ORIGINAL ARTICLE

Etiological Spectrum of Hyponatremia in Tuberculous Meningitis: Insights from Referral Institute in Northern India

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Abstract

Background: Tuberculous meningitis (TBM) causes death or severe disability in 50% of afflicted patients. Among the various complications, such as hydrocephalus, hyponatremia, cranial nerve palsies, seizures, and myeloradiculopathy, hyponatremia occurs more frequently, affecting 35–65% of patients with central nervous system (CNS) tuberculosis.

Aim: To study the prevalence of hyponatremia in patients with TBM and its correlation with the clinical profile, severity of illness, and outcome.

Methodology: In suspected TBM patients with low sodium levels, serum and urine osmolality were measured by an osmometer [Advanced Instruments, Inc. (2020)] to evaluate the possible causes. Patients underwent brain imaging to look for hydrocephalus, vasculitic infarcts, and basal exudates. They were followed up with serum sodium levels until the completion of treatment and repeat brain imaging during follow-up to assess radiological recovery.

Results: A total of 100 suspected TBM patients with a mean age of 32.6 years and predominantly female (57%) were included. The most common symptoms were fever, headache, altered sensorium, and seizures. The VI cranial nerve was most frequently involved. A total of 31% of patients presented in stage III, with a significant association with mortality ($p = 0.002$). Hyponatremia was seen in 57% of patients. The GeneXpert Ultra-based polymerase chain reaction (PCR) test for tuberculosis was positive in 41% of patients. Basal exudates ($p = 0.001$) and tuberculomas ($p = 0.004$) on brain imaging had a significant association with mortality. On follow-up, 41% of patients had improvement, 35% expired, and 10% had a static condition. Repeat sodium levels along with brain imaging were assessed ($p = 0.001$).

Conclusion: There was a high prevalence of hyponatremia in TBM patients, as seen in 57% of patients, which occurred most frequently due to syndrome of inappropriate antidiuretic hormone secretion (SIADH), followed by cerebral salt-wasting syndrome (CSWS), and was related to the severity of TBM.

Keywords: Basal exudates, Hyponatremia, Syndrome of inappropriate antidiuretic hormone secretion, Tuberculous meningitis.

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Introduction

Tuberculous meningitis (TBM) is a serious public health problem in developing countries, as it causes significant mortality and residual neurological sequelae. As per the Indian national guidelines for extrapulmonary tuberculosis (TB),[1] it constitutes 1% of all TB cases. In drug-resistant TB meningitis patients coinfected with the retrovirus, the death rate is nearly 100%. Among chronic central nervous system (CNS) infections, CNS TB is the most common because of the high prevalence of TB in the community.

In 2020, worldwide, there was a substantial fall (18% drop) in the number of people newly diagnosed with TB and notified to health authorities compared with 2019, in contrast to the marked increase in notifications between 2017 and 2019.[2]

Globally, an estimated 10.6 million people suffered from TB in 2021. Among all TB cases, 6.7% were among people living with human immunodeficiency virus (HIV). Geographically, most TB cases in 2021 were in the World Health Organization (WHO) regions of South-East Asia (45%), Africa (23%), and the Western Pacific (18%).[3] When it comes to TBM, it has an incidence of 0.1 million every year and is the most devastating and disabling form, with >50% mortality or neurological sequelae among survivors.[4]

The Government of India launched the Pradhan Mantri TB Mukt Bharat Abhiyaan on 9th September 2022, which aimed at the early identification of cases from such population groups and the early initiation of treatment,[5] as TB causes the largest number of deaths among all infectious diseases in India. India has 20% of the world's population but accounts for >25% of the total TB cases worldwide.

If untreated, TBM may lead to altered sensorium, coma, and death. In patients who survive TBM, neurological sequelae, such as intellectual disability, hydrocephalus, sensorineural hearing loss, cranial nerve palsies, seizures, stroke-associated neurological deficits, and coma, occur.[2]

Intravascular volume disturbances are frequent in TBM. Among TBM patients, nearly 45% present with hyponatremia in the study by Misra et al.,[6] and it can independently predict death or disability. In another study by Hieu et al.,[7] the death rate among TBM patients was approximately one-third of cases.

