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Imaging Features of Intracranial Tuberculosis on MRI: Correlation with Clinical Results and Laboratory Investigations

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Abstract

Aims and objectives: *To study the imaging features of intracranial tuberculosis on MRI and to correlate there MRI features with clinical findings and laboratory investigations.*

Material and methods: *This study was carried-out in the department of Radiodiagnosis at MMIMSR (Ambala). We included 50 patients with signs and symptoms suggestive of tuberculosis referred from various wards. Complete history was taken at the time of presentation. A thorough clinical examination was carried-out.*

Results: *It was observed that majority of the patients, i.e., 20 (40%) of patients were in the age group of 21 - 30 years. There was no gender predilection seen in our study. Out of 50 patients, 26 (52%) were female and 24 (48%) were male patients with male to female ratio of 1:1. Headache 37 (74%) and fever 37 (74%) were the chief clinical symptoms. Leptomeningitis (76%), tuberculomas (68%) and hydrocephalus (46%) were the most common manifestations of intracranial tuberculosis. 32 (95%) patients showed multiple tuberculoma lesions with or without conglomeration.*

Conclusion: *Diagnostic imaging like MRI plays a vitol role in diagnosis because of its proficiency in detecting intracranial lesions earlier than CT. Along with conventional MRI imaging, DWI, and proton magnetic resonance spectroscopy techniques also help in better tissue characterisation in intracranial TB.*

Key words: *Intracranial tuberculosis, magnetic resonance imaging, tuberculoma, hydrocephalus, leptomeningitis.*

Introduction

Tuberculosis, caused by *Mycobacterium tuberculosis*, continues to be a major public health problem in India. According to the World Health Organisation report, 4.8 lakh casualties were caused by tuberculosis in India in the year 2015, which is equal to 36 deaths per 1 lakh population[1].

Intracranial involvement is a severe form of this infection due to its high mortality rate and severe neurological complications. Involvement of CNS is seen in 2% - 5% of all patients with tuberculosis and in 10% cases of acquired immunodeficiency syndrome (AIDS)-related tuberculosis[2]. Intracranial manifestations of neurotuberculosis include meningeal and parenchymal diseases. Intraparenchymal disease usually presents as solitary or multiple tuberculomas and in other possible forms such as tuberculous abscesses, miliary TB, cerebritis and tuberculous encephalopathy[2].

MRI is a clinical aid in the initial detection of disease and is visually illustrated as unremarkable contrast between gray and white matter and ischaemia, infarct, abscess and haemorrhage. Contributing to this is MRI's inherent sensitivity as well as its capability to directly image in any place without reformatting, and to be undistorted by bony structures[3].

The most common radiographic findings associated with intracranial TB on MRI include enhancement of basal cisterns, tuberculoma, hydrocephalus, meningeal enhancement and infarction, usually of the basal ganglia[4].

Accurate and rapid diagnosis of intracranial TB, is necessary for the early institution of appropriate therapy for successful treatment, cure, better prognosis and prevention of long term sequelae[5]. Culture detects TBM in about 29.8% of patients as compared to rest of the microbiological modalities[6].

The BACTEC™ 960 system includes a liquid culture medium (BBL™ MGIT™ Mycobacteria Growth Indicator Tube), a growth supplement and an antibiotic. The Xpert MTB/RIF assay (Cepheid Inc.,Sunnyvale, CA, USA) is a cartridge based, semi automated, rapid molecular assay based on a real time heminested PCR test that detects the presence of *M. tuberculosis* complex bacilli and simultaneously determines the susceptibility to rifampicin[7]. The purpose of this study was to define the imaging spectrum and to improve the characterisation of lesions in CNS tuberculosis on MR

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imaging and to further correlate the clinical and imaging findings with GeneXpert MTB/RIF test and MGIT culture in the Indian population.

