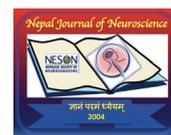


Sulcal FLAIR hyperintensity - Finding beyond meningitis



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Abstract

Introduction: The aim of this study was to enumerate the causes of Fluid-attenuated inversion recovery (FLAIR) hyperintensity in the sulcal space which could be due to cerebrospinal fluid (CSF) or non-CSF related pathologies.

Methods and Materials: This is an observational retrospective study done in 100 patients in the department of Radio-diagnosis of Patna Medical College and Hospital, Patna from September 2019 to September 2020.

Results: The mean age of patients was 40.13 +/- 13.88 years (Range 19 to 75 years). Male to female ratio was 1.32:1.00 (57:43). The most common cause of FLAIR sulcal hyperintensity was infection in 66% cases followed by meningeal tumor deposits in 16%, vascular cause in 6%, subarachnoid hemorrhage in 5%, mass effect in 5%, dermoid rupture in 1% and hyperoxygenation in 1%.

Conclusion: Presence of sulcal hyperintensity on FLAIR images is a very strong sign to an underlying brain pathology. A keen observation of the same on FLAIR sequence in association with other findings can increase diagnostic confidence and thus lead to better patient care.

Key words: Cerebrospinal fluid, Ivy sign, Meningitis, MRI, Sulcal FLAIR hyperintensity.

Introduction

Fluid-attenuated inversion recovery (FLAIR) was first described by Hajnal et al.¹ in 1992 and this technique consists of an inversion recovery pulse which nulls out the signal from cerebrospinal fluid (CSF) and a long echo time to produce a heavily T2-weighted sequence. In addition, factors that affect the T1-weighted relaxation time of CSF may interfere with its suppression and result in CSF hyperintensity. FLAIR sequence has greater sensitivity in

detecting lesions within the subarachnoid space and brain parenchyma, especially near the brain-CSF interface when compared with the conventional T2-weighted and proton density-weighted MRI sequences.²

When disease occurs within the subarachnoid space, alteration of relaxation time of CSF takes place causing lesser CSF suppression and thus hyperintensity on these sequences. Sulcal FLAIR hyperintensity is noted in many pathologic conditions such as meningitis, leptomeningeal spread of malignant disease and subarachnoid hemorrhage.³ Other than CSF pathology, non-CSF pathology too occurs like vascular cause. Less common causes of subarachnoid FLAIR hyperintensity are acute stroke, Moyamoya disease and mass effect.⁴

Artifact leading to FLAIR hyperintensity should also be kept in mind which includes CSF flow artifact, vascular motion artifacts, head motion artifact, inhaled oxygen, cross talk artifact and magnetic susceptibility artifact.⁵ Thus, analysis of the distribution of sulcal FLAIR hyperintensity and the associated imaging findings related to the primary pathologic condition can help in making the diagnosis.

In our paper we have considered all patients during one year who came up with FLAIR sulcal hyperintensity, however magnetic resonance imaging (MRI) with artifact related causes of sulcal hyperintensity on FLAIR sequence were excluded as described in the methodology section. The final diagnosis was established by clinical correlation, CSF examination and/or follow up.

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Methods and Materials

Our study is an observational retrospective study with cross sectional data analysis done in the department of Radio-diagnosis of Patna Medical College and Hospital, Patna from September 2019 to September 2020.

