

# A Prospective Study of Role of Magnetic Resonance Imaging in Central Nervous System Infections

Sunil Kumar Agrawal<sup>1</sup>, Shubhangi Gupta<sup>2</sup>, Ila Pahwa<sup>3</sup>, Sangita Singhal<sup>4</sup>, Bindu Agrawal<sup>5</sup>

<sup>1</sup>Professor & Head, Department of Radiodiagnosis, Muzaffarnagar Medical College, Muzaffarnagar, U.P., <sup>2</sup>3rd Year Resident, Department of Radiodiagnosis, Muzaffarnagar Medical College, Muzaffarnagar, U.P., <sup>3</sup>Professor & Head, Department of Medicine, Muzaffarnagar Medical College, Muzaffarnagar, U.P., <sup>4</sup>Professor & Head, Department of Pediatrics, Muzaffarnagar Medical College, Muzaffarnagar, U.P., <sup>5</sup>Professor, Department of Radiodiagnosis, Muzaffarnagar Medical College, Muzaffarnagar, U.P.

## Abstract

**Background:** Infections of the central nervous system (CNS) are important cause of morbidity and mortality world- wide owing to their life threatening complications. Today with the availability of excellent antimicrobials, many of these disorders are potentially treatable, making early recognition imperative. MRI has revolutionised the manner in which diagnosis is made. **Aims:** Our aim was to study the characteristic MRI imaging features of various CNS infections and correlate them clinically. **Subjects and Methods:** Total 50 (of all age groups) patients who were clinically suspected of CNS infections were subjected to MRI examination using MAGNETOM ESSENZA SIEMENS operating at 1.5 Tesla. MRI findings were analysed & correlated clinically. **Results & Conclusion:** CNS infections were almost equally distributed among males and females and were mostly found in paediatric population. Neurocysticercosis and Tuberculosis were the two most common CNS Infections diagnosed. Basilar meningitis is highly suggestive of tuberculosis.

**Keywords:** Encephalitis, Magnetic resonance imaging, meningitis, pyogenic, tuberculosis.

**Corresponding Author:** Dr. Shubhangi Gupta, 3rd Year Post Graduate Resident, Department of Radiodiagnosis, Muzaffarnagar Medical College, Muzaffarnagar (251203), Uttar Pradesh, India.

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## Introduction

Infections of the central nervous system (CNS) are important cause of morbidity and mortality world- wide.<sup>[1]</sup> Common aetiologies include bacterial, viral, fungal and parasitic infections, some of which are ubiquitous, while others primarily occur within endemic regions. The prognosis mainly depends on rapid identification of the site; extent; & nature of inflammation and pathogen to install effective antimicrobial treatment as early as possible. Whereas analysis of CSF, biopsy, and laboratory analysis remain the gold standard to identify the infectious agent for instance in meningitis, neuroimaging is crucial in clearly depicting inflammatory lesions of brain and spine.<sup>[2]</sup> Visualization of typical lesion patterns often allows a rapid diagnosis and subsequent therapeutic decisions. MRI is the superior for assessment of intracranial infections & allows for more sensitive evaluation of parenchymal & leptomenigeal disease.

This study was undertaken to evaluate various imaging features of CNS infections and to correlate clinically.

### Aims & Objectives:

1. To study the characteristic MRI imaging features of various CNS infections.
2. To see the correlation between clinical and radiological findings

## Subjects and Methods

This study was conducted on clinically suspected cases of CNS infections referred to the Department of Radiodiagnosis at Muzaffarnagar Medical College, Muzaffarnagar. Detailed clinical history with CNS examination findings & relevant laboratory investigations were noted. After taking informed consent, patients were subjected to MRI examination and spectrum of MRI findings were recorded.

**Study design:** Hospital based observational study (longitudinal, prospective) over a period of eighteen months.

**MR Imaging examinations** were performed using Magnetom Essenza Siemens one whole-body scanner operating at 1.5 Tesla.

**MRI Protocol:** Protocol consisted of localizers in coronal, axial, oblique and sagittal plane after proper positioning of the patient. The following sequences were used:

In the axial plane: Turbo spin echo T2- weighted sequence, Spin echo T1-weighted sequence, FLAIR- Fluid attenuated inversion recovery, GRE sequence.

