International Journal of Contemporary Pediatrics
Goyal G et al. Int J Contemp Pediatr. 2017 Jan;4(1):116-122
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: http://dx.doi.org/10.18203/2349-3291.ijcp20164589

Original Research Article

Clinical and laboratory profile of children with tubercular meningitis admitted in tertiary care centre of Kumaun region, Uttarakhand, India

Garima Goyal*, Ajay Kumar Arya

Department of Pediatrics, Government Medical College, Haldwani, Uttarakhand, India

Received: 21 September 2016
Revised: 22 September 2016
Accepted: 24 October 2016

***Correspondence:**
Dr. Garima Goyal,
E-mail: dr.garimagoyal24@gmail.com

ABSTRACT

Background: Tuberculous meningitis (TBM) is an important cause of hospital admission, death and neurological disability in India. Delay in diagnosis is an important cause of morbidity and mortality.
Methods: 80 cases with meningitis 1 month-16 year were enrolled, 44 diagnosed as TBM, studied in detail about clinical and laboratory profile. The data was analyzed by statistical test SPSS 21 and Chi square test was applied.
Results: Peak age incidence 11-16 year. Youngest patient 3 month. Male: female ratio 0.8:1. Majority of patients had fever, altered sensorium, vomiting, headache, seizures. Neurological deficit found was loss of speech, right sided weakness, and diplopia. 36.3% patients were comatose. Cranial nerve palsy was present in 57% cases, 6th nerve being the commonest. Papilloedema common than optic atrophy. Motor deficit like hemiplegia (4.5%). Maximum cases were in stage III BMRC. CSF findings were reduced glucose level in 47.7% of cases (mean 54.5 mg/dl), elevated protein (mean 116.9 mg/dl), lymphocytic pleocytosis (mean value 102.46) (>60% lymphocytes). CT scan abnormality in 75% cases. Hydrocephalus being the most common finding.
Conclusions: TBM one of most serious illness. Majority patients presented in stage 3 with neurological deficit, as this are tertiary care centre and due to difficult geographic terrain patient presents at late stage. High index of suspicion to diagnose TBM at early stage. CSF analysis continues to be a key in establishing the diagnosis. One should not wait for the microbiological proof to start the therapy. Mantoux and radiological tests can be good supportive investigation. Early diagnosis and treatment can make complete recovery even in comatose patients.

Keywords: Cerebrospinal fluid, Laboratory profile, Neurological deficit, Tuberculous meningitis

INTRODUCTION

Tuberculosis remains global health problem in children in developing countries. It is estimated that childhood TB constitutes 10-20% of all TB cases in high burden countries, accounting for 8-20% of TB-related deaths.[1,2]

Among the various forms of tuberculous infection, TBM continues to be most fatal type of systemic tuberculosis. It is associated with high morbidity and mortality. Delay in diagnosis results in neurological deficit and mortality. Herelies the importance of early diagnosis and treatment. Until new, affordable, sensitive and specific diagnostic assays become available, clinician must depend on the discriminative clinical and laboratory features of the disease for successful diagnosis and treatment.[3] Therefore cases of TBM were enrolled and detail analysis was done on clinical and laboratory profile of these patients.

METHODS

Prospective observational study was conducted in the department of paediatrics at Government Medical College, Haldwani (Nainital). 80 patients above 1 month up to 16years presenting with signs and symptoms of meningitis were selected for the study. Out of 80 cases, 44 patients were diagnosed as tuberculous meningitis. A meticulous history was taken from all the cases.

History of vaccination and history of contact with a known patient with tuberculosis in the house or neighbouhood or school was elicited and history of coexistent tuberculosis was also elicited. A thorough physical examination was done and search for extra meningeal tuberculous foci was carried out.

Observations were made on mode of presentation. A criteria was applied for diagnosis of TBM. It was not stringent in all cases and in some patients diagnosis was based on clinical suspicion and history of TB in past or evidence of co-existing TB.

