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Original Research Article

Role of CT in Evaluation of CNS Tuberculosis in Pediatric Age Group

Roushan Kumar[1], Ezzat Khalda[2]

[1]Assistant Professor, Department of Radio-diagnosis, Shree Narayan Medical Institute and Hospital, Saharsa, Bihar.

[2]Professor and HOD, Department of Radio-diagnosis, Shree Narayan Medical Institute and Hospital, Saharsa, Bihar.

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Corresponding author: Dr. Roushan Kumar
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Abstract

Background: This study was conducted to document the spectrum of pathological changes seen on CT scan as part of investigative battery for diagnosis of neurotuberculosis in children, to establish a clinico-radiological correlation as regards, initial presentation. Among the 30 patients selected none presented with TB spine. In the subsequent CT scans during the follow up of cases of intracranial TB, the resolution of the lesions on antitubercular therapy or any complication or sequelae noted was correlated with the clinical outcome. The Aims of this study is to identify the CT findings of the CNS tuberculosis in pediatric age group, correlate the CT imaging feature with clinical findings chest X-ray and biochemical, cytological as well as microbiological analysis of CSF aspirate, evaluate the pre-contrast and post contrast CT images to identify the complications of CNS tuberculosis such as abscess, infact, hydrocephalus etc and CT monitoring of the effect of therapy upto resolution or manifestation of any further complication during the course of therapy.

Methods: Thirty consecutive Pediatric patients presenting during the period of January 2023 to December 2023 to the Shree Narayan Medical Institute & Hospital, Saharsa, and Bihar especially from the department of Pediatrics were taken for the present study. The age group was from 1 year to 15 years. 20 patients were males and 10 were females. In all these patients the clinical and the laboratory parameters were suggestive of intracranial tuberculosis.

Results: The maximum number of cases in this study were in 4-6years (33.34%) and 7-9 Years (26.67%) year's age group. The male incidence in this study was (66.67%). The overall male: female ratio is 2:1 but the ratio in 1-3 years and 4-6 years age group is 1.5:1 & 4:1 respectively. Chest X-ray in this study showed abnormality suggestive of pulmonary tuberculosis in (26.67%). In our study hydrocephalus was found in 20/30 patients, of which two patients had posterior fossa tuberculomas. All cases (100%) of meningitis had hydrocephalus at presentation ranging from mild to severe degree. In this study focal parenchyma lesions were seen in 12 of the 30 cases (40%). All of these cases had isolated parenchymal lesions. The intracranial tuberculosis may present as a space occupying lesion in various morphological patterns. Single ring lesions in 8/12 (66.67%) patients were among common morphological pattern in this study. Two patients (16.67%) had multiple disc/ring lesions and 2 (16.67%) had conglomerate lesions.

Conclusion: New lesions developed in a couple of patients while they were still on antitubercular therapy treatment. These patients suffered from residual disability at 6 months of therapy. No definite pattern of resolution was found for any particular CT presentation on the initial scan.

Keywords: CT Scan, CNS, Antitubercular therapy, Hydrocephalus.

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Introduction

CNS tuberculosis usually results from hematogenous spread, while direct spread from intra- or extracranial focus is rare. [1] The clinical and radiologic manifestations of CNS tuberculosis may mimic other infectious and noninfectious neurological conditions, such as brain tumors. Therefore, familiarity of infectious diseases specialists with the imaging presentations of CNS tuberculosis is essential for prompt and accurate diagnosis of this entity. Most important favourable prognostic factor is early diagnosis and institution of treatment. High index of suspicion is a prerequisite for diagnosis. Early diagnosis may be delayed in children because they are usually less than 3 years of age with insidious onsent of non-specific signs and symptoms and a normal chest x-

ray in 28-43% of cases. In addition, delay also occurs due to lack of health facilities close to patient's residence i.e. in remote rural tribal areas. Meningitis is the most common manifestation of CNS tuberculosis which is most frequently seen in the children and adolescents.[2] Tuberculous meningitis is mostly due to the hematogenous spread of Mycobacterium tuberculosis; however, it can also occur secondary to extension and/or rupture of a subpial or subependymal focus (i.e., Rich focus) to the subarachnoid spaces or into the ventricular system . Tuberculous meningitis often has an insidious course with a nonspecific clinical presentation in early stages, especially in children. Therefore, the imaging plays a key role in the timely diagnosis and decreasing the morbidity and mortality.

