

An Uncommon Complication: Hypoglossal Nerve Palsy After Carotid Endarterectomy

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Abstract

Carotid artery stenosis is the important causes of ischemic stroke, and it may result in death. Many complications can occur during carotid endarterectomy (CEA), which is the standard revascularization procedure for treating severe carotid artery stenosis. CEA is applied for prevention of stroke occurred by atherosclerotic plaque which is located at the common carotid artery bifurcation and, commonly, internal carotid artery. In addition to serious complications, such as stroke, infection, myocardial infarction, postoperative bleeding and death, non-fatal complications, such as cranial nerve palsy, can also occur rarely. Cranial nerve injury is a well-recognized complication of carotid endarterectomy. Although major injuries are rare, in several series where thorough pre and postoperative neurologic examinations were performed, the incidence of cranial nerve dysfunction after carotid surgery ranged from 9.7% to 39%. Here we present the an 80-year-old male patient with hypoglossal nerve palsy after carotid endarterectomy as it is a rare complication.

Keywords: hypoglossal nerve, palsy, carotid endarterectomy, complication

Introduction

Stroke is known as the third most commonly occurring death causality in the world and may result in long-term loss of labor especially in developed countries^{1,2}. In total, 20%–25% of ischemic strokes are caused by atherosclerosis occurring in the carotid artery. Atherosclerotic plaque formation in the carotid artery is a pathological process that increases with age. Atheroma plaque usually begins in the posterior wall of the common carotid artery and progresses toward the internal carotid artery. An atheroma plaque is formed by endothelial damage and takes place in the intima. As the plaque increases, the internal carotid artery lumen is obliterated. The standard revascularization method for severe carotid artery stenosis is carotid endarterectomy, and cranial nerve palsy after endarterectomy has been reported in 3%–23% of cases³.

The most common cranial nerve palsy occurring after carotid endarterectomy is the hypoglossal nerve palsy. It is thought to be the result of nerve retraction during surgery⁴.

PURPOSE: We presented this case to indicate the rare occurrence of isolated hypoglossal nerve palsy after carotid endarterectomy.

Case Report

An 80-year-old male patient was admitted to the emergency department with a complaint of loss of power in the right arm and leg. The patient's medical history was remarkable for hypertension, coronary artery disease, and bypass surgery. Physical examination revealed arterial blood pressure of 135/80 mmHg; his pulse rate was 88 beats/min with a regular rhythm and body temperature was 36.6°C. Neurological examination revealed the following pathological findings: right central facial paralysis, right 4/5 hemiparesis, and extensor plantar response on the right side.

Cranial computed tomography performed in the emergency department revealed no acute hemorrhagic lesion. Diffusion magnetic resonance imaging (MRI) performed with the preliminary diagnosis of acute ischemic cerebrovascular disease revealed no diffusion restriction in the sections obtained from the infratentorial region that would suggest acute infarction. In the sections taken from the supratentorial region, acute infarct areas that were hyperintense on diffusion MRI examination and hypointense on apparent diffusion coefficient mapping were observed in the left parietal and occipital regions. The patient was referred to the neurology department

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with the diagnosis of acute ischemic cerebrovascular disease. Carotid artery color Doppler ultrasonography revealed mixed-type plaque formation in the left internal carotid artery resulting in a stenosis of >70%. Routine biochemistry, hemogram, and hormone tests revealed no pathological findings, except a glucose level of 163.8 mg/dl. Anti-aggregant therapy was initiated with acetylsalicylic acid at 100 mg/day, and anti-hypertensive therapy was initiated with candesartan/hydrochlorothiazide at 16/12.5 mg/day. Consultation with a cardiovascular surgeon was performed for treating existing stenosis in the carotid artery. Endarterectomy was performed