Case definition of CNS TB by either clinical or microbiological criteria

Clinical case definition abnormal neurological signs or symptoms and more than 2 of following

- History of contact with TB
- Mantoux test reactive (5TU): 10 mm induration or 5mm induration if child had close contact with infected adult
- CSF abnormalities without evidence of any other infection
- Abnormalities on cranial CT consistent with CNS TB.

Microbiologic case definition one of the following

- Isolation of M tuberculosis from CSF
- Abnormal neurologic signs and symptoms
- CSF, or cranial CT consistent with CNS TB, and isolation of mycobacterium Tb from any site
- Cases were classified according to the british medical research council (BMRC) into 3 stages and early treatment was started in all suspected cases.

A method of staging the severity of the disease was developed by the British Medical Research Council (BMRC).[4,5]

- Stage 1: prodromal phase with no definite neurological symptoms or signs
- Stage 2: signs of meningeal irritation with slight or no clouding of consciousness and minor (cranial) nerve palsies or neurological deficits
- Stage 3: severe clouding of consciousness, stupor, coma, convulsions, gross paresis or involuntary movements.

All enrolled cases were subjected to array of investigations. Routine investigations included complete blood count, erythrocyte sedimentation rate, blood sugar, LFT, KFT, Mantoux test, MP, Widal test. Gastric aspirate analysis was done. CSF analysis was done under aseptic precaution. It was analysed for biochemistry, cell count, differential count, Gram stain, AFB stain, culture and sensitivity. Cranial CT scan was done in all patients. Data was entered in Microsoft excel sheet and analysis done by SPSS software version 21 and applying Chi square test. P value <.05 was considered significant.

RESULTS

Prospective study was taken up in the department of Pediatrics at Government Medical College Haldwani for a period of one year from November 2013 to November 2014. A total 80 cases clinically suspected to be meningitis in the age group 1 month to 16 years were included in this study, 44 diagnosed as TBM.

Clinical profile

Out of 44 patients with TBM it has been observed that 20 were male (45%) and 24 were female (55%) (Table 1). Male:female is 0.8:1.

Table 1: Sex wise distribution.

Sex	Number	% of patients
Male	20	45
Female	24	55
Total	**44**	**100**

Out of 44 patients, 33 patients were Hindu and 11 were Muslims (Table 2). Patients aged 1 month-16 years were included in the study (Table 3). The majority of patients were in the 11-16 years age group (40.9%). Youngest patient affected with TBM in this study was 1 female of 3 months of age.

Table 2: Religion wise distribution.

Religion	Number	% of patients
Hindu	33	75
Muslim	11	25
Total	**44**	**100**

Past history

Past history of TB was present in 5 cases. Co-existing TB, in the form of miliary TB in 3 cases, tuberculous pleural effusion in remaining 3 cases. 2 cases have left sided hydropneumothorax, 2 cases had progressive pulmonary complex. 5 patients had extra-pulmonary TB, abdominal TB in 3 cases and 2 cases had TB lymphadenitis (Table 4).

Table 3: Age wise distribution.

Age	No. of males	Percentage	No. of females	Percentage	Total number	% of patients
1-3 months	0	0	1	4.1	1	2.2
3 months -1 years	1	5	1	4.1	2	4.5
1-5 years	4	20	5	20.8	9	20.4
6-10 years	9	45	5	20.8	14	31.8
11-16 years	6	30	12	50	18	40.9
Total	**20**	**45.4**	**24**	**54.5**	**44**	**100**

Symptoms

Fever was present in 100% of patients followed by altered sensorium, present in 54.5%. Vomiting (50%) > headache (45.4%) > seizures (40.9%). Cough present in only 27.2% patients. Abdominal pain (11.3%) > altered bowel habits (2.2%) > loose motion (2.2%). Other neurological deficit found was loss of speech in 6.8%, right sided weakness 4.5%, diplopia 2.2%, blurred vision 2.2% (Table 5).

Table 4: History of past or evidence of coexisting tuberculosis.

Category	Past tuberculosis		Coexisting tuberculosis	
	No.	Percentage	No.	Percentage
Pulmonary	2	4.5	10	22.1
Extra pulmonary	3	7.1	5	11

Table 5: Analysis of symptoms.