Intracranial infection by mycobacterium tuberculosis presents as meningitis or space occupying lesion. The diagnosis is based on history of contact, and Montoux test (which may be negative in 6.4-64% of cases). The clinical features and hematological investigation are in no way specific. The more specific tests like antigen detection in the CSF and DNA probe are available in research institutes only. The history of chronic illness and the fact that most chronic meningitis turn out to be due to mycobacterium tuberculosis infection, the clinical diagnosis of neurotuberculosis is made. Therefore, radiology has an important role in work up of these cases and hence the early diagnosis of the patients.

Skull x-ray may show calcification in 6% of cases and signs of raised intracranial tension in 20% of cases. Both these changes are neither specific nor diagnostic. Previously neuroangiography, pneumo enephalography and isotope scanning were used to assess the abnormalities, but the findings are not specific in majority of cases. Hence, they are obsolete in the management of neurotuberculosis, though they can suggest the diagnosis.

Similarly granulomatous spondylitis is most commonly caused by Mycobacterium tuberculosis. It accounts for 6% of new extrapulmonary tuberculosis.

Material and Methods

Thirty consecutive Pediatric patients presenting during the period of January 2023 to December 2023 to the Shree Narayan Medical Institute & Hospital, Saharsa, and Bihar especially from the department of Pediatrics were taken for the present study. The age group was from 1 year to 15 years. 20 patients were males and 10 were females. In all these patients the clinical and the laboratory parameters were suggestive of intracranial tuberculosis.

The clinical suspicion was based on:

1. History and Signs symptoms of the patients.
2. A positive Montoux test (>10 mm in diameter)
3. General physical examination, and
4. Neurological examination.

Laboratory supportive evidences were:

1. CSF – in cases of TBM. Each sample was examined macroscopically for cobweb formation, analyzed bio-chemically and cytologically;
2. Demonstration of AFB in CSF or elsewhere in the body e.g., lymph nodes, gastric aspirate etc.

Methods of Study

1. History – A detail history of patient was taken.

2. Physical examination –

a) Thorough general physical examination.
b) Complete neurological examination.

3. Laboratory investigation –

a) Montoux Test
b) CSF study.
c) Demonstration of AFB in sputum or gastric aspirate

4. Chest X-ray

5. CT – CT evaluation was done in the CT scan unit of the Department of Radiodiagnosis, Shree Narayan Medical Institute and Hospital, Saharsa, Bihar. Patients were evaluated by SEIMENS SOMATOM SP fourth generation SPIRAL CT scanner involving pre contrast or non-contrast CT (NCCT) and then post contrast or contrast enhanced (CECT) studies as required.

The limitation of all plain radiographic techniques is the two dimensional representation of three dimensional structures. The linear attenuation co-efficient of all structures in the path of x-ray beam form the image.

Computed tomography obtains a series of different angular x-ray projections that are processed by a computer to give a section of specified thickness. The CT image comprises a regular matrix of picture elements (pixels). All of the tissues contained within the pixel attenuate the x-ray projections and result in a mean attenuation value for the pixel. This value is compared with the attenuation of O Hounsfield unit (HU), and the scale is 2000 HU wide. Air typically has an HU number of – 1000; fat is approximately – 100 HU; soft tissues are in the range + 20 to + 70 HU; and bone is usually greater than + 400 HU. Modern multislice helical CT scanners can obtain images in subsecond times and imaging of the whole body can take as little as single breath hold of 20-30 seconds.

No specific preparation is required for CT examination of the brain. But nausea and emesis being a side effect of contrast medium it is preferred to scan the patient's empty stomach and hence preventing aspiration of food material. Studies usually require intravenous contrast medium that contains iodine, so enhancing the arteries and veins and defining their relationships to a greater extent.

The contrast medium used is the low osmolar nonionic monomer "omnipaque". Each of these molecules are tri-iodinated monionizing compounds and therefore in solution they provide 3 atoms of iodine to one osmotically active particle (the entire molecule) producing an iodine: particle ratio of 3: 1. Radio-opacity is dependent on the iodine concentration of the solution and is therefore dependent on the number of iodine atoms per molecule and the concentration of the molecule in the solution. Low osmolality reduces the pain of intra-arterial injections. Adverse reactions of contrast media include both idiosyncratic anaphylactoid reactions and nonidiosyncratic reactions. Minor reactions include flushing, nausea arm pain, pruritus, headache, vomiting and urticaria which are of short duration, self-limiting and require no treatment. Intermediate reactions include hypotension and bronchospasm which usually responds to chlorphenaramine (for urticaria), diazepam (for anxiety), salbutamol inhalation or hydrocortisone or adrenaline if required for bronchospasm. Severe life threatening reactions include convulsions, unconsciousness, laryngeal oedema, severe bronchospasm, cardiac arrhythmias, and pulmonary edema. Management includes maintenance of air ways, oxygen cylinder, artificial respiration, external cardiac massage and electrical defibrillation and drugs like diuretics, barbiturates and diazepam (for convulsion), hydrocortisone (for bronchospasm), chlorphena ramine, adrenaline (0.01 ml/k body weight Sc or 1M repeated at 10-20 minutes interval).

