

Case Report


Cerebral sinus venous thrombosis in hypothyroidism, hyponatremia and hypertension - A case report

Nagjere Shirisha^{1*}, E.A. Ashok Kumar²

¹Post Graduate, ²Professor

Department of Medicine, MNR Medical College and Hospital, Fasalwadi, Sanga Reddy, Telangana, India

*Corresponding author email: sirilavender@gmail.com

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Abstract

45-years-old male, hypertensive presented to the emergency in a state of unconsciousness after an attack of GTCS. Investigations revealed hypothyroidism, hyponatremia and Cerebral Sinus Venous Thrombosis (CSVT) on CT scan and CT Venogram. Other routine investigations were normal. This rare presentation of CSVT with hypothyroidism and hyponatremia was corrected with Eltroxin alone.

Key words

Cerebral sinus venous thrombosis, Hypothyroidism, Hyponatremia, Hypertension.

Introduction

Hypothyroidism is widely accepted as a cause of hyponatremia. Hypothyroidism predisposes to both euvoletic hyponatremia and hypercoagulability, former due to decreased cardiac output and diminished free water restriction and the latter through increase in factor VIIa levels. Hyponatremia is often asymptomatic when mild to moderate in severity and subacute to chronic in its time course of development. However, significant hyponatremia

(<120 mmol/L) of rapid onset is frequently symptomatic and can be life-threatening.

Cerebral sinus venous thrombosis (CSVT) should be considered in the differential diagnosis of all unexplained CNS disorders of sudden onset. Each component of the Virchow's triad (endothelial damage, stasis and hypercoagulability of blood) may in turn have several contributory factors/ causes to produce the final manifestation of CSVT. Studies have

suggested hypercoagulable state in hypothyroidism [1]. CSVT can present as sudden onset of headache, seizure or neurologic deficit which is difficult to be attributed to any one vascular territory, or unexplained vascular headaches, which is persistently unilateral or sometimes diffuse. Suspected cases should be thoroughly examined and investigated with CT or MR Venogram to confirm the diagnosis.

Case report

A 45 years old male patient presented on 18/10/15 with complaints of irrelevant talk of 6 hours duration. Sudden in onset, progressed to drowsiness gradually, with one episode of generalized seizure. There was no history of nausea, vomiting, headache, blurring of vision, fever. No history of weakness of any of the limbs. No history of cranial nerve and bladder or bowel involvement. No history of breathlessness, chest pain or palpitations. Patient had history of trauma on 1/10/15; he had fracture of lower third of right fibula, for which he was operated upon. Open reduction and internal fixation with semi tubular plate was done on 5/10/15 under spinal anesthesia (bupivacaine 0.5% and fentanyl) and was discharged on 8/10/15. He was a known hypertensive of 10 years duration on treatment with Telmisartan 40 mg/daily, Metoprolol 25 mg/daily. He was not a known case of diabetes, asthma, tuberculosis or epilepsy. He was a chronic smoker, not an alcoholic. There were no known drug allergies.

On examination, patient was drowsy, heavy built and well nourished. His pulse rate - 75/min, regular, B.P - 130/80 mm Hg, JVP – not raised, non pitting edema present, skin was dry, there was no hair found on all the 4 limbs. No pallor, icterus, cyanosis, clubbing were present. Cardiovascular system – ejection systolic murmur was present of grade II intensity in aortic area. Respiratory system exam was Normal. Gastrointestinal system exam was normal. Central nervous system examination – patient was drowsy, hoarseness of voice was present, cranial nerves were normal. Motor

system- revealed normal tone, power 4/5 in all the four limbs, DTR absent in all the limbs, plantars were not elicitable. Sensory system was normal, and there were no signs and symptoms of raised ICT and cerebellar involvement. ENT examination was normal. External Nose, anterior nasal cavity, posterior rhinoscopy was normal. Oral cavity, oropharynx, indirect laryngoscopy was normal