Symptoms	Number	% of patients
Fever (F)	44	100
Seizures (S)	18	40.9
Abdominal pain (AP)	5	11.3
Headache (H)	20	45.4
Vomiting (V)	22	50
Altered sensorium (AS)	24	54.5
Cough (C)	12	27.2
Loss of speech (LS)	3	6.8
Irritability (I)	1	2.2
Weight loss (WL)	7	15.9
Weakness (W)	6	13.6
Diplopia	1	2.2
Blurred vision (BV)	1	2.2
Right sided weakness	2	4.5
Altered bowel habits+LM	1+1	2.2+2.2

Duration of illness

Majority of patients sought medical advice within 3weeks (Table 6). Maximum duration in my study was 8 months and minimum duration was 2 days.

Table 6: Duration of illness.

Duration	Number	% of patients
0-7 days	9	20
7-14 days	8	18
14-21 days	13	30
21-28 days	2	4.5
1 -3 months	9	20
> 3 months	3	7

Contact history

Contact history of tuberculosis was present in 16 patients (36%) only. 64% patients have no history of contact with tuberculosis inspite of having TBM (Figure 1).

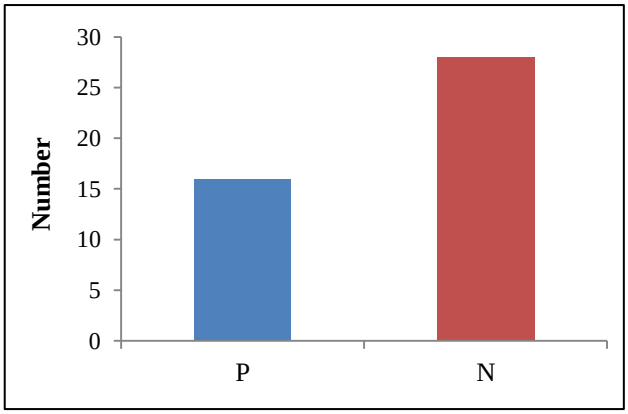

Figure 1: Contact history.

Signs

In 93% patients, consciousness was altered at presentation out of which 40.9% were drowsy at presentation, 36.3% patients were unconscious at presentation, 15.9% were irritable (Table 7).

Signs of meningeal irritation were present in 79.5%.

Cranial nerve involved in this study was optic (4.5%), ophthalmic (9%), abducens (31.8%), facial (13.6%), glossopharyngeal and vagus nerve (2.2%). Abducens nerve involvement was most common.

Fundus was normal in 45.4% patients, Edema was found in 29.5%, atrophy in only 2.2%.

Motor involvement in form of hemiplegia was found in only 2 patients. No case with quadriplegia was found. Two patients had tender lymphadenopathy.

Table 7: Analysis of signs.

Sign	Number	Percentage
Altered consciousness (AC)	41	93
Fundus changes	24	54.5
Cranial nerve (CrN) involvement (2,3,6,7,9,10)	25	57
Motor sign	2	4.5
Altered consciousness (AC)		
Drowsy	18	40.9
Irritability	7	15.9
Unconsciousness	16	36.3
Meningeal irritation (MI)		
MI +ve	35	79.5
MI –ve	9	20.4
Cranial nerve (CrN)		
CrN 2	2	4.5
CrN 3	4	9
CrN 6	14	31.8
CrN 7	6	13.6
CrN 9, 10	1	2.2
Fundus (F)		
Normal (FN)	20	45.4
Edema (FE)	13	29.5
Atrophy (FA)	1	2.2
Motor		
Hemiplegia (HP)	2	4.5
Quadriplegia (QP)	0	

Stage of TBM at presentation

Maximum cases were found in the third stage (19 cases). Minimum cases were in first stage (8 cases) (Table 8).

Table 8: Stage of disease, according to British Medical Research Council Criteria.

Stage	No of patients	% of patients
Stage I	8	18
Stage II	17	39
Stage III	19	43

Immunisation status

BCG scar was present in 81% patients. 19% children were unimmunised.

