Original Article

Utility of contrast-enhanced fluid-attenuated inversion recovery in magnetic resonance imaging of intracranial lesions

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Abstract

Aim: The aim of this study is to determine utility of contrast-enhanced fluid-attenuated inversion recovery (FLAIR) imaging by comparing results with contrast-enhanced T1-weighted imaging (T1WI) in various intracranial lesions.

Materials and Methods: Forty-nine patients with a known intracranial lesion or with clinical suspicion underwent the gadolinium-enhanced magnetic resonance (MR) imaging using 1.5T. Postcontrast axial, coronal, and sagittal T1 fat-saturated, axial FLAIR images were acquired after administration of gadobenate dimeglumine. The MR imaging parameters for the postcontrast T2-FLAIR images were 6000–9000/90–110/1845–2030 ms/150 (repetition time/echo time/inversion time/flip angle), and the acquisition time was 2 min 12 s. All images were acquired with a section thickness of 5 mm, an intersection gap of 2 mm, and a field of view of 256 mm × 144 mm. The images were transferred to a workstation and reviewed.

Results: We found that postcontrast FLAIR images are useful by showing better meningeal involvement in various pathologies and enhancement of the solid component in intra-axial lesions. However, it was not much helpful in extra-axial lesions and lesions with mild postcontrast enhancement and lesions with perilesional edema.

Conclusion: Postcontrast FLAIR is a useful adjunct to postcontrast T1W images in equivocal cases and for additional information.

Keywords: Intracranial lesions, postcontrast fluid-attenuated inversion recovery, postcontrast T1-weighted imaging

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INTRODUCTION

Intravenous contrast agents are commonly used to evaluate patients suspected with intracranial lesions. These lesions may show enhancement through various mechanisms. Contrast-enhanced T1-weighted imaging (CE-T1WI) is preferred sequence at most institutions.[1]

Access this article online	
Quick Response Code:	Website: www.wajradiology.org
	DOI: 10.4103/wajr.wajr_4_17

Fluid-attenuated inversion recovery (FLAIR) is a type of inversion recovery pulse sequence having a long repetition time (TR) and echo time (TE) and an inversion time (TI) and hence effectively nulls signals from the cerebrospinal fluid (CSF).[1] Because of mild T1 effect due to long TI, there is an enhancement on postcontrast study. Therefore, lesions showing enhancement on CE-T1WI also show

How to cite this article: Bhargava R, Patil AM, Bakshi V, Kalekar TM, Gandage SG. Utility of contrast-enhanced fluid-attenuated inversion recovery in magnetic resonance imaging of intracranial lesions. West Afr J Radiol 2018;25:34-8.

enhancement on contrast-enhanced FLAIR (CE-FLAIR) images. Many clinical trials have revealed that CE-FLAIR offers more information than CE-T1WI alone. FLAIR imaging is sensitive for detecting parenchymal lesions and also in detecting extra-axial diseases.[2] We describe the utility of CE-FLAIR imaging by comparing results to those with CE-T1WI in various intracranial lesions.

MATERIALS AND METHODS

A retrospective study done on forty-nine patients (20 women, 29 men) of all age groups with a known intracranial lesion or with clinical suspicion underwent the gadolinium-enhanced magnetic resonance (MR) imaging using 1.5T scanner (Avanto; Siemens Medical Solution, Erlangen, Germany) over a period of 7 months (from January 2016 to July 2016). Clearance from the institutional board was obtained before the study. Informed consent was taken from each patient before the study. Following the acquisition of routine multiplanar and multi-echo sequences, namely, sagittal and axial T1 SE, coronal and axial T2 SE, axial gradient-recalled echo and diffusion-weighted images, axial FLAIR fat-saturated (FS). Postcontrast axial, coronal, and sagittal T1 FS, axial FLAIR images were acquired after administration of gadobenate dimeglumine (Multihance, 0.1 mmol/kg of body weight over 1 min). The MR imaging parameters for the postcontrast T2-FLAIR images were 6000–9000/90–110/1845–2030 ms/150 (TR/TE/TI/flip angle), and the acquisition time was 2 min 12 s Moreover, the MR imaging parameters for T1-weighted images were 700/8–10/90 (TR/TE/flip angle), and the acquisition time was 1 min 42 s. All images were acquired with a section thickness of 5 mm, an intersection gap of 2 mm, and a field of view of 256 mm × 144 mm. The images were transferred to a workstation and reviewed by three experienced radiologists in a single sitting. Each of the radiologists independently evaluated the pre- and post-contrast FLAIR and T1WI side by side at the same time. In cases of disagreement, the final judgment was reached by consensus of the three reviewers. Various lesions were assessed for number, conspicuity, and degree of contrast enhancement on both pre- - and post-contrast FLAIR and T1W sequences. No categorization of the enhancement was done; however, visual degree of enhancement was taken into consideration. All lesions were not subjected to biopsy; however, many of them were histologically proven. Sensitivity and specificity of postcontrast FLAIR in various pathologies were calculated.