T2 weighted imaging sequence in sagittal plane & coronal plane.

Contrast enhanced T1 weighted sequence was done wherever indicated (contrast agent: Gadolinium as intravenous contrast media in dose of 0.1mmol/kg of body

weight).

Diffusion weighted and ADC imaging was performed using echo planar imaging sequence.

MR spectroscopy was done whenever indicated.

The positive findings were recorded. MRI differentials were correlated with clinical differentials based on clinical & biochemical analysis.

## Results

CNS infections were almost equally distributed among males (52%) and females (48%) with slight preponderance in males. Neurocysticercosis (42.00 %) & tuberculosis (34.00%) were the two most common CNS infections diagnosed followed by pyogenic in 8 (16.00%) & viral in 4 (8.00%) subjects. Mean CSF glucose in our study was significantly lowered in tubercular (39.8 mg/dl) and pyogenic infections (23.8 mg/dl). Mean CSF protein in our study was raised in tubercular group (158.7mg/dl) and pyogenic (175.3 mg/dl). In the viral group, mean CSF protein (63.5 mg/dl) was slightly raised and the mean glucose level (75 mg /dl) was within normal limits. Lymphocytic pleocytosis was seen in viral and tubercular group. Polymorphonuclear pleocytosis was seen in the pyogenic group. Imaging features of neurocysticercosis, tubercular, pyogenic & viral infections were also analysed. [Table 1-3].

**Table 1: Distribution of various imaging findings in diagnosed Neurocysticercosis.**

Neuroimaging feature	Neurocysticercosis cases (n= 21)	Percentage
Cystic lesion (T1 hypointense & T2/FLAIR hyperintense)	15	71.42
Cystic lesion with eccentric T2 hypointense dot (scolex)	13	61.90
Ring enhancement	5	23.80
Size <20 mm	21	100
Perilesional edema	15	71.42
T1 & T2 hypointense with blooming on GRE/SWI	6	28.58
Extracranial cysticercosis	2	9.52

**Table 2: Imaging analysis of cases with tubercular infection (n = 17) & pyogenic infection (n = 8)**

Imaging parameter	No. of patients of tubercular infection (n= 17)	No. of patients of pyogenic Infection (n = 8)
Leptomeningeal enhancement	12 / 70.58	8 / 100
Pachymeningeal enhancement	1 / 5.88	0 / 0.00
Tuberculoma	16 / 94.11	-
Enhancing basal exudates	10 / 58.82	0 / 0.00
Infarct	1 / 5.88	5 / 62.50
Hydrocephalus	6 / 35.29	3 / 37.50
Abscess	0 / 0.00	0 / 0.00
Ventriculitis	0 / 0.00	0 / 0.00

**Table 3: Imaging analysis of cases with viral infection of the central nervous system (n=4)**

Imaging parameter	No. of patients	Percentage
Leptomeningeal/Pachymeningeal involvement	1	25.00
Temporal lobe involvement	0	0.00
Thalamus	2	50.00
Brain stem involvement	1	25.00
Cerebellar involvement	1	25.00
Presence of T2/FLAIR hyperintensity	4	100.00
Presence of diffusion restriction	2	50.00
Presence of hemorrhage	1	25.00
Presence of Hydrocephalus	None	0.00
Presence of infarcts	1	25.00

## Discussion

Mean CSF glucose in our study was significantly lowered in tubercular and pyogenic infections as compared to other groups. Mean CSF protein in our study was raised in pyogenic and tubercular group. The mean CSF protein was slightly raised and the mean glucose level in viral group was seen to be within normal limits.

