References


Response

From our experience using HMPAO alone in about 800 acute and subacute stroke cases, we have found (as have Baird and Donnan) that increased HMPAO uptake within 48 hours of stroke onset is uncommon and tends to occur in regions without infarct on CT performed at a later date. Also, we are concerned about whether 99mTc-HMPAO images can be trusted to represent cerebral blood flow in acute stroke. As described in our response to Limburg and van Royen,1 we have not yet observed hyperfixation of 99mTc-HMPAO relative to 133Xe measured blood flow in strokes less than 10 days from onset of symptoms. In one case studied 44 hours from onset (out of three studies within 48 hours with 99mTc-HMPAO and 133Xe single-photon emission computed tomography [SPECT]), we observed increased HMPAO uptake (25% above that of the unaffected homologous region) in the posterior two thirds of the middle cerebral artery territory, which was matched by a similar increase in 133Xe blood flow in an area without infarction on CT at day 6 (the other two cases showed both low HMPAO uptake and blood flow). Furthermore, in the subacute phase we have thus far observed hyperfixation only in areas of infarction as seen on CT. Thus, according to our data, hyperfixation of HMPAO occurs only after stroke enters the late subacute phase and most likely only in areas of infarction. Acute or early subacute increased uptake of HMPAO most likely represents hyperemia and does not overestimate blood flow, as in the case of hyperfixation.

Most of our acute cases of hyperemia with HMPAO in areas without or with only small infarcts on CT were restudied with HMPAO. In several of these patients we observed in the chronic phase a slight, diffuse reduction of cerebral blood flow in the whole area or subregions of focal hypoperfusion despite a normal CT scan or magnetic resonance imaging. We wonder whether Baird and Donnan have found this also. Perhaps these hypoperfusions represent incomplete infarction with loss of solitary or small groups of neurons yet with preservation of gross tissue structure, a type of infarct undetectable on CT but visible on SPECT.

B. Sperling, MD
N.A. Lassen, PhD
Bispebjerg Hospital
Copenhagen, Denmark

Reference


Stroke Triggered by an Asthma Attack

Incidence of cardioembolic stroke is estimated at 15% to 19% of stroke patients studied by transesophageal echocardiography (TEE). A patent foramen ovale (PFO) has been reported in 72% to 85% of patients with ASA assessed by TEE. Valsalva maneuvers, cough, defecation, and sexual intercourse may enhance or provoke interatrial right-to-left shunt and are suspected of playing a role in stroke in some patients. We describe a patient with ASA and PFO who suffered an embolic stroke in the middle cerebral artery (MCA) territory that was triggered by a severe asthma attack. To our knowledge, no such case has been previously reported.

A 64-year-old woman was admitted for a severe asthma attack complicated by a right hemiparesis and loss of consciousness. Her history included moderate hypertension and asthma. She had no known phlebitis and did not take hormonal therapy. On admission, neurological examination showed stupor, a right lateral conjugate gaze palsy, right central facial paralysis, and a progressive right hemiplegia within a few hours. General examination showed cyanosis, pulmonic sibilances, blood pressure of 160/100 mm Hg, and heart rate of 108 beats per minute. Laboratory tests, including extensive coagulation tests, were normal, but PAO2 was 67 mm Hg. Brain computed tomography (CT) showed a mild hypodensity in the whole territory of the left MCA territory and a spontaneous hyperdensity of the MCA proximal segment. Doppler ultrasonography of the extracranial arteries was normal. Transcranial Doppler showed a normal flow blood in the right MCA but failed to detect any signal in the left MCA. A 12-lead electrocardiogram (ECG) and 24-hour ECG Holter monitoring showed no embolicigenic cardiac arrhythmia. Transthoracic echocardiography was normal. Contrast TEE combined with a microbubbles test detected an ASA (type 1c, following the criteria of Hanley et al) associated with a PFO. Valsalva maneuvers and cough tests could not be performed because of the important disability of the patient. No other cardiac abnormalities or signs of pulmonary hypertension were noted. Treatment was acetylsalicylic acid (500 mg/day) and oxygen 3 L/min. Twelve hours after the onset, she was alert, and neurological examination showed a global aphasia and right hemiplegia. After 1 week, the control transcranial Doppler demonstrated recanalization of the occluded left MCA. Brain CT showed a large left MCA infarct. Two weeks after admission, the patient exhibited symptoms of pulmonary embolism confirmed by blood gas analysis and pulmonary scintigraphy. Phlebography showed an old deep-vein thrombosis in her right leg. Over the next 4 weeks, she mildly recovered the walk while keeping a motor aphasia and right brachial monoplegia.

The abrupt stroke onset and spontaneous recanalization of the occluded artery strongly suggested a mechanism of cerebral embolism. In the absence of carotid disease or embolicigenic cardiac arrhythmia, the MCA infarct was likely caused by emboli released from an ASA or a PFO. Right-to-left shunting through a PFO may exist in basal conditions or with some diseases that increase the right atrial pressure, such as right ventricular infarction, primary pulmonary hypertension, and pulmonary lesions such as contemporary pulmonary embolism. In our patient, the medical history and echocardiography showed no such conditions. The pulmonary embolism occurred 2 weeks after stroke onset so that it could not be incriminated as a primary factor favoring a right-to-left shunting through a PFO. However, the latter may be transiently elicited by the cardiac cycle, inspiration, Valsalva maneuvers, cough, straining at stool, weight lifting, or sexual intercourse. In our patient, the severe asthma attack with forced expirations combined with hypoxia likely increased the interatrial shunting and possibly released sequestered emboli from the right atrium or ASA. As mentioned in two recent reports, the source of emboli is often undetected. Likewise, in our patient, a mechanism of paradoxical embolism remained uncertain because without history of prior phlebitis, the old deep-vein thrombosis demonstrated 2 weeks after admission in the paralyzed leg could not be implicated with certainty. We therefore hypothesize that ASA, a frequently PFO-associated cardiac abnormality, was the likely source of emboli since a true paradoxical embolism could not be definitely ascertained.

This case highlights the role of ASA and PFO in cerebral embolism triggered by asthma attack, which can be added to the list of Valsalva-inducing activities involved in stroke onset.

Patrice Laloux, MD
Michel Ossemann, MD
Department of Neurology
Baudouin Marchandise, MD
Department of Cardiology
Mont-Godinne University Hospital
Belgium
Stroke triggered by an asthma attack.
P Laloux, M Ossemann and B Marchandise

Stroke. 1993;24:1262-1263
doi: 10.1161/01.STR.24.8.1262

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/24/8/1262.citation