RESULTS

All the cases varied within the age group of 24–81 years, with most of these lesions seen in the males (n = 29).

Out of the 49 cases of intracranial lesions [Table 1], 14 cases were extra-axial lesions and rest 35 cases were intra-axial lesions. Intra-axial lesions were further classified on the basis of etiology into infective and neoplastic. Seventeen cases were due to infective etiology and rest 18 cases were due to neoplastic etiology including both of benign and malignant etiology.

The extra-axial lesions included acoustic schwannoma, primitive neuroectodermal tumor, epidermoid cyst, meningioma, olfactory neuroblastoma, and chordoma. The lesions showed more enhancements on postcontrast T1WI in comparison with the postcontrast FLAIR images which showed variable enhancement in addition to the hyperintensity due to T2 effect of FLAIR, depending on the lesion. We did not find any extra-axial lesions which showed enhancement characteristic better on postcontrast FLAIR than in postcontrast T1WI.

The infective intra-axial lesions mainly included those of the granulomatous etiology such as neurocysticercosis and tuberculomas. All these lesions showed peripheral enhancement on postcontrast T1WI and also on postcontrast FLAIR images which were almost equal in signal intensity. The lesions on both sequences were better evaluated in comparison with T2WI. The lesions on postcontrast T1WI were better delineated due to the absence of perilesional edema which was seen on FLAIR images as hyperintensity resulting in nonvisualization of small peripherally enhancing lesions [Figure 1]; however, seven lesions showed

Table 1: Incidence of various intracranial pathologies in our study

	Number of pathologies
Extra-axial lesions	
Meningioma	3
Schwannoma	2
Epidermoid	2
Pituitary lesion	2
Bone lesions	4
Primary	1
Intra-axial lesions	
Primaries	16
Secondaries	2
Tuberculomas	10
NCC	3
NCC/tuberculoma	4
Others	
Total	49

NCC – Neurocysticercosis

more hyperintense enhancement than the perilesional edema on postcontrast FLAIR [Figure 2]. This helped us to see the extent of edema as well as enhancement. Furthermore, one case of tuberculoma with associated meningitis showed better meningeal enhancement on postcontrast FLAIR images which was not seen well on postcontrast T1WI [Figure 3].

The neoplastic intra-axial lesions include the primary tumors such as gliomas (both high and low grade), astrocytoma, and metastases. Enhancement on both postcontrast T1 and FLAIR images was comparable. Eight cases showed better enhancement on postcontrast FLAIR mainly in the solid component of lesion in comparison to the postcontrast T1WI [Figure 4]; however, no additional lesions were identified on the postcontrast FLAIR images. Of the two cases of metastases, enhancement was similar on both postcontrast T1 and FLAIR images.

Thus, postcontrast FLAIR images were seen to be better in the visualization of the meningeal enhancement and also improved delineation of the solid component in the various intracranial lesions. However, we could not found it useful in extracranial lesions.

DISCUSSION

Gadolinium is the most commonly used intravenous contrast agent for imaging the brain. It helps in improved lesion detection and better characterization. Gadolinium causes shortening of both T1 and T2 of the tissues in which it has accumulated. It is due to the T1 shortening that there is contrast enhancement of a lesion on clinical MR images.[2]

T1WI are usually acquired in multiple planes before and after the administration of gadolinium. The areas which were previously hypointense on precontrast study and develop hyperintensity on postcontrast study are said to have enhanced. This enhancement occurs due to the accumulation of gadolinium in the extracellular space which influences the MR relaxation time of nearby tissue protons.[2]