Disability-free survival in TBM is not entirely dependent on the activity of antitubercular drugs. Brain infarction, severe hyponatremia, tuberculoma formation, and hydrocephalus are the most common life-threatening complications that can occur both before and during antituberculosis treatment. The need to distinguish between cerebral salt-wasting syndrome (CSWS) and syndrome of inappropriate antidiuretic hormone secretion (SIADH) is important because the treatment of one might be detrimental to the other.

This study primarily emphasized the need for the early diagnosis of the cause of hyponatremia, which can significantly alter the neurological outcome of the patient.

Methodology

The study was conducted based on the collection of data from suspected TBM patients aged older than 12 years admitted to the Post Graduate Institute of Medical

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Education and Research (PGIMER), Chandigarh, India, during the study period from August 2021 to June 2023 after obtaining permission from the Institutional Ethics Committee vide INT/IEC/2021/SPL-1301 dated 14th September 2021. Suspected TBM patients who fulfilled the Lancet Consensus Criteria[8] were enrolled in the study, and their cerebrospinal fluid (CSF) was collected. The collected CSF samples were analyzed using the polymerase chain reaction (PCR)-based GeneXpert Ultra method to diagnose TBM.

At the initial evaluation of patients, a detailed history, including symptoms of raised intracranial pressure, seizures, diarrhea, and vomiting, was recorded in the case record form. TBM severity [British Medical Research Council (BMRC)] was categorized, and the conscious level was also assessed using the Glasgow Coma Scale (GCS). Clinical examination was focused on hemodynamic assessment, such as hypotension, tachycardia, and dry mucous membranes, which helped differentiate between the major causes of hyponatremia in TB meningitis.

Biochemical Tests

All suspected TBM patients at the time of admission underwent routine blood investigations, such as complete hemogram, serum sodium and potassium levels, renal function tests, and liver function tests. Serum samples were analyzed on the random-access autoanalyzers Beckman Coulter (models AU5800 and AU480) and Cobas 8000 used in the emergency biochemistry laboratory. Serum and urine sodium level measurements were based on the potentiometry principle.

In patients with low sodium levels who were treatment-naive for TBM, serum and urine osmolality were measured by an osmometer [Advanced Instruments, Inc. (2020)] using the freezing-point depression method to establish the type of hyponatremia and evaluate the possible causes.

Hyponatremia was categorized based on serum sodium levels into severe (<120 mEq/L), moderate (120–129 mEq/L), and mild (130–134 mEq/L) groups. In addition, a volume status assessment was performed to differentiate the common causes of hyponatremia in TBM, such as CSWS and SIADH.[9,10]

Patients underwent brain imaging, such as noncontrast computed tomography (CT) of the head at admission, followed by magnetic resonance imaging (MRI) of the brain to look for hydrocephalus, vasculitic infarcts, basal exudates, and gyral enhancement.

Patients were followed up every 6 weeks, and serum sodium levels and liver function tests were measured until the completion of treatment. They also underwent repeat MRI of the brain to assess radiological recovery.

Statistical Analysis

Data were entered into an Excel sheet after collection from the study participants. The cleaned data were exported to SPSS version 21 (SPSS Inc., Chicago, IL, USA) for Microsoft Windows to carry out all statistical calculations.

Continuous data, including demographic details and clinical and laboratory parameters, were described in terms of mean ± standard deviation (SD) and range or median and interquartile range. Discrete categorical data to denote the prevalence of hyponatremia in TBM patients were estimated as percentages and presented as *n* (%) with a 95% confidence interval. For predictors of outcome compared between patients with hyponatremia and those without hyponatremia in the TBM study subjects, the Chi-square (χ^2) test and Fisher's exact test (<5) were performed. Comparison among etiologies, such as CSWS and SIADH, in TBM patients was also evaluated using the Chi-squared test. A probability value (*p*-value) of <0.05 was considered statistically significant.

