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CLINICAL PROFILE OF PATIENTS WITH TUBERCULOUS MENINGITIS

General Medicine

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ABSTRACT

Back ground: tuberculous meningitis kills or disables more than half of those affected. The diagnosis of TBM is difficult. Delay in diagnosis and treatment are regarded as the major contributing factors in the high mortality reported.

Aims and objectives: the study is aimed to prove that:

A) To identify different clinical presentation of patients with TBM

B) To identify the comorbidities in patients with TBM

C) To identify the complications and outcome of TBM

Methods: 50 cases of TBM aged 18 years above were studied in detail about clinical and laboratory profile.

Results: patients ranged from 18-70 years. Peak incidence in age group 20-30 years (38%). Majority of patients had fever, headache, vomiting, altered sensorium and neck stiffness. 18% patients were comatose. Cranial nerve palsy was present in 40% cases, 6th nerve being the commonest. Motor deficit like hemiplegia/paresis (8%), quadriplegia (2%) was present in 10% cases. Maximum cases were in stage 2 BMRC.

Csf findings were reduced glucose level in 70% of cases (mean 36.9mg/dl) elevated protein (mean 267.6 mg/dl), lymphocytic pleocytosis with mean value of 143.4 (60% lymphocyte). Majority i.e. 36% patients have leptomeningeal enhancement in their MRI brain followed by acute infarct which is seen among 28% of patients.

ADA level in TBM was elevated compared to pyogenic meningitis group with mean value of 15.29 +/- 7.9. At cut value of 10 the sensitivity of CSF ADA in TBM was 75% & 90% respectively. CT scan was abnormal in 36.6% out of 30 cases.

76% improved with therapy out of which 3 patients (6%) developed with sequel (hemiparesis in one optic atrophy in other one). 3 cases (6%) didn't improve in the hospital. Apart from this, 6 cases (12%) expired in the hospital. Stage 3 found to have high mortality rate.

Conclusion: TBM continuous to be a serious illness mostly affecting young adults. CSF analysis continuous to be a key in establishing the diagnosis. One should not wait for the microbiological proof to start therapy. Early diagnosis and treatment make complete recovery even in comatose patients.

KEYWORDS

tuberculous meningitis, adenosine deaminase, cerebrospinal fluid

INTRODUCTION

The total number of tuberculosis (TB) cases in the world is increasing. It is estimated that most of these new cases will be in South East Asia fuelled by rapid spread of HIV. The physician need to be aware of these changes, as less common form of tuberculosis such as TBM (Tuberculous Meningitis) will be encountered more often[1].

In population with a high prevalence, CNS tuberculosis differ from pulmonary and other extra pulmonary tuberculosis in that, the peak age is from 0 to 4 years. In population with lower prevalence most cases are in adults. Risk factors include alcoholism, diabetes mellitus, malignancy and recent corticosteroid use but co-infection with HIV now dwarf all these.

The main reason for the spread of tuberculosis is poverty, with resulting homelessness, malnutrition and break down of public health infrastructure[1]. Involvement of CNS by tuberculosis is the most hazardous type of systemic tuberculosis because of its high mortality rate and possible serious neurological complications and sequelae. CNS tuberculosis occurs in 2.5% of all patients with TB and in 10% those with AIDS – related TB[2].

Delay in the diagnosis and treatment are regarded as major contributing factor in the high mortality reported. The diagnosis of TBM relies on isolation of mycobacterium TB from the CSF. Unfortunately culture is too slow and insensitive to aid clinical decision making. Direct ZN staining of the CSF for acid fast bacilli remains the cornerstones of rapid diagnosis but this technique lacks sensitivity. Consequently the decision to treat patient for TBM is frequently empirical, irrespective of the diagnostic facilities available to clinicians. Until new, affordable, sensitive and specific diagnostic assays becomes available, clinician must depends on the discriminative clinical and laboratory features of the disease for successful diagnosis and treatment[3].

AIM

- To Study Clinical Profile Of Patients With Tuberculous Meningitis

OBJECTIVES

- To Identify Different Clinical Presentations of patients with Tuberculous meningitis
- To identify the comorbodities in patients with Tuberculous meningitis
- To identify the complication and outcome of Tuberculous meningitis

MATERIAL AND METHODOLOGY

The study was conducted at Sir T. General Hospital, Bhavnagar over a period of 12 months. Fifty patients aged 18 years and above presenting with signs and symptoms of meningitis, were selected for the study after taking permission from IRB(HEC), Government Medical Collage, Bhavnagar.