Material and methods

After approval from the institutional ethics review committee, a hospital-based descriptive, observational study was carried-out in the department of Radiodiagnosis at MMIMSR (Ambala) from December 2017 to September 2019. The study comprised of 50 patients presenting with signs and symptoms suggestive of tuberculosis referred from various wards. Complete history was taken at the time of presentation. A thorough clinical examination was carried-out.

The patients included in the study were those giving valid informed consent and the ones presenting with symptoms of tuberculosis for the first time like cough, fever and weight loss including the signs of meningeal irritation. The patients that were excluded from the study were those having contraindication to MR imaging such as aneurysmal clips claustrophobia, cochlear implants, compromised renal function (eGFR < 30ml/min) and cardiac pacemaker.

All the patients were subjected to detailed general, systemic and neurological examination. All relevant laboratory investigations like Hb, TLC, DLC and ESR were carried-out. CSF analysis and X-ray chest was performed in all cases.

MR imaging was conducted with the help of a 1.5T MR (Acheiva Philips medical systems Netherlands BV) with the application of standard head coil and an actively shielded whole body magnetic field gradient. Head was set and patient was fixed to avoid movement during and in between acquisition of images. All routine MRI sequences, i.e., T1W1 (TR - 542 ms, TE - 15 ms, slice thickness 5 mm) axial, T2W1 (TR - 4,047 ms, TE - 100 ms, slice thickness - 5 mm) axial, DW sequence (TR - 3,678 ms, TE - 116 ms, max b factor 1,000) and SWI sequences were taken along with T2 sagittal and FLAIR coronal sequence. A Gadolinium based agent was administered in dose of 0.1 mmol/kg and then contrast enhanced T1W sequence was taken in axial, coronal and sagittal planes. During analysis of MRI images, we documented the distribution, location and extension of lesion, signal intensity, diffusion characteristics and pattern of post contrast enhancement. Size of the parenchymal lesion was measured along its long axis. Images were transferred to a separate workstation where specialised software was used to analyse the images. Single voxel spectroscopy was done wherever required. They were corrected for artifacts.

Observation and Results

- Majority of the patients, i.e., 20 (40%) of patients were in the age group of 21 - 30 years. We included both children and adults. Our patients fell in the age group 7 years to 72 years. The minimum age of the patients was 07 years whereas the maximum age was 72 years. The mean age of our study group was 32.5 ± 16.5 years. Least incidence of CNS TB was seen in the age group of 0 to 10 years.
- There was a no significant gender predilection seen in our study. Out of 50 patients, 26 (52%) were female and 24 (48%) were male patients with male to female ratio of 1:1.
- Salient predominant findings of intracranial tuberculosis on MRI have been summarised in Table II.
- Headache 37 (74%) and fever 37 (74%) were the chief clinical symptoms associated with intracranial tuberculosis. Altered sensorium was seen in 12 (24%) patients and vomiting in 27 (54%). Associated clinical findings of tuberculosis like weight loss, cough and fever were seen in 40 (80%), 42 (84%) and 37 (74%) patients respectively.
- According to Table I, only 15 out of 50 patients had positive findings on chest X-ray. Hence, it was seen that tuberculoma can occur in patients without any active lesion in lungs.
- CSF culture, smear microscopy and GeneXpert for AFB-positivity in the present study were 44%, 8% and 16% respectively. The diagnosis was made by combination of clinical findings and other biochemical and radiological investigations. The use of single test as sole diagnostic test would lose a large number of patients with TBM as negative test does not rule-out TBM.
- Distribution of various manifestations of intracranial tuberculosis is given in Table II.
- 32 out of 34 patients of tuberculomas showed multiple lesions with or without conglomeration.
- Supratentorial hemisphere was more commonly involved in 30 (88%) cases of tuberculoma. Right parietal lobe was involved in 16 (47%) cases.
- Majority of the patients 22 (65%) had tuberculoma lesions with hypointense centre on T2W images with similar signal on DWI.
- All the cases of tuberculoma showed different grades of oedema, grade I oedema was noted in 4 (11.7%) cases, grade II oedema was noted in 28 (82.3%) cases and grade III oedema was noted in 2 (5.8%) cases.

