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A Case of Severe COVID 19 Disease with Disseminated Tuberculosis and Prolonged Cerebral Salt Wasting Syndrome.

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Abstract

We herein report a case of a child with tuberculous meningitis with pulmonary TB(Disseminated Tuberculosis) and COVID-19 coinfection complicated by Cerebral Salt Wasting. The symptoms of TB and COVID-19 can be similar, for example cough and fever. Not only can this create diagnostic confusion, but it could worsen the stigmatization of TB patients, given the fear of COVID-19. Children with TB are a vulnerable group and hence prone to suffer collateral damage. Our patient presented to our hospital with Severe COVID 19 infection and was simultaneously diagnosed to have Disseminated TB. Concurrently, the child was found to have findings suggestive of Cerebral salt wasting which persisted for more than three weeks.

Keywords: NIL

INTRODUCTION

BACKGROUND

COVID-19 is caused by SARS-CoV-2 infection. Tuberculosis (TB) still ranks as the leading cause of death from a single infectious disease globally. COVID 19 pandemic has led to grave consequences for existing and undiagnosed TB patients, particularly in a country like India, where TB is endemic and health services are overburdened. TB patients often have underlying co-morbidities and lung damage that may make them prone to develop severe COVID-19 disease. Mortality in COVID-19 has been linked to the presence of the 'cytokine storm'.

Overproduction of proinflammatory cytokines leads to extensive tissue damage resulting in multiorgan failure and subsequent death. A major proportion of the neurological complications of TBM are also likely to be due to an exaggerated host inflammatory response. Patients with tubercular meningitis (TBM), may have higher frequency of hyponatremia compared to other CNS infections due to leptomeningeal inflammation, hydrocephalus, raised intracranial tension and ventriculitis. Cerebral salt wasting syndrome is a relatively rare cause of hyponatremia and is often confused with the syndrome of inappropriate antidiuretic hormone (SIADH). CSWS was first reported by Peters in 1950, is defined as the occurrence of an elevated diuresis and natriuresis after cerebral injury, leading to hyponatremia and hypovolemia. Meningitis may result in stress response which may cause dysregulation of hypothalamo pituitary adrenal axis.

Differentiating cerebral salt wasting syndrome from SIADH is of paramount importance as the management of both conditions is significantly different but their presenting features are similar. In our case, the CSWS persisted for more than three weeks.

CASE PRESENTATION

A 14 years old girl presented with Fever for 20 days, Headache for 15 days, projectile vomiting for 5 days, and one episode of GTCS seizure. She was admitted in post ictal drowsy state in Pediatric Ward. The preceding history revealed progressive loss of weight and decreased appetite. There was no history of either household contact with an adult TB source case or COVID-19. She had history of weight loss. All immunizations, including BCG, was up to date. She belonged to low socioeconomic class. No history of rash, ear ache or ear discharge, or any weakness or focal neurological deficit, No history suggestive of any cranial nerve involvement. Clinical examination revealed normal vital functions with Spo2 of 97% in room air. Neurological examination revealed a depressed level of consciousness, with a Glasgow Coma Scale of 11 (E- 4, V-2 and M-5), no meningeal signs, tone was normal, power could not be elicited beyond 2/5 as the girl was drowsy, pupils were bilaterally normally sensitive and reactive to light, globally brisk deep tendon reflexes and bilateral extensor plantar responses. Fundus Examination revealed papilledma.The initial respiratory examination was normal. Examination of the other systems proved unremarkable. Since she was tested positive for SARS CoV-2 by TRUNAT test at admission, hence her sputum/gastric aspirate could not be examined for Acid Fast Bacilli or CBNAAT. She was managed conservatively in COVID positive ward till her RTPCR for SARS CoV-2 report was found to be negative six days later, after which she was further managed in pediatric ward. She was started on 4 drug Anti Tuberculosis Treatment with steroids before shifting to COVID ward in view of Chest X ray findings, raised ESR and prolonged history of fever with weight loss and raised CSF protein and Adenosine Deaminase levels. She improved clinically till day 10, although she had low levels of sodium in her blood and high sodium levels in urine inspite of receiving 3% normal saline. But she again had seizures and depressed sensorium on day 10 for which she needed to be intubated and put on mechanical ventilation for next 2 days. After extubation, she was found to have Left lower zone Pneumonia on post-extubation chest X-ray, and hence was started on second line intravenous antibiotics as per the Institutional PICU protocol. The patient also had low BP readings and hence was managed as a case of Cerebral salt wasting instead of SIADH (in which patient is essentially euvolemic). She received measured doses of added salt and fluids titrated according to her output. Fludrocortisone

0.1 mg per day was then started 2 days after extubation after which the patient had normal serum and urine sodium levels within next 2 days and was discharged after documentation of three consecutive normal values of serum sodium and urine sodium each 24 hours apart. Thus, a final diagnosis of tuberculous meningitis leading to cerebral salt wasting syndrome was made and the patient is on regular follow-up and is doing well on ATT.