This study received approval from the ethics committee (IEC) of the institute human research approval. Informed consent was obtained from each patient, according to the Declaration of Helsinki, and the IEC approved proforma. Patients were recruited during their work-up and the MRI scans during the FLAIR sequence were done as part of evaluation. Study was designed to enroll 100 consecutive patients with sulcal hyperintensity on FLAIR images. 8 patients were excluded due to motion artifacts and dental filling artifacts. Patients with metallic implant, claustrophobia and pacemaker were excluded from our study. MR imaging was performed on the 1.5 T Signa MR imaging system (General Electric Medical Systems, Milwaukee, WI). Unenhanced MR examinations included FLAIR images (TR/TEeff range, 8000/80–130; inversion time, 2000 msec; echo train length, 8), fast spin-echo T2-weighted images (8000/90–100; echo train length, 8), and spin echo T1-weighted images (TR range/TE range, 350–500/16–20), with the axial plane; section thickness, 5mm; matrix size, 256 × 256; field of view, 24 × 24 cm. Contrast-enhanced T1-weighted images with parameters identical to unenhanced T1-weighted images after the IV injection of 0.1 mmol/kg of gadopentetate dimeglumine (Magnevist; Berlex Laboratories, Wayne, NJ) were obtained. T1-weighted contrast study was done in all cases and depending on the probable cause MRI Angiography and MR venography was done in selective cases. Two radiologists [one senior resident (AS) and one consultant (RG)] retrospectively and independently reviewed these scans. The findings were subsequently transferred to a Microsoft Excel 2012 sheet and statistical analysis was done using IBM SPSS for Windows version 22.0 (SPSS, Inc., Chicago, IL, USA).

Sulcal hyperintensity on FLAIR imaging was defined as hyperintensity in the CSF space of one or more cortical sulci or cerebellar sulci. The pattern of the sulcal hyperintensity was further classified as focal or diffuse. Cisternal, ventricular and ventricular foraminal hyperintensities were not included in our study, thus lowering the chances of artifactual causes of FLAIR hyperintensity.

Non-pathological causes of FLAIR sulcal hyperintensities were excluded from our study. CSF flow related artifacts were excluded by the characteristic location of their occurrence, that is at the basal cisterns, third and fourth ventricles, and at ventricular foramina. Similarly, head motion artifacts were excluded by their location at the cerebral convexities. To null the effect of

oxygen, the FiO₂ was reduced to lower levels (30%) during the time of scan in patients on supplemental oxygen (100% FiO₂). The vascular pulsation artifacts were excluded by their appearance and location, reproducing the size, shape, and alignment of the nearby vessel along the phase-encoding direction of the image. Metallic susceptibility artifact is produced at tissue interfaces due to incomplete CSF nulling. These are mostly produced by metals and sometimes by the air in paranasal sinuses. Patients with metallic implants were excluded from our study.

Discrepancies between reviewers' interpretations were resolved by consensus.

Results

Mean age of patients was 40.13 +/- 13.88 years (Range 19 to 75 years). Male to female ratio was 1.32:1.00 (57:43).

Out of 100 patients, 66 patients had infections, 16 had meningeal deposits, 6 had vascular cause, 5 had sulcal hyperintensity due to mass effect, 5 had subarachnoid hemorrhage, 1 had dermoid rupture and 1 was due to hyperoxygenation (Table 1).

Cause	Percent (%)
Meningitis	66
Meningeal tumor deposits and lymphomatosis	16
Vascular	6
Subarachnoid bleed	5
Mass effect	5
Fat	1
Hyperoxygenation	1

Table 1: Etiology of sulcal FLAIR hyperintensity.

Out of 66 patients with infection, 63(%) had tubercular meningitis, 2(%) had bacterial meningitis, 1(%) had CMV meningitis. Among 16 patients with meningeal tumoral deposits, 4 had carcinoma lung, 4 had carcinoma breast, 2 had carcinoma prostate, 3 had leukemic infiltration, 2 had lymphoma and 1 had choroid plexus carcinoma. Among 8 females, carcinoma breast (50%) was the most common cause of leptomeningeal spread whereas carcinoma lung (25%) and lymphoma (25%) were the causes in other 4 patients. Among 8 male patients, 2 (25%) had carcinoma lung as primary, 1 (12.5%) had carcinoma prostate, 3 (37.5%) had leukemic infiltration and 1 (12.5%) had meningeal deposit from choroid plexus carcinoma.

