Hiponatremia pada Pasien Muda dengan Tuberkulosis Paru dan Meningoencefalitis Tuberkulosis

Hyponatremia in Young Patients with Pulmonary Tuberculosis and Tuberculous Meningoencephalitis

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Abstract

Tuberkulosis paru (PTB) adalah infeksi pernapasan yang jarang terjadi dan jarang menyebabkan hiponatremia, suatu kondisi yang ditandai dengan rendahnya kadar natrium dalam darah. Pada kebanyakan kasus, gejala hiponatremia yang diakibatkan oleh PTB adalah ringan hingga sedang, sering kali tidak menunjukkan gejala yang nyata, dan dapat diatasi secara efektif melalui pengobatan dengan obat anti TB. Hiponatremia didiagnosis ketika kadar natrium serum turun di bawah 135 mmol/L, dan dianggap parah ketika kadarnya turun di bawah 125 mmol/L. Di antara ketidakseimbangan elektrolit, hiponatremia adalah salah satu yang paling sering diamati, dengan prevalensi berkisar antara 1 hingga 4% dalam manifestasi yang parah. Jurnal ini melaporkan kasus seorang pasien laki-laki yang datang dengan penurunan kesadaran, yang diduga terkait dengan hiponatremia sedang, meningoencefalitis tuberkulosis, tuberkulosis paru, dan bradikardia sinus. Gejala pasien yang terkait dengan hiponatremia relatif ringan, sehingga perlu dilakukan evaluasi lebih lanjut untuk memastikan penyebab ketidakseimbangan elektrolit. Hal ini terutama penting bagi pasien yang diduga menderita tuberkulosis yang juga mengalami komplikasi yang berkaitan dengan hiponatremia.

Kata Kunci: Infeksi Tuberkulosis; Hiponatremia; SIADH

Keywords: Tuberculosis Infection; Hyponatremia; SIADH

Pulmonary tuberculosis (PTB) is an uncommon respiratory infection that rarely leads to the development of hyponatremia, a condition characterized by low levels of sodium in the blood. In most cases, symptoms of andiated hyponatremia are mild to moderate, often lack noticeable symptoms, and can be effectively reversed through treatment with anti-TB medications. Hyponatremia is diagnosed when the serum sodium level falls below 135 mmol/L, and it is considered severe when the level drops below 125 mmol/L. Hyponatremia is one of the most frequently observed among electrolyte imbalances with a prevalence ranging from 1 to 4% in its severe manifestation. This journal reports the case of a male patient who presented with decreased consciousness, suspected to be associated with moderate hyponatremia, tuberculous meningoencephalitis, pulmonary tuberculosis, and sinus bradycardia. The patient's symptoms associated with hyponatremia were relatively mild, prompting further evaluation to ascertain the root cause of the electrolyte imbalance. This is particularly important for patients with suspected tuberculosis who are also experiencing complications related to hyponatremia.

Keywords: Tuberculosis Infection; Hyponatremia; SIADH
Introduction

Pulmonary tuberculosis (PTB) is an infrequent cause of hyponatremia (Ganiger et al., 2019), with most cases being mild to moderate and asymptomatic, but reversible with anti-TB treatment. Hyponatremia is defined as having serum sodium levels below 135 mmol/L (Mentrasti et al., 2020), and it is considered severe when levels drop below 125 mmol/L (Dash et al., 2019). It is one of the most prevalent electrolyte disorders, with severe forms occurring in 1-4% of hospitalized adults and milder forms in 15-30% (Ewa et al., 2021; Jafari et al., 2013). Screening for hyponatremia is crucial due to its incidence in active tuberculosis cases, ranging from 11% to 51% (So & Villanueva, 2021).