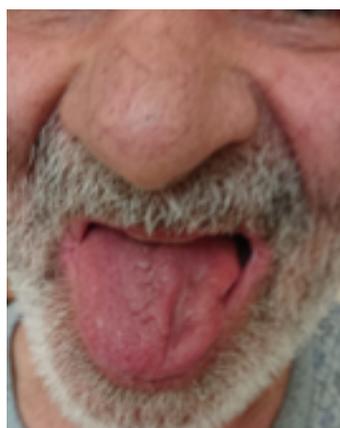


Figure 1. The patients tongue with difficulty in movement, swallowing and talk due to isolated left N hypoglossus paralysis

on the left carotid artery by a cardiovascular surgeon, and the patient was discharged.

After discharge, the patient presented to our outpatient clinic with complaints of restraint in tongue movements and swallowing difficulty in addition to his pre-existing neurological complaints (Fig. 1). In Cranial Magnetic Resonance

(MR) images; Axial T2A and T1 weighted (Fig. 2) image had a normal brainstem and fluid attenuated inversion recovery (FLAIR) and T2-weighted image (Fig. 3) images left posterior circulation infarction.

Neurological examination revealed 4/5 hemiparesis in the right upper and lower extremities, extensor plantar response on the right side, atrophy in the left half of the tongue, and deviation of the tongue to the left side on protrusion. The patient was hospitalized in the neurology department with the diagnosis of hypoglossal nerve palsy. The patient was given physical therapy, and total parenteral nutrition was initiated because of swallowing and feeding difficulty. The ongoing anti-aggregant and anti-hypertensive therapies were continued. During the follow-up of the patient, it was observed that the severity of the existing tongue atrophy and swallowing difficulty decreased. The patient was discharged from the hospital on the 10th day of hospitalization with a recommendation of follow-up at an outpatient clinic.

Discussion

Stroke is the third most common cause of death in the world and the most common cause of fatal neurological events. Risk factors for ischemic stroke are divided into two groups: alterable and inalterable. Alterable risk factors include hyperlipidemia, hypertension, smoking, diabetes mellitus, alcohol, obesity, physical inactivity, heart disease, high homocysteine levels, carotid stenosis, presence of anti-cardiolipin antibodies, and the use of oral contraceptives, whereas unalterable risk factors include race, age, genetics, and gender.

Obstructive stroke is observed in approximately 85% patients who are hospitalized and treated for stroke, and ca-