Five patients had sulcal hyperintensity due to subarachnoid hemorrhage (SAH). Aneurysmal rupture was the cause in 3, arteriovenous malformation (AVM) in 1 and deep venous thrombosis in 1 patient. All patients had focal sulcal FLAIR hyperintensity. Six patients had vascular causes of sulcal hyperintensity which included

leptomeningeal collaterals due to arterial thrombosis (2 patients), Moyamoya disease (2 patients), Takayasu arteritis (1 patient) and dural venous thrombosis (1 patient). Dural vein thrombosis caused focal SAH which in turn produced focal sulcal hyperintensity on FLAIR sequence. Five patients had sulcal hyperintensity due to mass effect from primary brain tumor all of which were focal sulcal FLAIR hyperintensity.

Eighty patients had diffuse sulcal hyperintensity and 20 had focal sulcal hyperintensity. Among the 80 diffuse sulcal hyperintensity cases, 64 patients had infection, 12 had meningeal tumor deposits and 4 had vascular cause. In the 20 focal sulcal hyperintensity cases, 2 patients had vascular cause (1 as dural venous thrombosis, 1 slow flow after arterial thrombus), 5 Subarachnoid hemorrhage, 2

infection, 4 meningeal tumor deposits, 5 due to mass effect, 1 due to dermoid rupture and 1 due to hyperoxygenation (Table 2).

Cause	Diffuse	Focal
Meningitis	64	2
Meningeal tumor deposits and lymphomatosis	12	4
Vascular	4	2
Subarachnoid bleed	0	5
Mass effect	0	5
Fat	0	1
Hyperoxygenation	0	1
Total	80	20

Table 2: Causes of diffuse and focal sulcal hyperintensity.

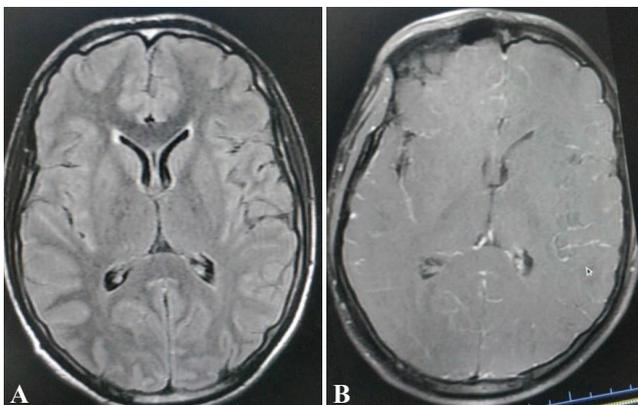


Figure 1: 26-year-old female with meningitis. 1a: Axial FLAIR shows diffuse leptomeningeal hyperintensity. 1b: Axial T1 weighted contrast enhanced MR image showing leptomeningeal enhancement.

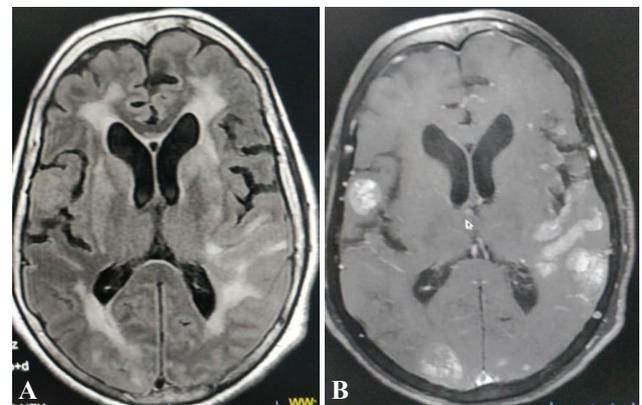


Figure 2: 70-year-old female with leptomeningeal metastasis and intraparenchymal metastasis from breast carcinoma. 2a: Axial T2W FLAIR image shows diffuse leptomeningeal hyperintensity in parieto-occipital region. 2b: T1W contrast enhanced axial image shows diffuse, thick leptomeningeal enhancement in parieto-occipital region with multiple intraparenchymal variable sized lesions.

