Cervical Carotid Artery Vasospasms
Causing Cerebral Ischemia
Detection by Immediate Vascular Ultrasonographic Investigation

Christian Arning, MD; Alexandra Schrattenholzer, MD; Lutz Lachenmayer, MD

Background—The etiology of cerebral ischemic accidents in young adults often remains unclarified.

Case Description—A 32-year-old woman presented after multiple episodes of left monocular visual impairment and right-sided focal signs. MRI revealed a low-flow infarction on the left; color-coded duplex sonography (CCDS), however, showed normal vascular findings. During the inpatient rehabilitation, a renewed visual impairment occurred; an immediate CCDS examination now demonstrated a filiform stenosis of the left internal carotid artery (ICA) 4 cm above the origin and indirect signs of a severe stenosis of the right ICA. Results of a follow-up examination 18 hours later were again normal. Six weeks later, on reoccurrence of visual impairment, a reversible stenosis of the left ICA was again demonstrated. A search for possible causes of vasospasm was unsuccessful. After treatment with calcium antagonists the patient was free of complaints (with the exception of 3 very short attacks of visual impairment) during the following 12 months.

Conclusions—Cervical carotid artery vasospasms can apparently occur spontaneously without a mechanical trigger. Because their detection is difficult, vasospasms may go undetected.

Key Words: carotid arteries ■ cerebral ischemia ■ ultrasonics ■ vasospasm ■ young adults

Vasospasms of the cranial arteries are a well-known cause of cerebral ischemia in cases of subarachnoidal hemorrhage and have also been reported in isolated cases of migraine,1 vasculitis,2 and eclampsia.3 Extracranial vasostrictions may arise from mechanical manipulations during operative interventions,4 vasopuncture,5 and catheter examinations,6 as well as in cases of ergot poisoning.7 It was previously suspected that extracranial vasospasms could be of some importance in the pathogenesis of migrainous strokes,8 but this possibility could not be confirmed until now.

Case Report

In September 1995, a 32-year-old woman experienced acute visual impairment with light flashes and dark spots in the left eye and later a 1-sided hemiparesis and sensory impairment on the right side followed by bilateral forehead pain. The symptoms disappeared within 2 hours. All examinations, including cranial CT and extracranial and intracranial vascular sonography carried out 2 days later, were negative. Apart from a moderate consumption of nicotine (nonsmoker since the beginning of November 1996), no vascular risk factors were present. The patient was otherwise healthy, and there was no history of migraine-like headaches. She was not taking oral contraceptives or any other drugs.

The symptoms recurred 5 weeks later with an additional dysarthria, and the patient was admitted to hospital immediately. On admission a slight vascular murmur was detected in the left side of the neck. Again, the symptoms regressed rapidly. In addition to the previous diagnostic investigations, MRI of the head was carried out but results were not significant; the vascular sonography was not repeated. In the following 12 months similar symptoms occurred temporarily (about 5 times at irregular intervals); in these episodes the patient was not seen by a physician.

Approximately 13 months after the first episode, the patient was again admitted. The symptoms were now more strongly pronounced than in earlier episodes, and there was also a neuropsychological syndrome, with lack of drive and mental slowness, as well as an aphasia. This time the symptoms did not disappear rapidly but remained, at first, constant. Intensive diagnostic examinations were performed in the next few days but did not produce any etiologic clarification. Vascular sonography was again without pathological findings. MRI showed an extensive subcortical increase in signal intensity in the left parietal region. This finding was best interpreted as a low-flow infarction of hemodynamic origin, and because of the negative vascular findings on Doppler sonography and MR angiography, the possibility of an inflammatory lesion was considered. Treatment with prednisone for 3 weeks was begun. The cerebrospinal fluid was analyzed twice (on days 4 and 19); results were without significance both times. Also, no indications for

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From the Department of Neurology, Barmbek Hospital, Hamburg, Germany.
Correspondence to Dr Christian Arning, Abteilung Neurologie, Allgemeines Krankenhaus Barmbek, D-22291 Hamburg, Germany.
E-mail christian.arning@t-online.de
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a possible inflammation were found in the blood. Virology (including a test for human immunodeficiency virus) was also negative, as were bacteriological-serological diagnoses (including tests for *Borrelia burgdorferi* and *Treponema pallidum*), blood count investigations, and extensive immunologic diagnoses (including tests for antinuclear antibodies, immunoelectrophoresis, C-reactive protein, and lysozyme). Furthermore, no coagulation disorder was found.

