

Cerebral ischemia from thoracic goiter

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Most ischemic cerebral disturbances result from narrowing or occlusion of brain vessels. Other causes include cardiac disorders, subclavian steal syndrome, thrombocytosis, changes in hemoglobin of fibrinogen concentration and hypotension. The extracranial circulation can account for cerebral ischemia, from transient ischemic attacks (TIA) to stroke, due to modifications of anatomy and flow of arteries at the cervicothoracic level. The responsibilities of carotid, vertebral and subclavian arteries have been well documented. The role of the thyroid gland and its vessels has been discussed less often. It could be considered in the following two cases.

Case 1

A 39-year-old truck driver had been unable to work for 2 years because of frequent TIA causing left-sided hemiparesis. Computed tomography (CT) scans showed an area of low density in the right parietal lobe (Fig. 1). Angiography of the cervical and cerebral vessels revealed no stenosis or other anomaly. Blood pressure, blood count and biology were normal. However the patient had a cervicothoracic goiter, without any symptom of thyroid disorder or any sign of compression. No treatment of this goiter had ever been considered. A new angiogram of the aortic arch showed both carotids originating from a common trunk shared with the right subclavian artery. The carotids were pushed apart but were not compressed. The right thyroid arteries were enlarged (Fig. 2). It was decided to proceed with thyroidectomy, as frequent TIA had left the patient with a motor deficit and decreased sensitivity on the left side and as no other cause of this disorder could be found. In March 1979, subtotal bilateral resection of the goiter was performed through a cervicotomy. It was benign. One month later the patient had recovered his sensitivity and his motricity had improved. In 1994 he was leading a normal life, driving a truck and had not had any more trouble for 15 years.

Case 2

A 48-year-old lady had a motor cycle accident in 1985, with cerebral commotion and no after-effect. In May 24, 1988, she became unconscious, with no tongue biting or urinary loss. She recovered after 3 h. Clinical examination was normal. Electroencephalograph (EEG) showed right temporal paroxymal waves. Brain CT scans showed diffuse edema. Chest radiograms and CT scans suggested the existence of a mediastinal goiter, hypervascularized on its surface with aspects of deep necrosis (Fig. 3). Thyroid hormone assays were normal. A scintigram showed no fixation. An angiogram of the aortic arch showed an enlargement of the thyrocervical trunk with voluminous inferior arteries. In June 1993, a cervico-sternotomy revealed a mediastinal retrovascular goiter, pedunculated on the left cervical thyroid lobe and crossing the mediastinum to the right. The cervical gland appeared normal otherwise. The mediastinal mass was resected. It was benign. Twenty-two months later the patient had had no more attacks, with no further treatment. Clinical examination, EEG and cerebral magnetic resonance imaging (MRI) were all normal.

Although cerebral disorders in relation to an impaired superior caval venous return (which did not exist in these two cases) are known and accidents from modified cervicothoracic arterial patterns have been explained and detailed, the importance of thyroid gland and arteries as causes of cerebral ischemia has not often been discussed. In 1978, Le Brigand and associates [2] operated on a patient with a cervicothoracic goiter who had transient attacks of right hemiplegia with aphasia when he extended his neck and threw his head backwards. Thyroidectomy cured him, but the duration of follow-up was not given. In 1986, Gadisseux and associates [1] described a case of recurrent TIA resolved after the removal of a voluminous retrotracheal goiter. Six months after surgery, the patient was free of symptoms. Previously, Mueller and Hinck [3] and Trevino [4] had brought attention to thyrocervical collateral channels secondary to the obliteration of the subclavian arteries beyond the vertebral orifices.

Though it is admitted that a proximal steal in the common carotid artery in the absence of distal stenosis, for example when using this vessel for bypass, does not give rise to any cerebral symptom, these two cases suggest the contrary: the neurological disorders disappeared after the resection of the thoracic goiters. Arterial compression and steal probably added up in the two patients. The follow-up is in favor of this mechanism in spite of hemodynamic lack of evidence. In practical terms, these cases advocate the necessity of chest radiograms and of heart-to-brain angiograms in patients presenting with otherwise unexplained cerebral symptoms

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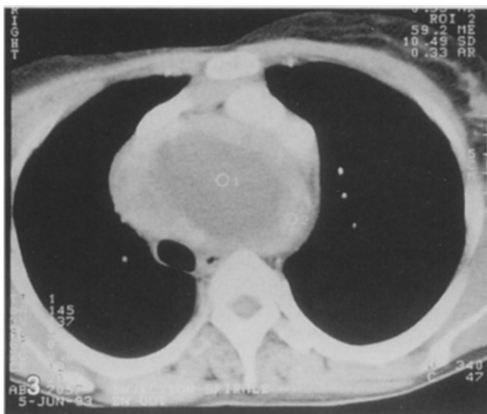
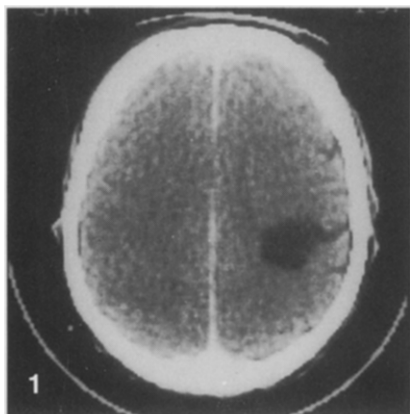


Fig. 1. Case 1: Low density area in the brain right parietal lobe

Fig. 2. Case 1: Cervicothoracic goiter, carotid anomaly and enlargement of thyroid arteries

Fig. 3. Case 2: Mediastinal mass, both hypervascularized (02) and necrotic (01)



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