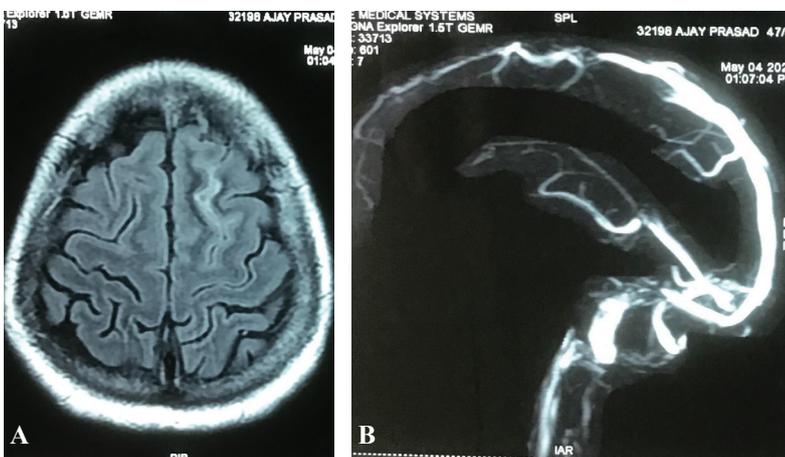


Figure 3: 45-year-old alcoholic patient with subarachnoid bleed due to partial thrombosis of Superior Sagittal Sinus. 3a: Axial T2W FLAIR image shows focal sulcal hyperintensity in the left frontal region. 3b: MR Venography shows partial thrombosis of the middle segment of superior sagittal sinus and irregular anterior segment.

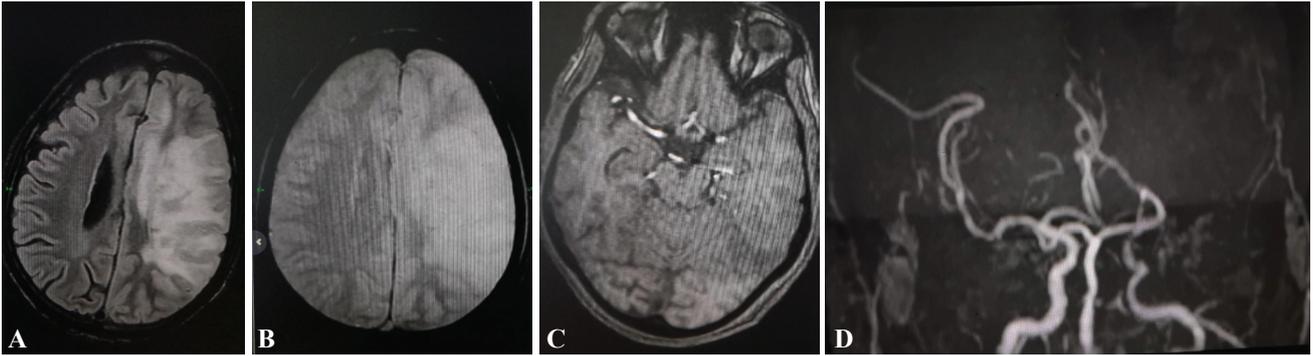


Figure 4: 55-year-old male patient with acute right MCA aneurysm.

4a: Axial T2W FLAIR image shows hyperintensity in the right frontoparietal region with loss of gray-white interface and sulcal hyperintensity is also noted in the involved area.

4b: Axial GRE sequence shows no blooming.

4c: Axial MR Angiography image shows abrupt cut off of right middle cerebral artery.

4d: VRT MR Angiography image shows abrupt cut off of right middle cerebral artery.

