Thyroid Steal Syndrome Secondary to Active Hyperthyroid State

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Background

- Most transient ischemic attacks (TIAs) are the result of blockage or narrowing within the intracranial blood vessels.
- However, deviation of blood from the cerebral circulation due to pathology involving extracranial blood vessels can also result in TIAs.
- Thyroid steal syndrome (TSS) is a condition characterized by episodes of transient cerebral ischemia that occur secondary to the “steal,” or diversion, of blood from the brain to the thyroid due to a goiter and, less commonly, thyroid hormone derangements (1).
- Patients with this condition commonly present with a history of multiple TIAs and are found on angiography to have enlargement of the thyroid arteries in the setting of a voluminous goiter or thyroid hormone derangement (1-3).

Case Presentation

- 45-year-old male with a past medical history of insulin-dependent diabetes mellitus and peripheral artery disease presented to the emergency department with complaints of two episodes of left hemiparesthesia and hemiplegia. The patient reported that the first episode lasted 30-40 minutes and the second lasted two hours.
- The patient was found to be tachycardic with a heart rate of 129 beats per minute and hypertensive with a blood pressure of 139/77 mmHg. There were no focal neurological deficits on the initial physical exam.
- Labs showed thyroid-stimulating hormone (TSH) of <0.015 mIU/L (normal range: 0.465-4.68 mIU/L), free T4 of 4.52 ng/dL (normal range: 0.78-2.19 ng/dL), thyrotropin receptor antibody of 4.64 IU/L (normal range: 0.00-1.75 IU/L), and thyroid microsomal antibody of 299 IU/mL (normal range: 0-34 IU/mL).
- Computed tomography angiography (CTA) of the neck reported greater than 70% stenosis of the right clinoid internal carotid artery (ICA), up to 50% stenosis of the left petrous, cavernous, and clinoid ICA, and greater than 50% stenosis of the intradural right vertebral artery (Figure 1).
- Cerebral digital subtraction angiography (DSA) demonstrated mild atherosclerotic changes but ruled out significant flow limiting extra or intracranial stenosis (Figure 2).
- However, the study revealed prominent bilateral thyrocervical trunks with a prominent vascular blush of the thyroid gland (Figures 3-4).
- These findings paired with laboratory findings indicating hyperthyroidism led to the consideration of the diagnosis of TSS.
- The patient was initiated on methimazole and propranolol for treatment of hyperthyroidism.

Case Presentation cont’d

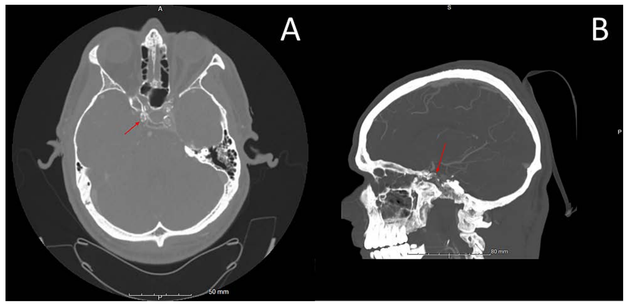

Figure 1. CTA of the head (A) axial cut and (B) sagittal cut demonstrating atherosclerotic changes in the right clinoid ICA (red arrow) resulting in approximately 70% stenosis per Warfarin-Aspirin Symptomatic Intracranial Disease criteria

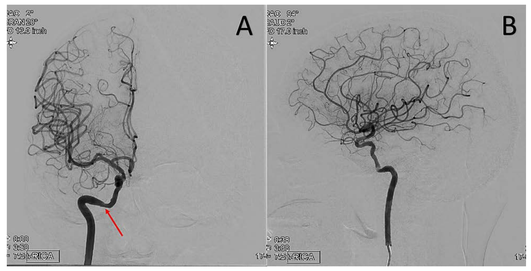

Figure 2. Right ICA angiogram, (A) AP view and (B) lateral view demonstrating atherosclerotic changes at the right lacerum ICA (red arrow) with no hemodynamically significant flow limiting stenosis in the clinoid segment.

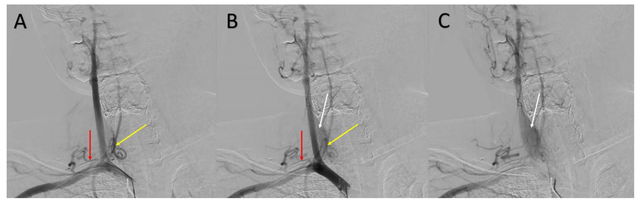

Figure 3: Brachiocephalic artery angiogram progressing from A to C, AP view, demonstrating prominent right thyrocervical trunk (red arrow), right inferior thyroid artery (yellow arrow) and thyroid blush (white arrow).

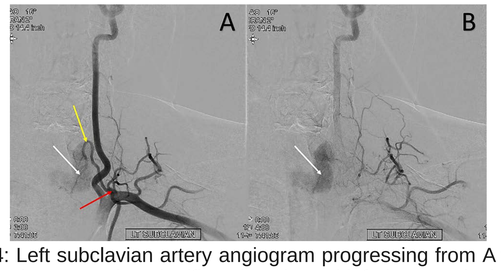

Figure 4: Left subclavian artery angiogram progressing from A to B, AP view, demonstrating significantly enlarged left thyrocervical trunk (red arrow), left inferior thyroid artery (yellow arrow) and thyroid blush (white arrow).

Discussion

- TSS was first described in 1982 in a patient who presented with TIA after treatment of hyperthyroidism rendered the patient hypothyroid. The patient was found to have significant dilation of the arteries supplying the thyroid on cerebral angiogram.
- After cessation of antithyroid medications, the patient was rendered euthyroid and the symptoms of TIA ceased (1). In this case, it was theorized that the increased thyroid blood flow was stimulated by the high levels of TSH (>100 mIU/ml) (1,4).
- The other three documented cases of TSS were all in patients who presented with recurrent TIAs and were found to have a voluminous thyroid goiter (2,3). In all three of these cases, the arteries supplying the thyroid were found to be enlarged by cerebral angiogram and surgical removal of the thyroid resulted in long-term prevention of symptom recurrence (2,3).
- Contrary to prior reported cases of TSS, in our case, the increased thyroid blood flow and vascularity that resulted in the steal phenomenon did not occur due to a high TSH level or a thyroid goiter, as our patient had neither.
- The patient’s high levels of thyrotropin receptor antibody likely explain the increased thyroid vascularity and blood flow, as these antibodies are able to mimic the effect of TSH (5).

Conclusion

- TSS should be considered in the context of any patient presenting with transient neurological symptoms who are found to have a large thyroid goiter on exam or laboratory evidence of thyroid hormone derangement.
- It is important to consider TSS as the possible etiology in patients with recurrent TIAs even after CTA demonstration of carotid stenosis, as CTA can misestimate carotid stenosis especially when the reported stenosis is 50-70% (6).
- Correctly making the diagnosis of TSS is crucial, as the condition is treated by addressing the underlying cause. This means surgical intervention in the case of a thyroid goiter and medical management in the case of thyroid derangements (1-3).

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