

Ischemic stroke: a rare complication of a large multinodular goiter

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Summary

Mass effect from a goiter is a serious complication with potentially life-threatening consequences. In rare instances, a goiter can compress nearby vessels, compromising cerebral blood flow, which can lead to an ischemic stroke. Ischemic strokes generally occur due to atherogenic or embolic phenomenon, albeit a rare etiology can be due to a mechanical obstruction of great vessels of the neck that provide blood supply to the brain. An unusual example of a similar obstruction is the mass effect of an expansive goiter on the carotid artery (CA) in the neck. We present a rare case of a 90-year-old female who had a historically untreated goiter for 13 years. She presented with symptoms of acute stroke, including right-sided weakness and dysarthria. CT angiogram of the neck revealed a massively enlarged thyroid gland causing compression and intermittent obstruction of the blood flow in the left common CA. Subsequently, the patient underwent a total thyroidectomy. Postoperatively, she had a remarkable recovery of her symptoms of right-sided weakness and dysarthria. Acknowledging stroke as a grave mechanical complication of a large multinodular goiter is crucial for timely and appropriate management to avoid serious consequences.

Learning points:

- The natural history of euthyroid multinodular goiters include progressive, abnormal goiter which occurs when it obstructs the carotid artery (CA) and compromises cerebral blood flow.
- Timely diagnosis and surgical management of an enlarging goiter compressing the CA can reduce morbidity from an ischemic stroke.
- Ischemic stroke is a rare and dangerous complication of a giant multinodular goiter.

Background

Hyperplasia of the thyroid gland, referred to as a goiter, is a common endocrine disorder. Goiters can sometimes grow into surrounding structures, causing respiratory compromise (1), esophageal compression (2), vocal cord paralysis, or vascular compromise. The indirect association of acute cerebral ischemia and thyroid disorders has been well described in the literature, with cases of hyperthyroidism-induced atrial fibrillation resulting in cardioembolic stroke (3). However, there is a

scarcity of literature describing an enlarged multinodular goiter causing stroke by mechanical obstruction of the vascular flow to the brain. We present a unique case, which demonstrates a giant goiter causing partial compression of the left common carotid artery (CA) leading to a debilitating left frontal lobe infarct presenting as stroke. An urgent thyroidectomy, which relieved the obstruction on the common CA, lead to an impressive recovery of symptoms in our patient.

Case presentation

A 90-year-old Ecuadorian female with a past medical history of multinodular goiter and bilateral hearing loss presented with symptoms concerning acute stroke. On the morning of the presentation, the family initially noticed the patient to be confused and drooling from the mouth with right upper and lower extremity weakness. The new onset of neurological symptoms prompted the family to bring her to the hospital. However, she was outside the tissue plasminogen activator window. Further history revealed that the patient had a thyroid mass that was slowly growing over 13 years. The thyroid mass was biopsied 8 years ago in Ecuador and was told to be benign. Though the patient had complaints of intermittent dysphagia due to the enlarging goiter, she denied any choking sensation, dysphonia, or dyspnea. She did not have any further workup or intervention for the thyroid mass. Review of symptoms was negative for cold or heat intolerance, constipation, diarrhea, tremors, palpitations, or weight changes. On admission, the patient's vital signs were within normal limits, and she was saturating 98% on room air. Physical examination revealed a well-appearing elderly woman with no acute distress. She was oriented to name and was following only simple commands. A complete neurological exam was not performed due to the patient's inability to follow commands in the setting of her hearing loss. She had mild dysarthria without facial droop and right upper and lower extremity weakness. Neck examination revealed an asymmetrically enlarged soft, non-tender, nodular thyroid gland approximately three times the normal size. Pemberton's sign could not be assessed due to right arm weakness. The remainder of the physical exam was unremarkable.

