Cerebral Blood Flow and Perfusion Reserve Capacity in Hemodynamic Carotid Transient Ischemic Attacks Due to Innominate Artery Stenosis

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Ischemia in the carotid artery territory due to atherosclerotic stenosis of the innominate artery is rare. We report a case in which transcranial Doppler ultrasonography (TCD) and SPECT with acetazolamide challenge proved the hemodynamic mechanism. The patient presented with three hypotensive TIAs in the right middle cerebral artery territory. Angiography showed a tight innominate artery stenosis and subclavian steal but no coexistent carotid or vertebrobasilar lesion. SPECT showed disturbed regional blood flow in the middle cerebral artery territory along with an exhausted perfusion reserve capacity. After angioplasty, flow velocities were normal and the perfusion reserve was restored. SPECT should be used to select patients at risk susceptible to benefit from angioplasty.

Key Words: innominate artery stenosis; transient ischemic attack; single-photon emission computed tomography; ultrasound

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Atherosclerotic occlusion or stenosis of the innominate artery is rare. In the Joint Study of Extracranial Arterial Occlusions (*1*), frequencies were 0.6% and 4.2%, respectively. These lesions may be asymptomatic or cause ischemic syndrome in the right upper limb, vertebrobasilar system and, less frequently, in the carotid artery territory in the absence of coexistent carotid disease (*2–4*). Although embolic origin has been reported in some cases (*5*), the mechanism of neurologic deficit is commonly hemodynamic (*6,7*). We report a patient in whom SPECT images with acetazolamide challenge and transcranial Doppler ultrasonography (TCD) supported the hypothesis of transient carotid hemodynamic insufficiency. Percutaneous transluminal angioplasty of the innominate artery improved cerebral perfusion.

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CASE REPORT

A 43-yr-old man with a history of alcohol and tobacco addictions developed acute lipothymia and weakness in the left upper and lower limb lasting for 5 min. He did not complain of headaches and had no alteration of consciousness or disorientation in time and space. The attack occured 1 hr after lunch. On admission, neurological and neuropsychological examinations were normal. General examination revealed a right cervical bruit. Blood pressure was 110/70 mmHg on the left. Information about radial and brachial pulses was unavailable at this time. Laboratory tests disclosed an increased rate of LDL cholesterol (170 mg/dl). Brain CT with contrast was normal. Electrocardiogram (ECG), ambulatory 24-hr ECG and transthoracic/transesophageal echocardiography were normal. Cervical Doppler ultrasound was likewise normal. The patient was then discharged without any treatment.

Five months later, the patient suffered two additional transient attacks of left brachiocrural paresis and lipothymia lasting for 3 and 5 min, respectively, without any other symptoms. The attacks were not precipitated by exercise, change of head position or sudden rising from a seated position. The first attack occurred when the patient was at rest 1 hr after breakfast and the second 2 hr after lunch. He was admitted to our department 4 wk later. Neurological and neuropsychological examinations were normal. General examination showed no right radial and brachial pulses without ischemic syndrome in the upper limb. A bruit was heard in the right supraclavicular fossa. Blood pressure was 120/80 mmHg on the left. Blood pressure monitoring revealed several episodes of nocturnal and postprandial hypotension down to 80/50 mmHg. Fundoscopic examination was normal. Laboratory tests disclosed a high rate of LDL cholesterol (167 mg/dl). Brain CT was normal. After angiographic demonstration of a tight stenosis of the innominate artery, percutaneous transluminal angioplasty (PCTA) was successfully performed 6 days after admission without complication. Transient ischemic attacks did not recur over a follow-up period of 12 mo.

METHODS

Doppler Ultrasonography

Cervical and TCD ultrasonography were performed with an EME TC2000 transcranial ultrasound system with a 2-MHz probe

(Überlingen, Germany). The systolic velocities were recorded according to standard techniques for extracranial internal carotid, subclavian, vertebrobasilar and intracranial vessels (*8,9*) and compared with normal values (*9,10*). For the ophthalmic and vertebral arteries, the patient served as his own control because the velocities are dependent on the insonation angle and the recorded segment of the artery. Brachial artery compression and release tests could not be obtained to seek a flow reversal of the vertebral artery recorded. Hypotensive provocative tests were not carried out for ethical reasons.