Generally all studies are performed with the patient supine and images are obtained in the transverse or axial plain.

Occasionally, direct coronal images are obtained in the investigation of cranial and maxillo-facial abnormalities, in these cases the patient lies prone with the neck extended and the gantry appropriately angled.

Serial CT section of cranium was obtained in each case using Reid's base line as anatomical landmark. Patients were evaluated with axial scans of 5 mm slice thickness with interslice gap of 5 mm. Thin sections were taken in areas of interest as required as in posterior fossa to reduce beam hardening artifact and for increasing the conspicuity of small lesions.

The direct contrast scans were performed after giving intravenous contrast material in the dose of 1-2ml/kg body weight. The scan time varied from 20 to 30 seconds. The children were sedated as and when required after taking full precautions.

Follow up CT scans were done in all patients for six months exept in those cases who died earlier.

In a few patients, CT scans were done prior to two months and later than 6 months as the clinical situation demanded. These scans were evaluated in detail and the findings were recorded on proforma (specimen enclosed).

Results

Table 1: Age and Sex Distribution (n=30)

Age group (in yrs)	Total	Females	Males	Total (%)	Female (%)	Males (%)
1-3	5	2	3	16.67%	40%	60%
4-6	10	2	8	33.33%	20%	80%
7-9	8	3	5	26.67%	37.5%	62.5%
10-12	5	2	3	16.67%	40%	60%
13-15	2	1	1	6.6%	50%	50%

Table 2: Clinical Features (n=30)

Clinical Presentation	No. of Cases	Percentage
Fever	16	53.33%
Meningeal Signs	15	50%
Altered Consciousness	11	36.67%
Vomiting	11	36.67%
Convulsions	18	60%
Visual disturbance	4	13.33%
Headache	12	40%

Table 3: Clinical Signs (n=30)

Signs	No. of Cases	Percentage
Signs of meningeal irritation	15	50%
Hemiparesis	2	6.67%
Decerebrate posture	2	6.67%
Papilledema	12	40%
Sensorial changes		
• Comatosed	3	10%
• Disoriented/drowsy	6	20%
• Irritable	9	30%
• Normal	12	40%
Cranial nerve palsies	9	30%

Table 4: Analysis of Chest X-ray (n=30)

	No. of Cases	Percentage
Normal CxR	22	73.34%
Abnormal CxR	8	26.67%
• Miliary TB	1	
• Hilar/mediastinal lymph glands	6	
• Consolidation	1	

Table 5: Biochemical Analysis of CSF (n=15)

	No. of Cases	Percentage
Raised CSF proteins	14	93.75%
Reduced sugar	11	73.34%

Table 6: Grade

Grade	No. of Cases (n = 30)	Percentage
Normal (upto 30%)	10	33.34%
Mild enlargement (30-39%)	13	43.34%
Moderate enlargement (40-46%)	4	13.33%
Severe enlargement (>47%)	3	10%

Table 7: Distribution of Degree of Hydrocephalus in Meningitis (n=18)

Grade of hydrocephalus	No. of Cases	Percentage
Mild enlargement (30-39%)	13	72.72%
Moderate enlargement (39-46%)	3	16.67%
Severe enlargement (>47%)	2	11.12%

Table 8: Relationship of Hydrocephalus with Duration of illness (n=18)

Grade of hydrocephalus	Upto 1 month	1-3 months	4-6 months
Sever > 47%	-	1	1
Moderate 39-46%	1	1	1
Mild 30-39%	5	8	-

Table 9: Distribution of hydrocephalus according to clinical stage in meningitis (n =18)

	Degree of hydrocephalus		
MRC Stage	Mild	Moderate	Severe
Stage I	1	-	-
Stage II	12	1	-
Stage III	0	2	2