Table I: Distribution of cases according to chest X-ray findings.

X-ray findings (N = 50)	No of cases	Percentage %
Hilar adenopathy	9	18
Miliary pattern	3	6
Apical infiltrates	9	18
Pleural effusion	2	4
Nodular shadowing	2	4
Normal	35	70

Table II: Distribution of abnormal MRI findings in study subjects.

Findings (N = 50)	No of cases	Percentage%
Leptomeningitis	38	76.0
Tuberculomas (Ring/solid/nodular enhancing lesions)	34	68
Hydrocephalus	23	46
Abscess	4	8
Infarcts/vasculitis	6	12
Ventriculitis	2	4
Basal exudates	35	70
Mass effect/midline shift	6	12

- All 4 (100%) cases of tubercular abscess had smooth enhancing walls, mass effect and oedema. All showed central diffusion restriction with peripheral SWI regular hypointense rim. In 3 cases, single lesion was encountered and multiple lesions were seen in the remaining one case. All lesions measured approx. 4 cm x 3.5 cm. All cases showed mass effect with midline shift. Diffusion restriction was seen in all the cases. In all 4 patients with brain abscesses, post-contrast T1-weighted spin-echo images delineated the capsule better than any other sequence. Likewise, in patients with meningitis; FLAIR images provided no more information than conventional spin-echo images, including the postcontrast T1-weighted spin-echo images. However, it was noteworthy that the cisternal lesions in the patient with tuberculous meningitis were more clearly seen on FLAIR images than on T2- or proton density-weighted images, as they appeared hyperintense relative to CSF on FLAIR images.
- 28 (70%) cases showed exudates in mainly suprasellar cistern followed by interpeduncular cistern. Grade I refers to non enhancing exudates which were seen in 2 (5%) patients. Maximum cases 32 (80%) had grade II basal exudates, i.e., hyperintense exudates outlining the cisterns. Grade III refers to enhancing exudates with enlargement of cisterns which were seen in 6 (15%) patients.
- Single and multivoxel MRS was performed in patients with emphasis on Cho/cr ratio, lipid and lactate peaks. All the patients had Cho/cr < 3, with mean ratio of 23 ± 0.5 Out of 20 patients with tuberculoma, 16 had raised lipid peak at 0.9, 1.3 and 2.0 ppm.

Table III: Distribution of tuberculomas.

Tuberculomas	Number of cases	Percentage of cases
Tuberculomas in isolation	8	23.5%
Tuberculomas associated with meningitis	26	76%
Total	34	100%

Table IV: Signal characteristics of tuberculomas on MRI.

Tuberculomas (N = 34)	Signal intensity	No. of cases	Percentage
T1	Hyper	0	0%
	Hypo	32	94.1%
	ISO	1	5.8%
T2 centre	Hyper	10	29.4%
	Hypo	22	64.7%
	ISO	2	5.9%
T2 capsule	Hyper	4	11.8%
	Hypo	1	2.9%
	ISO	29	85.3%
DWI restriction	N	24	70.4%
	Y	10	29.4%
Flair	Hyper	34	100%
SWI Capsule	Hypo	33	97%
Post-contrast enhancement	Solid	4	11.7%
	Ring	28	82.3%
	Target	2	5.8%

Discussion

Intracranial tuberculosis is one of the most devastating manifestations of TB and a challenging public health issue of considerable importance and magnitude in India. The tubercular patients constituted a good number of radiological workload.

There was no particular age group selected in this study. Jyothi *et al* (2016)[8] found that there was no specific age group for tuberculomas.

The results of the gender distribution are in concordance

with the study done by Jyothi *et al*[8] on 75 patients that also showed no obvious difference in the distribution of cerebral tuberculosis among the male (57%) and female (43%).