Investigation

A contrast-enhanced CT scan of the neck and soft tissues was performed. The scan demonstrates massive, multinodular enlargement of the thyroid gland which enhanced heterogeneously. Innumerable internal calcifications and enhancing discrete nodules are seen in the mass. The constellations of these imaging findings were compatible with a goiter. The mass resulted in right lateral deviation of the trachea and esophagus. Additionally, the bilateral common carotid arteries were displaced laterally (Fig. 1).

An MRI scan of the brain was performed to assess for acute infarction. MRI of the brain demonstrates wedge-shaped diffusion-weighted hyperintensity in the left frontal lobe involving the left cortex and subcortical white



Figure 1

Contrast-enhanced CT of the neck soft tissues: coronal (A) and axial (B) soft tissue windows of the neck demonstrate a large, heterogenous multinodular mass (asterisk) within the neck soft tissues causing displacement of the trachea (straight arrow) and adjacent vasculature (curved arrows).

matter as well as the pre-central gyrus (Fig. 2). Expected chronic microangiopathic changes were identified. The large regions of restricted diffusion, combined with no evidence of vascular disease on CT Angiogram (CTA), suggest a non-embolic process, more compatible with acute chronic infarction. Embolic workup of the patient proved no likely embolic source.

Due to the presence of acute infarction, a CTA of the head and neck was obtained. The CTA demonstrates mild to moderate luminal narrowing of the proximal left common carotid artery without complete obstruction (Fig. 3). Distal dolichoectasia of the vessel suggests a chronic process, possibly due to repeated positional extrinsic compression and stenosis. There is displacement of the aerodigestive tract without stenosis. While the vessel is opacified with contrast during the examination, mechanical compression of the vessel with flexion or rotation of the neck possibly leads to obstruction of flow and decreased parenchymal perfusion, especially to the brain.

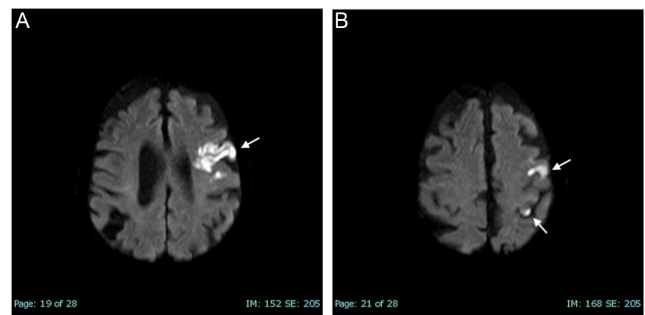


Figure 2

Non-contrast MRI of the brain: multiple axial diffusion-weighted images of the brain (A and B) wedge-shaped regions of restricted diffusion in the left frontal lobe and precentral gyrus, consistent with acute infarction (arrows).

