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# Hyponatremia Unveiled: Tuberculous Effusion Masquerading as SIADH - A Rare Presentation

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**Abstract:** ***Tuberculous effusion is one of the rare causes of hyponatremia. Although other mechanisms have been described, Syndrome of Inappropriate secretion of ADH(SIADH) is usually the most common among them. We report a case of 75-year-old who had two admissions in view of altered sensorium before diagnosing tuberculous effusion causing SIADH. This report emphasizes on the uncommon presentation of a fairly common disease.***

**Keywords:** Tuberculous effusion, hyponatremia, Syndrome of Inappropriate secretion of ADH, SIADH, altered sensorium

## 1. Introduction

Tuberculosis is one of the most common diseases with high prevalence of mortality and morbidity in developing nations. With high prevalence uncommon manifestation of both, pulmonary and extrapulmonary tuberculosis, is often encountered. Hyponatremia is one such complication which occurs with tuberculosis. This report highlights the occurrence of tuberculous effusion as an unusual culprit of severe symptomatic hyponatremia.

## 2. Case Report-

A 75-year-oldpreviously healthy male presented in medical emergency with complaint of irrelevant talk and gradual deterioration in his sensorium for last 15 days. Patient also complained of decreased appetite with nausea but had not vomited. On further probing, patient complained of dry cough with right sided pleuritic type of chest pain for last 2 months which was not associated with fever, night sweats or weight loss. Past family history was insignificant.

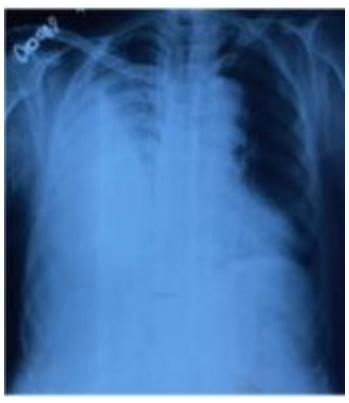
The patient had history of similar episode of confusion and irrelevant talk and loose motion 20 days back for which he was admitted in a day care centre and was managed with IV fluids and IV antibiotics. Patient was discharged the next day with diagnosis of acute gastroenteritis causing hyponatraemia. He was readmitted in our emergency department with above mentioned complaints after 5 days.

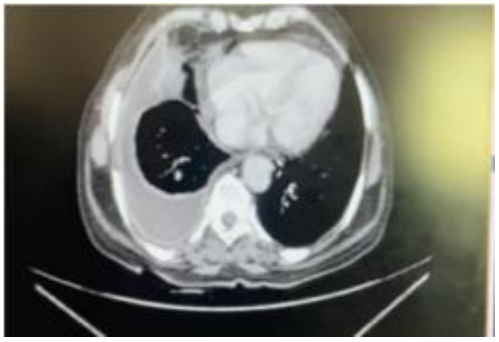
On physical examination, patient was non-edematous and hisvitals were within normal limits. Patient was conscious but confused and talking irrelevantly. Rest of the neurological examination was unremarkable. Respiratory system examination revealed stony dullness on percussion in the mammary, infra-mammary, axillary, infra-axillary, interscapular and infrascapular region on right side. Auscultation revealed decreased air entry on the same regions.

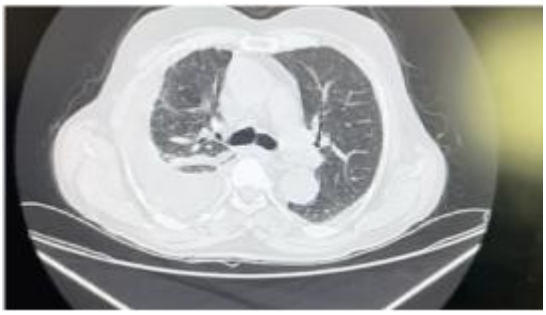
After admission, an emergent CT scan of brain without contrast was done to rule out bleed or gross neurological abnormality. Routine blood workup with serum electrolyte studies revealed a $Na^+$ of 121 mmol/L, $K^+$ of 4.3 mmol/l, $Cl^-$ of 89mmol/l, random blood glucose of 100 mg/dl, urea 43mg/dl and creatinine of 1.0 mg/dl. Complete blood count was within normal limit. Further workup for hyponatremia revealed serum osmolality of 261 mOsm/kg, urine osmolality of 323mOsm/kg, urine sodium of 63.4mmol/L. Thyroid profile and plasma cortisol levels were within normal limits. Based on these results, a diagnosis of SIADH was made. Patient was managed with hypertonic saline and salt capsules. Chest radiograph on admission showed right sided massive pleural effusion.

Over the course of hospital stay, symptoms of patient improved after improvement in sodium levels. Pleural fluid analysis showed cell count of 200/cumm with lymphocytic predominance, glucose of 20mg/dl, protein of 4.3g/dl and ADA of 99.0 U/L. chest CT with contrast was done which revealed pleural effusion with compressive collapse on right side. It did not reveal any other significant lung pathology.

Patient was discharged on day 5 of admission with Anti tubercular therapy, salt capsule and tolvaptan. Patient was followed up in outpatient department after 1 month of discharge with significant decrease in pleural effusion and no episodes of symptomatic hyponatremia in between.







## 3. Discussion

Tuberculosis (TB) is considered as one of the common illnesses in developing countries such as India which can present with various clinical manifestations. TB can induce hyponatremia via several mechanisms containing local invasion to the adrenal glands (adrenal insufficiency[1]) , local invasion to hypothalamus or pituitary gland[2], Tubercular meningitis[3] and inappropriate ADH secretion via pulmonary infection[4].Multiple studies have noted that nearly 11% of patients with active TB (pulmonary or non-pulmonary) are affected with hyponatremia, and it is apparent that the main cause of serum sodium depletion in these patients is SIADH[5].SIADH is a considerable complication of pulmonary infection, inflammatory and neoplastic disorders, although its prevalence and mechanism are poorly regarded.SIADH must be considered in every case with hyponatremia with low serum osmolality condition, a normal acid-base state, urine osmolality over 100 mOsm/kg, and urine sodium concentration more than 40 meq/L.SIADH is commonly reversible with effective PTB treatment in most cases.Few studies demonstrated that the ADH level was not detectable following full anti-TB therapy[6].Sharma et al. suggested hyponatremia as predictors of development and outcome in patients with acute respiratory distress syndrome due to tuberculosis[7].It is also recommended that patients with TB (especially older age patients) should be closely observed for electrolyte imbalance.[7]

## 4. Conclusion

Hyponatremia in tuberculosis is still underdiagnosed with missed opportunities prevalent in health facilities, and thus there is an urgent need to raise the index of suspicion at all levels of care to identify these patients.Early diagnosis and prompt treatment of is lifesaving because SIADH, cerebral salt wasting syndrome and adrenal insufficiency all require different regimens, especially in older adults.

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