The major complaint in nearly in all patients was cough. In tuberculomas, one-third of the patients have evidence of tuberculosis elsewhere. Thus, a past history of tuberculosis is common and is of great value in further diagnosis.

The predominant radiographic pattern on X-ray chest of some of the patients in our study is depicted in Table I. Out of 50, only 15 (30%) patients showed findings on chest X-ray. Rest 30 patients showed no obvious abnormality. Sometimes the focus is quite small and not visible on radiographs. This was in concordance with the study conducted by Kumar *et al*[9] and Aurangzeb *et al* (2016)[10] that stated that only 30% of TBM patients showed chest X-ray positive results. This was in discordance with the study done by Yarami *et al* (1998)[105] that stated hilar adenopathy as the major finding in X-ray.

The diagnosis of TBM was difficult and mainly based only on clinical and preliminary cerebrospinal fluid (CSF) findings without definitive microbiologic confirmation. Few of the patients that did not show positive results on CSF culture, were seen responding well on ATT.

Most common imaging findings of TB on MRI are summarised in Table II. These findings are consistent with the study by Uysal *et al* (2001)[11] who found enhancement of meninges present in up to ninty per cent of cases and considered it to be the most important feature of tubercular meningitis. Similar results were also interpreted by Ojha *et al* (2019)[12] who found leptomeningeal enhancement in about seventy eight per cent of the cases. Shahzad *et al* (2019)[13] stated mass effect/midline shift in 15% of cases. Andronikou *et al* (2004)[14] showed sensitivity of basal enhancement to be as high as 89% in making the diagnosis of tubercular meningitis. Tuberculoma was found in 34 (68%) patients which is not in agreement with the study by Kumar Raju *et al* (2015)[15] in which tuberculoma was found in 25% of patients. Hydrocephalus was seen in 23 (46%) of our patients. The incidence, predictive factors and impact of hydrocephalous in tubercular meningitis was studied by Raut *et al* (2013)[16] who in their study of 80 patients with tuberculous meningitis showed that 52 (65%) had hydrocephalus at presentation. Factors associated with hydrocephalus include advanced stage of disease, severe disability, duration of illness > 2 months, diplopia, seizures, visual impairment, papilloedema, cranial nerve palsy, hemiparesis. Our findings are also similar to a community-based study by Davis *et al* (1993)[17] which analysed 54 patients with definite or probable tuberculous meningitis and observed ventricular dilatation on CT or MRI in 52% of patients. Azeemuddin *et al* (2019)[18], concluded tuberculoma (40%) hydrocephalus (26%), and leptomeningitis (41%) as the most prominent findings in CNS tuberculosis. Similar findings were noted in our study.

Clinical manifestations of tuberculomas depend upon their location, most being located in the supratentorial compartment. The pleomorphism is mainly related to individual differences in size and topography of the lesions. Features of raised ICP are seen in tuberculomas due to their mass effect on adjacent structures.

However, in a study conducted by Garg, Meena *et al* (2017) seizures (86.6%) were seen in the major proportion of the tuberculoma patients followed by headache (73.3%) and fever (60%). Kumar *et al* (2016)[9] also stated convulsions as the most common clinical feature of tuberculomas in about 66.6% patients. In our study 34 patients were diagnosed as having tuberculoma on MRI.

In our study, on T1W images, the intensity of the lesions varied from isointense to hypointense and, on T2W, from hypointense to hyperintense with predominantly hypointense centre. Diagnosis of tuberculoma was based on MRI appearances, clinical findings and past history. Intensity on T2 weighted images is variable and is dependent on the relative proportion of macrophages, cellular infilterates and fibrosis. Granulomas, which were frankly hyperintense on TW images, exhibited increased cellular infilterates scanty macrophages and very little fibrosis, while the hypointense lesions showed greater number of macrophages, more fibrosis and gliosis.