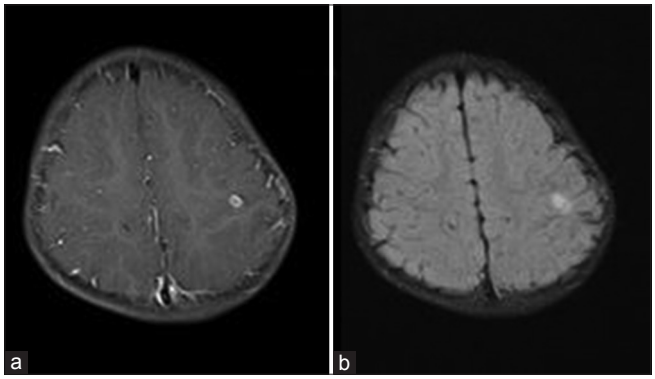

Figure 1: Known case of tuberculoma with peripheral ring enhancement seen better on postcontrast T1 (a) while subtle enhancement on postcontrast fluid-attenuated inversion recovery (b) not well delineated due to surrounding perilesional edema

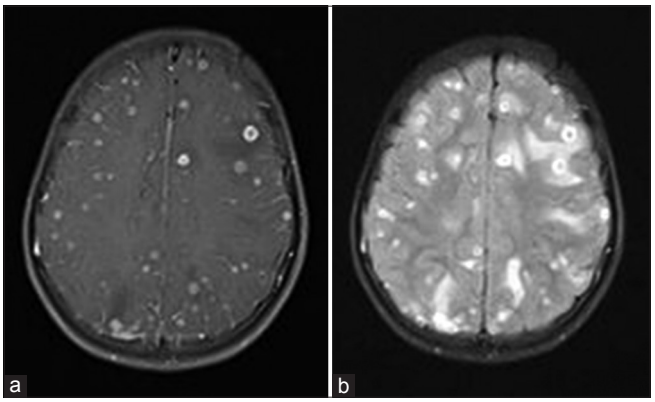

Figure 2: Known case of tuberculoma, multiple ring-enhancing lesions well demarcated on postcontrast T1 (a), postcontrast fluid-attenuated inversion recovery (b) showed some of lesions enhancing more than the perilesional edema and some small lesions not well appreciated due to surrounding edema

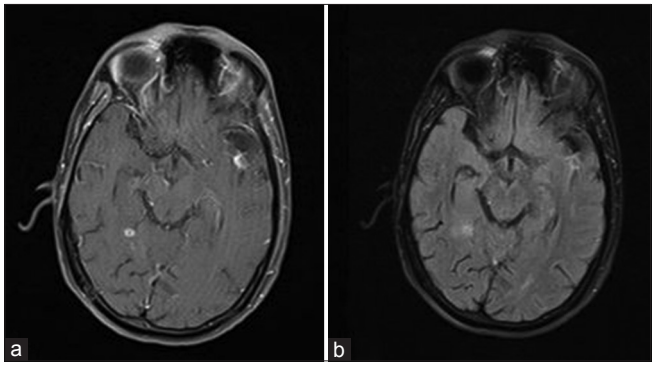

Figure 3: Known case of tuberculoma with meningeal involvement, the lesion is well seen on postcontrast T1 (a), while the meningeal enhancement (arrows) is better seen on postcontrast fluid-attenuated inversion recovery (b)

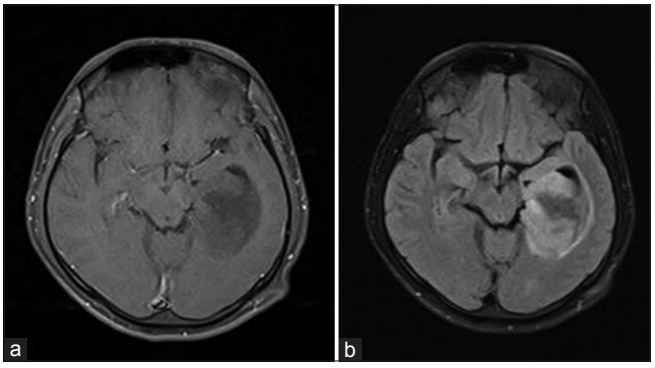

Figure 4: In a case of low-grade astrocytoma, minimal enhancement is seen on postcontrast T1 (a) while postcontrast fluid-attenuated inversion recovery (b) which better delineates the solid and cystic areas of the lesion even in such minimal enhancing lesions

For intra-axial brain lesions to enhance, the blood–brain barrier should be disrupted so that gadolinium can enter the extracellular space. While for extra-axial lesions, enhancement is mainly seen in those lesions which have relatively high vascularity.[3]

