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A STUDY OF ELECTROLYTE IMBALANCE (SODIUM AND CHLORIDE) IN CENTRAL NERVOUS SYSTEM TUBERCULOSIS – AN OBSERVATIONAL STUDY

Pulmonary Medicine

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ABSTRACT

This study investigates the Sodium and Chloride abnormalities in Central Nervous System Tuberculosis. Conducted in the Department of Pulmonary Medicine at Government Medical College, Amritsar, 50 diagnosed cases of Central nervous system tuberculosis were analysed. This study shows significant prevalence of Sodium and Chloride abnormalities. Early detection and treatment of underlying electrolyte abnormality can potentially reduce mortality and morbidity associated with Central nervous system tuberculosis and reduce duration of hospitalization.

KEYWORDS

Central nervous system tuberculosis, sodium, chloride, tuberculosis

BACKGROUND

Tuberculosis (TB) stands as one of the most persistent and pervasive infectious diseases known to humanity, with history dating back to ancient civilizations. Tuberculosis, caused by the bacterium Mycobacterium tuberculosis (MTB), is transmitted through the air when infected individuals expel the bacteria by coughing, sneezing, or talking. While the disease mainly affects the lungs (pulmonary TB), it can also occur in other parts of the body (extrapulmonary TB). Approximately one-fourth of the global population is infected with Mycobacterium tuberculosis. [1]

Involvement of the central nervous system (CNS) is recognized as one of the most concerning complications of tuberculosis (TB). Autopsy findings indicate that 5%-15% of individuals exposed to Mycobacterium tuberculosis (Mtb) progress to active pulmonary TB, with 5%- 10% of these cases developing neurological TB.[2]

Managing CNS tuberculosis is challenging in any hospital setting and becomes even more difficult when patients are co-infected with HIV.[3] TBM can often be diagnosed based on clinical grounds, but microbiological confirmation is challenging. The clinical features, including imaging characteristics, resemble those seen in patients without HIV infection. Intracerebral tuberculomas are more common in those with HIV co-infection.[4] In about 50% of cases, a definitive microbiologic diagnosis is not obtained, necessitating empirical anti-tuberculosis treatment (ATT) by the physician. Various infectious and non-infectious disorders affecting the central nervous system (CNS) include partially treated pyogenic meningitis, fungal meningitis, sarcoidosis, meningeal metastases, and meningeal lymphomatosis, closely mimic TBM, presenting significant diagnostic challenges.

MATERIALS AND METHODS

This observational study was carried out on 50 patients of central nervous system tuberculosis patients. For these 50 patients blood samples were collected and their serum values of sodium and chloride were evaluated. The patients were enrolled at department of Pulmonary Medicine, Govt. Medical College, Amritsar. This study was conducted after approval from Institutional Thesis and Ethics Committee, Government Medical College, Amritsar.

Inclusion Criteria

1. Patient consenting for the study.
2. Age 18 years or older
3. Patients admitted with diagnosis of central nervous system tuberculosis.

Exclusion Criteria

1. Patients not giving consent for the study.
2. Patients <18 years of age.
3. In CSF examination, presence of other causes of meningitis (Cryptococcus, Streptococcus pneumonia, Neisseria meningitis, Haemophilus influenzae).
4. Patients who are transferred out (Migration).

METHODOLOGY

Two millilitres of blood were drawn using all standard safety precaution and analysed for sodium and chloride levels. If hyponatremia/hypochloremia were present evaluated for the etiology of hyponatremia and patients was labelled as having SIADH or CSWS based on the volume status; Euvolemia (absence of supine heart rate more than 100 and systolic BP less than 100 mmHg) was labelled SIADH and hypovolemia (presence of supine heart rate more than 100 and systolic BP less than 100 mm Hg) was labelled as CSWS, plus having any two or more of the following: Plasma sodium concentration ≤135 mmol/L, Plasma osmolality ≤280 mOsmol/kg, Urine osmolality ≥100 mOsmol/kg, Urinary sodium concentration ≥30mmol/L, absence of clinical or biochemical features of adrenal and thyroid dysfunction or no history of diuretic use within the past three months.

Statistical Analysis

Data were analyzed using SPSS 20 software (IBM Corporation, Armonk, NY, USA). Continuous data were presented as mean with standard deviation (SD), and categorical data as percentages. Numerical variables were compared using Chi-Square, Post hoc ANOVA, Student's t-test, and Spearman's correlation. The p value determined the level of significance. Results were compared to previous studies for conclusions.

