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Massive Cerebral Venous Sinus Thrombosis as an Unexpected Complication of Severe Hyperthyroidism

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Abstract

Cerebral venous sinus thrombosis (CVST) is a rare but potentially fatal neurological condition frequently associated with prothrombotic states. Identifying the underlying cause of CVST is crucial but can be challenging, especially when it is reported as uncommon. Here, we present a case of a previously healthy 31-year-old Malay man who presented with massive CVST complicated with status epilepticus and arrhythmia in the setting of undiagnosed severe hyperthyroidism. The patient subsequently improved with intensive care therapy, thyroid storm protocol treatment, and antiplatelet medications. This case highlights the need to rule in hyperthyroidism in patients with atypical neurological symptoms, particularly CVST, even in the absence of associated risk factors.

Keywords
hyperthyroidism, thyrotoxicosis, cerebral vein thrombosis, thyroid crisis

Introduction

Cerebral venous sinus thrombosis (CVST) is a rare but potentially fatal form of stroke that can present with a multitude of clinical features. The most common implicated risk factors for CVST are prothrombotic conditions, which can be hereditary, such as haemophilia, or acquired, such as pregnancy, puerperium and hormonal replacement therapy, which are all considered more specific in females.[1] Hyperthyroidism has been described as a rare risk factor for CVST, accounting for less than 2% of all cases, without a precise understanding of the pathophysiological basis.[1] This article aims to describe a case of a young gentleman who presented with massive CVST complicated by status epilepticus in the setting of undiagnosed severe hyperthyroidism, and to review existing literature on the association between the two conditions.

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Case Report

A 31-year-old Malay man with no known medical illness presented to the Emergency Department (ED) with generalized body weakness and high-grade fever in the last two days, associated with headache, myalgia, arthralgia, and incoherent speech. He denied any substance abuse, traditional medicine usage or supplements intake and had no history of head trauma. Further history was taken with irrelevant findings. There was no recent history of palpitations, increased hunger, or weight loss. There is no anxiety, nervousness, and irritability. He also denied having any tremors, irregular bowel movements, fatigue, or increase sweating prior to this recent presentation.

Upon arrival at the ED, he had neurological deficits in the forms of hypotonia, hyporeflexia, and hemiparesis of the left side of his body, which were suggestive of a lower motor neuron lesion. Of note, there was no neck stiffness, and Kernig's and Brudzinski's signs were negative. In addition, he developed fast AF in the ED, with a heart rate of 180 beats per minute, for which he was given a loading dose followed by a maintenance dose of intravenous (IV) amiodarone. He also had a hypoglycaemic event with a blood glucose of 3.3 mmol/L, which quickly reverted with 50 ml of IV dextrose 50%.

Because of the neurological deficits, a Computed Tomography (CT) scan of the brain was performed. The CT scan showed presence of extensive thrombi within the superior sagittal, inferior sagittal, straight, right transverse, and right sigmoid sinuses (Figure 1). This was consistent with the radiological diagnosis of CVST. He was then admitted to the medical ward for further observation and was diagnosed with meningitis with fast AF.

Unfortunately, in the ward, he developed generalized tonic-clonic seizures for four episodes, which were aborted with IV phenytoin. After the ictal phase of a seizure, he remained drowsy, failed to regain consciousness, and had worsening of the AF up to 200 beats per minute. Despite the administration of IV amiodarone, his heart rate remained high at 180-200 beats per minute. He was then intubated for airway protection because of a low Glasgow Coma Scale and haemodynamic instability. Post-intubation, he had hypotension that required three times of synchronized cardioversion of 50 kJ and subsequently two times of 120 kJ. Therefore, he was admitted to the intensive care unit (ICU) from the medical ward following these episodes of status epilepticus and unstable atrial fibrillation (AF).

He was immediately arranged for ICU admission for systemic organs support and close monitoring. A full infective screening was sent, and a lumbar puncture (LP) was performed. The LP showed an opening pressure of 10 cmH_2O and a closing pressure of 8 cmH2O, while the cerebrospinal fluid (CSF) appeared clear and colourless. The CSF analysis results came back as unremarkable, which ruled out meningitis. However, empirical IV meropenem and acyclovir were administered and continued to cover for the possibility of infection.

In the ICU, a thyroid function test (TFT) sent earlier showed evidence of severe primary hyperthyroidism with suppressed TSH of <0.005 mIU/L, elevated T4 of >100 pmol/L, high anti-thyroid peroxidase and normal anti-thyroglobulin level. The calculated Burch-Wartofsky Point Scale was 90 points, which was highly suggestive of thyroid storm. Considering this possibility, the following medications were started: oral Lugol's iodine 10 drops three times daily (TDS), oral propylthiouracil 200 mg four times daily, oral propranolol 40 mg TDS, and IV hydrocortisone 200 mg once daily (OD). In addition, despite mild thrombocytopenia, dual antiplatelet therapy was started with oral aspirin 100 mg OD and oral clopidogrel 75 mg OD to treat the CVST

He was ventilated for one week in the ICU and was extubated well before being transferred to the medical ward. The seizures and AF eventually ceased while the TFT significantly showed improvement with TSH of

<0.005 mIU/L and reducing T4 of 22.85 pmol/L with the initiation of the thyroid storm protocol treatment. He was discharged well without any neurological deficits after five days in the ward, with oral carbimazole 20 mg OD, oral propranolol 60 mg TDS, and oral levetiracetam 500 mg twice daily. He was given a one-month follow-up appointment at the endocrine clinic.

Conclusion

In conclusion, our case reinforces the previous evidence that indicates a possible association between CVST and hyperthyroidism. However, this association and its exact physiological basis require ascertainment in a larger prospective study. Given the association demonstrated in our case and published data, we strongly suggest ruling in hyperthyroidism by default in patients who present with CVST. Early diagnosis of hyperthyroidism is crucial but can simply be established to initiate the appropriate therapy, which can potentially improve the clinical outcome of the patients.

Conflict of Interest Statement

The authors of this manuscript certify that there is no conflict of interest nor any financial interest in the subject matter or materials discussed in this manuscript.

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