Embolic Stroke by Compression Maneuver During Transcranial Doppler Sonography

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Background and Purpose Embolic complications during ultrasound examinations are a rare cause of neurological deficits. The present case documents the occurrence of embolism by a nonobstructive compression maneuver during transcranial Doppler examination, resulting in a minor stroke.

Case Description A 63-year-old man suffered from recurrent transient ischemic attacks. Duplex sonography showed a small echogenic plaque at the right carotid bifurcation. During transcranial Doppler studies with a reverberating compression maneuver of the right common carotid artery low in the neck, multiple emboli signals were detected, and the patient developed a left-sided hemiparesis with slurred speech. Funduscopic examination revealed cholesterol emboli in the inferior temporal arteriole of the right eye. These findings suggested embolization as the cause of the stroke.

Conclusions Compression maneuvers should not be performed in patients with recent neurological symptoms, even in the case of only small lesions in the extracranial carotid territory. (Stroke. 1994;25:1056-1057.)

Key Words • Doppler • embolism • ultrasonics

Compress tests in the region of the extracranial carotid system are an integrated component of transcranial Doppler sonography (TCD). Embolic complications during ultrasound examinations are a rare cause of neurological deficits. However, a few cases of cerebral embolism after carotid sinus pressure have been reported. The present case documents the occurrence of embolism by a compression maneuver during TCD examination, resulting in stroke in a 63-year-old male patient.

Case Report

A 63-year-old man suffered for 1 month from recurrent transient ischemic attacks (TIAs) with left-sided hemiparesis. The patient had a history of coronary heart disease, long-standing cigarette smoking, and hypertension. Neurological examination on admission revealed no pathological findings. Duplex sonography showed an extracranial carotid system to be free of arteriosclerotic lesions except for a small echogenic plaque at the right carotid bifurcation (Fig 1). Subsequently TCD studies were performed using the transtemporal approach. Frequency spectra with a low mean blood flow velocity of 35 cm/s were detected at a depth of 55 to 65 mm on the right side. Reverberating compression maneuvers of the right common carotid artery were performed to discriminate the right middle cerebral artery from the posterior cerebral artery. During these compression maneuvers multiple emboli signals appeared on the scope (Fig 2). Immediately after the high-frequency signals were detected, the patient developed a left-sided hemiparesis and speech impairment. Funduscopic examination performed 24 hours later showed asymptomatic retinal cholesterol emboli in the inferior temporal arteriole. Cerebral angiographic studies confirmed the duplex sonographic findings, demonstrating a small plaque in the region of the right carotid bifurcation. The other parts of the extracerebral and intracerebral carotid system appeared free of arteriosclerotic lesions.

The patient was treated conservatively with platelet antiaggregants. He recovered nearly completely within 2 weeks and showed only a slight weakness of the left arm at the time of discharge.

Discussion

Cholesterol emboli, dislodged by a reverberating compression maneuver of the common carotid artery, resulted in a minor stroke in this patient. In our neurosonographic laboratory such compression tests are performed only in selected patients with very complex hemodynamic flow patterns or when discrimination of certain basal cerebral arteries is not possible. Compression tests are always carried out by an experienced physician according to the recommendation of Aaslid: nonobstructive reverberating compression of the common carotid artery low in the neck after duplex examination has shown the site of compression to be free of arteriosclerotic plaques. Despite these precautionary measures, vibrating compression led to the dispersion of cholesterol emboli obviously originating from the small plaque at the bifurcation. This is the first example of cerebral embolism resulting from compression of the common carotid artery proved by TCD. Spencer et al showed that microemboli dislodged during carotid endarterectomy are detected reliably by TCD. Transient signals with a high amplitude (up to 10 dB greater than the background Doppler signal) and a relatively narrow spectrum, occurring randomly during the cardiac cycle, are characteristic of emboli. To the ear, signals indicat-
ing emboli are harmonic in tone, with a chirping or
whistling quality. This acoustic quality enables an ex-
perienced investigator to differentiate clearly between
emboli signals and artifacts.

Funduscopic examination 24 hours after the acute
event suggested that stroke in this patient had been
cased by cholesterol emboli. We found characteristic
yellow fragments in the right inferior temporal arte-
riole, whereas the retinal arteries of the left eye appeared free
of cholesterol crystals. In 1961 Hollenhorst7 first de-
scribed orange-, yellow-, and copper-colored fragments
seen at retinal arteriolar bifurcations and suggested that
these were cholesterol crystals rising from ulcerated
arteriosclerotic lesions of cardiac valves, aorta, or the
carotid arteries. In 1963 David et al8 found doubly
refractile cholesterol crystals in the eye of a patient with
a fatal ipsilateral cerebral infarct after undergoing sur-
gical exploration of the ipsilateral carotid artery; it was
elected, however, not to open the artery because of
normal blood flow measurements. Histochemical and
optical characteristics of the material were identical to
those found in the patient’s atheroma of the carotid
artery and of the embolus of the middle cerebral artery.
The retinal cholesterol emboli in our patient were
considered to be asymptomatic because ocular symp-
toms such as monocular visual loss and monocular visual
field loss on examination were not detected. This is in
accordance with the findings of Bruno et al9 who
described 70 cases of asymptomatic retinal cholesterol
emboli associated with hypertension, cigarette smoking,
and the presence of heterogeneous or echolucent ca-
rotid plaque on ultrasound.

In 1981 Beal et al4 reported the first case of cerebral
atheromatous embolism after carotid sinus pressure. A
79-year-old man had experienced paroxysmal atrial
tachycardia, which was converted to sinus rhythm by
firm compression of the left carotid sinus for approxi-
mately 4 seconds. One minute after sinus massage he
developed a right hemiparesis and slurred speech. He
died of sepsis 2 weeks after the ictus. Neuropathologic
examination showed typical atheromatous material in
the middle cerebral artery containing an abundance of
cholesterol clefts. In this case the firm compression
maneuver led to the fatal stroke. Friedman4 described
two cases of TIA’s resulting from carotid duplex scan-
ing. Both patients suffered recurrent TIAs during the
weeks before undergoing ultrasound examination. They
experienced brief TIAs, identical to those experienced
previously, under manipulation of the ultrasound trans-
ducer, which was applied with the usual pressure.
During surgery, deep ulcers with fresh thrombus over
the surface were detected at the carotid bifurcations of
these patients.

In conclusion, our case and the two cases of Friedman
suggest that ulcerated lesions in patients with recurrent
TIAs are potentially unstable and that manipulation of
an ultrasound transducer or compression maneuvers
might precipitate embolization from a friable ulcer. In
these cases reverberating maneuvers are probably more
dangerous than a gently performed obstructive manual
compression lasting several seconds.

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