Lesion detection depends on number of factors such as lesion size, image contrast, and location. It can be improved by giving large doses of contrast material that increases the signal of the lesion.[4] T1WI are primarily used for postcontrast brain MR imaging in the detection and characterization of intracranial lesions. FLAIR images are equivalent to T2WI except for dark CSF which is due to T1 effect present because of the long TI. After administration of gadolinium, there is T1 shortening which is seen as hyperintensity on FLAIR images; therefore, lesions showing enhancement on postcontrast T1WI will also enhance on postcontrast FLAIR images.[5,6] FLAIR should be performed before and after the gadolinium administration to overcome the confusion arising from the hyperintensity observed when the FLAIR sequence is performed only after gadolinium administration as this hyperintensity can be due to either T2 lengthening or T1 shortening.[7] Ercan *et al*. showed that postcontrast FLAIR images are better in comparison to postcontrast T1WI in detecting the number of lesions, its conspicuity, and enhancement probably due to delayed enhancement in such lesions.[2] However, in our study, none of the cases on postcontrast FLAIR were seen to be better for detecting more number of lesions in comparison to postcontrast T1WI. However, solid component was better evaluated on postcontrast FLAIR images in eight of the cases, thus helping in better guidance of biopsy in the solid portion of the lesion. Cystic areas were also better seen on FLAIR images in ten cases [Figures 4 and 5]. Postcontrast fast FLAIR images were also more effective for showing meningeal neoplasms as meningeal carcinomatosis.[8,9] However, we did not come across any such case in our study which showed a significant difference in contrast enhancement on FLAIR and T1.

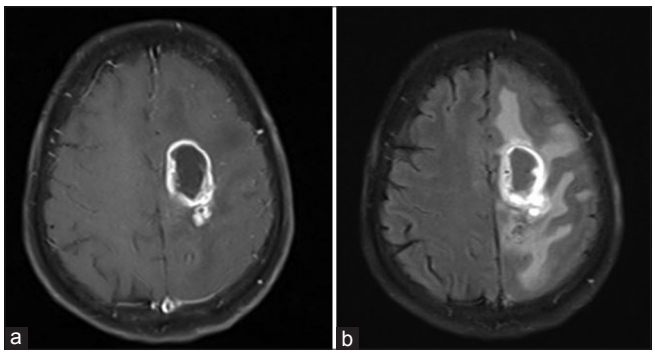

Figure 5: Postcontrast T1 fat-saturated (a) and postcontrast fluid-attenuated inversion recovery fat-saturated (b) show an irregular peripherally enhancing lesion with central areas of necrosis and surrounding perilesional edema seen better on postcontrast fluid-attenuated inversion recovery

Enhancement within the pituitary gland or in nasal mucosa/turbinate on FLAIR images is subtle due to T2 prolongation, and therefore, these lesions are difficult to identify on postcontrast FLAIR images,[1] and we also did not find any additional benefit of postcontrast FLAIR.

Lesions which did not showed any enhancement on postcontrast T1WI also showed no enhancement on postcontrast FLAIR images. In postcontrast T1WI, the subtle enhancement was easier to detect because the edema appeared hypointense while it is difficult to differentiate subtle enhancement with hyperintense perilesional edema on FLAIR, and hence, T1WI was considered better than CE-FLAIR images in such parenchymal lesions, which was similar to findings of Mathews *et al*.[10] Furthermore, postcontrast FLAIR images may show difficulty in detecting lesions which have long T2 and appear hyperintense. Therefore, in such cases, T1WI is a better option when compared with the postcontrast fast FLAIR imaging.

In the evaluation of parenchymal metastases, we did not find any additional lesions on postcontrast FLAIR images, and lesions were seen of similar intensity to those seen on the postcontrast T1WI. This was in contrast to study conducted by Ercan *et al*.[2]

CONCLUSION

Contrast-enhanced FLAIR MR imaging is a useful tool in diagnosis and characterization of some lesions and should be considered when CE-T1WI findings of the brain are inconclusive, or it is necessary to detect minimal amounts of contrast enhancement. However, we would also like to emphasize that it cannot substitute postcontrast T1 FS sequences. CE-FLAIR imaging has many advantages for intracranial disease manifestations. It may be used as an adjunct rather than substitute to postcontrast T1WI in equivocal cases. It has been also found to be better in the delineation of cystic components in neoplastic lesions as well as in diagnosis of meningeal involvement in infective processes. It thus helps to increase the diagnostic confidence and improve patient care. Our study, however, included wide variety of focal lesion, and the role of postcontrast FLAIR in individual pathologies needs to be further assessed separately.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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