Mantoux test

It was reactive only in 68%.

Laboratory profile

Gastric aspirate/sputum aspirate for acid fast bacilli (AFB)

Negative result was found in majority of patients (90%). GA for AFB was positive in only 10% cases.

Hemogram

There was evidence of anaemia in 25% patients. In the majority of cases, TLC was >11,000 (54%), whereas 41% had normal counts. Mean cell count 12342.5. High ESR value was present in 70% cases. Mean ESR value was 32.

CSF profile

A lower level of glucose (<50) were present in 47.7% of cases, whereas, only 6 cases had glucose > 70mg/ dl. The mean value of CSF glucose was 54.54 mg/dl ±16.5. All except 6 patients had CSF glucose <60% of the corresponding blood sugar (85%).

Table 9: CSF parameters.

CSF-cells	Number	Percentage
0-50	18	40.9
50-100	9	20.4
100-200	10	22.7
> 200	7	15.9
CSF-Protein	**Number**	**Percentage**
< 50	9	20.4
50-100	12	27.2
100-200	14	31.8
> 200	9	20.4
CSF Sugar	**Number**	**Percentage**
0-50	21	47.7
>50	23	52.2

Twenty five cases had cobweb appearance of CSF and protein was found to be elevated in 35 cases. 9 cases had > 200 mg CSF protein, maximum being 380mg. The mean value of CSF protein was 116.9 mg/dl±77.4.

Pleocytosis (more than 5 cells/cumm) was seen in 36 (82%) cases. It was lymphocytic in 10 (22%) and mixed in the rest. The remaining 9 (20%) did not show any pleocytosis. Ninety percent patients had predominantly lymphocytosis with >60% lymphocytes. In majority of cases CSF cells were <50 (40.9%) with a mean value of 102.46 ±101.76 and range of 5-400.

Mycobacterium tuberculi could not be isolated in any of 44 cases.

Radiological profile

CT head was done in all patients. CT head was normal in 11 patients. Commonest finding was hydrocephalus

(25%) followed by basal enhancement (23%), edema (20%).

Two patients had tuberculoma. One patient had communicating hydrocephalus with infarct at basal ganglia. Incidental finding was hydrocephalus with calcification (Table 10).

Table 10: Radiological profile.

Radio	Number	Percentage
Basal enhancement	10	23
Edema	9	20
Hydrocephalus	11	25
Hydrocephalus with infarct basal ganglia	1	2.2
Tuberculoma	2	4.5
Normal	11	25

DISCUSSION

The disease can occur at any age but is uncommon in children younger than 6 months and rare in those who are younger than 3 months of age. The peak incidence in the present study was found during adolescence in the age group of 11 to 16 years (43%), followed by 31.8% cases in the age group of 6- 10 years. Youngest was 3 months old. Higher incidence was observed by Malla et al in the similar age group 10-16 years (70 %). This is probably due to pubertal growth spurt creating a relative immunodeficiency due to demand supply mismatch of nutrients.[6]

According to the present study, there is a female (55%) predominance over the males (45%), while earlier studies done by Kumar B et al, Mishra UK et al noted a male predominance with 70%, 74%, respectively whether this is due to the socio cultural bias prevalent in the region against the female child and discrimination for food or else needs further evaluation.[7,8]

Clinical profile

In this study, 80% of children had symptoms for >1 week and 32% for >3 weeks before admission.

Duration of illness was 14-21 days in majority of cases (30%), followed by a duration of >1 month (27% of cases) which was consistent with earlier studies done by Kaur H et al.[9] The presence of undiagnosed TBM is considered an important factor in the spread of the disease.