Tuberculosis can affect various organs, including the adrenal glands, hypothalamus, pituitary gland, meninges, or lungs, leading to hyponatremia through adrenal insufficiency (AI) or inappropriate antidiuretic hormone (ADH) secretion. Major causes of hyponatremia in tuberculosis include AI, syndrome of inappropriate ADH secretion (SIADH), and cerebral wasting syndrome (CSWS). SIADH should be considered when hyponatremia is accompanied by low serum osmolality, average acid-base balance, urine osmolality above 100 mOsm/L, and urine sodium levels above 40 meq/L. The exact cause is not fully understood, but it is believed to be associated with hypoxia, hypovolemia, or reduced blood volume. On the other hand, CSWS leads to hyponatremia and decrease extracellular fluid volume due to renal salt depletion (Oh & Seo, 2020). SIADH physiologically involves inappropriate ADH secretion or increased renal sensitivity to ADH, resulting in kidney water retention and euvolemic hyponatremia hyponatremia (Ye et al., 2019). However, increased serum ADH does not exclude CSWS (Cui et al., 2019), as it can also increase as a physiological response to hypovolemia. The distinct pathophysiology’s of SIADH and CSWS require early differentiation for appropriate treatment, as they necessitate different therapeutic approaches (Hannon & Thompson, 2019).

Case Report

The 33-year-old male presented to the hospital with decreased consciousness. According to the patient’s family, he suddenly became unconscious at 6:30 PM. Before this, the patient complained of a one-week fever accompanied by a headache, sore throat, and a two-week cough. Over time, the patient experienced a headache that did not improve with paracetamol and ibuprofen. The patient also became lazy and withdrawn before eventually losing consciousness. This headache was accompanied by nausea and vomiting, but the patient’s family could not describe how the vomiting occurred, although it was stated that the vomiting was continuous. When in the emergency department, the patient vomited once, in the form of food. The cough was productive with white-colored sputum. There was no history of diarrhea, hypertension, diabetes mellitus, or previous TB from the patient and his family. The patient also did not consume alcohol or any regular medications. The patient’s treatment history included normal saline, omeprazole, ondansetron 4mg, antrain, ceftrixone, and dexamethasone.

On physical examination, the patient appeared weak with a Glasgow Coma Scale (GCS) score of E1M2V5, blood pressure of 96/63 mmHg, heart rate of 49 beats per minute, respiratory rate of 20 breaths per minute, and body temperature of 36.8°C. The patient’s saturation was 98% with a nasal cannula at 3 litres per minute. Conjunctival pallor, icteric sclera, cyanosis, and dyspnea were not observed. The pupils were isochoric, and light reflexes were present in both eyes, measuring 2 mm. There was neck stiffness detected. Upon examination of the thorax, vesicular breath sounds were heard bilaterally, and no rales or wheezing were present. A heart examination revealed a single S1S2 heart sound. The abdomen was distended, and bowel sounds were normal, but tenderness evaluation was complex. On examination of the four extremities, the warm acral sensation was detected, and no oedema was found in any of the extremities. However, there were signs of laterality on the left side of the body.

Laboratory investigations revealed a hemoglobin level of 12.1 g/dl, leukocyte count of 13,500/μl, platelet count of 472,000/μl, hematocrit level of 37%, blood glucose level of 104 mg/dl, and negative COVID-19 antigen. The albumin level was 3.2 mg/dl, sodium level was 128.7 mEq/L, potassium level was 4.37 mEq/L, chloride level was 97.9 mEq/L, total bilirubin level was 0.79 mg/dl, and direct bilirubin level was 0.25 mg/dl. Chest X-ray and head CT-scan examinations were performed, and the results are shown in Figure 1.

Based on the anamnesis, physical examination, and supporting investigations, the patient has been diagnosed with decreased consciousness with moderate hyponatremia due to Tuberculous Meningoencephalitis and Pulmonary Tuberculosis with Sinus Bradycardia. Previously, the patient received the following management: Nasal cannula oxygen at 3 LPM, NS infusion at 20 drops per minute, Injection of Omeprazole 2 x 40 mg, Injection of Ondansetron 3 x 4 mg, Injection of Antrain 3 x 1 gram, Injection of Ceftriaxone 2 x 2 grams, Injection of Dexamethasone 4 x 5 mg. The patient has a DC (disposable catheter) and NGT (nasogastric tube) in place. Advice from the neurology department includes the administration of Injection Dexamethasone 4 x 10 mg IV, Injection Ranitidine 2 x 50 mg IV, and consultation with the Cardiovascular Thoracic Surgery (TS Cardio) department.