Table 10: Relationship of hydrocephalus with final outcome in meningitis (n=18)

Degree of hydrocephalus	Group I	Group II	Group III
Mild	5	8	0
Moderate	-	1	2
Severe	-	1	1

Table 11: Distribution of PVL with degree of hydrocephalus (n = 9)

Grade of hydrocephalus	PVL
Severe hydrocephalus	3
Moderate	4
Mild	2

Table 12: Relationship of PVL with final outcome

	Group I	Group II	Croup III
PVL	2	3	4

Table 13: Location of Exudates (n=18)

Subarachnoid spaces	Exudates present	
	No. of cases	Percentage
Cerebellopontine cistern	5	33.33 %
Prepontime cistern	8	53.33%
Perimesencephalic	18	100%
Supraseller	12	80%
Cisterna ambiens	18	100%
Quadrigeminal cistern,	12	80%
Sylvian fissures	12	80%
Sulci.	5	33.34%

Table 14: Grading of the Exudates

Grade	No. of patients	Percentage
Mild	6	33.34%
Moderate	7	38.89%
Severe	5	27.78%

Table 15: Relationship of the degree of Exudates with outcome (n=18)

Degree of exudates	Final Outcome		
	Group – I	Group – II	Group – III
Mild	2	4	-
Moderate	3	3	1
Severe	-	3	2

Table 16: Location of Focal Parenchymal Lesions (n=12)

Location	No. of Cases	Percentage
Supra tentorial	9	75%
Left frontal	1	
Right frontal	1	
Left parietal	3	
Right parietal	4	
Left occipital	-	
Right occipital	-	
Infratentorial	2	16.67%
Both supra and infratentorial	1	8.33%

Table 17: Morphological patterns of granulomas (n=12)

Pattern	No. of Patients	Percentage
Single (small and large) lesion	8	72.22%
Multiple ring lesion	2	16.67%
Conglomerate lesion	2	11.11%

Table 18: Pattern of changes on treatment (n=12)

Pattern of Changes	No. of patients	Percentage
Complete resolution	4	33.34%
Decrease in size/Ca++/Focal atrophy	6	50%
Unsatisfactory response/no decrease in size	2	16.67%

Ca++ = Calcification.

Table 19: Radiological outcome pattern of lesions (n=12)

Patterns of lesions	Radiological outcome		
	Group I	Group II	Group III
Multiple small solid/ring lesion	-	1	1
Single lesion	4	4	-
Conglomerate	-	1	1

Discussion

The maximum number of cases in our study were in 4-6years (33.34%) and 7-9 Years (26.67%) year's age group. Wallace in 1980 reported that 9 children of intracranial tuberculosis studied by him ranged from 7-56 months.

The male incidence in our study was (66.67%). The overall male: female ratio is 2:1 but the ratio in 1-3 years and 4-6 years age group is 1.5:1 & 4:1 respectively. Humphires[3] and Ramchandran[4] found a male incidence of 55% and 63% respectively. There is a male preponderance noted in most Indian studies [4] which may be because of colored feeling towards male children in Indian Society. It may not be representing the true sex incidence of the disease. The marked male preponderance in 1-3 years and 4-6 years age group in our study may be because of same the reason.

Common presenting symptoms were convulsions (60%), fever (53.33%) and vomiting (36.67%). All these are non-specific findings and in no way point out the diagnosis of intracranial tuberculosis. [5] Neurological examination revealed signs of meningeal irritation in 50%, papilledema 40% and cranial nerve palsies in 30%. All these findings may suggest the diagnosis in appropriate clinical context but are nonspecific [5].

Family history/history of contact with a tubercular subject was found in total of 12 cases (40% in our study). Tandon and Bhargava in 2015[21] reported positive family history in only 7/50 (14%) cases of intracranial tuberculomas studied by them. 12 cases (40%) had TB elsewhere in the body and in the rest diagnosis was based on image morphology.

Chest X-ray in our study showed abnormality suggestive of pulmonary tuberculosis in (26.67%). Our findings are suggestive of that Dieter Enzmann[6] and Weckner et al. in 2020[7] who reported abnormal chest films in 25-50% cases. Total of 12/30 (40%) showed concomitant tuberculosis outside CNS. Jinkins in 2021[8] reported that 28.8% patients showed concomitant tuberculosis outside CNS in his series. Gee T. Gloria et al. 2022[9] have quoted a figure of 32.50% with cerebral tuberculomas.