On investigations

Complete Blood Picture: Hb – 8.7 gms/dl, RBC - 4.9 million/mm³, WBC - 11,200/mm³, DC – Neutrophils - 62%, Eosinophils - 2%, lymphocytes - 34%, Monocytes - 2% and Basophils - 0%, Platelets - 2.4 lakhs/mm³, Complete urine examination – Normal, ESR - 30 mm 1st hour, RBS - 107 mg/dl, Blood Urea - 21 mg/dl, Serum creatinine - 1.2 mg/dl, LFT – Normal, Serum electrolytes Na – 96 mMol/L, K - 2.5 mmol/L, Cl - 90 mMol/L. Thyroid function test - T.S.H – 165 uIU/ml, FT3 – 0.70 pg/ml, FT4 - 0.15 ng/dl. TPO antibodies 40 IU/ml, TgAb- 30 IU/ MI. Bleeding time 3min, clotting time 4 min and prothrombin time was normal, INR 1.2. Serum homocysteine - 5.6 µmol/l (normal 2.2 - 13.2 µmol/l), ANA < 1:160, lupus anticoagulant – 30 sec (N-28-40 sec), Anti cardiolipin antibodies IgM – 10.5 mpl and IgG- 10 mpl, IgA - <10 apl. USG of thyroid was normal. FNAC of thyroid revealed normal thyroid structure. Serum osmolality – 201 mosm/L. ECG showed T wave inversions in inferolateral leads (**Figure - 1**). 2D Echo – EF was 69%, Valves were normal, Asymmetric Septal Hypertrophy, Good LV/RV Contraction, Decreased diastolic compliance, and there was no pericardial effusion. USG of abdomen – Right kidney - 9.3x4.2 cm, Left kidney - 9.5x 4.2 cm. Chest radiograph was normal. CT brain – showed empty delta sign (**Figure - 2**), suggesting CSVT. As CT Brain showed empty delta sign, suspecting CSVT, CT Venogram was ordered (MR Venogram contraindicated because of metal plates) CT venogram showed filling defect in Right lateral sinus region (**Figure - 3**). CT venogram showed thrombosis of Right lateral sinus (**Figure - 4, 5**).

Figure – 1: ECG showing T wave inversions in inferolateral leads.

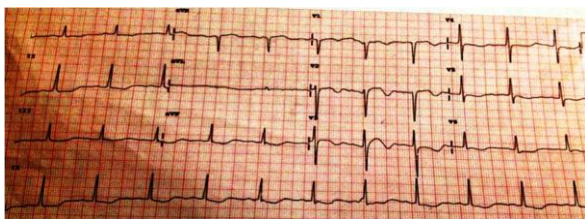


Figure - 2: CT brain – showing empty delta sign.

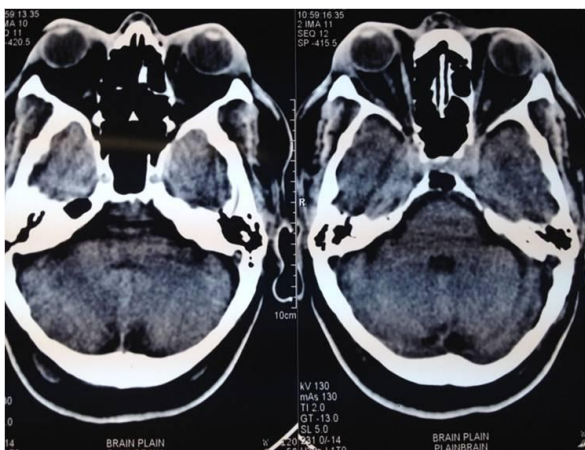


Figure - 3: CT venogram showing filling defect in right lateral sinus region.

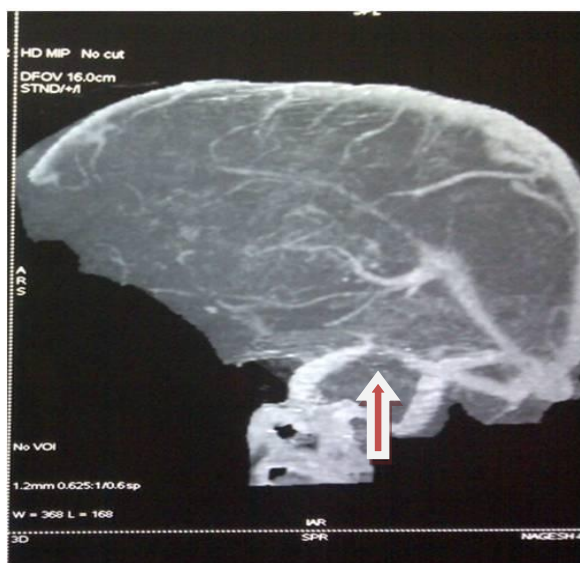


Based on history, clinical examination and investigations patient was diagnosed as a case of Severe hypothyroidism, hyponatremia, CSVT, hypertension.