TBM occurs with a variety of nonspecific clinical manifestations. In children, apathy, lack of interest in play, irritability, restlessness at night, minor headaches, loss of appetite, nausea, vomiting, and abdominal pain are the usual presenting symptoms.[10]

Fever, vomiting, changes in personality, seizures and headache was present in most of our cases, whereas cough, weight loss, and night sweats, which are commonly associated with pulmonary TB, were present in 43% of our patients. Fever was the predominant symptoms in the present study which was present in all the cases. Malla et.al6 observed 100% incidence of fever, while it was 66% in the study carried out by Farinha NJ et al.[11]

Altered sensorium was present in 54.5% of cases on presentation. Next common presentation was vomiting (50%) and headache (45.4%). Seizure was present in 50% of cases. This was similar to the studies carried out by Malla et al and Rajesh B et al.[6,12]

History of contact with tuberculosis was present in 36% of cases, similar to studies presented by Gupta BK et al and Gurses et al (42%).[13,14] However Waecker and Connor reported that an adult source of contact was identified in 70% of cases of children with CNS TB.[15] Thus, it is important that the patients should be questioned persistently about contact with a person with TB and that family members should be examined for TB, especially because of the delay from development of symptoms to diagnosis of TBM.

History of evidence of coexistent and past history pulmonary tuberculosis was noted in 27% cases similar to a study done by Gupta BK et al (32% cases).[13]

The protective efficacy of BCG vaccination is not known and ranges from 0 to 80%.[16] However, it has been stressed that in childhood, the vaccination protects against serious complications, such as TBM.[16-18]

In the present study, only 81% cases were immunised. Whereas in other study done by Yaramis et al and Farinha NJ et al children immunised were 12% and 16%, respectively.[11,19] Though the immunisation coverage was found to be adequate the protective efficacy of vaccination seems to be inadequate.

The Mantoux test result was positive in 68% of the cases. However, an additional 6% of patients had reactions of 5 to 10mm in duration. These results are similar to those reported previously in the literature: PPD was found positive in 62% in TBM in study by Titone L et al.[20] This underlies the importance of this cheap, readily available test in diagnosis of TBM.

In present study, 93% cases were in altered consciousness; signs of meningeal irritation were present in 79.5% cases, similar to other studies. Papilloedema was present in 29.5% of cases, optic atrophy in (2.2%) but none of the cases revealed choroid tubercles on fundoscopy. Girgis et al had a 7% cases with papilloedema, 4% cases with optic atrophy in their study.[21]

Cranial nerve palsies were present in 57% of the cases, 6th nerve being the most common nerve involved (31.8%), followed by 7th nerve (13.6%) and 3rd nerve (9%). Much lower % in study done by Yaramis A et al who noticed cranial nerve palsies in 26% of cases, 7th nerve (10%) being the most common followed by 6th (9%) and 3rd.[19]

In the present study, hemiplegia/paresis noticed in 4.5% of cases which was consistent with Thwaites et al, in which incidence was 8%, whereas Hosoglu et al, noticed in 22.8% of cases but in adult patients only.[3,22]

An early diagnosis of TBM is highly relevant to its treatment and prognosis. In this study, majority of children (43%) were diagnosed at stage III of TBM followed by stage II (39%) then in stage I (18%).

The severity of this presentation was probably attributable to the low socioeconomic and educational levels of most of our patients' families, who did not seek medical assistance until the terminal stages of the disease. Therefore high index of suspicion should be there so that early diagnosis of TBM could be made. And it could also be due to the difficult geographic terrain which makes access to healthcare difficult.

In this study gastric aspirate for AFB was positive in only 10% cases corroborative with study done by Yaramis A et al (9%).[19]

Laboratory Profile

Routine

Most patients had a normal haemoglobin but anaemia was present in 25% cases, and elevated study found leukocytes in 54% cases. Mean Leukocytes was 12342. This was consistent with studies done by Malla et al.[6] An elevated ESR was present in 70% of cases (ESR >20) which is consistent with earlier studies.[22]

CSF parameters

CSF analysis was diagnostic in almost all the cases. Although all CSF examinations were abnormal, the ranges of values for that percentage lymphocyte, protein, glucose level varied considerably.[23]