Results

Our study included 100 patients with TBM, of whom 43 (43%) were male and 57 (57%) were female. The mean age of the patients in our study was 32 years, with two-thirds of patients (67%) below 40 years of age. The age of the patients turned out to be a significant predictor of mortality (*p*-value = 0.015), whereas gender was not a significant predictor of mortality (*p*-value = 0.369).

Among the 100 patients, the most common presenting symptom was fever in 97 (97%), followed by headache in 81 (81%), altered mental sensorium in 73 (73%), and vomiting in 56 (56%) patients. In addition, 27 (27%) patients had seizures on presentation.

Hyponatremia was prevalent in 57% of the patients, among whom most were euvolemic (29%), 26% were hypovolemic, and 2% were hypervolemic. However, no significant association between the type of hyponatremia and the outcome was observed (*p*-value = 0.693).

Among the cranial nerve palsies, cranial nerve VI was affected most frequently in 18 (18%) patients, followed by cranial nerve III in eight (8%), cranial nerve IV in four (4%), and cranial nerve II in three (3%) patients; these findings were not statistically significant.

In this study, 41 (41%) patients had definitive cases, 48 (48%) were highly probable, and 11 (11%) had a possible diagnosis of TBM.[8] The GeneXpert Ultra test was positive in 41 (41%) patients, which accounted for a definitive diagnosis of TBM. Sixty (60%) patients had rifampicin-sensitive disease, two (2%) patients were rifampicin resistant, whereas 38 (38%) had indeterminate resistance, as highlighted in Table 1.

TBM patients presented with different clinical stages. Most patients, 40 (40%), were in stage II, 31 (31%) were in clinical stage III, and 29 (29%) were in stage I. Clinical stage had a significant association with mortality (*p*-value = 0.002).

On brain imaging, 57 (57%) patients had hydrocephalus, 38 (38%) had basal exudates, 24 (24%) had cerebral vasculitic infarcts, 17 (17%) had tuberculomas, 13 (13%) had arachnoiditis, and 11 (11%) patients had a miliary pattern on chest X-ray. Among these findings, only basal exudates (*p*-value = 0.01) and tuberculomas (*p*-value = 0.004) had a significant correlation with patient outcomes.

The clinical-radiological predictors of poor outcome in this study were low GCS at admission, the presence of altered mental sensorium, a higher clinical stage of TBM, and the presence of infarcts and exudates on MRI brain findings at the time of admission.

In our study, the probable etiology of hyponatremia was assessed based on volume status (clinical and laboratory parameters). Among the 57 (57%) patients with hyponatremia, 29 (29%) belonged to the SIADH group, and 26 (26%) belonged to the CSWS group. However, the etiology of hyponatremia in our study was not a predictor of mortality (*p*-value = 0.693). CSWS was more common in males, whereas SIADH was more common in females. Seizures occurred with almost equal frequency in both the CSWS and SIADH groups. Among the radiological parameters, hydrocephalus occurred with equal frequency in both groups. Exudates and infarcts appeared to be more common in the SIADH group (Fig. 1).

The Glasgow Coma Scale (GCS) score calculated at the time of admission was found to be a significant predictor of mortality (*p*-value = 0.001) (Fig. 2).

The occurrence of clinical parameters, such as hypotension, dry mucous membranes, and tachycardia, had a significant association when comparing the CSWS and SIADH groups (*p*-value = 0.001). However, the laboratory parameters, such as serum uric acid, urea, hematocrit, and albumin, did not have a significant association, whereas the occurrence of anemia in both groups had statistical significance (*p*-value = 0.039) (Table 2).

Modified antitubercular therapy (ATT) was started in 10 patients who had initially been started on first-line antitubercular therapy,

Table 1: Comparison of clinical, radiological parameters with and without Hyponatraemia in patients with Tubercular meningitis (n = 100)