Caseating granulomas with central liquefaction were described as hypo on T1 and hyper on T2 with peripheral hypointense rim on T2 and rim enhancement on contrast.

Salgado *et al* (1989)[19] conducted a study on 6 patients. They found that lesions were hypointense in all cases on T1. T2 lesions varied with the stage of the disease.

Wassey *et al* (2003)[20] in a study of 100 patients found mainly multiple lesions. After contrast enhancement more than 450 lesions were seen on CT and MRI images in all 100 scans. A hypointense core with a hyperintense rim was the most common signal characteristic on T2W MRI. The central hyointensity on T2 wighted and FLAIR images reflected extensive necrosis and hypercellularity. Mass effect and midline shift were present in 18 patients. The present study is in concordance with the study by Gupta *et al* and Wassey *et al*.

In our study, on DWI imaging out of 34 cases of tuberculoma, 10 patients' lesions showed restricted diffusion likely due to liquefaction. Mishra *et al* (2004)[21] and Batra *et al* (2004)[22] showed similar kind of results in the study by stating that those lesions which showed central hyperintense signal on T2 were hyperintense on DWI too. Vasudev *et al* (2004)[23]

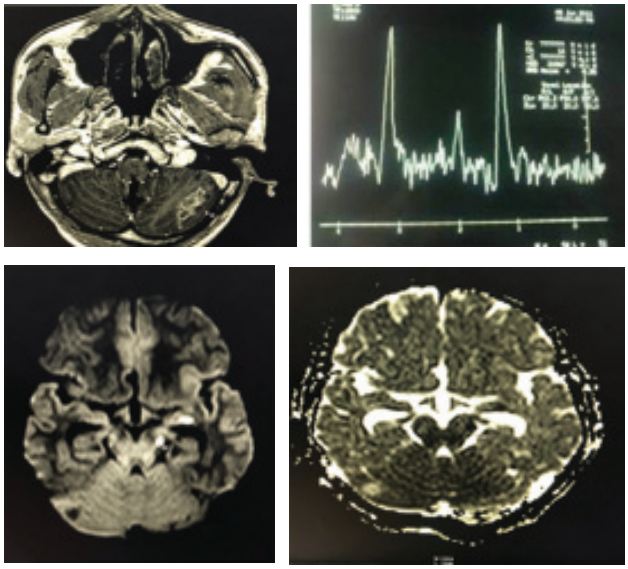

Fig. I: *A 25-year-old male patient with TBM, tuberculomas and infarct: Post-contrast T1 axial image shows nodular enhancing lesions in inferior aspect of left CP angle with leptomeningeal enhancement in cerebellar folia. MRS shows lipid peak at 1.3 ppm. DWI/ADC mapping show restrictred diffusion in left cerebral peduncle of midbrain and medial temopral lobe.*

stated that diffusion restriction is not a criterion of diagnosing tuberculoma.

Our results of tuberculoma on DWI are in concordance with the study by Batra *et al* (2004)[22] Maheshwarappa *et al*[24] and Mishra *et al*[21] and contradictory to the results of Vasudev *et al*.

Perilesional oedema was seen to be of lesser severity (grade I or II) in brainstem lesions because of the compactness of the tissues in those areas. Perilesional oedema was seen to be of higher grade (grade II and grade III) in patients with conglomerate lesions.These findings were in concordance with the study done by Jyothi *et al* (2016)[8] that stated grade I oedema was noted in 9 (15.6%) cases, grade II oedema was noted in 29 cases and grade III oedema was noted in 20 (34.3%) cases.

Intracranial abscess was found in 4 patients contributing 8% of the total 50 cases of intracranial tuberculosis. All the lesions were smooth and thin walled and more than 3 cm in size. Abscess showed central hypointense signal due to necrosis with hyperintense rim on T2W1 showed thick ring enhancement on post-contrast images. Diffusion restriction was seen in all the cases. Similar results were obtained in studies by Gupta *et al*[25] and Mohindra *et al*[26].