The biochemical analysis of CSF revealed raised proteins in 93.75% and reduced sugar in (73.34%) cases. The cytology showed raised total cell count in 15/15 cases (100%). The white cell count ranged 50-800 cells/cu. mm. Lymphocytic predominance was noted in 12/15 cases (80%). Humphires[3] also reported increased WBC counts. Lymphocytic predominance in his series was seen in 83% in the first lumber puncture specimen. In our study hydrocephalus was found in 20/30 patients, of which two patients had posterior fossa tuberculomas. All cases (100%) of meningitis had hydrocephalus at presentation ranging from mild to severe degree. Weckner et al[7] also reported hydrocephalus demonstrated by CT in all 30 cases (100%) studied by him. Mild, moderate and severe hydrocephalus was present in 13/18 (72.23%), 4/18 (22.23%), 3/18 (16.67%) patients respectively. Bhargava et al in 2022[10] reported hydrocephalus in 83.05% cases of meningitis (in both adults and children). They graded it as mild (8.47%), moderate (22.03%) and severe (52.54%), on the basis of cella media index. Wallace et al[11] also reported ventriculmegaly in 7/9 patients studied by them and found it to be the commonest abnormality in intracranial tuberculosis in children. Bullock et al[7] reported hydrocephalus in 76% patients with severe TBM.

In our study, we found that only one patient in clinically stage I, had mild hydrocephalus. Rest of the patients of mild hydrocephalus were clinically in stage – II, whereas all cases in stage III at presentation had moderate to severe ventriculmegaly. Hence larger ventricular size was usually associated with severer clinical disease at presentation. Our study reveals that all patients with moderate to severe hydrocephalus with exudates showed moderate to severe disability after at least 6 months of therapy. Only 5/13 patients with mild hydrocephalus with exudates recovered completely and the rest suffered from moderate residual disability. Hence, hydrocephalus in our study was not associated with good outcome. Wallace et al 2021[11] also reported that ventriculmegaly was predictive of poor outcome. Only one of the seven patients with hydrocephalus in their study, recovered neurologically. The results of Bullock et al 2022[12] are in variance from ours. He reported that ventricular enlargement alone or basal enhancement did not constitute a bad prognostic sign. Kingley in 2017[13] observed that larger ventricular size is associated with severe disease at presentation. They also said mild clinical disease had mild residual disability. The patients with severe disease initially either died or had

severe disability, which is in agreement with our findings.

Correlation of the degree of hydrocephalus with duration of illness in cases of meningitis reveal a positive correlation. Bhargava et al[10] in 2022 reported a linear correlation between the degree of ventriculmegaly with duration of illness. Periventricular lucency (PVL) was found in 9 to the total 20 cases with hydrocephalus (45%) at presentation. It was associated mostly with moderate to severe hydrocephalus and in two patients with mild hydrocephalus which could be due to acute presentation. Seven patients had associated exudates and two had posterior fossa tuberculoma. PVL associated with mild hydrocephalus completely resolved on ATT. Moderate to severe disability was seen in the remaining 7 cases of hydrocephalus with PVL at 6 months of therapy. Both the cases of hydrocephalus and PVL due to tuberculoma recovered clinically but with radiological sequelae after an early shunt surgery. Hence, it suggests that PVL has a poor outcome. Bhargava et al in 2022.[10] reported PVL in 61.66% cases of hydrocephalus. Of which 72.79% had severe and 27.02% had moderate hydrocephalus. Bullock et al[12] reported PVL in 64.74% cases and found it to be associated with poor outcome.

Hyperdense exudates in basal cisterns were seen in perimesencephalic cistern and cisterna ambiens in all cases (100%) with TBM. Prepontine in 53.33%, suprasellar cistern and sylvian fissures contained exudates in 80% cases each. Grading of exudates (according to Bhargava et al 1982) showed mild exudates in 6/18 (33.34%) moderate in 7/18 (38.8%), severe in 5/18 (27.78%). The patients with moderate to severe grade of exudates recovered with moderate to severe disability. Majority of patients with mild exudates also showed residual disability. This is not in total agreement with other case studies. Bharagava et al[10] in 2022 observed that severe grade of exudates occurred only in children while mild to moderate exudates were seen in all ages. Also, that the severe exudates showed no clinical improvement on follow up even if ventricular size reduced to normal after shunt surgery. This is in agreement with our findings.