SPECT Evaluation of Cerebral Perfusion

Regional cerebral blood flow (rCBF) was measured by brain SPECT after the intravenous injection of 20 mCi (740 MBq) ^{99m}Tc-HMPAO under resting conditions with eyes open in a dim, quiet room. Acquisition was carried out 10–30 min after injection of the tracer with a single-head rotating gamma camera (General Electric, Milwaukee, WI) and a standard low-energy, high-resolution collimator using 64 projections of 30 sec each. The matrix size was 64 × 64 pixels (pixel size 6 mm). The raw data were first compressed to permit reconstruction of eight consecutive parallel axial slices every 12 mm parallel to and above the orbitomeatal line.

Filtered backprojection with linearlization was performed for data reconstruction using a Shepp-Logan filter (with Hanning window) which is a modified version of the ramp filter. Because attenuation is only mild in the cerebral cortical areas studied, correction of scatter and attenuation was not used. Furthermore, the semiquantitative analysis used in this study makes ratios of counts not affected by such a method. Visual and semiquantitative analysis were performed. Fourteen symmetrical ROIs (3 × 3 pixels) were automatically located on the cortical ribbon, four in the anterior, middle, and posterior hemispheric areas and one in each cerebellar hemisphere with a computerized program developed in our laboratory (*11*). Thus, the ROIs covered the prefrontal, frontal, frontoparietal, parietal, parieto-occipital and occipital regions. Because of the poor spatial resolution of the device, ROIs were not placed in subcortical regions. In each ROI, the differences in total count were expressed as a percentage of the values from the symmetrical ROI in the contralateral hemisphere (*12–16*). Interhemispheric difference (IHD) of at least 10% was considered to be significant (*12,13,16*). To assess cerebrovascular reserve capacity, acetazolamide (1 g) was given intravenously 10 min before the SPECT examination in accordance with the method used in previous studies (*17–21*). Stimulations were performed before (AZ1) and after (AZ2) angioplasty.

RESULTS

Cerebral arterial angiography demonstrated a tight stenosis of the innominate artery without floating thrombus as well as a retrograde flow in the right vertebral artery and slow flow in the right internal carotid artery (Fig. 1A). There was no extracranial or intracranial carotid or vertebrobasilar atherosclerotic disease. After PCTA, the innominate artery's tight stenosis was removed, but the residual lumen was estimated to be narrowed by one-third (Fig. 1B).

Cervical Doppler ultrasound and TCD demonstrated reduced flow velocities in the right subclavian artery, common carotid artery (CCA), internal carotid artery (ICA), middle cerebral artery (MCA) and ophthalmic artery (OA).

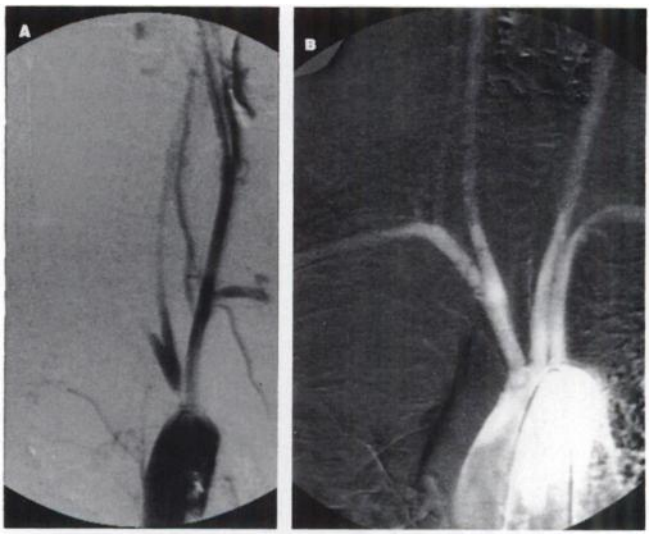

FIGURE 1. Aortic angiogram shows a tight stenosis of the innominate artery (A) and its partial removal after angioplasty (B).

By contrast, flow velocities were increased in the left MCA and vertebral artery (VA) (Table 1). The flow in the right VA was anterograde and so reduced that precise calculation of flow velocity was impossible. Two days after angioplasty, flow velocities, though remaining abnormal, increased in the right subclavian artery and ICA and were normal in the CCA, MCA, OA and VA. Moreover, the velocities increased in the basilar artery and decreased within the normal range in the left CCA, ICA, MCA and VA. Therefore, angioplasty restored normal intracranial flow (Table 1).