Patients who presented with seizure & headache as presenting complaints & on MRI revealed Neurocysticercosis, no further laboratory investigations were done. Abnormal body movements was the chief presenting complaint observed in 81% patients and headache was seen in 33% of our patients. None of these patients had fever or altered sensorium as the presenting complaint. This was similarly observed by Robert Y Shih et al,<sup>[3]</sup> who postulated that patients develop seizure (50-70%), headache (43%) and hydrocephalus (30%) as scolices begin to die and being frequently asymptomatic during early stages of infection. We found all the 21 cases in our study was of parenchymal NCC type. None of the case showed other types of NCC like intraventricular / subarachnoid / spinal. In a study conducted by Zhao JL et al,<sup>[4]</sup> also postulated that the parenchymal NCC is the most common form of NCC followed by intraventricular type, subarachnoid type.

We found T1 hypointense & T2/FLAIR hyperintense lesions (cystic lesions) (15/21) with eccentric T2 hypointense dot / focus (scolex) (13/21) with perilesional edema (15/21) and few showing ring enhancement (5/21) as the most common imaging finding in our study. These findings favoured colloid vesicular stage of NCC. Our findings corroborated to larger extent with study by Lucato LT et al,<sup>[5]</sup> in which they described Colloid vesicular stage findings.

On imaging analysis of tubercular CNS infection, presence of tuberculomas (16/17) was the most common finding followed by leptomeningeal enhancement (12/17), enhancing basal exudates (9/17) [Figure 2a] and hydrocephalus (6/17) [Figure 2b]. This correlated well with Jerome H et al,<sup>[6]</sup> who found out most common findings are meningeal enhancement, hydrocephalus, basal exudates, infarcts and tuberculomas in their study. Similar results were also interpreted by Gupta K et al,<sup>[7]</sup> who found basal

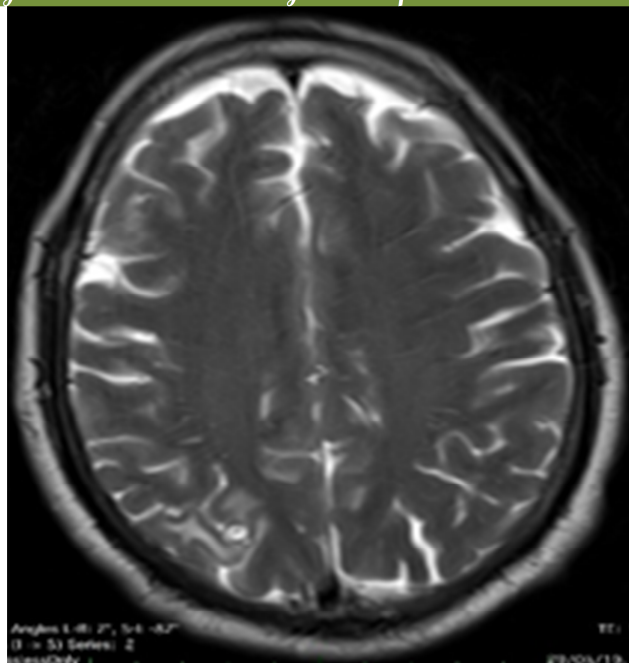
meningitis as the universal finding in tubercular meningitis. Hydrocephalus was noted in 6 [35.29%] of our patients. The incidence, predictive factors and the impact of hydrocephalus in tubercular meningitis was studied by Raut T et al,<sup>[8]</sup> who in his study of 80 patients with tuberculous meningitis showed that 52 (65%) had hydrocephalus at presentation.