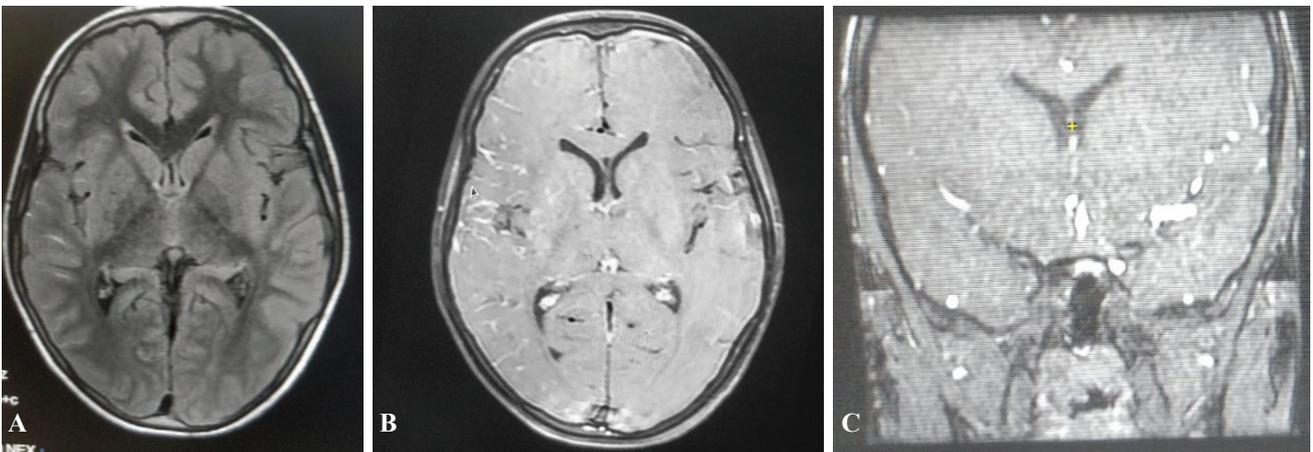


Figure 5a: Axial FLAIR image shows sulcal hyperintensities in the right fronto-temporo-occipital region.

5b: T1-weighted post contrast image at the same level shows leptomeningeal enhancement in the same region.

5c: Coronal MR angiography shows near complete stenosis of the right supraclinoid ICA.

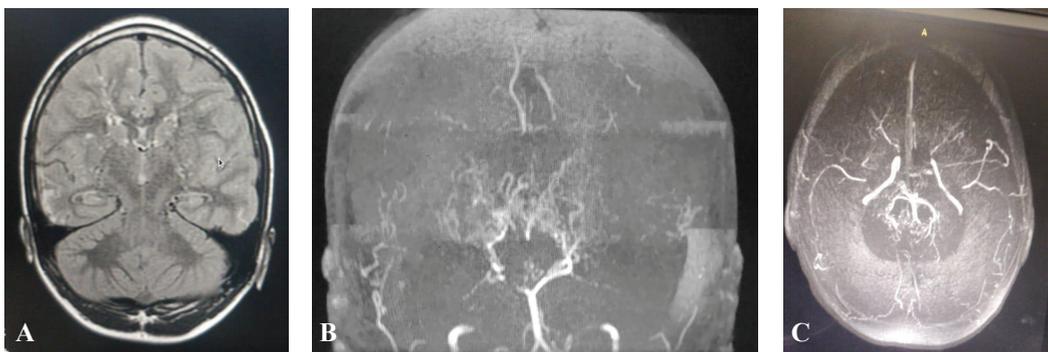


Figure 6: 9-year-old female with Moyamoya disease.

6a: Coronal FLAIR image shows multiple collaterals producing sulcal hyperintensities in bilateral cerebral hemispheres, right more than left.

6b: Coronal VRT MR angiography shows stenosis of bilateral supraclinoid segments of ICA and proximal segments of middle and anterior cerebral arteries with multiple mesh-like network of collaterals giving the characteristic puff-of-smoke appearance.

6c: VRT MR angiography image showing stenosed bilateral ICA and proximal middle and anterior cerebral arteries.

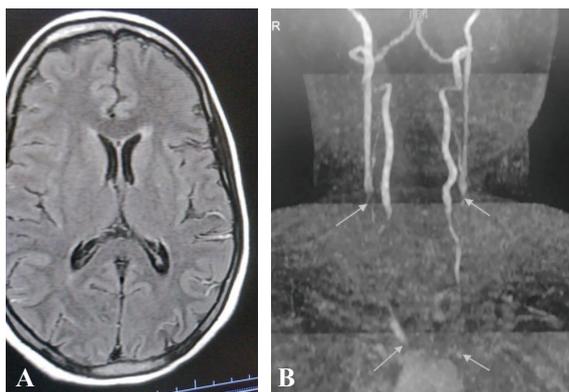


Figure 7: 25-year-old female with Takayasu arteritis.
7a: Axial T2W FLAIR image shows diffuse sulcal vascular hyperintensity.
7b: MR angiography shows significant narrowing of bilateral common carotid artery.