INVESTIGATIONS

Initial laboratory investigations showed a white cell count of 12500/mm3 (neutrophils 84%, lymphocyte 8 %), haemoglobin of 11.7 g/dL and platelets of 267×109/L. The ESR was 30 and C- reactive protein was 6.41mg/dL. CSF examination revealed raised protein levels

(301.3mg/dL), CSF sugar was 61 mg/dL with a corresponding blood sugar of 104 mg/Dl, had 10 Leucocytes/HPF and of which100% were lymphocytes, Adenosine deaminase (ADA) was 10.5U/L but did not grow any microorganisms on culture. Montoux test was reactive. HIV ELISA was non-reactive, WIDAL test was negative, Rapid Malaria Antigen test was also negative. eGFR was 138ml/min /1.73m2, Serum Sodium was 122mEq/l and Chloride 90mEq/l, Serum Calcium 8.2mg/dL. Chest X ray had miliary shadows all over the lung fields. CT head showed hydrocephalus and hence she was started on Acetazolamide. She continued to have low serum sodium values during initial 8 days of hospital stay inspite of giving 3% normal saline.

She tested Negative for COVID on day 6 of admission. Urine sodium done on day 8 of hospitalization was 142mEq/l which later worsened to 339 mEq/l on day 14. Urine routine examination was normal. On day 10, the serum sodium was 107 mEq/l, chloride was 88 mEq/l, her CSF was reexamined which was found hazy on gross appearance, and still had 10 leucocytes of which 100 % were lymphocytes, CSF proteins were still raised (165.9 mg/dl). Chest X ray was repeated which was normal but later developed ventilator associated Left Lower Zone Pneumonia after 2 days of mechanical ventilation. Urinary tract infection was found on day 13 of hospitalization. On day 23 of

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admission, she had normal serum and urine sodium levels. At discharge her Serum Sodium was 133mE/l and urine sodium were 49mE/l.

DISCUSSION

COVID-19 is predicted to have a devastating impact on other global infections, most notably tuberculosis (1). Children and adolescents are a vulnerable population that have been overlooked in the COVID-19 and tuberculosis responses worldwide (2,3). Cerebral salt wasting syndrome or renal salt wasting is seen after a few days of brain injury, in patients with a normal thyroid and adrenal gland function, having a defective kidney sodium transport mechanism that leads to a decreased extracellular volume (4). It is supposed to be one of the major causes of hyponatremia amongst the neurosurgical cases (5). Our patient already had hyponatremia at admission which signifies that the brain injury probably was a few days old. CSW is similar in presentation to SIADH, but the effective arterial blood volume is decreased in cerebral salt wasting syndrome while increased in SIADH (6). Our patient had hypotension at presentation along with signs of raised intracranial tension. Since the treatment of both conditions is different, an accurate diagnosis is necessary (6). The main diagnostic features of cerebral salt wasting syndrome are a brain lesion and a loss of sodium and chloride by the kidneys without having any stimuli for it (7). Even though its cause is still not known, researchers have concluded that low sodium in patients with brain disease might be due to cerebral salt wasting syndrome (8). Bettinelli et al studied 110 patients with brain disorders having cerebral salt wasting syndrome and concluded that one of the main underlying cause was found to be TB meningoencephalitis, similar to our case (9). Treatment of cerebral salt wasting syndrome includes fluid along with sodium replacement, which is done via hypertonic saline (8) Hedge treated his case of cerebral salt wasting syndrome with hypertonic 3% saline which not only improved the patient's consciousness but also his sodium levels (5). In our case, the patient responded to Oral Fludrocortisone which was given along with 3% Normal saline and salt supplementation.

CONCLUSION

This case illustrates that patient with Tubercular Meningitis with co- infection with COVID 19 could have a prolonged Cerebral Salt Wasting Syndrome, although Tuberculosis did not worsen the COVID 19 disease in this patient. Also, patients with low serum sodium and high urine sodium with some intracranial pathologies are likely to be suffering from cerebral salt wasting syndrome instead of SIADH, as a wrong diagnosis could lead to an inappropriate treatment and might increase the morbidity of patients.

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