On analysis of 8 patients of pyogenic etiology, leptomeningeal enhancement was the most consistent feature seen in all of our 8 (100%) patients of pyogenic meningitis [Figure 3a]. The predominant location of the leptomeningeal enhancement was in the cortical sulci in all (100%) of them as opposed to the basilar enhancement seen in our study in tubercular meningitis. Our result is similar to the study done by Oliveira CR et al,<sup>[9]</sup> who in their study of MRI findings in 75 CSF bacterial culture positive infants showed leptomeningeal enhancement to be the most common finding present in 57% of the cases. Pachymeningeal enhancement was not seen in any of our patients. The absence of pachymeningeal enhancement in pyogenic meningitis observed in our study correlated well with the study by Kioumehri F et al.<sup>[10]</sup> They studied the enhancement pattern in 83 patients with meningeal enhancement on MR scans. Pachymeningeal enhancements observed by them were attributed to proven cases of meningeal carcinomatosis (83% of cases) and in 100% of the reactive cases (due to trauma, shunt, surgery). Infarcts were seen in 5 (62.50%) of our patients. The location of the infarct was white matter in two, thalamus [Figure 3b & 3c] in two and basal ganglia in three patients. Associated hydrocephalus was seen in 3 (37.50%) patients. All the patients had reduced GCS & poor clinical outcomes (there were 3 deaths). These findings correlate well with the study of Wang et al,<sup>[11]</sup> in whom the authors described poor outcomes in this specific group of patients.

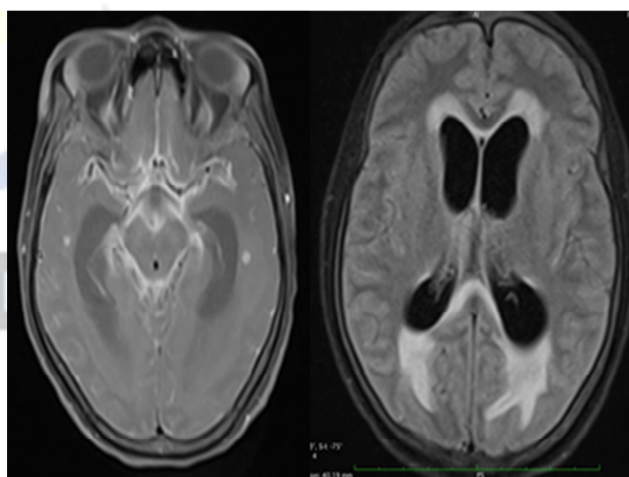
Diagnosis of viral encephalitis (kept as diagnosis of exclusion) was suspected based on fever with altered sensorium with or without focal neurological deficit or seizure in whom bacterial, fungal or tubercular meningitis were excluded and CSF showed mild protein rise with pleocytosis. On imaging analysis, we found thalamic involvement in 2 (50%), brain stem involvement in 1 (25%) and cerebellar involvement in 1 (25%) case. Concurrent leptomeningeal enhancement was noted in 1 (25%) and infarct was also seen in 1 (25%) case.

In our study, there was one patient with bilateral thalamic T2/FLAIR hyperintense signal [Figure 4a & 4b] with concurrent mild leptomeningeal enhancement. No hemorrhage was seen in bilateral thalamus in this patient. Another patient [Figure 5a, 5b & 5c] had T2/FLAIR hyperintensities in bilateral thalamus, midbrain, pons & medulla extending to bilateral middle cerebellar peduncle which also showed restriction of diffusion and patchy areas of blooming (hemorrhage) in these areas.

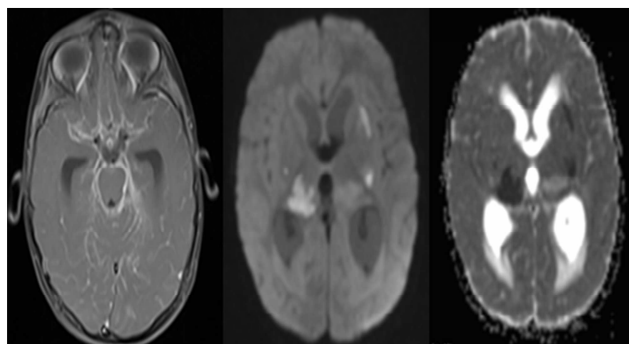
Both patients were suspected of Japanese encephalitis. Our imaging findings are somewhat related with study of Kumar S et al,<sup>[12]</sup> who documented the MRI features in seven patients of Japanese encephalitis. In all the patients MRI revealed bilateral thalamic lesions, which were hemorrhagic in five.