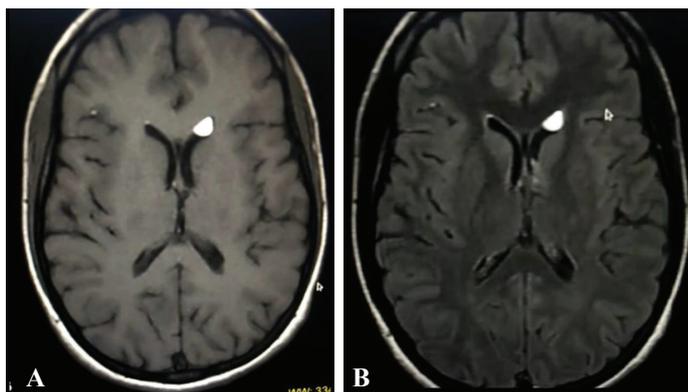


Figure 8: 20-year-old male with ruptured dermoid.
8a: Axial T1W image shows few hyperintense foci in bilateral lateral ventricle and right sylvian fissure.
8b: Axial T2W FLAIR image shows similar hyperintense foci in bilateral lateral ventricle and right sylvian fissure.

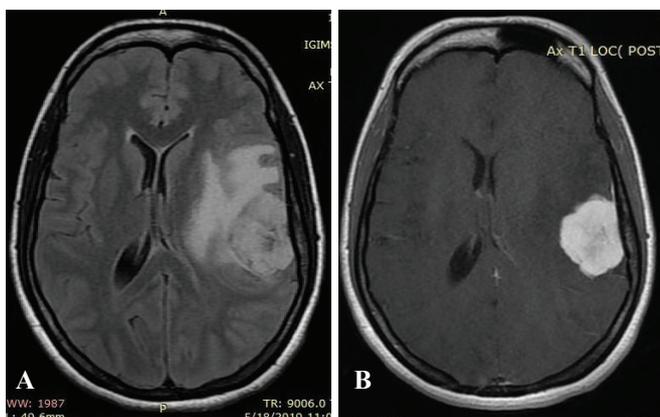


Figure 9: 45-year-old male with convexity meningioma.
9a: T2-weighted FLAIR image shows extra-axial hyperintense lesion in left parietal region with adjacent perilesional edema and effaced sulcal spaces showing sulcal hyperintensity.
9b: Axial T1W contrast shows intensely enhancing extra-axial left parietal meningioma.

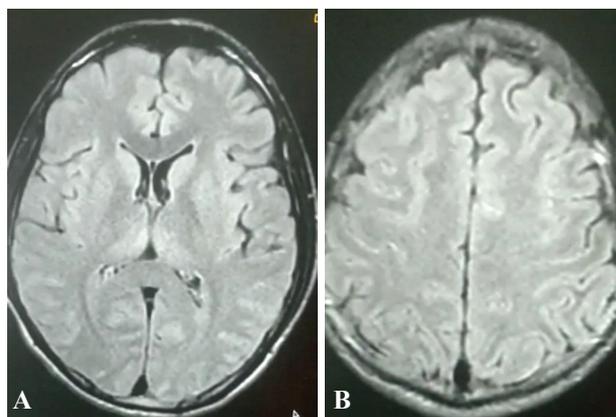


Figure 10: A 41-year-old male patient who was on oxygen therapy was imaged.
10a: Axial FLAIR images show sulcal hyperintensity along bilateral occipito parietal regions.
10b: Axial section of FLAIR sequence through high frontoparietal region reveals patchy sulcal hyperintensity in bilateral cerebral hemispheres.

