in the 2nd and 3rd decade of life, it is often diagnosed [16]. Our study classified the patients into 2 groups (based on enhancement of lesions with contrast or not),

correlated with ADC value measurement in some suspicious cases to be progressive: Group A: active cases with lesions enhanced post contrast (8 patients) and Group B: inactive cases with lesions non-enhanced post contrast (16 patients). Then both groups were subdivided into: A): 21 patients (Relapsing remitting cases) and B): 3 patients (Progressive cases). These results were in agreement with Markowitz, who found that relapsing remitting form is more prevalent than progressive form [17].

Conventional MRI and advanced MRI techniques (diffusion weighted MR imaging & Susceptibility-weighted imaging (SWI)) used to examine the twenty four patients with well-documented definite or clinically probable MS. ADC values were measured in selected white matter lesions that show scattered hypointense dots or hypointense rim in SWI. Our study has shown that in both lesions, enhancing and non-enhancing, plaques with dispersed hypointense dots on SWI showed that greater ADC values were substantially in these plaques than in other isointense lesions, as found more in chronic 2ry progressing patients. This study was also agreeing with multiple studies that have shown that iron accumulates inside microglia and macrophages along the borders of these lesions, generating rims [18, 19]. A recent study discovered iron deposition at the edges of MS lesions using Seven Tesla post-mortem MRI. The authors found that SWI hypointense rim was associated with accumulation of iron in microglia and macrophages histologically expressing the pro-inflammatory markers p22phox and CD86 around the margin of slowly progressive developing lesions, whereas non-rim lesions decreased in size with time [20]. The existence of SWI iron rims may serve as a biomarker of the disease and may be a clue of progressive tissue injury, according to the authors' conclusion [20]. It's probable that hypointense rim lesions are only observed in multiple sclerosis and not in other diseases (Figure 3). Future researches could address this more. Our study was a single-center retrospective study with a relatively small

number of cases without pathologic correlation, despite the large number of lesions analyzed. Also, some patients refusing contrast injection, GD- SWI is relatively a new sequence; so some MRI centers refuse to implicate it in their protocol of MRI sequences although this sequence is very fast and it is not time consuming; additionally in the context of MS patients, it requires further study to standardize and implement the results acquisition and interpretation.

CONCLUSION

Gd-SWI sequence is better than Gd-T1 SE in the accuracy of detection of MS-typical contrast-enhancing lesions, involving their morphologies and locations. Additionally; increasing the Gd-SWI sequences use in clinical practise can help us to learn more about MS, allowing us to analyze BBB dysfunction in addition to the central vein sign described previously.

Conflict of Interest

The authors of this manuscript declare no relevant conflicts of interest, and no relationships with any companies, whose products or services may be related to the subject matter of the article.

Financial Disclosures

None.

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