Cerebral Infarction Due to Carotid Occlusion Caused by Cervical Vagal Neurilemmoma
Report of a Case
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Background—We report a case of a 71-year-old woman with cerebral infarction due to occlusion of the internal carotid artery (ICA) caused by a neck tumor.

Case Description—In 1998, the patient complained of mild hoarseness, and a diagnostic workup showed a cervical mass that was considered a benign neck tumor. In September 2000, she developed right-sided weakness. Diffusion-weighted MRI showed a high-intensity area in the territory of the left middle cerebral artery. Carotid angiography and ultrasonography revealed occlusion of the left ICA, which was due to compression by the neck tumor. Superficial temporal artery–middle cerebral artery anastomosis was performed to prevent critical reduction of cerebral blood flow in the left ICA territory; this was followed by tumor resection. The occluded ICA recanalized after tumor resection. Microscopic examination showed that the tumor was a vagal neurilemmoma.

Conclusions—This is the first case of cerebral infarction due to left ICA occlusion by a cervical neurilemmoma. Even when the neck tumor is benign, it may occlude the ICA and thereby cause cerebral infarction. (Stroke. 2002;33:1428-1431.)

Key Words: carotid arteries ■ cerebral infarction ■ head and neck neoplasms ■ neurilemmoma

Cerebral infarction is considered one of the important complications of neoplasia. A neoplasm may cause cerebral arterial occlusion by tumor embolism, arterial encasement, or hypercoagulability, such as disseminated intravascular coagulation, and nonbacterial thrombotic endocarditis. Mechanical obstruction of the cerebral and cervical arteries by tumor may be one possible cause of cerebral infarction. However, most head and neck tumors are benign and less invasive, and cerebral infarction due to mechanical obstruction by such tumors is uncommon. A cervical neurilemmoma is a rare tumor, and to our knowledge, there are no reports of a neurilemmoma causing cerebral infarction. Here we report a case of cerebral infarction due to carotid artery occlusion caused by a vagal neurilemmoma.

Case Report
A 71-year-old woman was admitted to our hospital on October 1, 2000 because of weakness on the right side. Two years before admission, she complained of mild hoarseness. CT scan showed a cervical mass, which was considered a benign tumor. During these 2 years, she had had no neurological symptoms other than mild hoarseness. On September 18, 2000, the patient felt weakness of the right upper limb for the first time and was admitted to our hospital 2 weeks later. Physical examination showed no apparent abnormalities. Neurological examination revealed left vagal nerve palsy. Past history included no vascular risk factors such as hypertension, hyperlipidemia, smoking, or diabetes. The results of laboratory studies were normal. Electrocardiogram showed a normal sinus rhythm. Echocardiography was normal, and no thrombus was seen in the left atrium. Cervical MRI showed a multi-cystic mass located between the left jugular vein and left carotid artery (Figure 1, A and B). Carotid ultrasonographic findings indicated occlusion of the left internal carotid artery (ICA) by the mass. T2-weighted MRI of the brain showed diffuse high-intensity lesions in the white matter, bilaterally (Figure 2A), and diffusion-weighted MRI showed prominent high intensity area in the border zone of the left ICA territory (Figure 2B). Left carotid angiogram showed complete obstruction of the left ICA due to external compression by the tumor mass (Figure 3A). Right carotid angiogram showed no significant stenotic lesions in right ICA or right middle cerebral artery (MCA), except for a congenital defect of the proximal portion of the right anterior cerebral artery (ACA) (A1). The right ACA was fed through leptomeningeal anastomosis by the right MCA, which provided...
flow to the left MCA via the anterior communicating artery (Figure 4A). In the left vertebral angiogram, the posterior communicating artery was barely observed, and the posterior branches of the left MCA were fed by the left posterior cerebral artery through leptomeningeal anastomosis (Figure 4B). Moreover, cerebral hypoperfusion with decreased vasodilatory capacity in the left MCA territory was confirmed by single-photon emission computed tomography under acetazolamide challenge.