All patients were subjected to detailed history and thorough clinical examination. Investigation like complete hemogram, liver function test, Renal function test, Electrolyte, Chest x ray, CSF analysis , MRI, etc were carried out in each patient.

INCLUSION CRITERIA :

Patients giving informed & written consent

Age >12 Year

Patients with suspected cases of tuberculous meningitis

CSF picture suggestive of tuberculous meningitis

EXCLUSION CRITERIA :

Age <12 Year

Pregnancy

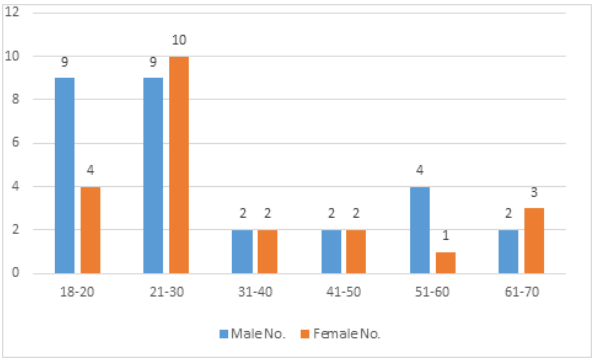
CSF picture not suggestive of tuberculous meningitis.

OBSERVATION AND RESULT

Table No – 1: Age Wise Distribution of Cases (N=50)

Age in years	Male		Female		Total	
	No.	Percent	No.	Percent	No.	Percent
18-20	9	32.14	4	18.18	13	26
21-30	9	32.14	10	47.36	19	38
31-40	2	7.14	2	10.53	4	8
41-50	2	7.14	2	5.26	4	8
51-60	4	14.29	1	2.88	5	10
61-70	2	7.14	3	15.79	5	10
Total	28	100	22	100	50	100

Patients aged 18 and above were included in the study, youngest patient being 18 years and the oldest patients was of 70 years. The majority of patients were in the second to third decade. There were 18 patients in this age group (64.28%).



Age in Years

Fig 1: Age Wise Distribution of Cases

Table No. – 2: Duration of Illness (N=50)

s	No. of Cases	Percentage
1-7 days	4	8
8-14 days	14	28
15-30 days	23	46
1-3 months	6	12
> months	3	6

Most of the cases i.e.41 (82%) sought Medical advice within a month. The minimum duration of illness was 5 days, and maximum duration was 7 months.

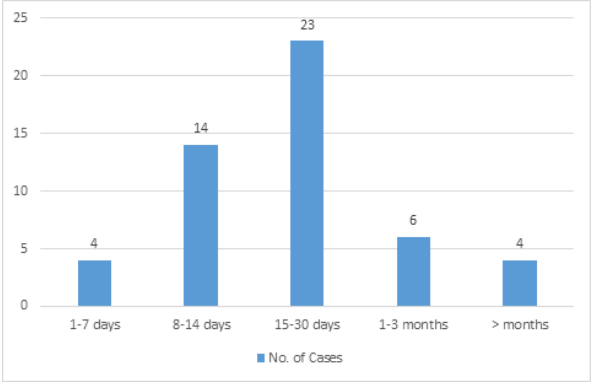


Fig 2: Duration of Illness

Table No. – 3: CSF Parameters (N=50)

	CSF Parameters	No. of Cases	Percentage
i	**Glucose (mg/dl)**		
	a) 0-40	35	70
	b) 41-70	11	22
	c) > 70	4	8
ii	**Protein(mg/dl)**		
	a) < 50	0	0
	b) 51-100	13	26
	c) 100-300	28	56
	d) > 300	9	18
iii	**Cells/mm^3**		
	a) 0-20	1	2
	b) 21-100	28	56
	c) 101-200	10	20
	d)201-300	6	12
	e) > 300	5	10
iv	**Low CSF chloride**		
	< 119	39	78
v	**CSF AFB**		
	Smear positivity	0	0

A lower level of glucose (<40) were present in 70% of cases.

All except 6 patients had CSF glucose <50% of the corresponding blood sugar (85%).

80% of cases had cobweb appearance of CSF and protein was found to be elevated in all cases. 9 cases had > 300mg CSF protein, maximum being 2g. The mean value of CSF protein was 267.6mg/dl

CSF cells were elevated in all cases with a mean value of 143.4 and range of 18- 600. Majority were in the range of 21-100 cells/mm^3. All cases had > 60 % lymphocyte predominance.