Kingsley et al[14] found that patients with mild exudates presented in clinical stage I while all patients with moderate severe exudates came in clinical stage II or III. Nevertheless, 3 patients with stage II and 2 patients in stage III had no meningeal enhancement on admission in their study of 25 cases. They observed that blindness and hypothalamic involvement in patients with exudates in chiasmatic and perimesencephalic cisterns. In our study, we also observed blindness in 1 & blurred vision in 12 patients with thick exudates in chiasmatic and perimesencephalic cisterns. Bullock et al[12] reported basal enhancement in 62.2% cases. But they found ventriculomegaly alone or basal exudates did not constitute a bad prognostic sign, which does not completely agree with our observations. We did not encounter a normal CT scan in any of our cases with TBM but Bullock et al[12] and Bhargava et al[10] reported normal CT scan in few cases and reported most favourable outcome in this group.

Parenchymal granulomas in CNS tuberculosis are always secondary to an extracranial source. In children involvement results from haematogenous spread of primary infection, while in adults CNS infection results from reactivation.[6]

In our study focal parenchyma lesions were seen in 12 of the 30 cases (40%). All of these cases had isolated parenchymal lesions. Jinkins in 2021[15] found isolated parenchymal form in 71.25% and compound parenchymal meningeal lesion in 11.25% cases. This is not in accordance with our study. Multiple lesions were present in 13.34% cases in our study. Bhargava in 2020[16] and Dieter in 2024[6] found multiplicity of lesions in 50-55% cases. This variation could possibly be due to lesser number of patient selected for our study. Jinkins in 2021[15] quoted that multiplicity of lesions on CT has been identified in 10-34% cases only. Which is consistent with our findings.

Supratentorial lesions were found in 9/12 cases (75%) while infratentorial lesions in only 2/12 cases (16.67%). 4/12 cases (33.34%) showed multiple supra and infratentorial lesions. Wallace in 2021[11] quoted that in adults, tuberculomas were most commonly reported in supratentorial compartment. But in our study 9/12 cases have supratentorial lesions most of which were single lesions. Only 2 had isolated infratentorial lesions and both these cases had more than one coalescing lesion. One had both supra and infratentorial lesions.

Right parietal region was found to be commonest site for tuberculomas in our study. However, Dyk A van [19] reported no predilection for any site. Bhargava in 2020[17,18] reported that no part of the brain is spared however largest number of lesions were in cerebral hemispheres in MCA territory.

The intracranial tuberculosis may present as a space occupying lesion in various morphological patterns. Single ring lesions in 8/12 (66.67%) patients were among common morphological pattern in our study. Two patients (16.67%) had multiple disc/ring lesions and 2 (16.67%) had conglomerate lesions. Bharagava et al [17,18] observed small disc and ring pattern in 18/25 (72%) cases studied by them.

In our study, target sign was found in only one patient at presentation and developed in three patients while on therapy. Dyk A Van [19] found target sign in 12/58 (20.68%) lesions studies by him. One case of multiple cerebral tuberculoma initially, developed exudates, hydrocephalus and new large ring lesions while he was still on ATT. Witham Robert in 2019[20] described a case of miliary cerebral pattern with no evidence of TBM. Kingsley in 1987 has reported that abnormal meningeal enhancement may develop upto one month after ATT. Jinkins in 2021[8] reported that 6.8% children with TBM have concomitant miliary TB of the brain. Conversely only 1% of children with miliary TB had or developed TBM subsequently. Four showed spot calcification and 2 were of target type.

It was observed in our study that 4/12 (33.34%) lesions that showed complete resolution after at least six months of therapy were small single solid disc/ring like. One case of multiple small disc lesions subsequently developed meningitis at 2 months of therapy while he was still on treatment. Tandon and Bhargava in 2015[21] reported that immature tuberculomas (small disc and ring lesions) respond well to medical treatment. Most multiple & large ring lesions in our study showed residual radiological sequelae after six months of therapy while they were clinically normal although still on treatment (ATT+ anticonvulsants).

Tandon and Bhargava [21] reported that smaller lesions measuring less than 1cm showed complete resolution after 4-6 weeks of Chemotherapy (often supplemented with corticosteroids) and a complete resolution was seen in several cases within 8-10 weeks. This is in agreement with our findings although complete resolution took at least 6 months. Tandon and Bhargava [21] reported no case of recurrence of a lesion that was followed to complete resolution. Our findings also show that lesions that recovered completely with no radiological sequelae did not recur till 6 months of therapy.