Discussion

Fluid attenuated inversion recovery (FLAIR) sequences are now part of our standard brain protocol. It is basically a T2W sequence to which an inversion recovery pulse is applied so that signal from water (CSF) gets nullified¹. As compared to T2W sequence, FLAIR improves the detection of lesions at CSF-brain interface and within the subarachnoid space. FLAIR images are sensitive to factors that affect T1 relaxation time of CSF and T2-weighted prolongation in tissue.

Failure to suppress the CSF signal leads to sulcal hyperintensity which occurs due to meningeal pathologies

including meningitis, meningeal deposits from primary carcinoma and round cell tumors, and subarachnoid hemorrhage. There are few other causes of sulcal hyperintensity namely vascular and mass effect. Many artifacts also cause sulcal hyperintensity on FLAIR which we have not included in our study as mentioned in the methodology section.

MENINGITIS

FLAIR MRI is sensitive technique for the detection of meningitis.³ The cause of subarachnoid hyperintensity in these patients is CSF protein and cellular concentrations in subarachnoid space which result in shortening of the

T1 relaxation time and thus complete nullification of CSF does not take place.⁶ However, contrast-enhanced T1-weighted imaging is more sensitive in the detection of meningitis in comparison to unenhanced FLAIR.⁷

In our study 66% of patients had FLAIR sulcal hyperintensity due to meningitis (Figure 1a, 1b). Out of these, 64 patients had diffuse sulcal hyperintensity and 2 had focal sulcal hyperintensity.

Thus, CSF pathology accounts maximum for FLAIR sulcal hyperintensity and more so when the involvement is diffuse. Out of the 64 cases, 63 had tubercular meningitis which was the most common etiological agent, 2 had pyogenic meningitis and 1 had CMV meningitis. These were confirmed by CSF examination.

LEPTOMENINGEAL METASTASES AND LYMPHOMATOSIS

Leptomeningeal spread of malignancy can be diagnosed without the use of IV gadolinium in patients with known or suspected neoplastic disease using FLAIR sequence.^{8,9} Many studies have been done which compared FLAIR sequence findings with that of post Gadolinium T1 weighted contrast technique and some concluded FLAIR to be superior to contrast-enhanced T1-weighted images in detecting leptomeningeal spread of tumor.^{3,10}

In our study 16 patients had meningeal tumor deposit as the cause of FLAIR sulcal hyperintensity accounting for the second commonest cause of it (Figure 2a, 2b).

The pattern of sulcal involvement was predominantly diffuse. One differentiating feature between infective etiology and carcinomatosis was the smooth or nodular hyperintensity in neoplastic disease while predominantly smooth sulcal hyperintensity in cases of meningitis. Presence of associated features like ring enhancing or conglomerated lesions, basal exudates, CSF and clinical correlation aided in diagnosing infective meningitis. In meningeal carcinomatosis, neoplastic lesions can be seen which show solid, nodular or irregular thick rim enhancement with perilesional edema. Presence of systemic disease and known primary malignancy on other imaging modalities further help establish the diagnosis.

SUBARACHNOID HEMORRHAGE

FLAIR imaging is equally or more sensitive than CT in the evaluation of acute SAH, but compared with the findings at lumbar puncture, the findings on FLAIR imaging are not definitive in excluding acute SAH.¹¹⁻¹³ The FLAIR sequence is particularly useful in visualization of acute SAH in areas where CT may be limited because of beam-hardening artifacts (Figure 3a, 3b).¹⁴

T1-weighted shortening of bloody CSF occurs due to the higher protein content. T2-weighted prolongation also occurs as a result of the high protein content of blood and inflammatory products in both dilute and

dense blood-CSF mixtures.¹⁴ The mix of blood with high-oxygen-tension CSF in case of SAH causes delay in formation of paramagnetic deoxyhemoglobin due to which oxyhemoglobin remains for last causing complexity and thus signal of SAH differs from intraparenchymal hemorrhage.¹⁴

In our study, 5 patients had sulcal hyperintensity due to subarachnoid bleed which was focal in all cases.