In the present study, out of 50 patients, 40(80%) patients presented with features of meningitis. Out of the 40 patients presenting with meningitis (with or without tuberculomas) features of infarction were present in only 6

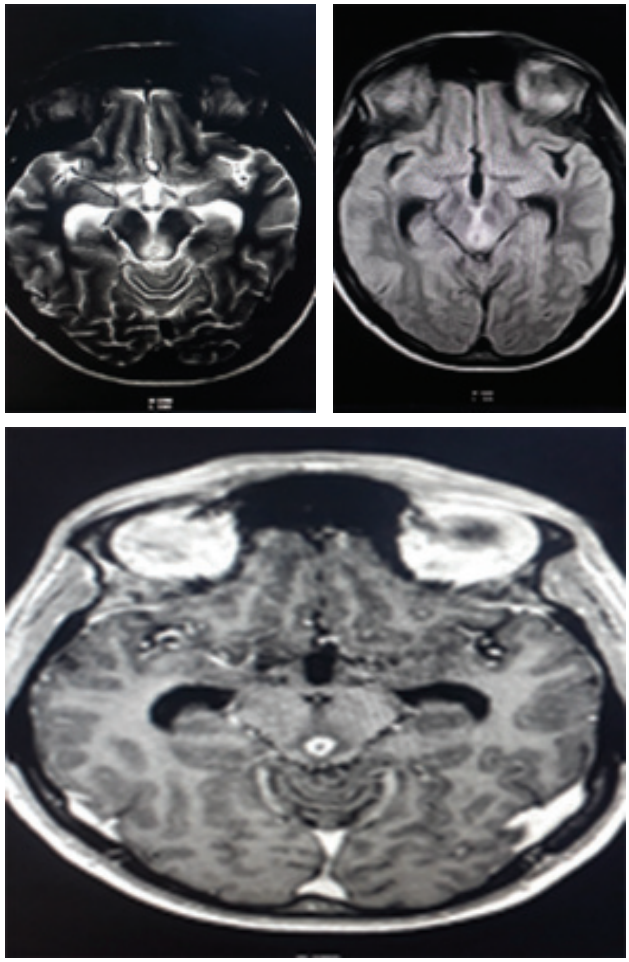

Fig. II: *A 34-year-old patient with midline tuberculoma: T2W/FLAIR images show a hypointense granuloma with hyperintense rim in the region of cerebral aqueduct. Post-contrast T1W image shows ring enhancement in post-contrast images. Mild hydrocephalus is seen.*

(15%) of cases.Thick area of enhancement was seen in the region of the basal cistern and the sylvian fissure. Basal meningeal enhancement, in fact, demonstrated thick basilar exudates. The interpeduncular fossa, pontine cistern, ambient cistern, suprasellar cisterns, and Sylvian fissures were the sites of predilection for the thick basilar exudates. MRI depicted diffuse enhancing lesions in the interpeduncular fossa, pontine cistern, and the perimesencephalic and suprasellar cisterns.

Meningeal hyperintensities are quite subtle on T1 and T2 images and are not detected easily abnormal meningeal enhancement was seen in 95% of the patients in the form of enhancement of pia-arachnoid after administration of a contrast material and are seen in the subarachnoid spaces of the sulci, basal cisterns, along the inner table of the skull, and in dural folds of the falx and tentorium.Tentorial and cerebellar meningeal enhancement was less commonly seen. Out of 23 cases of hydrocephalus,

communicating hydrocephalus was present in 21 (91%) patients due to inflammatory exudates occupying the basal cisterns and circle of Willis. Non communicating hydrocephalus was seen only in 2 (8%) cases due to obstruction of the cerebral aqueduct by a subependymal tuberculoma as shown in Table/Fig. VI. Similar findings were stated by Jyothi *et al* (2016)[8] in their study.