In 2 of our patients with ring lesions, we found that central portion of ring became hypodense while on medical therapy and clinically our patient did well. Tandon and Bhargava [21] reported similar findings in brainstem tuberculoma but it had to be operated due to clinical deterioration. They also exemplified a response characterized by decrease of edema and not of those of the granuloma itself. We found this pattern in 3 patients who at two months of theraphy (ATT + steroids) showed decrease in edema only. Decrease in size was observed at or after six months of treatment. One case showed appreciable decrease in size and enhancement only after 1½ years of therapy.

Two cases with conglomerate lesions were seen. Poor response to medical therapy was observed in one, who was subjected to surgery due to worsening clinical state. While another responded to chemotherapy and showed dense residual calcification. Tandon and Bhargava [21] also reported that very large lesions may show no response to medical therapy. One case with parenchymal lesions to start with developed basal exudates and new lesions at 3 months of ATT. This patient did not recover completely clinically & radiologically even at 1½ years after ATT. Jinkins in 2021[8] reported that compound lesions had a poor outcome with residual morbidity/death in all but one case studied by him. He attributed it to multicompartmental attack of the disease. Bhargava in 2022[10] reported tuberculomas along with TBM in only 10% cases.

Our study revealed that 7 patients developed new lesions while on antitubercular therapy for neurotuberculosis. Four of these patients had exudates ± hydrocephalus to begin with and three had multiple tuberculomas. The patients with exudates developed infarcts while they were still on ATT. One case with multiple tuberculomas initially developed exudates and mild hydrocephalus after two months of therapy. His initial multiple disc lesions regressed in size and edema around them. At six months he developed large multiple ring lesions in the supratentorial compartment. But, paradoxically this patient revealed steady clinical recovery. He is still under observation for further management. Moderate to severe disability at six months was observed in patients who developed new lesions while on therapy.

Tang et al reported development of multiple intracranial tubercular abscess in a case of TBM after adequate ATT for 14 weeks. Malone et al in 2019[22] and Lees in 2020[23] also reported intracranial tuberculomas developing during therapy for TBM. Teoh et al 1987 reported paradoxical development of intracranial tuberculomas on treatment for systemic tuberculosis. Jinkins in 1991 also quoted that parenchymal lesions, exudates and hydrocephalus may develop during treatment to the point of non-enhancement. Chandramukhi et al in 2007[24] postulated that the reason for tuberculoma failing to disappear during treatment could be due to continually released antigen during therapy that got sequestrated in some macrophage instead of getting eliminated and caused new lesions at a later date.

In our study infarcts were seen in (26.67%) patients. All these patients had variable degrees of exudates and hydrocephalus at initial stage. Four had infarcts at presentation. Two of these each were in the MCA and ACA territory in the region of basal ganglia. Two had severe basal exudates

while the other two had minimal exudates in sylvian fissures.

The remaining four cases with exudates initially developed infarcts on therapy. Overall, the commonest territory was MCA and ACA in the basal ganglia regions. It was seen in 3 cases each in our study. Right was preferred side. Two cases showed multiple infarct in both MCA and ACA. No posterior circulation infarcts were noted. We observed gradual dilatation of the ipsilateral ventricle in follow up scans in basal ganglia infarcts. Similar findings were seen by Bhargava et al.[10] Leiguarda Ramon in 2007[16] reported infarcts in 38% cases with TBM. Anterior circulation infracts were seen in 23 out of 25 cases and only 2 had vertebrobasilar involvement. Bhargava et al[17] in their study, observed infarcts in 28.33% cases. Both ACA & MCA territory was involved in equal number of cases (3 each). In our study also both ACA and MCA territory was equally involved. Hseith et al in 2022[25] said that 75% infarcts in TBM occurred in "TB zone" i.e., head of the caudate nucleus, anteromedial limb of internal capsule and genu. This region is supplied by medical striate, thalamochoroidal, and thalamo perforating arteries. They also said that 17-33% cases with TBM showed vascular lesions. His findings are in agreement with ours.

In our study the final outcome of most of the patients with infarcts revealed moderate to severe disability irrespective of the time of appearance. Leiguarda Ramon et al in 2007[26] also reported poor outcome in his studies because no patient with infarction recovered completely.

Conclusion

Our study shows that exudates, hydrocephalus with PVL and infarcts do not have a good outcome even after at least 6 months of treatment. Since, most of them suffered from residual moderate to severe disability. Most tuberculomas had good initial clinical status and they recovered clinically, although, they showed radiological sequelae.