Based on the imaging studies and clinical course, the condition was diagnosed as cerebral infarction in the border zone of ICA territory due to ICA occlusion by the parapharyngeal mass. The right-sided weakness worsened suddenly on October 14, 2000, after the patient stood up. The right hemiparesis progressed and failed to respond to medical therapy, finally becoming complete hemiparesis, which was
also associated with speech disturbances. Because symptoms were progressive, anastomosis of the superficial temporal artery and MCA was performed to preserve cerebral blood flow before removal of the tumor and to prevent further deterioration of the neurological condition. The hemiparesis started to improve gradually postoperatively. Two weeks after the bypass surgery, the tumor was completely resected. The mass was located in the left carotid sheath and originated from the left carotid artery.

Figure 3. Angiogram of the left carotid artery. (A) Note the complete obstruction of the left ICA (arrow) probably due to the mass. (B) Note recanalization of the left ICA after tumor resection.

Figure 4. Right carotid angiogram. (A) Note the lack of significant stenotic lesions in the right ICA or right middle cerebral artery (MCA), except for a congenital defect of the proximal portion of the right anterior cerebral artery (ACA). The right ACA was fed through leptomeningeal anastomosis by the right MCA and thereby the left MCA was fed via the anterior communicating artery. (B) The posterior communicating artery was barely evident on the left vertebral angiogram and posterior branches of the left MCA were fed by the left posterior cerebral artery through leptomeningeal anastomosis.
vagal nerve. It was approximately 70×55×45 mm in size and compressed the left ICA. Histopathological examination indicated that the tumor was a benign neurilemmoma without any malignant cells or vascular invasion. After resection of the tumor, the left ICA was recanalized without any other ischemic events. Recanalization of the artery was confirmed by carotid ultrasonography and angiography (Figure 3B). Single-photon emission computed tomography performed 1 month after surgery showed improvement of cerebral perfusion in the left cerebral hemisphere. During the 3-month period after tumor resection, the right hemiparesis further improved, and the patient was able to move her hand and foot voluntarily, although hoarseness and dysphagia were still present.

Discussion
We described a case of cerebral infarction with ICA occlusion due to compression by a cervical vagal neurilemmoma. Cerebral infarction is one of the complications of neoplasm. Several factors could induce ischemic stroke in patients with neoplasm. For example, occlusion of the cerebral artery due to compression by head and neck tumors could cause ischemic stroke. Few reports have described cerebral infarction due to occlusion of the cerebral artery by tumors. In the present case, occlusion of the left ICA was caused by a neck mass, and the collateral blood flow to the left ICA territory was insufficient to preserve cerebral perfusion. Thus, cerebral infarction was presumably caused by ICA occlusion and poor collateral blood flow. Two different mechanisms seem to cause mechanical obstruction of carotid artery: (1) arterial encasement by the tumor and (2) compression of the arterial walls. Meningioma, pituitary adenoma, and malignant fibrous histiocytoma have been reported to cause occlusion of the carotid artery. Direct invasion of these tumors into the arterial wall is the common mechanism of infarction. In the present case, ICA was occluded by mechanical compression, which resulted in hemodynamic infarction.

Cervical vagal neurilemmoma in the head and neck area is rare. To date, 95 neurilemmomas in the cervical region have been reported in the literature. The tumor is often associated with 1 or more clinical signs and symptoms, such as hoarseness, dysphonia, dyspnea, dysphagia, syncopal episodes, and Horner’s syndrome. To our knowledge, this is the first case of a cervical neurilemmoma causing cerebral ischemia. Unexpectedly, the occluded artery recanalized after tumor resection, and MRI did not show any new infarcts after recanalization. Our case suggests that in patients with cerebral infarction caused by occlusion of ICA due to cervical tumor, the occluded artery can potentially recanalize after tumor resection.

References
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