In this study, a lower level of glucose (<50) was present in 47.7% of cases whereas it was in 94.5% cases in a study by Kaur et al.[12] Twenty five of cases had cobweb appearance of CSF, and protein was found to be elevated in 80% cases. Nine cases had > 200 mg CSF protein. Range was 29-380 mg/dl. The mean value of CSF protein was 116.9±77.4 mg/dl. Majority (31.8% cases) had protein in the range of 100-200 mg. Similar results were observed by Yaramis et al, with mean protein 142±29 mg/dl with maximum cases with protein 100-200 mg/dl (62%).[19]

In majority cases, CSF cells were <50 (40.9%) with a mean value of 102.46. and range of 5-400. 90% patients had predominantly lymphocytosis with >60% lymphocytes. Similar to Yaramis et al study, 83% cases were predominant with lymphocytes.[19] Mean CSF cell count was higher in study by Malla et al 158±133.[6] Pinto et al noticed neutrophilic pleocytosis in 32.4% cases which was associated with worse outcome.[24]

CSF AFB

Microbiological proof is the gold standard of diagnosis. Even though all the cases analyzed thoroughly, AFB could not be demonstrated in any of these, by staining method. This proves that though AFB isolation would confirm the diagnosis beyond any doubt, there is no need to wait for this proof in order to begin therapy.

Imaging

CT scan was done in all cases; in 25% of cases it was normal. Out of the remaining 33 cases, 11 cases showed evidence of dilated ventricles (25%), classical basal exudates was demonstrated in ten young patients, edema was noted in 9 patients. Infarction at basal ganglia was noticed in one case. Two cases had tuberculoma (4.5%). One incidental finding in the present study was hydrocephalus with calcification. An abnormal CT scan in 75% also emphasizes the growing importance of radio diagnosis. It is readily available therefore supportive in diagnosis of TBM.

Higher incidence of hydrocephalus was found by Farinha NJ et al (94%), whereas it was 80% in Yaramis et al study, but lower incidence for tuberculoma (2% of cases) as compared to present study.[11,19]

CONCLUSION

Thus, the present study provides the sociodemographic, clinical and laboratory profile of TBM in children presenting in tertiary care centre of Kumaun region. Our study noted higher incidence of neurological deficit and stage 3 of TBM at presentation. Since this region belongs to hilly region and due to difficult geographic terrain, patients presented at very late stage of disease with complications, so high index of suspicion is required to diagnose TBM so that early diagnosis could be made resulting in lower morbidity and mortality.

Laboratory diagnosis was supported by mantoux positivity in 67% cases, highlighting the relevance of this cheap easily available and simple test in diagnosis of TBM. High ESR and suggestive CSF findings were present in almost all cases. The presence of TB at sites other than CNS, associated pulmonary TB and positive imaging findings have highlighted the importance of radio diagnosis in our study.

CSF analysis, continue to be the key in establishing the diagnosis. The abnormal CSF can have range of values of different parameters. Though the microbiological proof is the gold standard of diagnosis, one should not wait for this proof to begin the therapy.

