Carotid Body Tumor: Unusual Cause of Transient Ischemic Attacks

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Carotid body tumors are an unusual cause of transient ischemic attacks. The cases reported in the literature have been limited to the postoperative period. We report a patient with bilateral carotid body tumors and transient ischemic attacks as the presenting syndrome. A low-perfusion state could explain his symptomatology. (Stroke 1988;19:102-103)

Carotid body tumors (CBTs) are not commonly found in clinical practice. They usually are noted as a painless mass laterally placed in the neck and sometimes with cranial nerve involvement; dizziness and headache have also been described. We have not found any report in the literature with transient ischemic attacks as a presenting syndrome.

Case Report

A 61-year-old man came to the emergency room having complained for the previous 2 hours of paresthesias in his right arm and face and difficulty in speaking correctly. The patient had also experienced local neck discomfort for the previous 15 years on turning his head to the left. Vascular risk factors such as hypertension, diabetes, or hematologic abnormalities were not present. Family history revealed a sister who died 4 years previous following surgery for an aortic paraganglioma.

The general examination showed a conscious, oriented, and slightly agitated patient. Blood pressure and cardiac rhythm were normal, cardiac and carotid auscultation revealed no bruits, and funduscopy showed no vascular change. Peripheral pulses were present and normal. On palpation a painless mass in his left neck, movable only laterally, became evident; neither pulsations nor bruits were noted. Another, smaller mass was found on the right side.

The neurologic examination showed a transient nonfluent aphasic patient with a right faciobrachial hemiparesis; the rest of the exploration was within normal limits. An hour after the patient had entered the hospital, both signs and symptoms disappeared except for the mild agitation.

On the day he was admitted to the hospital, the patient suffered five episodes of transient nonfluent aphasia with a right faciobrachial hemiparesis, each of which lasted about 30 minutes. There was no change in blood pressure or cardiac rhythm. Complete blood count, urinalysis, serum electrolytes, SMAC 12, lipids, cholesterol, and prothrombin time were all normal. Electrocardiographic and echocardiographic studies showed no cardiac rhythm alterations or other embolic risk factors. Brain computed tomography (CT scan) showed normal sulci and ventricular size with no evidence of infarct or blood. Carotid angiography revealed a vascular mass at the level of the left carotid bifurcation (Figure 1); another, smaller vascular mass was evident in the right carotid bifurcation. No alterations were detected in the intracranial carotid circulation. Contrast-enhanced cervical CT scan confirmed the vascular nature of the two masses.

The left tumor was 7 x 5 cm and partially surrounded the left internal carotid artery. Longitudinal arteriography on the internal and common carotid arteries revealed severe narrowing of the internal carotid artery lumen. There was no disruption of the endothelium, and no plaques were found. The tumor and an internal carotid fragment were resected. Cerebral circulation was reestablished with an end-to-end anastomosis between the common and internal carotid arteries. Pathologic study showed a benign CBT attached to the arterial wall with the endothelium intact. The excised carotid fragment did not reveal any atherosclerotic changes.

At the onset of the postoperative period, the patient was aphasic and very agitated. This situation persisted for 4 days, after which he became asymptomatic. A second operation was performed 4 months later on the right side without arterial resection. Pathologic study showed another benign paraganglioma. The patient has remained asymptomatic since the last operation 20 months ago.

Discussion

The pathology of the carotid body is limited to tumor formation. CBTs are very uncommon, and the differential diagnosis should include other more frequent neck masses such as brachial cysts, solitary lateral aberrant thyroid masses, neurofibromas, mixed parotid tumors, lymph nodes, carotid artery aneurysms, and arteriovenous fistulas.1,2 CBT usually presents as a painless, slow-growing, compressible mass in the upper neck that may pulsate and have a bruit.3 Local discomfort, recent increase in

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size, headache, dizziness, dysphagia, tinnitus, hearing loss, and voice change are other symptoms. Cranial nerve involvement is not uncommon, with the vagus nerve most frequently involved. Glossopharyngeal and hypoglossal paralysis are also seen. Raeder’s syndrome has been described only once. CBTs are bilateral in 4.4% of cases, the frequency increasing to 31.8% in familial cases. Although cited by Robertson, we have not found any reports of transient ischemic attack as a definite clinical syndrome before surgery for a CBT. The cerebrovascular accidents cited in literature were postoperative and were also reported as one of the main causes of death.

In our patient, there were several factors suggesting a direct relation between CBT and symptomatology. Although speculative, an ischemic mechanism based on a carotid low-perfusion state appears to be the most probable explanation of the symptoms. The recurrence of similar clinical patterns reflecting the same brain region distal to the obstructed carotid artery, as well as the fact that surgical revascularization led to cessation of symptomatology, suggests a low-perfusion state that was corrected by surgery. The absence of evidence of atherosclerotic changes on angiography, the lack of vascular risk factors, and the preservation of the carotid artery endothelium at the level of the tumor make other possibilities such as embolism related to CBT rather improbable.

Even though cervical CT scan is very useful for studying CBTs, this exploration does not exclude the need for angiography. Due to the possibility of bilateral CBTs, both carotid arteries must be studied.

References


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