Brief Communication

Cerebral venous sinus thrombosis with autoimmune thyroiditis

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ABSTRACT

Cerebral Venous Thrombosis (CVT) is a multifactorial condition which is described as idiopathic in 12.5% of patients. Hyperthyroidism has been associated with CVT in many case reports, and increased levels of factor VIII and von Willebrand factor (vWF) have been proposed as the possible link in this association, but only few rare case reports have described an association of hypothyroidism with CVT. We report here a case of autoimmune thyroiditis presenting with CVT.

Key words: Cerebral vein thrombosis, Hashimoto's thyroiditis, hypothyroidism

Introduction

Cerebral venous thrombosis (CVT) is idiopathic in around 12.5% of patients.[1] Studies have described links between thyroid disorders and thrombophilia. High concentrations of factor VIII and von Willebrand factor (vWF) contribute to a hypercoagulable state in hyperthyroidism. Lower vWF concentrations in hypothyroidism may protect against venous thrombosis.[2] . Studies have suggested hypercoagulable state in hypothyroidism.[3] Rare case reports have described association of CVT with autoimmune hypothyroidism.[4] We report here a case of Hashimoto thyroiditis with hypothyroidism who presented with CVT.

Case Presentation

45-year-old female presented in emergency with complaints of headache since 1 month, vomiting since 1 day, and weakness with numbness and tingling in right half of body since 12 h. Her weakness progressed to quadriparesis overnight. She had no history of fever, head injury, seizures. No history of oral contraceptive use and prolonged immobilization. Her menstrual and obstetric history was unremarkable. On examination she was drowsy and disoriented. On neurological examination, reflexes were present and muscle tone was normal but power was 1/5 on right side and 3/5 in left half of body. Fundoscopy showed papilledema.

Normal hemogram and renal and liver function tests. Thyroid function tests were suggestive of subclinical hypothroidism T3 - 0.94 ng/ml, T4 - 8.36 μg/dl, TSH - 13.92 mIU/ml. TPO antibodies were elevated (>1,300 IU/ml) . Thrombophilia profile including prothrombin time index/international normalized ratio , serum homocysteine (5.6 mol/l), ANA, lupus anticoagulant, IgM and IgG cardiolipin antibodies, and APLA antibodies were negative. USG of thyroid showed bilateral bulky lobes with altered echotexture s/o thyroiditis. FNAC of thyroid nodule showed lymphocytic infiltration consistent with a diagnosis of Hashimoto thyroiditis. CT head showed hyperdensity in the region of superior sagittal sinus. MRI brain showed loss of flow void of superior sagittal and left transverse sinuses with altered signal appearing hyperintense on T1W and fluid-attenuated inversion recovery (FLAIR) images suggesting thrombosis.

She was put on heparin and warfarin. Her headache

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improved after 4 days of treatment and patient was discharged after 7 days with no residual neurological deficit.

Discussion

Both hyperthyroidism and hypothyroidism have been associated with CVT .

Hypothyroidism favors a procoagulant by decreasing fibrinolysis (high levels of alfa2-antiplasmin and plasminogen activator inhibitor-1),[5] inducing hyperhomocysteinemia,[6] and high C-reactive protein (CRP).[7] Decreased fibrinolytic capacity,[8] high CRP levels,[9] and coagulation factors abnormalities can occur even in subclinical hypothyroidism.[3]

Elevated plasma thrombin-activatable fibrinolysis inhibitor levels were observed in patients with mild and overt hypothyroidism, and levothyroxine treatment was effective in reducing these levels.[10]

Hypothyroidism also contributes to endothelial injury and slow venous flow.

Endothelial dysfunction was found in the microvasculature of patients with overt and subclinical hypothyroidism.[9] Chronic low-grade inflammation and impaired nitric oxide availability in the endothelium have been demonstrated in Hypothyroidsm.[11]

There is also evidence of increased prevalence of antiendothelial cell antibodies in hypothyroidism.[12]

This case emphasizes the need for thyroid evaluation in all patients with CVT and other venous thromboembolic event even in the absence of clinical signs of hypothyroidism.

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