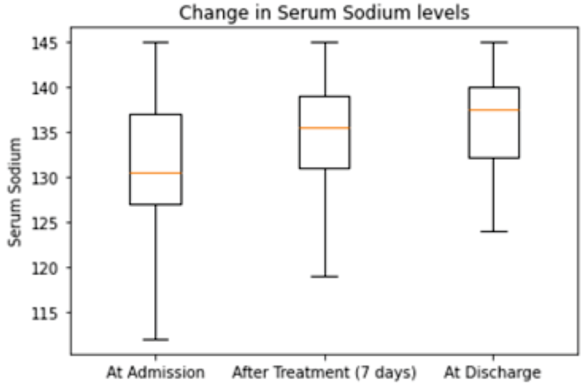
OBSERVATION AND RESULTS

Our study shows that in 44% percent patient have Normal Serum Sodium levels (135-145 Meq/L) while the rest 56% shows Hyponatremia (<135 meq/L) at the time of Admission, 68% percent patient have Normal Serum Sodium levels (135-145 Meq/L) while the rest 32% shows Hyponatremia (<135 meq/L) after 7 days of treatment and 86% percent patient have Normal Serum Sodium levels (135-145 Meq/L) while the rest 14% shows Hyponatremia (<135 meq/L) at the time of discharge. In patients with central nervous system tuberculosis, specifically tuberculous meningitis (TBM), serum sodium levels at the time of admission are often lower than in other types of meningitis. According to a study by Na et al., the initial serum sodium level in TBM patients was significantly lower compared to those with bacterial meningitis (BM) and viral meningitis (VM), with an average level of 136.9 ± 5.9 mmol/L. [5] Further studies have shown that hyponatremia is a common finding in TBM patients. Misra et al. reported that 44.7% of TBM patients had hyponatremia, categorized as mild (130-134 mEq/L), moderate (120-129 mEq/L), or severe (<120 mEq/L).76 Another study by Ye et al. found that 45.6% of TBM patients had hyponatremia, with the majority presenting with moderate hyponatremia.[6]

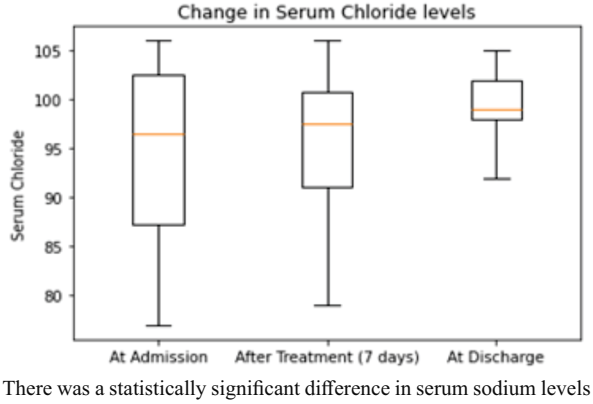
Our study shows that in 52% percent patient have Normal Serum Chloride levels (96-106 Meq/L) while the rest 48% shows Hypochloremia (<96 meq/L) at the time of admission,66% percent

patient have Normal Serum Chloride levels (96-106 Meq/L) while the rest 34% shows Hypochloremia (<96 meq/L) after 7 days of treatment for Tubercular meningitis and 86% percent patient have Normal Serum Chloride levels (96-106 Meq/L) while the rest 14% shows Hypochloremia (<96 meq/L) at the time of discharge. In the study conducted by Harrison HE et al on disturbances of ionic equilibrium in patients with tuberculous meningitis assessed plasma chloride levels in 12 patients. The results indicated that plasma chloride levels were generally low, with values ranging from 85 to 100 mEq/L, which is below the normal range of 98 to 106 mEq/L. This hypochloremia was attributed to the loss of chloride in the urine and the cerebrospinal fluid, as well as to the dilutional effect of water retention. [7]

Our study shows that 42% percent patients have Normal Sodium levels, in 28% cases Hyponatremia is caused by Cerebral salt wasting, in 14% cases Hyponatremia is caused by SIADH and in the rest of 16% cases Hyponatremia is caused by miscellaneous causes. In a study conducted at the Department of Neurology, Jinnah Postgraduate Medical Centre (JPMC) in Karachi, Pakistan, sodium levels were investigated to determine the frequency of hyponatremia among patients presenting with tuberculous meningitis (TBM). The study included 160 patients diagnosed with TBM. Among these patients, 40% (64 individuals) were found to have hyponatremia. Further analysis revealed that 14.4% (23 patients) of those with hyponatremia had syndrome of inappropriate antidiuretic hormone secretion (SIADH), while 25.6% (41 patients) exhibited cerebral salt wasting syndrome (CSWS). [8] In a prospective hospital-based study involving 76 patients diagnosed with tuberculous meningitis (TBM), hyponatremia was found in 44.7% of cases, totalling 34 patients. Further investigation into the causes of hyponatremia revealed distinct etiologies: 17 patients were diagnosed with cerebral salt wasting syndrome (CSWS), 3 patients had syndrome of inappropriate secretion of antidiuretic hormone (SIADH), and miscellaneous causes accounted for hyponatremia in 14 patients. This study underscores the notable incidence of hyponatremia among TBM patients and highlights the variability in underlying mechanisms contributing to this electrolyte disturbance. [9]



A Friedman test was conducted to assess differences in serum sodium levels across three conditions: At Admission, After Treatment (7 days), and At Discharge (N= 50).