Figure – 4: CT venogram showing thrombosis of right lateral sinus.



Figure – 5: CT venogram showing right lateral sinus thrombosis.



The patient was kept on Tab Eltroxin 100 mcg OD, IV Antibiotics, Tab Nifedipine 10 mg TID.

Day 2: 19/10/15: Na – 98 mMol / L, K - 2.7 mMol/L, Cl - 63 mMol/L

Day 3: 20/10/15: Na – 103 mMol / L, K - 3.2 mMol/L, Cl - 84 mMol/L

Day 4: 21/10/15: Na – 107 mMol / L, K - 3.8 mMol / L, Cl - 86 mMol/L

Day 5: 22/10/15: Na – 113mMol / L, K - 3.7 mMol / L, Cl - 94 mMol/L

Hyponatremia gradually returned to normal with Eltroxin alone.

On the day of discharge

Patient was well oriented, Hoarseness of voice improved, Deep tendon reflexes returned to normal, serum sodium was 125 mMol/L.

Discussion

Euvolemic hyponatremia, the most commonly encountered dysnatremia in hospitalized patients, is also encountered in patients with hypothyroidism. The principal abnormality in hyponatremia caused by hypothyroidism with normal fluid intake appears to be the inability to maximally suppress antidiuretic hormone. This is most likely due to reduced cardiac output in this disorder, which can lead to the release of antidiuretic hormone via the carotid sinus baroreceptors [2]. The glomerular filtration is also decreased in hypothyroidism [3]. This can directly diminish free water excretion by diminishing water delivery to the diluting segments [4]. Decreased water delivery may be particularly important in those cases in which hyponatremia develops, despite appropriate suppression of ADH secretion. Regardless of the mechanism, the net effect of the impairment in water excretion is the retention of ingested water and a reduction in the plasma sodium concentration by dilution. It should be treated with thyroid supplements only and there is no need to replace with hypertonic saline, as this is dilutional hyponatremia and total body sodium is normal.

The results of Koide, et al. [5] indicate that neither vasopressin nor aldosterone plays a dominant role in the pathogenesis of the hyponatremia in patients with hypothyroidism. It appears that thyroid-hormone deficiency itself causes the derangement of tubular-cell function, which results in the development of the impaired water excretion and hyponatremia.

Hypothyroidism favors a procoagulant by decreasing fibrinolysis (high levels of alfa2-

antiplasmin and plasminogen activator inhibitor-1) [6], inducing hyper homocysteinemia, and high C-reactive protein (CRP). Decreased fibrinolytic capacity, high CRP levels, and coagulation factors abnormalities can occur even in subclinical hypothyroidism [1].

Increased levels of plasma thrombin-activatable fibrinolysis inhibitor levels were observed in patients with mild and overt hypothyroidism, and levothyroxine treatment was effective in reducing these levels [7].

Hypothyroidism also contributes to endothelial injury and slow venous flow. Endothelial dysfunction was found in the microvasculature of patients with overt and subclinical hypothyroidism [8]. Chronic low-grade inflammation and impaired nitric oxide availability in the endothelium have been demonstrated in hypothyroidism.

Patients with sub clinical hypothyroidism had significantly higher level of Factor VII: C. The greater increase in Factor VII: C compared to that of Factor VII: Ag as shown by the increase in their ratio, might reflect the presence of activated Factor VIIa. This might mean a hypercoagulable state which could contribute to the increased prevalence of venous thrombosis [9]. A hypercoagulable state might be another argument in favor of thyroxine replacement treatment in subclinical hypothyroidism especially in patients with additional risk factors for vascular disease. Antithrombotic prophylaxis in patients with severe hypothyroidism, however, should be viewed with caution because of a possible hyper fibrinolytic state in such patients [10].

Hence, treatment with thyroxine treats hyponatremia and also reverses hypercoagulable state.

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