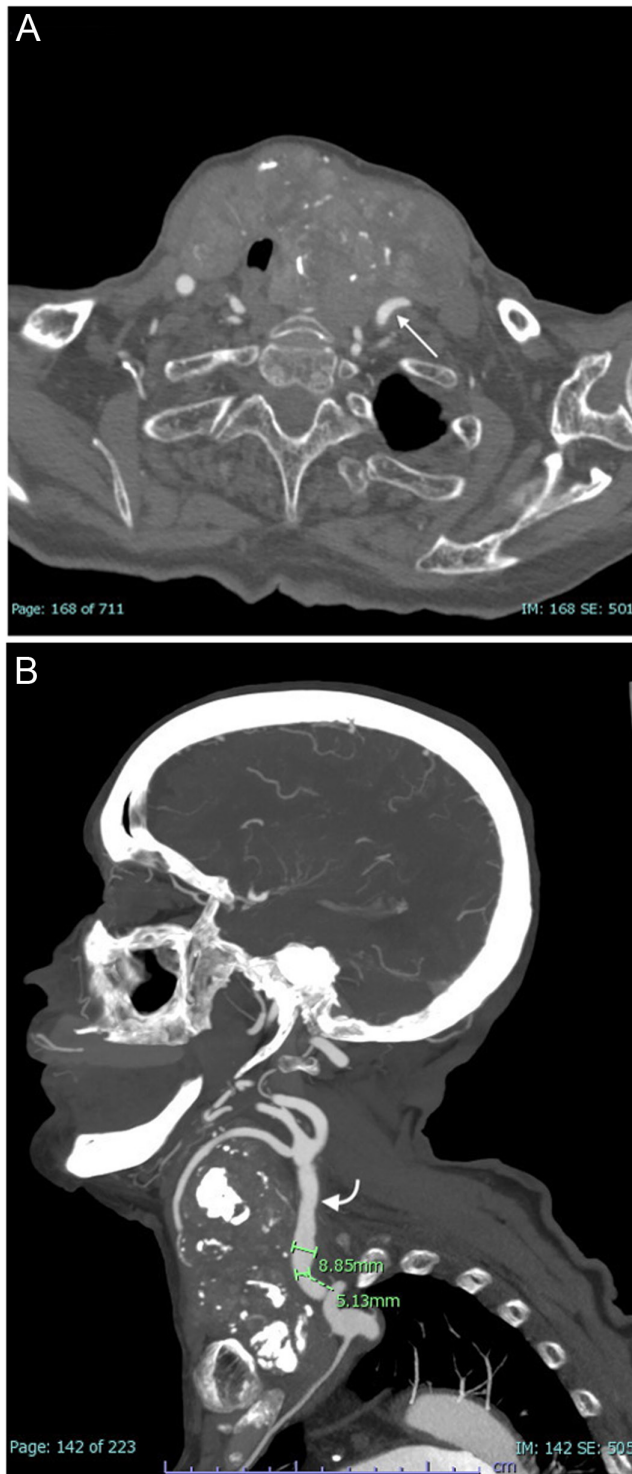


Figure 3
CT angiogram of the neck: windowed axial (A) view with stenosis of the left common carotid artery (arrow). Sagittal (B) image demonstrates approximately 40–50% stenosis of the proximal left common carotid artery (calipers). Note the dolichoectasia of the distal common carotid artery without obstruction of flow distally (arrow).

Workup was performed to evaluate the other possible causes of stroke, such as cardiac thrombus and atrial fibrillation, using an ECG, telemonitoring, Holter monitoring, and transthoracic echocardiography, which were unremarkable. EEG was negative for epileptiform abnormalities. Ultrasound duplex of upper extremities was negative for deep venous thrombus.

Thyroid hormones revealed a thyroid-stimulating hormone level of 0.045 mIU/L (normal range 0.350–4.70), normal levels of free thyroxine 1.2 ng/dL (normal range 0.7–1.9), and normal levels of total triiodothyronine 102 ng/dL (normal range 79–149).

Treatment

It was assumed that compromised blood flow of the compressed left common CA by the massive goiter with flexion or rotation of the neck was contributing to her otherwise unexplained ischemic stroke. Additionally, due to the history of chronic dysphagia the patient underwent a total thyroidectomy within 24 h of admission. During the procedure, the left thyroid lobe was found to be significantly larger than the right lobe measuring 15 cm in superior to inferior dimension. It was nodular and hypertrophic, compressing the lateral aspect of the trachea and left common CA. The thyroid gland was carefully removed, and pressure was released from the common CA. The gross pathology showed a distorted thyroid gland weighing 43 g and measuring 13.5 × 12 × 4.5 cm (Fig. 4). It had a diffuse heterogeneous nodular appearance consistent with multinodular goiter, negative for malignancy. Patient had an uneventful postoperative course and her symptoms of stroke resolved. Patient was eventually discharged on 50 µg of levothyroxine daily.

Outcome and follow-up

Two months post-surgery, the patient was seen in the family medicine clinic. Her physical examination revealed full range of motion in all extremities without dysarthria. Thyroid function tests were within normal limits on levothyroxine 50 µg.