There was a statistically significant difference in serum sodium levels across the three conditions, $\chi^2(2) = 129.25$, $p < 0.001$. The mean ranks of serum chloride levels were significantly different between At Admission (mean rank = 130.68), After Treatment (7 days) (mean rank = 133.8), and At Discharge (mean rank = 135.46).

A Friedman test was conducted to assess differences in serum chloride levels across three conditions: At Admission, After Treatment (7 days), and At Discharge (N= 50). There was a statistically significant difference in serum chloride levels across the three conditions, $\chi^2(2) = 110.41$, $p < 0.001$. The mean ranks of serum chloride levels were significantly different between At Admission (mean rank = 93.42), After Treatment (7 days) (mean rank = 95.46), and At Discharge (mean rank = 96.8).

CONCLUSION

Central nervous system tuberculosis, a debilitating and often lethal form of tuberculosis, presents significant challenges for clinicians and researchers. Diagnosing this form of Tuberculosis is particularly difficult in resource-limited settings. It primarily relies on analyzing sufficient volumes of cerebrospinal fluid (CSF), with at least 5 ml needed for accurate assessment. Given any CNS symptoms, clinicians should promptly perform lumbar punctures, as these symptoms might indicate TBM, which has a high fatality rate. To speed up diagnosis using CSF samples, the CBNAAT method should be used preferentially. Rapid diagnosis and treatment are crucial for improving prognosis, reducing mortality, and minimizing neurological deficits.

Early detection and treatment of underlying electrolyte abnormality can potentially reduce mortality and morbidity associated with CNS tuberculosis and reduce duration of hospitalization. Further research into the prevalence of Sodium and Chloride abnormalities can add to the lacunae of knowledge. Longitudinal studies can be used to assess the incidence of electrolyte abnormality which develop over the course of illness.

REFERENCES

[1]. Mirzayev, F., Viney, K., Linh, N. N., Gonzalez-Angulo, L., Gegia, M., Jaramillo, E., ... & Kasaeva, T. (2021). World Health Organization recommendations on the treatment of drug-resistant tuberculosis, 2020 update. European Respiratory Journal, 57(6).
[2]. Busl, K. M., & Bleck, T. P. (2013). Bacterial infections of the central nervous system. Current infectious disease reports, 15, 612-630.
[3]. Thwaites, G. E., & Hien, T. T. (2005). Tuberculous meningitis: many questions, too few answers. The Lancet Neurology, 4(3), 160-170.
[4]. Thwaites, G. E., Duc Bang, N., Huy Dung, N., Thi Quy, H., Thi Tuong Oanh, D., Thi Cam Thoa, N., ... & Farrar, J. J. (2005). The influence of HIV infection on clinical presentation, response to treatment, and outcome in adults with tuberculous meningitis. The Journal of infectious diseases, 192(12), 2134-2141.
[5]. Na, S., Kim, T., Song, I. U., Chung, S. W., Kim, S. H., Oh, Y. S., ... & Kim, W. (2021). The association between serum sodium level and tuberculous meningitis compared with viral and bacterial meningitis. Scientific Reports, 11(1), 10906.
[6]. Ye, Q., Peng, X., Zhang, X., Cao, Q., Tao, K., & Wang, L. (2022). Clinical analysis of 103 cases of tuberculous meningitis complicated with hyponatremia in adults. Neurological Sciences, 1-7.
[7]. Harrison, H. E., Finberg, L., & Fleishman, E. (1952). Disturbances of ionic equilibrium of intracellular and extracellular electrolytes in patients with tuberculous meningitis. The Journal of Clinical Investigation, 31(3), 300-308.
[8]. Bhati, M. R., Prajapat, N. K., & Sharma, A. K. (2022). A study of electrolyte disturbance in patients with tubercular meningitis in central Rajasthan. European Journal of Molecular & Clinical Medicine, 9(7), 2442–2450.
[9]. Chen, W., Huang, L., Tang, Q., Wang, S., Hu, C., & Zhang, X. (2020). Progress on diagnosis and treatment of central nervous system tuberculosis. Radiology of Infectious Diseases, 7(4), 160-169.