Parameters	*Total*	*Hyponatremia 57 (57%)*	*Without hyponatremia 43 (43%)*	*p-value*
Age-group				0.151
<20 years	23 (23%)	15 (26.3%)	8 (18.6%)	
21–30	32 (32%)	17 (29.8%)	15 (34.9%)	
31–40	18 (18%)	12 (21.05%)	6 (14%)	
41–50	15 (15%)	7 (12.2%)	8 (18.6%)	
51–60	7 (7%)	3 (5.2%)	4 (9.3%)	
>60	5 (5%)	3 (5.2%)	2 (4.7%)	
Gender				0.67
Male	47 (47%)	27 (47.36%)	20 (46.5%)	
Female	53 (53%)	20 (35.08%)	23 (53.5%)	
Cerebral imaging				
Hydrocephalus	50 (50%)	30 (52.6%)	20 (46.5%)	0.709
Basal exudates	38 (38%)	27 (47.3%)	11 (25.6%)	5.97
Vasculitic infarcts	24 (24%)	16 (28.07%)	8 (18.6%)	1.43
Tuberculomas	17 (17%)	11 (19.29%)	6 (14%)	5.76
Arachnoiditis	13 (13%)	6 (10.52%)	7 (16.3%)	1.32

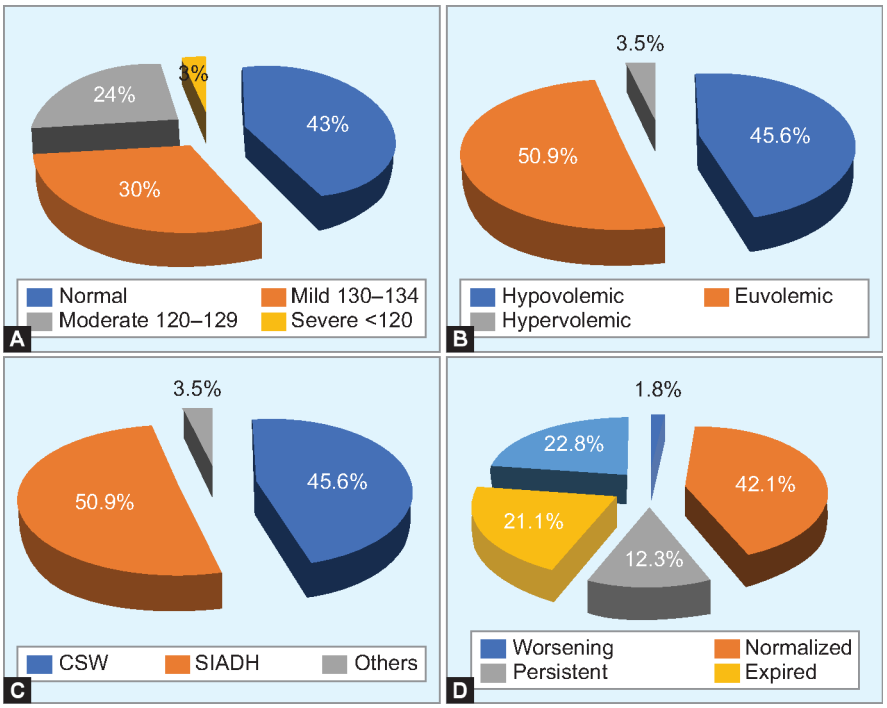

Fig. 1A to D: Pie-diagram illustrates various types of Hyponatramia on basis of laboratory values of sodium (A), volume status (B), etiology of hyponatraemia (C), outcome of patients with hypo-natraemia (D)

and ventriculoperitoneal (VP) shunting was performed in 15 patients among the 50 patients with hydrocephalus. Neither of these parameters had a significant association with the final outcome (p-values = 0.2 and 0.5, respectively).

In terms of study outcomes, 48 (48%) patients showed improvement, 35 (35%) patients died, 10 (10%) patients remained in a static condition, and seven (7%) patients were lost to follow-up. Parameters that had a significant association with mortality included patient age, clinical stage, GCS, hydrocephalus, basal exudates on MRI of the brain at presentation, follow-up MRI findings, and follow-up sodium levels (p-value = 0.001) (Table 3).

Discussion

In India, the disease burden of tuberculosis (TB) is estimated at 2.6 million people contracting the disease, and approximately 4,00,000 people die every year.[5] The enormous burden of the disease is faced by developing and underdeveloped countries because of poverty, poor sanitation, overcrowding, and limited access to health care.