On the second day of hospitalization, the pulmonologist added oral OAT therapy consisting of isoniazid 400 mg, rifampicin 600 mg, ethambutol 1000 mg, and pyrazinamide 1500 mg. Additionally, streptomycin injection was administered once. The neurologist increased the dosage of dexamethasone from the previous hospital, prescribing dexamethasone four times a day, with each dose being 10 mg. TO manage the patient’s hyponatremia, NaCl 0.9% at a rate of 20 drops per minute was given. On the fourth day, the patient’s consciousness improved with a Glasgow Coma Scale (GCS) measurement of 325. The patient still experienced shortness of breath but showed improvement. From vital sign observations, the pulse rate began to increase. However, the sodium electrolyte level
remained low at 128.9 mmol/L. The neurologist advised administering NaCl 3% at a rate of 7 drops per minute to correct the sodium level. The next day, the patient’s consciousness further improved with a GCS measurement of 4x6. The sodium level had increased to 132.3 mmol/L. The patient’s nasogastric tube (NGT) was removed, and they began swallowing exercises with soft porridge. On the seventh day, the patient became communicative with GCS measurements of 4x6. The therapy involving ceftriaxone, omeprazole, and dexamethasone was discontinued. On the ninth day, the patient’s general condition improved, the shortness of breath subsided, and there were no seizures. The patient was discharged while continuing the OAT therapy.

In summary, the patient showed significant improvement during the hospitalization period. The pulmonary doctor adjusted the OAT therapy, and the neurologist monitored and managed the patient’s consciousness and sodium levels effectively. With the progress made, the patient’s shortness of breath resolved, and they were free from seizures. After a successful recovery, the patient was discharged with instructions to continue the OAT therapy.

Discussion

The discovery of hyponatremia in young adults affected by TB meningitis remains extremely rare, despite its strong association with young age and elevated infection parameters. A recent study revealed that patients diagnosed with TB and exhibiting moderate to profound hyponatremia before or at the time of diagnosis experienced a significantly higher mortality rate (Lee et al., 2022). These findings underscore the importance of early recognition and appropriate management of hyponatremia in individuals with TB meningitis, as it can significantly impact on patient outcomes (Bal et al., 2022).

Hyponatremia can arise from two factors: Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) and Cerebral Salt Wasting (CSW) (Yoshida et al., 2022). SIADH occurs when the normal secretion of AVP (antidiuretic hormone) is disrupted due to hypothalamic or pituitary gland injury or dysfunction (Wilke, 2021), while CSW is characterized by hypovolemia and negative salt balance (Liangos & Madias, 2021). The utilization of Fractional excretion of Uric Acid and the fractional excretion of phosphate has consistently proven to be effective in distinguishing between SIADH and CSW (Rudolph & Gantioque, 2018).

In this case, the patient’s condition improved after receiving hypertonic saline, which is the fundamental treatment for SIADH involving the replacement of urinary salt and water loss using 0.9% or 3% Sodium chloride. To achieve the optimal therapeutic response, patients may require doubling or tripling their adrenal steroid dose (Miller et al., 2020). Mineralocorticoid supplementation has been proven safe and effective for treating SIADH (Gurnurkar et al., 2018). Therefore, management in the patients case, includes administering a Dexamethasone injection. Once the underlying pathology is corrected, SIADH typically resolves within 3-4 weeks, eliminating the need for long-term therapy (Chaya et al., 2018; Misra et al., 2019).

In addition to treating the symptomatic manifestations, it is crucial to address the underlying disease in this patient, which is TB meningitis (Imran et al., 2020). The management of TB
involves continuing the TB treatment protocol as prescribed by the pulmonary physician. Alongside managing hyponatremia, it is essential to ensure that the patient receives appropriate and timely treatment for TB to effectively combat the infection (WHO, 2019). Following the established TB treatment guidelines set forth by the pulmonologist will aid in eradicating the TB bacteria and reduce the risk of complications associated with TB meningitis. Therefore, a comprehensive approach that includes treating the underlying TB disease and the symptomatic hyponatremia is necessary for the patient's overall recovery and well-being (Adrogué et al., 2022).

Conclusion
This case report highlights a case of hyponatremia caused by TB meningitis. Based on the patient's condition, we concluded that the hyponatremia was due to SIADH. However, future assessments can be based on the fractional excretion of phosphate for a more precise diagnosis, as it has consistently and accurately differentiated between SIADH and CSW. This additional diagnostic tool can provide valuable information to determine the underlying cause of hyponatremia in TB meningitis patients, enabling tailored treatment approaches for improved patient outcomes.

Conflict of interest
The authors declare no conflict of interest.

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Reference


126