References

1. R. B. Rock, M. Olin, C. A. Baker, T. W. Molitor, and P. K. Peterson, "Central nervous system tuberculosis: pathogenesis and clinical aspects," Clinical Microbiology Reviews, vol. 21, no. 2, pp. 243–261, 2008.
2. Andronikou, J. Wilmshurst, M. Hatherill, and R. VanToorn, "Distribution of brain infarction in children with tuberculous meningitis and correlation with outcome score at 6 months," Pediatric Radiology, vol. 36, no. 12, pp. 1289–1294, 2006.
3. Humphires, M.J. et al.: Factors of prognostic significance in Chinese children with tuberculous meningitis. Tubercle 1990; 71 (3): 161-168.
4. Ramachandran, R.S., Ramanathan, K. and Indra, G.: Tuberculous meningitis. A review of 288 cases in children. Ind. J. Pediatr. 1970; 37: (266): 85-88.
5. Thomas, M.D., Chopra, J.S., and Walia B.N.S.: Tubercular meningitis – a clinically study of 232 cases. Jr. Aso. Phys. Ind. 1977; 25: 633-639.
6. Enzmann Dieter R. et. Al.: Computed tomography of granulomatous basal arachnoiditis Radiology 1976; 120 341-344.
7. Weckner, J.J. Jr and Connor, J.D.: Central nervous system tuberculosis in children – a review of 35 cases. Paediatr. Infect. Dis. J. 2020; 9 (8): 539-453.
8. Jinkins J.R.: Computed tomography of intracranial tuberculosis, Neuroradiology. 2021; 33: 126-135.
9. Gee Gloria, T. et al.: Miliary tuberculosis involving the brain – MR finding. AJR. 2022; 159: 1075-1076.
10. Bhargava S, Gupta Arun Kumar and Tandon, PN. : Tuberculosis meningitis – CT Study. Br. J. Radiol, 2022: 55:189-196.
11. Walace, R.C. et al.: Intracranial tuberculosis in children: CT appearance and clinical outcome. Pediatr. Radiol. 2021; 21: 241-246.
12. Bullock, M.R.R. AND Dellen Van, J.R.: The role of cerebrospinal fluid shunting in tuberculous menintis. Surg. Neurol, 2022; 18: 274-277.
13. Kingley, D.P.E. et al.: Tuberculous meningitis – role of CT. J. Neurol. Neurosurg. Psychiatry, 1987; 50: 30-36.
14. Gupta Satya nd Chopra Kamlesh. Tuberculous meningitis in children Ind. J. Tub. 1981; Vol. XXVII No. 1: 6-11.
15. Jinkins J.R.: Computed tomography of intracranial tuberculosis, Neuroradiology. 2021; 33: 126-135.
16. Lehrer Harold: The angiographic triad and tuberculous meningitis. Radiology 1966; 87: 829-835.
17. Bhargava S, and Tandon PN,: Intracranial tuberculoma – A CT study. Br. J. Radiol. 2020; 53: 935-945.
18. Bhargava S, and Tandon PN,: Intracranial tuberculoma – lessons learnt from CT. Neurology (India) 2020: Vol. XXVII No. 206-21.
19. Syk Van A.: CT of intracranial tuberculoma with specific reference to the "target sign". Neuroradiology 1988; 30: 329-336.
20. Witham Robert R. et al.: Diagnosis of military tuberculosis by cerebral computed tomography. Arch. Intern. Med. 2019; 139: 479-480.

21. Tandon, P.N. and Bhargava Sneh. : Effect of medical treatment on intracranial tuberculoma – CT Study. Tubercle 2015; 66 (1): 84-97.
22. Malone, J.L, et al: Intracranial tuberculomas developing during therapy for tubercular meningitis. West. J. Med. 2019; 152 (2) : 188-190.
23. Lees, A.J., Macleod, A.F. and Marshall, John. : Cerebral tuberculoma developing during the treatment of TBM. Lancet. June 7, 2020.
24. Chandramukhi, A., and Shankar, S.K.: Intracranial tuberculoma – Area they really disappearing? Neurology (India) 36:395. 2007.
25. Hsiech, F.V., Chia, L-G and Shen, W.C.: Location of cerebral infarction in tuberculous meningitis. Neuroradiology. 2022; 34: 197-199.
26. Leiguarda Ramon, et al: Ischaemic infarction in 25 children with tuberculous meningitis. Stroke 2007; 19: 200-204.