The present study included 100 patients, with 43 males and 57 females, and the mean age was 32 years. Most of the patients (n = 32) included in our study were between 21 and 40 years of age, and age was established as a significant predictor of mortality (p-value = 0.015), in contrast to the study by Hieu et al.,[7] in which age was not a significant contributor to mortality.

In our study, 30% of patients had mild hyponatremia, 24% had moderate hyponatremia, while severe hyponatremia occurred in only 3% of cases, possibly because our institute provides tertiary care facilities to various northern states of India, especially for neurological illnesses, as the first point of contact, and patients came from far-flung areas with early referral for timely diagnosis and the excellent services provided by our institute (Figs 1 and 2).

About 30% of the patients with hyponatremia were hypovolemic, which was similar to the study by Misra et al.,[6] as most of the admitted patients belonged to a lower socioeconomic status and were less educated, resulting in a poorer understanding of the disease process.

A total of 41 (41%) patients had a definitive diagnosis of TBM, which differed from the study by Misra et al.,[11] in which 23% of patients had a definitive diagnosis. This is attributed to the higher sensitivity of the CSF GeneXpert Ultra test[12] performed in comparison with previous studies, which primarily depended on GeneXpert. Among cranial nerve involvement, three (3%) patients had involvement of cranial nerve II, eight (8%) of cranial nerve III, four (4%)

Table 2: Comparison of clinical, laboratory, cerebral imaging, staging in different etiologies of Hypo-natraemia in patients with Tubercular meningitis ($n = 57$)

Parameters	*CSWS (N = 26)*	*SIADH (N = 29)*	*Others (N = 2)*	*Total (N = 57)*	*p-value*
Clinical					
Gender					0.189
Male	15 (57.7%)	12 (41.4%)	0 (0%)	27	
Female	11 (42.3%)	17 (58.6%)	2 (100%)	30	
Dry mucous membrane	24 (92.3%)	2 (6.9%)	0 (0%)	26	0.001
Tachycardia (HR >100)	26 (100%)	4 (13.8%)	1 (50%)	31	0.001
Weight loss	15 (57.7%)	17 (58.6%)	0 (0%)	32	0.265
Seizures	17 (65.4%)	18 (62.1%)	1 (50%)	36	0.896
Blood investigations					
Hemoglobin >11 gm/dL	26 (100%)	23 (79.3%)	2 (100%)	51	0.039
Hematocrit >30	18 (69.2%)	25 (86.2%)	2 (100%)	45	0.329
Urea >40 mg/dL	15 (57.7%)	16 (55.2%)	2 (100%)	33	0.462
Uric acid <4 mg/dL	2 (7.7%)	7 (24.1%)	1 (50%)	10	0.395
S. albumin >3 gm/dL	18 (69.2%)	18 (62.1%)	1 (50%)	37	0.727
Cerebral imaging					
Hydrocephalus	13 (50%)	13 (44.8%)	2 (100%)	28	0.318
Exudates	9 (34.6%)	17 (58.6%)	1 (50%)	27	0.204
Infarcts	5 (19.2%)	11 (37.9%)	0 (0%)	16	0.204
Tuberculomas	4 (15.4%)	7 (24.1%)	0 (0%)	11	0.557
Arachidonitis	3 (11.5%)	3 (10.3%)	0 (0%)	6	0.876
Stage III TBM	9 (34.6%)	11 (37.9%)	0 (0%)	20	0.532