Under rehabilitation measures with physical therapy, ergotherapy, and logopedics, the deficiency symptoms improved slowly, although a slight, right-side hemiparesis remained. At 3½ weeks after admission, the patient complained of a sudden recurrence of the known visual impairment in the left eye. Vascular sonography was performed immediately and revealed a filiform stenosis of the left internal carotid artery (ICA) 4 cm cranial of its origin (Figure 1). On the right, a severe flow impediment of the ICA was detected; there was an ophthalmic collateral with rapid, unambiguous retrograde flow in the supratrochlear artery. Direct signs of a stenosis were not seen in the examined section of the right ICA. The patient was given an infusion of saline for circulatory support and heparin for embolus prophylaxis as well as prednisone. This time no further deficiency symptoms occurred. The next day, visual impairment was no longer present, and results of a sonographic follow-up (18 hours after the previous examination) were once again normal (Figure 2). To avoid the possibility of provoking a vasospastic reaction, angiography was not attempted.

The prednisone treatment was continued, with the dose being very slowly reduced. At a dose of 30 mg/d, the known visual impairment occurred again, 6 weeks after the preceding episode. Color-coded duplex sonography again revealed a filiform stenosis of the left ICA in the same position (Figure 3). This time, however, the findings for the right carotid and supratrochlear arteries were inconspicuous. Treatment com-
prised infusion of saline and heparin. The visual impairment lasted 2 days, during which other symptoms did not appear. On follow-up examinations on succeeding days, the stenosis was still detectable, albeit to varying extents, but finally normalized after 3 days (Figure 4). A routine ECG at this time showed slight recovery disorders over the posterior wall; later ECG measurements were inconspicuous. On the assumption of a recurrent vascular spasm, the patient was treated with vasodilators (first molsidomin and nifedipine, then nitrendipine). At the 12-month follow-up, the patient was free of complaints, with the exception of 3 very short attacks of visual impairment. A vascular sonographic follow-up was inconspicuous.

**Discussion**

Despite intensive diagnostic efforts, the etiology of cerebral ischemic accidents in young adults often remains unclear. It is worthy to note that in this case, even after the occurrence of a cerebral infarction of hemodynamic origin, the cause of the perfusion disorder was not recognized. Only during the ninth attack, which occurred in the hospital, very severe bilateral vascular stenoses of the ICAs were detected by immediate sonography. If the examination had been carried out after an interval of more than 1 day, it would have shown normal findings. The rapid regression of the stenoses within less than 18 hours excludes the possibility of a vascular stenosis due to arteriosclerosis or dissection; even so, the degree of stenosis, according to the usual hemodynamic criteria, amounted to at least 90%.

The further course with the renewed occurrence of a reversible stenosis of the left ICA at the same site again supports the interpretation of these findings as vasospasm. The detection of repeated spasm of the carotid artery in combination with the patient’s ipsilateral visual disorders supports the suggestion that a vasospasm had also occurred at
the time of her earlier symptoms but had not been detected due to its short duration. However, the cause of this functional narrowing remains unclear, because none of the usual causes of spasm could be found. Apparently the patient benefited from treatment of the symptoms: in a follow-up period of more than 12 months, 3 short-duration visual impairments have occurred but were not accompanied by further neurological deficit symptoms.

Another important aspect is the temporary change in the ECG, which may be interpreted as the effect of a coronary artery spasm. Findings of this type have been described in migraine patients. The diagnostic problems in the detection of vascular spasms in coronary arteries are well known: at the moment of investigation the arteries appear to be normal—the vasoconstriction has already disappeared. Vasoasms of carotid arteries can be so short lived that they cannot be seen with the usual diagnostic examinations, as in the present case. But they may also last for several days (like the second stenosis documented in this case) and may then be mistakenly interpreted as dissections.

In light of the above-mentioned diagnostic difficulties, it is possible that vasoasms may not be recognized in all patients. It remains to be determined whether this is a functional syndrome (eg, a migraine variant) or whether the spasms result from an as-yet unidentified angiopathy.

References

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