All data for the ROIs on the OM + 48 mm, OM + 60 mm and OM + 72 mm slices before and after PCTA in the right symptomatic and left asymptomatic cerebral hemisphere

TABLE 1
Doppler Ultrasonography Data before and after PCTA

Artery	Depth (mm)	Systolic velocity (cm/sec) Normal values	Before PCTA	After PCTA
R subclavian		70–90	35	50
L subclavian			185	180
R CCA		70–100	36	80
L CCA			70	60
R ICA		89 ± 23	31	45
L ICA			90	60
R MCA	52	94 ± 23	55	95
L MCA	52		140	100
R OA			20	35
L OA			50	38
R VA	70		—	90
L VA	70		220	90
BA	96	64 ± 13	70	100

PCTA = percutaneous transluminal angioplasty; CCA = common carotid artery; ICA = internal carotid artery; MCA = middle cerebral artery; OA = ophthalmic artery; VA = vertebral artery; BA = basilar artery.

TABLE 2
Interhemispheric Differences in the Right Symptomatic and Left Asymptomatic Cerebral Hemispheres

		OM + 48 cm			OM + 60 cm			OM + 72 cm		
ROI	Side	Basal	AZ1	AZ2	Basal	AZ1	AZ2	Basal	AZ1	AZ2
PF	R	1	−6	0	−5	−3	0	0	−2	−3
	L	0	8	0	6	5	0	1	3	4
F	R	0	−5	−1	−1	−4	−5	2	−2	−3
	L	0	7	2	2	5	7	−1	−3	4
FP	R	−5	−12	−5	−6	−12	−4	−7	−12	5
	L	6	14	7	7	15	5	9	15	−4
P	R	1	−3	−3	−5	−4	−2	−1	2	3
	L	0	5	4	6	5	4	2	−1	−1
PO	R	−2	−1	−1	3	−11	3	−2	−10	1
	L	3	2	2	−2	14	−1	3	13	0
O	R	6	−12	0	1	−10	0	−2	−8	0
	L	5	15	0	0	13	0	3	9	0

Interhemispheric differences (%) calculated in each ROI on the OM + 48 mm, OM + 60 mm and OM + 72 mm slices without (basal) and with acetazolamide testing before (AZ1) and after (AZ2) PCTA: PF = prefrontal, F = frontal, FP = frontoparietal, P = parietal, PO = parieto-occipital, O = occipital.

are given in Table 2. The other slices did not show any significant interhemispheric differences for the three SPECT examinations. SPECT performed 30 days after the last attack showed a mild but nonsignificant hypoperfusion in the right cortical frontoparietal area (Fig. 2A). On the second SPECT examination, the acetazolamide test induced a CBF increase in the left cortical regions and enhanced a more marked cortical hypoperfusion in the right frontoparietal and parieto-occipital regions (Fig. 2B). Three days after angioplasty, SPECT with acetazolamide test showed no significant asymmetry between either hemisphere and was considered normal (Fig. 2C).

DISCUSSION

Innominate artery stenotic lesions are commonly atherosclerotic and predominate in the initial third of the artery (*2*). Although all series of patients with innominate artery atherostenosis have mentioned a high frequency of coincidental carotid and vertebral artery disease (*1,4,22,23*), our patient did not present such a coexistent lesion. In a large series of 324 patients with subclavian steal phenomenon, Hennerici et al. (*4*) reported only six patients with hemispheric symptoms due to innominate obstruction without carotid disease. The most common neurologic symptoms are related to vertebrobasilar insufficiency (*3,4,22,24,25*). Hemiparesis, hemihypesthesia or monoparesis are rare and usually explained by coexistent extra- or intracranial carotid artery occlusive disease (*2,4,26*) which was absent in our case. The neurological deficit, Doppler ultrasonography and SPECT results were compatible with a focal ischemia in the right MCA territory. Despite a subclavian steal, the blood flow in the basilar artery was normal. Moreover, the absence of associated vertebrobasilar symptoms excludes the likelihood of vertebrobasilar insufficiency to explain the pure motor deficit. Lacunar syndrome is unlikely in the absence of hypertension, diabetes mellitus or angiographic findings of atherosclerotic disease of the basilar artery.