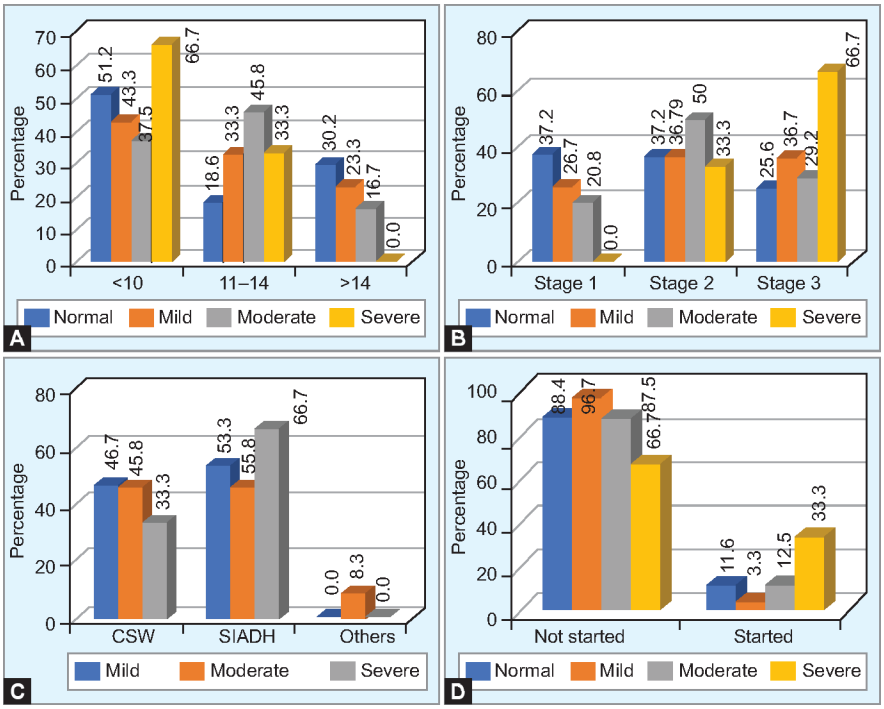

Fig. 2A to D: Patients with Hyponatraemia are categorized into mild, moderate, severe types on basis of sodium levels on Y-axis as constant. In different bar diagrams: (A) Comparison with level of consciousness with Glasgow coma scaling system(GCS) is established; (B) Comparison with various stages of TBM is described; (C) Various etiologies of Hyponatraemia like cerebral salt wasting, syndrome of in appropriate anti-diuretic hormone secretion is described; (D) Comparison is done with early initiation of anti-tubercular therapy versus late initiation with severity of Hypo-natraemia

of cranial nerve IV, two (2%) of cranial nerve V, and 18 (18%) of cranial nerve VI, but this did not predict mortality, in contrast to the study by Synmon and Kayal,[13] in which a significantly poor outcome was observed. The sixth cranial nerve was the most commonly involved in our study because of its longest intracranial course and basal brain predilection.

In this study, mortality was high in both hypovolemic and euvolemic states, at 14 and 16%, respectively, while 14.3% mortality was observed in the hypervolemic state. In the present study, two patients (among the three patients) with severe hyponatremia died. As far as gender is concerned, although female patients had a predisposition for moderate and severe hyponatremia, no significant difference in terms of mortality was observed. The Chi-squared test did not demonstrate any significant association between the severity of hyponatremia and overall survival (p-value = 0.621). However, mortality with respect to the clinical stage of the disease had a significant association (p-value = 0.001), which is expected because patients with stage III disease had more severe illness and, thus, higher mortality.

Correction of hypovolemic hyponatremia requires close serial monitoring, as the correction rate is unpredictable in this category. In contrast, in hypervolemia or SIADH, water restriction with appropriate drugs is the mainstay of treatment for the correction of hyponatremia to induce negative water balance. In the management of SIADH, loop diuretics, plentiful oral salt, and vaptans augment free water excretion and help normalize sodium levels. Thus, hypotonic fluids must be administered carefully to hospitalized patients, as they can precipitate severe hyponatremia and carry a high mortality risk, as reported in earlier studies.[6,11]

As the study was conducted during the coronavirus disease 2019 (COVID-19) pandemic, when access to tertiary health

Table 3: Comparison of clinical characteristics, radiological, staging of TBM between survivors and non survivors (*n* = 100)