**Figure 1: Axial T2-Weighted image showing Colloid Vesicular stage in Right parietal lobe**



**Figure 2a & 2b: a shows Axial Contrast enhanced T1 weighted image showing enhancing exudates in perimesencephalic cistern. b. Axial FLAIR image showing hydrocephalus**



**Figure 3a-c: a Axial Contrast enhanced T1 Weighted image shows leptomeningeal enhancement. b: Axial diffusion weighted image and c : apparent diffusion coefficient image shows diffusion restriction in right thalamus showing infarct.**

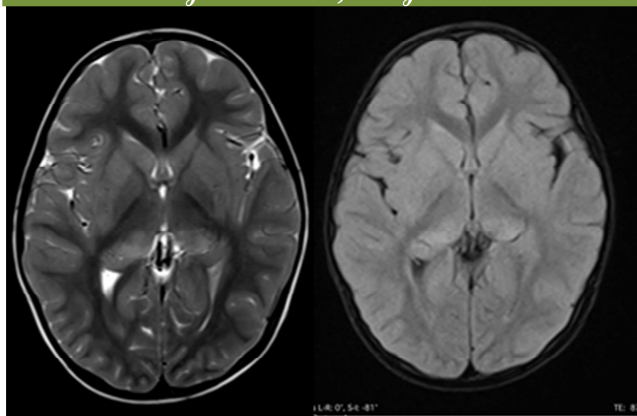


Figure 4: T2 Axial a) and FLAIR axial, b) depicting bilateral symmetrical thalamic involvement.

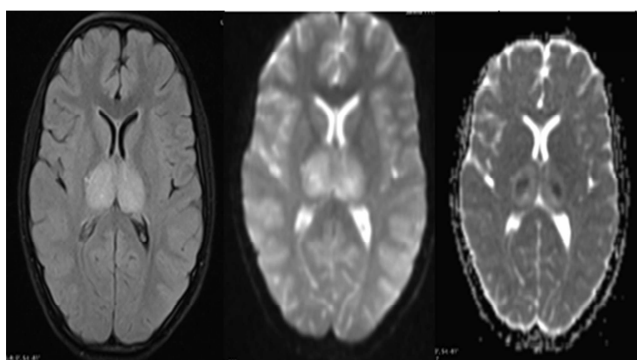


Figure 5a, 5b & 5c: Axial FLAIR a) showing symmetrical Bilateral Thalamic Hyperintensity. Diffusion weighted image b) and Apparent diffusion coefficient c) showing foci of diffusion restriction in Bilateral thalamic hyperintensity.

## Conclusion

In our study of 50 patients,

1. CNS infections were almost equally distributed among males (52%) and females (48%) with slight preponderance in males.
2. Neurocysticercosis (42.00 %) & Tuberculosis (34.00%) were the two most common CNS infections diagnosed followed by Pyogenic in 8 (16.00%) & Viral in 4 (8.00%) subjects.
3. Findings of lowered glucose and raised proteins may be seen in bacterial or tubercular infections of the central nervous system. Viral infection of the CNS present with normal CSF glucose and proteins, the latter however may sometimes be increased.
4. Neuroimaging in Neurocysticercosis revealed, Parenchymal NCC is the most common type of NCC, which in our study was seen in 100% cases. Colloid vesicular in 8 (38.09%) was the most frequent stage of NCC found in our study followed by nodular calcified stage in 6 (28.58%) cases.
5. Neuroimaging in tubercular infections revealed, basilar pattern of meningeal enhancement in tuberculosis is most common. When coupled with the presence of

tuberculomas, the findings are highly suggestive for the tubercular nature of infection.

6. On analysis of pyogenic infections, we concluded meningeal enhancement is the predominant feature in pyogenic meningitis with leptomeningeal enhancement seen more than pachymeningeal enhancement and cortical sulcal enhancement seen more than basal enhancement.
7. Neuroimaging in viral encephalitis revealed, hemorrhagic involvement of thalamus with/without basal ganglia and brainstem involvement may be seen in Japanese encephalitis.

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