NON-CSF PATHOLOGY VASCULAR CAUSES

The etiopathogenesis behind the vascular cause causing sulcal hyperintensity is the leptomeningeal or pial collaterals having slow flow due to which there is deoxyhemoglobin causing paramagnetic effect leading to FLAIR sulcal hyperintensity. The collaterals develop subsequent to stenosis in proximal vessels.^{15,16}

In our case series, 6 patients had sulcal hyperintensity due to vascular cause; 2 had acute stroke, 2 had Moyamoya disease, 1 had Takayasu arteritis and 1 had dural vein thrombosis.

ACUTE STROKE

FLAIR vascular hyperintensity is helpful in assessing the prognosis in acute stroke. The mismatch between the FLAIR hyperintensity and DWI is used to prognosticate the patient, as mismatch indicates larger penumbra, so intervention in the form of reperfusion therapy in the form of IV thrombolysis or thrombectomy would lead to better outcome. Focal vessel hyperintensity in acute stroke indicates better collateral flow and larger penumbra (Figure 4a-d).^{17,18}

MOYAMOYA DISEASE

Another cause of FLAIR vascular hyperintensity is Moyamoya disease or phenomenon where there is supraclinoid internal carotid artery (ICA) stenosis (Figure 5a-c, 6a-c). After development of the stenosis, firstly there is abnormal dilatation of lenticulostriate and thalamoperforating arteries, then second collaterals to develop are anterior choroidal and pericallosal, then dilatation of anterior and posterior ethmoidal arteries takes place, and lastly there is pial and leptomeningeal collateralization. It is the last set of collateral formation that is leptomeningeal collaterals and pial collaterals that causes the *ivy sign* (Figure 5b).^{19,20}

Slow flow in leptomeningeal collaterals which is contrasting on cortical surface gives impression of ivy growing on the rock, from which the name comes *ivy sign*.^{19,20}

TAKAYASU ARTERITIS

A patient with Takayasu arteritis showed bilateral common carotid and bilateral ICA narrowing due to wall

thickening leading to luminal stenosis and thus causing collateralization same as described above resulting in FLAIR sulcal hyperintensity (Figure 7a, 7b).

DERMOID

The technique of FLAIR imaging involves application of an inversion pulse which nullifies the signal intensity of CSF as these spins pass through the zero-point determined by the tissue-specific T1-weighted relaxation time. Any shortening of the CSF T1-weighted relaxation time negates this effect resulting in high signal. Fat containing tumors like lipoma, that appear hyperintense on T1W images therefore appear hyperintense on FLAIR sequences. Fat droplets from ruptured dermoid also appear hyperintense on FLAIR sequences due to the same reason (Figure 8).²

MASS EFFECT

In cases of mass effect without any apparent CSF abnormality, sulcal hyperintensity may be caused by the alteration of hemodynamics due to sulcal and vascular compression by the mass itself. In our case, convexity meningioma over the left parietal region exerted a mass effect on adjacent brain parenchyma causing effacement of the adjacent sulci with hyperintensity within them (Figure 9).

HYPEROXYGENATION

Oxygen is weakly paramagnetic which leads to reduction in T1 weighted relaxation time of sulcal CSF which leads to hyperintensity of the CSF within the sulci on FLAIR images.

In our case, the patient received oxygen therapy which led to focal patchy areas of sulcal hyperintensity on FLAIR images in bilateral cerebral hemispheres (Figure 10).

The limitation of this study is that we did not include artefact related causes of FLAIR sulcal hyperintensities which are important non-pathological causes of FLAIR sulcal hyperintensities and can at times be mistaken for pathologies.

Conclusion

FLAIR sequence is routinely used in MRI Brain, usually in the axial plane. Pathologies are picked up on this sequence as there is incomplete suppression of the CSF signal due to the pathologies. Thus, presence of any FLAIR sulcal hyperintensity on non-contrast MRI Brain should always be correlated with clinical details and associated findings on other sequences like post contrast study, MR angiography or venography, susceptibility weighted images, diffusion weighted images, T1 and T2 weighted images to reach the final diagnosis.

Conflict of Interest: None

Source(s) of support: None

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