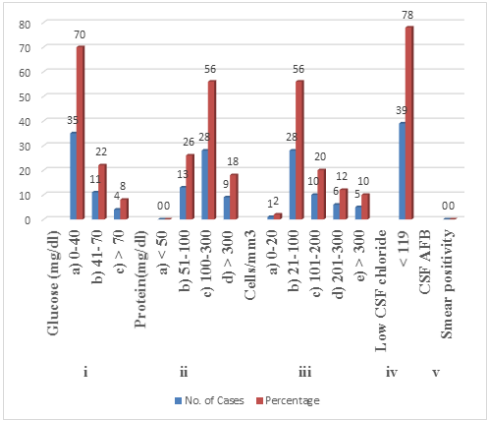


Fig 3: CSF Parameters

Table 4: MRI Findings of Patients (N=50)

MRI findings	No. of patients	% of patients
Leptomeningeal enhancement	18	36
Tuberculoma	10	20
Acute infract	14	28
Ventriculomegaly and Hydrocephalus	08	16

It is evident from table that majority i.e. 36% patients have leptomeningeal enhancement in their MRI brain followed by acute infract which is seen among 28% of patients

CSF ADA analysis

Table No. – 5: CSF ADA Levels in TBM **(N=50)**

Group	No. of cases	CSF ADA levels (U/L)		
		Mean	SD	Range
TBM	50	15.29	7.9	4 – 40

Mean value of CSF ADA protein is 15.29±7.9

SUMMARY

50 cases of tuberculous meningitis aged 18 years and above were included in the study. They were studied in detail about clinical and laboratory profile. The following observations were made:

- Patient ranged from 18-70 years. Peak incidence of cases were in 21-30 age group (38%).
- Fever and headache was present in 100%, vomiting in 84% and altered sensorium in 72% cases.
- Majority of patients presented with a duration of symptoms of 15-30 days(46%).
- Neck rigidity was present in 95% of cases, Kernig's sign in 50% cases but none had Brudzinski'ssign.
- Cranial nerve palsy was observed in 40% of cases, 6th nerve being the commonest in 16% of cases.
- Hemiparesis/ hemiplegia was present in 4 cases (8%), quadriplegia in 1 case (2%).
- According to BMRC and API Text book of Medicine 11th edition 2019, 27 cases were in stage II (54%), 26% in stage I, 20% in stage III.
- CSF glucose level was reduced(<40mg)in70% of cases with a mean valueof 36.9 mg/dl.
- CSF protein was elevated in all cases with mean value of 267.6mg/dl and majority were in the range of 100-300mg/dl.
- AllpatientshadelevatedCSFcellswith>60%lymphocytepredominance(mean 143.4 cells/ mm^3). Majority were in the range of 21-100 cells/mm^3.
- CSF ADA was elevated in TBM group than in the pyogenic group with a mean value of 15.29 ±8.2.

- At cut of value of 10 U/L the sensitivity and the specificity of CSF ADA in TBM was 75% and 90%respectively.
- MRI was done in 50 patients, in which majority i.e 36% patients have leptomeningeal enhancement followed by acute infarct is seen among 28% of patients.
- All cases were started on ATT and steroids and other antioedema measures and response to therapy by clinical improvement isnoticed.
- Out of 50, 38 cases (76%) improved, 30 cases without any sequelae, 3 cases with sequelae in the form of hemiparesis in two and optic atrophy in the other.
- 9 cases does not respond with therapy, of these 6 cases expired in the hospital, other 3 cases had multiple deficit at presentation and during followup
- Mortality cases were studied and found to have some correlation with stage of disease (stage III), and low CSF glucose (<2.2mmol/l) and altered sensorium.

CONCLUSION

- While the commonest form of the disease is pulmonary infection, one of the most dangerous forms is that effecting CNS.
- TBM has been a major problem and cause of death in developing countries. Mycobacterium TB is responsible for most cases.
- Recently there has been an increase in the incidence of tuberculosis and its complications like TBM due to HIV infection.
- Most cases of TBM are young children but can be acquired at later age also. In recent years there is an increase in the incidence of TBM in adults.
- The diagnosis of TBM is difficult. Delay in the diagnosis and treatment are regarded as the major contributing factors in the high mortality reported.
- So the present study was undertaken to study the various clinical and laboratory features of TBM
- Cranial nerve palsies or other motor deficit like hemiparesis can be a presenting feature in some patients 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. CSF ADA is a useful, diagnostic method to differentiate TBM from other meningitis. Though stage III of the disease, altered consciousness correlate with fatality, even comatose patients with localized neurological deficit can make complete recovery with prompt institution of antitubercular therapy
- MRI was done in 50 patients, in which majority i.e 36% patients have leptomeningeal enhancement followed by acute infarct is seen among 28% of patients.