Parameters	*Total*	*Survivors (N = 73)*	*Nonsurvivors Mortality (N = 27)*	*p-value*
Age				0.015
<20 years	23 (23%)	21 (28.8%)	2 (7.4%)	
21–30	32 (32%)	26 (35.6%)	6 (22.2%)	
31–40	18 (18%)	12 (16.4%)	6 (22.2%)	
41–50	15 (15%)	9 (12.3%)	6 (22.2%)	
51–60	7 (7%)	2 (2.7%)	5 (18.5%)	
>60	5 (5%)	3 (4.1%)	2 (7.4%)	
Gender				0.369
Male	47 (47%)	32 (43.8%)	15 (55.6%)	
Female	53 (53%)	41 (56.2%)	12 (44.4%)	
GCS group				0.001
<10	26	26 (35.6%)	20 (74.1%)	
11–14	23	23 (31.5%)	7 (25.9%)	
>14	24	24 (32.9%)	0 (0.0%)	
Hydrocephalus	50	31 (42.5%)	19 (70.4%)	0.023
Basal exudates	38	32 (43.8%)	6 (22.2%)	0.048
Vasculitic infarcts	24 (24%)	12 (16.4%)	12 (44.4%)	0.004
Tuberculomas	17 (17%)	16 (21.9%)	1 (3.7%)	0.036
Arachnoiditis	13 (13%)	11 (15.1%)	2 (7.4%)	0.505
Diagnosis				
Definitive	41	28 (38.4%)	13 (48.1%)	0.609
Clinical stage TBM				0.001
I	29 (29%)	27 (37%)	2 (7.4%)	
II	40 (40%)	31 (42.5%)	9 (33.3%)	
III	31 (31%)	15 (20.5%)	16 (59.3%)	
VP shunting	15	10 (13.7%)	5 (18.5%)	0.549
MRI status				0.001
Worsening	1	1 (1.4%)	0 (0.0%)	
Improvement	38	38 (52.1%)	0 (0.0%)	
Static	12	11 (15.1%)	1 (3.7%)	
LTFU	24	22 (30.1%)	2 (7.4%)	
Death	25	1 (1.4%)	24 (88.9%)	
Follow-up Na				0.001
Worsening	1	1 (1.4%)	0 (0.0%)	
Normalized	24	24 (32.9%)	0 (0.0%)	
Persistent	7	7 (9.6%)	0 (0.0%)	
Death	12	11 (15.1%)	1 (3.7%)	
LTFU	13	0 (0.0%)	13 (48.1%)	
Etiology of hyponatremia				0.693
CSWS	26	19 (26.0%)	7 (25.9%)	
SIADH	29	22 (30.1%)	7 (25.9%)	
Other causes	2	2 (2.7%)	0 (0.0%)	

Contd…

Contd…

Parameters	*Total*	*Survivors (N = 73)*	*Nonsurvivors Mortality (N = 27)*	*p-value*
Type of hyponatremia				0.693
Hypovolemic	26	19 (26.0%)	7 (25.9%)	
Euvolemic	29	22 (30.1%)	7 (25.9%)	
Hypervolemic	2	2 (2.7%)	0 (0.0%)	

services was jeopardized, more patients presented with stage III disease; therefore, patients presented in critical condition, leading to high mortality.

Hyponatremia in CNS infections can result from more than one mechanism, with SIADH and CSWS being the most common, as reported in a similar study by Misra et al.[6]

SIADH is a volume-expanded state because of antidiuretic hormone-facilitated renal water retention, in contrast to CSWS, in which there is reduced effective arterial blood volume secondary to renal salt wasting. Natriuretic peptides, including both brain natriuretic peptide and C-type natriuretic peptides, have been associated with the proposed mechanism in CSWS.[1]

In our study, 57 (57%) of the patients had hyponatremia at admission, similar to the study by Anderson et al.[14] Out of the 57 patients, the probable etiology of hyponatremia was SIADH in 29 (29%) patients and CSWS in 26 (26%) patients, in contrast to the study by Misra et al.,[6] in which CSWS occurred in 17% of patients and SIADH in 13%.

Conclusion

There was a high prevalence (57%) of hyponatremia in TBM patients. Hyponatremia in TBM occurred most frequently due to SIADH, followed by CSWS. There was high mortality (35%), and the predictors of outcome were patient age, clinical stage, GCS, the presence of hydrocephalus and exudates on MRI brain findings at presentation, improvement on follow-up MRI brain findings, and normalization of follow-up sodium levels, all of which were statistically significant parameters.

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