Cranial nerve involvement was seen in 3 (7.5%) patients due to ischaemia and nerve entrapment in the basal exudates was most commonly seen in 2nd cranial nerve. Taheri *et al* (2015)[4] stated cranial neuropathy in about 17 - 40% of cases, most commonly affecting the second, third, fourth, and seventh cranial nerves.

MRS: In the present study out of 20 patients,16 (80%) revealed lipid peaks and rest of the 4 (2%) did not show lipid peaks. 3 cases (15%) showed elevated lactate peak and 3 cases (15%) show elevated lipid and lactate peaks. The detection of these lipids could be due to lipid laden macrophages.The presence of lipid was seen in both lesions with increased and decreased signal intensity in the centre on T2W and DWI sequences which amplifies so that both solid caseation and liquefaction necrosis in tuberculoma can yield lipid peak on MRS.The presence of lipids peak in liquefied necrosis is related to presence of lipid in tubercular bacilli as well as breakdown products of gray-white matter. In solid caseation necrosis, infiltration with lipid-laden macrophages probably contributes to lipid signal.

In all 20 cases, the choline/cr ratio was less than two, indicating minor but significant neuronal loss. Gupta *et al* (2001)[25] stated that the MRS of the tuberculoma demonstrates lipid peaks at 0.9, 1.3, 2.0 and 2.8 ppm and phosphoserine peak at 3.7 ppm. Lipid as a hallmark of tuberculoma was first stated by Gupta *et al* (1996) [27]. Thus, our study is in concordance with the studies done by Gupta *et al* (2001)[25].

Thus, MRI is an excellent modality for confirmation of presence and complications of cerebral tuberculosis even in CXR negative, CSF culture negative and smear negative patients. Based on MRI findings, empirical treatment with ATT can be started.

Limitation

Our study has limitation of small sample size including only 50 patients without any control population which hinder evaluation of actual specificity and accuracy of these imaging modalities.