Discussion

Iodine deficiency, especially in endemic areas such as Africa and Latin America, is the most common cause of goiterogenesis (4). Although iodine deficiency is uncommon in the United States, it still affects up to one-third of the global population (4). Since our patient was from



Figure 4

The gross pathology showed a distorted thyroid gland weighing 43 g and measuring 13.5 × 12 × 4.5 cm.

a rural area in Ecuador, iodine deficiency was most likely the cause of her massive multinodular goiter. Commonly, goiters grow in an outward anterior direction, and large symmetrical goiters rarely grow posteriorly to compress the trachea and esophagus or laterally to impinge on the great vessels of the neck. However, these structures can be invaded if there is a substantial asymmetrical enlargement of one or both lobes of the thyroid gland and respiratory failure, dysphagia, or stroke can ensue, respectively (1, 2, 4). The intermittent obstruction of the left common CA in our patient could be due to the asymmetric growth of the left lobe, which was greater than the right. Hans *et al.* have reported similar clinical signs and symptoms of substernal goiters causing compression syndromes involving neurovascular structures, esophagus and trachea (5).

There are a few cases in the literature reporting the association between goiter and ischemic stroke (3, 6, 7). There was a case described by Fernando and Hassan that showed a 55-year-old female with left hemiparesis caused by complete occlusion of right internal CA by a giant goiter, in addition to retrosternal extension of the thyroid

mass (6). Silvestri *et al.* described the case of a 75-year-old female with a goiter and no major risk factors presented with extensive infarction in the right temporal-parietal region due to stenosis of the brachiocephalic trunk and right subclavian arteries. CT scan of the neck and thorax revealed that these arteries were compressed exteriorly by a right thyroid mass. The angiogram showed a normal blood flow in the right common CA, which disappeared on the rotation of the patient's head toward the right, most likely due to compression by the goiter. A thyroidectomy was performed in this case for the prevention of future strokes (3). Gadsseux *et al.* described a case with a congenital atrophic left vertebral artery and a giant retrosternal goiter (7). This patient was found to have recurrent transient ischemic attacks (TIA) due to compromised blood flow in the CA. There was complete recovery from symptoms after thyroidectomy. This patient's carotid artery ischemia was due to retrograde blood flow in the hypertrophied inferior thyroid artery rather than a goiter-related mass effect on CA. We hypothesize that our patient had recurrent TIAs from chronic compression of the left carotid artery with flexion of the neck and she likely had a hemodynamic stroke caused by hypoperfusion from severe obstruction (8). Hypoperfusion is the most common mechanism of cerebral ischemia in patients with internal CA occlusion (8). Therefore, thyroidectomy restored the hemodynamic balance that occurs from chronic compression (9). Embolic disease although possible is less likely as she had a full recovery after thyroidectomy. In addition, the large regions of restricted diffusion shown on her brain MRI are more consistent with acute chronic infarction.

Our patient had a long-standing goiter which over time gave rise to her presenting symptoms. She did not have risk factors and a negative stroke workup led to the indication for a total thyroidectomy. Her neurological symptoms recovered completely after thyroidectomy, due to early presentation to the hospital, and a timely decision for surgical intervention.

Identifying the mass effect from a goiter as the cause of a stroke can be a diagnostic challenge given the rarity of such a presentation, particularly in the emergency department setting, where physicians have limited time to work up patients suspected of having a stroke. More commonly encountered causes of stroke, such as thrombotic and embolic etiologies, may be worked up first, while less common causes, such as mass effect from a goiter, may be missed (10). This can result in a delay of appropriate treatment leading to poor outcomes. In our case, prompt recognition was key, and the recovery from neurological symptoms was complete without long-term



disability. In conclusion, recognizing stroke as a grave complication of a large multinodular goiter is crucial for timely and appropriate management.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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Patient consent

Written informed consent for publication of patient's clinical details was obtained from patient's daughter.

Author contribution statement

All the authors were involved in the writing of this manuscript. Dr Chiarolanzio was responsible for gathering the radiological images. All the authors read and approved the final version of the manuscript.

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