



# Contrasting FLAIR mechanisms in meningitis

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## Abstract

### Background

Fluid-attenuated inversion recovery (FLAIR) MRI has become a cornerstone in the evaluation of infectious leptomeningitis. Its diagnostic utility stems from two distinct pathophysiological mechanisms: the failure of CSF signal suppression on unenhanced scans and T1-shortening effects following contrast administration. This case presentation compares these mechanisms to optimize the early detection of meningeal pathology.

**Key words:** Autoimmune encephalitis; Meningitis; Post contrast FLAIR

## 1. Clinical presentation & MRI workup

A 32-year-old female was being worked up elsewhere for autoimmune encephalitis based on clinico-radiological features of altered behavioural changes with medial temporal signals. MRI plain studies were done twice. After admission in our institute, neuroradiology opinion was sought. The films were reviewed and contrast study was suggested to look at leptomeningeal changes. MRI contrast study was done in our institute showed diffuse post contrast sulcal FLAIR changes, suggestive of basal meningitis with few subpial nodular enhancing foci and early obstructive hydrocephalus. MR angiogram showed mild focal attenuation of left distal M1 MCA, concerning for secondary vasculitis.

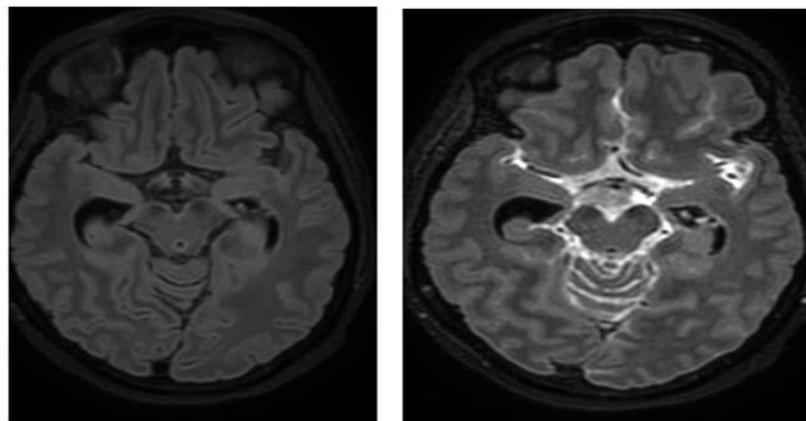
**Citation:** Shriram Varadharajan, Meena Nedunchelian. Contrasting FLAIR mechanisms in meningitis *Kauverian Med J.* 2026;3(3):59-61.

Academic Editor: Dr. Venkita S. Suresh

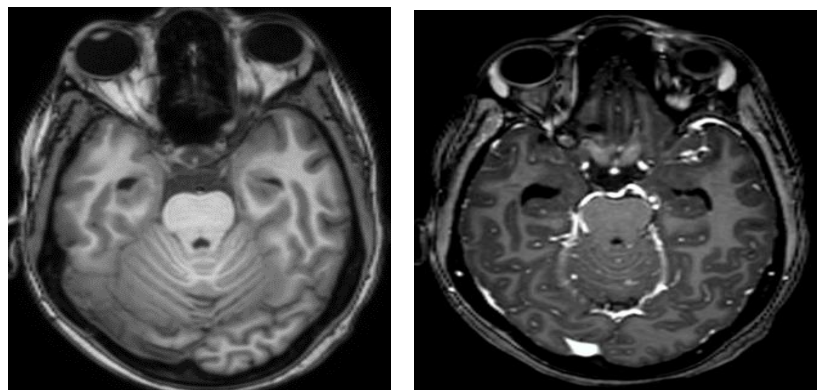
ISSN: 2584-1572 (Online)



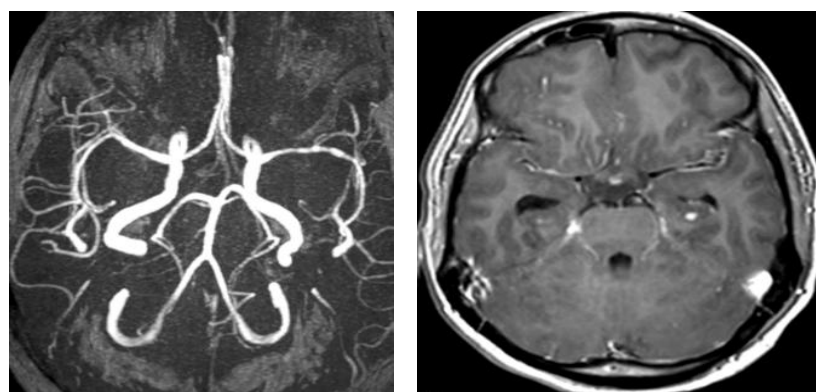
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**Fig (1):** Diffuse post contrast sulcal FLAIR changes, suggestive of basal meningitis and hydrocephalus.



**Fig (2):** Pre and Post contrast axial T1W images show subpial nodular enhancement along cerebellar folia and lack of meningeal enhancement as opposed to FLAIR



**Fig (3):** Axial MIP image TOF MRA shows asymmetrical irregular narrowing of the left MCA and contrast images show vessel wall enhancement s/o vasculitis

Differential diagnosis included granulomatous (tubercular and non-tubercular) meningitis, and partially treated meningitis. Further evaluation with lumbar puncture was done. Genexpert for mycobacterium tuberculosis turned out positive. This was a case of chronic granulomatous meningitis. Early vasculitis can also be picked up by vessel wall study so as to initiate aspirin with steroids if needed in the MDT also between Neurosciences and infectious diseases.

## 2. Discussion

Meningeal enhancement is an imaging finding commonly seen in diverse pathologies including but not limited to infections, autoimmune, inflammatory and neoplastic etiologies. Pathophysiology of post contrast meningeal enhancement: Break down of blood brain barrier leading to contrast leak and enhancement. Meningitis on imaging with subacute to chronic clinical presentations should raise suspicion of granulomatous /other non-granulomatous infections, partially treated meningitis and immunocompromised states with opportunistic infections like cryptococcus. However, basal cisternal enhancement is more specific for tubercular etiology. Autoimmune diseases of the brain usually present as a spectrum of imaging findings including white matter, optic nerves and spinal cord. Meningeal involvement though less common than other presentations, have

been reported in variants of MOGAD like FLAIR-variable unilateral enhancement of the leptomeninges (FUEL), which present as focal leptomeningeal enhancement rather than diffuse basal meningitis as in our case.

### 3. Neuroradiological - Learning points

Meningitis is a clinical diagnosis - role of imaging includes ruling out tonsillar herniation before lumbar puncture and diagnosis of complications of raised ICP, vasculitis with infarcts, hydrocephalus and parenchymal involvement.

Furthermore, pattern of meningeal involvement can provide clues to underlying disease.

- Use of appropriate MRI sequences is the key to early diagnosis
- Post contrast FLAIR is the most sensitive sequence for the diagnosis of meningitis
- Post contrast T1 often may not depict meningeal enhancement as clearly as FLAIR
- MR angiogram and Post contrast vessel wall imaging are useful for secondary vasculitis
- Autoimmune diseases can also present with leptomeningeal FLAIR enhancement especially in MOGAD (MOG Antibody disease), often unilateral along the sulcal spaces.

### 4. Acknowledgements

Dr. Swathi T, Prof Dr. K. Subramaniyan

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