References

1. World Health Organisation. Global Tuberculosis Report 2016 cited 30 June 2016. Available from: http://who global tuberculosis report.
2. Bathla G, Khandelwal G, Maller VG *et al*. Manifestations of cerebral tuberculosis. *Singapore Med J* 2011; 52 (2): 124-30.
3. Garg RK, Malhotra HS, Jain A. Neuroimaging in tuberculous meningitis. *Neurol India* 2016; 64 (2): 219-27.
4. Sanei Taheri M, Karimi MA, Haghighatkhah H *et al*. Central nervous system tuberculosis: An imaging-focused review of a reemerging disease. *Radiol Res Pract* 2015; 2015: 202806. doi:Io.1155/2015/202806.
5. Bernaerts A, Vanhoenacker F, Parizel P *et al*. Tuberculosis of the central nervous system: overview of neuroradiological findings. *Eur Radiol* 2003; 13 (8): 1876-90.
6. Venkataswamy MM, Rafi W, Nagarathna S *et al*. Comparative evaluation of Bactec 460TB system and Lowenstein Jensen medium for the isolation of *M. tuberculosis* from cerebrospinal fluid samples of tuberculous meningitis patients. *Ind J Med Microbiol* 2007; 25: 236-40.
7. Nhu N, Heemskerk D, Thu D *et al*. Evaluation of GeneXpert MTB/RIF for Diagnosis of Tuberculous Meningitis. *J Clin Microbiol* 2013; 52 (1): 226-33.
8. Jyothi JSA, Sreevani I. Spectrum of intracranial tuberculosis on magnetic resonanceimaging. *J Evolution Med Dent Sci* 2016; 5 (1): 71-8.
9. Ashok Kumar EA, Jijiya Bai P. A clinical study of CNS Tuberculomas. *IAIM* 2016; 3 (6): 101-6.
10. Aurangzeb S, Badshah M. Chest Radiographic Findings in Neurotuberculosis without pulmonary signs and symptoms. *J Col Physicians Surg Pak* 2008; 18 (1): 27-30.
11. Uysal G, Köse G, Güven A *et al*. Magnetic resonance imaging in diagnosis of childhood central nervous system tuberculosis. *Infection* 2001; 29 (3): 148-53.
12. Ojha M, Verma SR, Saran S *et al*. Imaging of CNS Infections with Clinico-pathological Correlation. *IJCMR* 2019; 6 (5): E6-E10.
13. Shahzad G, Nayab S, Shaikh AH *et al*. Tuberculoma Brain and its Early Radiological Presentation; Descriptive Analytical Study. *J Liaquat Uni Med Health Sci* 2019; 18 (02): 160-3.
14. Andronikou S, Smith B, Hatherhill M *et al*. Definitive neuroradiological diagnostic features of tuberculous meningitis in children. *Pediatr Radiol* 2004; 34: 876-85.
15. Kumar R, Kumari P, Verma N. Evaluation of neuroimaging studies in CNS infections and its correlation with clinical presentation in children in Bihar. *Int J Med Applied Sci* 2015; 4: 224-9.
16. Raut T, Garg RK, Jain A *et al*. Hydrocephalus in tuberculous meningitis: Incidence, its predictive factors and impact on the prognosis. *J Infect* 2013; 66: 330-7.
17 Davis LE, Rastogi KR, Lambert LC *et al*. Tuberculous meningitis in the southwest United States: A community based study. *Neurology* 1993; 43 (9): 1775-8.
18. Azeemuddin M, Alvi A, Sayani R *et al*. Neuroimaging findings in Tuberculosis: A single-center experience in 559 Cases. *J Neuroimaging* 2019; 29 (5): 657-68.
19. Salgado P, Del Brutto OH, Talamás O *et al*. Intracranial tuberculoma: MR imaging. *Neuroradiology* 1989; 31 (4): 299-302.
20. Wasay M, Kheleani BA, Moolani MK *et al*. Brain CT and MRI findings in 100 consecutive patients with intracranial tuberculoma. *J Neuroimaging* 2003; 13: 240-7.
21. Mishra AM, Gupta R, Jaggi RS *et al*. Role of diffusion *in vivo* mrs in differential diagnosis of ring enhancing intracranial cystic mass lesions. *J Computer Assisted Tomography* 2004; 28 (4): 540-7.
22. Batra A, Tripathi RP. Diffusion weighted MRI and MRS. In evaluation

of focal cerebral tubercular lesions. *Acta Radiologica* 2004; 45 (6): 679-88.

23. Vasudev MK, Jayakumar PN, Srikanth SG *et al*. Quantitative magnetic resonance technique in evaluation of intracranial tuberculosis. *Acta Radiologica* 2007; 48 (2): 200-6.
24. MaheshwarappaR, Agrawal C, Bansal J. Tuberculoma versus Neurocysticercosis: Can Magnetic resonance spectroscopy and diffusion weighted imaging solve the diagnostic conundrum? *JCDR* 2019; 13 (6): TC01-TC06.
25. Gupta RK, Vatsal DK, Husain N *et al*. Differentiation of tuberculous from pyogenic brain abscesses with *in vivo* proton MR spectroscopy and magnetisation transfer MR imaging. *Amer JNeuroradiol* 2001; 22 (8): 1503-9.
26. Mohindra S, Savardekar A, Gupta R *et al*. Tuberculous brain abscesses in immunocompetent patients: A decade long experience with nine patients. *Neurol India* 2016; 64: 66-74.
27. Gupta RK, Roy R, Poptani H *et al*. Finger printing of Mycobacterium tuberculosis in intracranial tuberculomas using *in vivo*, *ex vivo* and *in vitro* proton MR spectroscopy. *Magn Reson Med* 1996; 36: 829-33.