There should be high index of suspicion to diagnose TBM. TBM can be diagnosed relatively accurately at earlier stages with less CNS complications effectively declining the morbidity and mortality due to this disease.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Marais BJ, Hesseling AC, Gie RP, Schaaf HS, Beyers N. The burden of childhood tuberculosis and the accuracy of community-based surveillance data. Int J Tuberc Lung Dis. 2006;10:259-63.
2. Jain SK, Ordonez A, Kinikar A, Gupte N, Thakar M, Mave V, et al. Pediatric tuberculosis in young children in India: a prospective study. Biomed Res Int. 2013;2013:783698.
3. Thwaites GE, Chau TTH, Stepniewska K, Phu NH, Chuong LV, Singh DX, et al. Diagnosis of adult tuberculous meningitis by use of clinical and laboratory features. Lancet. 2002;360:1287-92.
4. Kennedy DH, Fallon RJ. Tuberculosis meningitis. JAMA. 1979;241:264-8.
5. Medical Research Council. Streptomycin treatment of tuberculous meningitis. Report of the committee on streptomycin in tuberculosis trials. Lancet. 1948;1:582-97.
6. Malla K, Rao KS, Malla T, Vedamony J, Ghimire P. Role of cerebrospinal fluid adenosine deaminase level in early diagnosis of various etiologies of meningitis in children. Nepal J Med Sci. 2012;1(2):97-102.
7. Bharat KG, Anchit B, Bandyopadhyay D. Adenosine deaminase levels in CSF of tuberculous meningitis patients. J Clin Med Res. 2010;2:220-4.
8. Misra UK, Kalita J, Roy AK, Mandal SK, Srivastava M. Role of clinical, radiological, and neurophysiological changes in predicting the outcome of tuberculous meningitis: a multivariable analysis. J Neurol Neurosurg Psychiatry. 2000;68(3):300-3.
9. Kaur H, Sharma K, Modi M, Sharma A, Rana S, Khandelwal N, et al. Prospective analysis of 55 cases of tuberculosis meningitis (TBM) in North India. J Clin Diagn Res. 2015;9(1):15-9.
10. Molavi A, LeFrock JL. Tuberculous meningitis. Med. Clin North Am. 1985;69:315-31.
11. Farinha NJ, Razali KA, Holzel H, Morgan G, Novelli VM. Tuberculosis of the central nervous system in children: a 20-year survey. J Infect. 2000;41(1):61-8.
12. Baheti R, Laddha P, Gehlot RS. CSF-adenosine deaminase (ADA) activity in various types of meningitis. J Indian Academy of Clin Med. 2001;2(4).
13. Gupta BK, Bharat V, Bandyopadhyay D. Sensitivity, specificity, negative and positive predictive values of adenosine deaminase in patients of tubercular and non-tubercular serosal effusion in India. J Clin Med Res. 2010;2(3):121-6.
14. Gurses H, Bilkay U, Ayas G. One hundred and sixty-five cases with primary tuberculosis and their relatives. Tuberkuloz ve Toraks. 1971;19:260-72.
15. Waecker NJ, Connor JD. Central nervous system tuberculosis in children: a review of 30 cases. Pediatr Infect Dis J. 1990;9:539-53.
16. Clarke A, Rudd P. Neonatal BCG immunization. Arch Dis Child. 1992;67:473-4.
17. Conference abstracts of Turkish health and treatment foundation: tuberculosis issue. Ankara, Turkey. Today's situation with tuberculosis in Turkey. 1988:3-10.
18. Smith AL. Tuberculous meningitis in childhood. Med J Aust. 1975;1:57-60.
19. Yaramiş A, Gurkan F, Elevli M, Söker M, Haspolat K, Kirbaş G, et al. Central nervous system tuberculosis in children: a review of 214 cases. Pediatrics. 1998;102(5):E49.
20. Titone L, Di Carlo P, Romano A, Maggio MC, Salsa L, Abbagnato L, et al. Tuberculosis of the central nervous system in children: 32 years survey. Minerva Pediatr. 2004;56(6):611-7.
21. Nabil I, Girgis, Sultan Y. Tuberculous meningitis abbasia fever hospital - Naval Medical Research Unit No. 3 - Cairo. Egypt from 1976-1996. Am J Trop Med Hyg. 1998;58(1):28-34.
22. Hosoglu S, Ayaz C, Geyik MF, Kökoğlu OF, Ceviz A. Tuberculous meningitis in adults: an eleven years review. Int J Tuberc Lung Dis. 1998;2(7):553-7.
23. Davis LE, Rastogi KR, Lambert SC, Skipper BJ. Tuberculous meningitis in the Southwest United States: a community based study. Neurology. 1993;43:1775-8.
24. Pinto VL, Lima MA, Rolla VC, Rebelo MC, Bóia MN. Atypical cerebrospinal fluid profile in tuberculous meningitis. Trop Doct. 2009;39(2):76-8.

Cite this article as: Goyal G, Arya AK. Clinical and laboratory profile of children with tubercular meningitis admitted in tertiary care centre of Kumaun region, Uttarakhand, India. Int J Contemp Pediatr 2017;4:116-22.