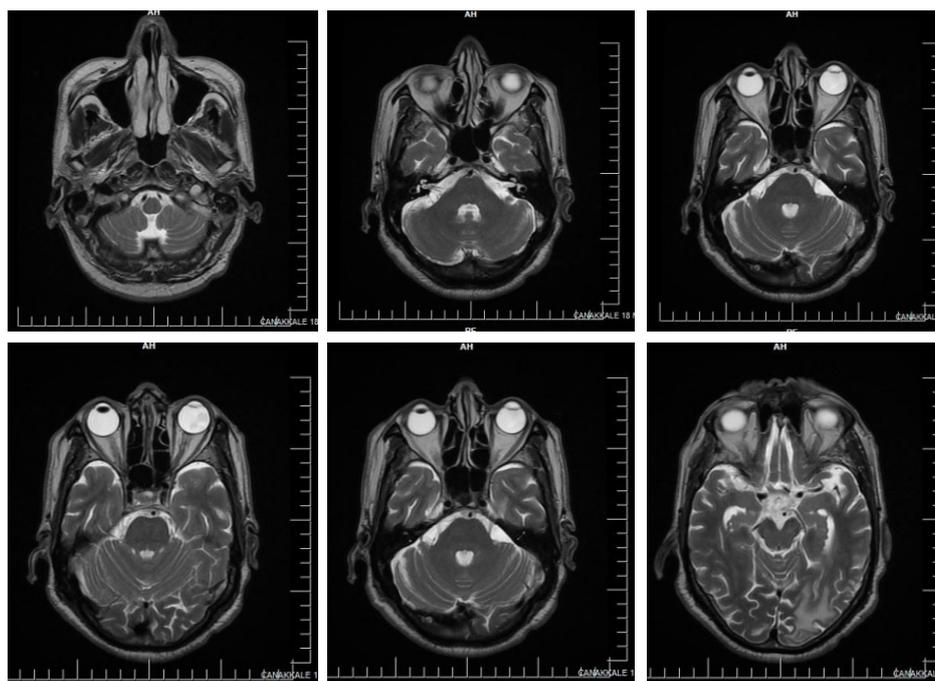


Figure 2. Brain magnetic resonance imaging (MRI) scan T2-weighted image of normal brainstem

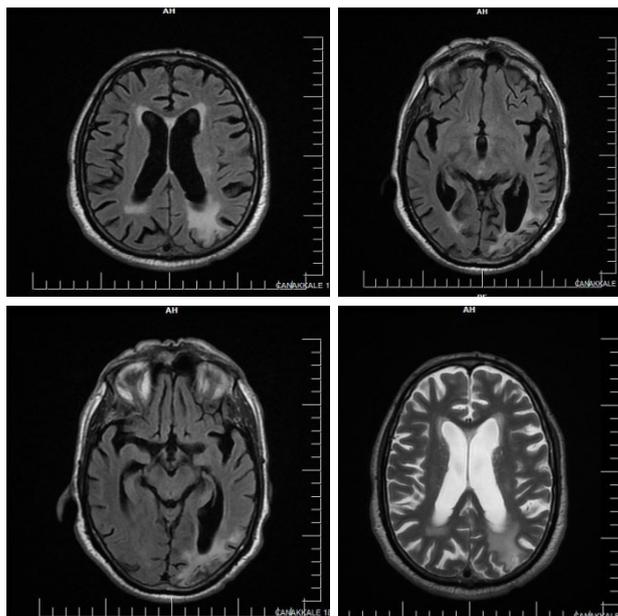


Figure 3. Fluid attenuated inversion recovery (FLAIR) and T2-weighted image images left posterior circulation infarction

rotid stenosis is responsible for 5%–12% of these attacks⁵. Carotid artery atherosclerosis is a systemic vascular disease due to possible complications of stroke and requires a multidisciplinary treatment. Carotid artery endarterectomy is currently accepted as the most effective method for preventing the development of stroke and transient ischemic attacks in patients with symptomatic/asymptomatic carotid artery stenosis⁶. Atherosclerotic plaques are most densely seen on the posterior wall of the carotid bifurcation in the internal carotid artery. Stroke occurs because of occlusion of the arteries by an embolus that detaches from the thrombus formed on the plaque or because of thrombosis of the atherosclerotic plaque resulting in occlusion of the vessel and decrease in brain perfusion⁷.

The incidence of cranial nerve palsy after carotid endarterectomy has been reported to be between 3% and 23%³. The most common cranial nerve palsy after carotid endarterectomy is hypoglossal nerve palsy. The hypoglossal nerve is a pure motor cranial nerve innervating the extrinsic and intrinsic muscles of the tongue. The hypoglossal nerve follows a vertical course in the neck after leaving the anterior condylar foramen and it is in close proximity with the internal carotid artery and internal jugular vein. This proximity is also the cause of nerve palsy after surgeries performed on cervical veins⁷. Hypoglossal nerve palsy occurring after carotid endarterectomy is thought to result from nerve damage secondary to nerve retraction during surgery.

In our patient, hypoglossal nerve palsy was detected after carotid endarterectomy. This was probably due to axonal injury as a result of overstretching of the nerve while attempting to preserve the hypoglossal nerve during carotid artery exploration.

Conclusion

Isolated hypoglossal nerve palsy, which is a rare complication occurring after carotid endarterectomy, should be kept in mind, and careful neurological examination should be performed after the operation to avoid this complication from being unnoticed.

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