The absence of floating thrombus and the repetitive TIAs in the same territory make a mechanism of emboli originating from the innominate stenosis unlikely. The simultaneous occurence of TIAs and hypotensive lipothymia is consistent with transient carotid hemodynamic insufficiency. Angiography, Doppler ultrasonography and SPECT with acetazolamide test showed reduced CBF in the right carotid system. Grosset et al. (*27*) reported one patient in whom SPECT showed similar reduced hemispheric perfusion ipsilateral to innominate occlusion. In our patient, only SPECT with acetazolamide test showed hypoperfusion reflecting an exhausted cerebrovascular reserve capacity in the right MCA territory. Indeed, the marked CBF increase in the left cortical regions without response in the right MCA

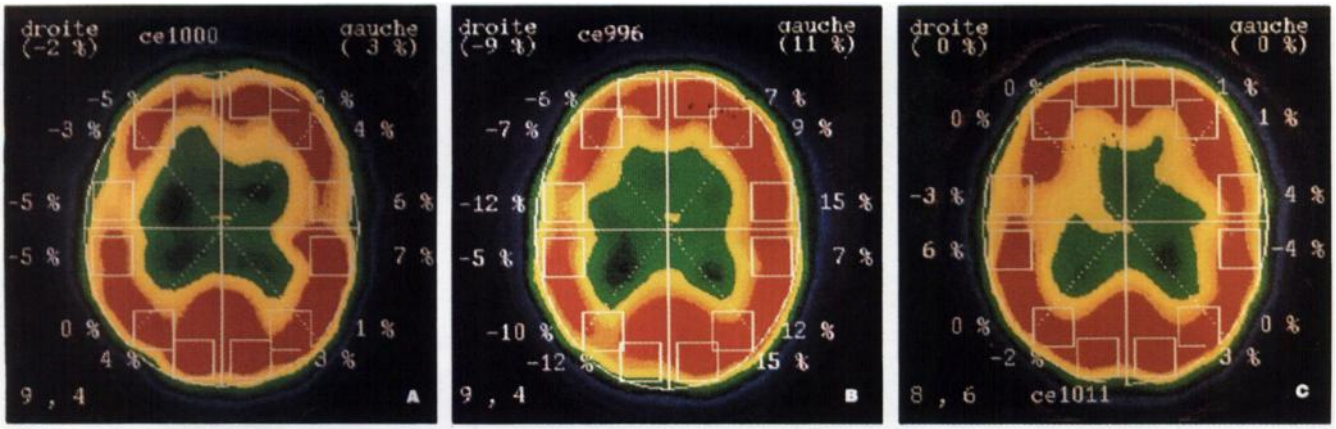

FIGURE 2. HMPAO-SPECT (OM + 60 cm) performed at baseline with and without (A) acetazolamide before (B) and after innominate artery angioplasty (C).

territory enhanced the interhemispheric difference with a right relative hypoperfusion. Acetazolamide stimulation offers a simple and reliable method for assessing the cerebrovascular reserve capacity in occlusive extracranial diseases (*17–21*). The significant restriction of compensatory vasodilatation makes the CBF directly dependent on blood pressure. In our patient, blood pressure monitoring revealed several periods of nocturnal and postprandial hypotension. We hypothesize that some episodes of more severe hypotension associated with tight innominate artery stenosis could be responsible for worsening of pre-existing hemodynamic insufficiency. Failure of autoregulation causing local hemispheric ischemia in the presence of a drop in systemic blood pressure in regions of already disturbed circulation has been stressed by Hennerici et al. to explain hemispheric events without coincidental carotid obstruction (*4*).

After PCTA, the flow velocities in the right extracranial carotid arteries and subclavian artery increased enough to restore normal CBF and vascular reserve capacity in the MCA territory, which resulted in cessation of ischemic attacks. This is also supported by the slight decrease of velocities in the left carotid arteries and MCA which likely supplied the right MCA through collaterals before angioplasty. Similar restoration of blood flow has also been well documented by Doppler and SPECT studies after carotid artery endarterectomy (*28*).

Thus, in patients with innominate artery stenosis without coexistent carotid occlusive lesion, HMPAO-SPECT studies of CBF and perfusion reserve capacity, despite semiquantitative analysis, may ascertain the presence of carotid hemodynamic insufficiency and should be used to select at risk patients who can benefit from angioplasty.

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