Response

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References


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Reference


Stroke Triggered by an Asthma Attack

Incidence of cardioembolic stroke is estimated at 15% to 19%1,2 and prevalence of atrial septal aneurysm (ASA) at 15% in stroke patients studied by transthoracic echocardiography (TEE). A patent foramen ovale (PFO) has been reported in 72% to 85% of patients with ASA assessed by TEE.3,4 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.5,6 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, pulmonary 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 blood flow 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 Ic, following the criteria of Hanley et al7) 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 conditions8 or with some diseases that increase the right atrial pressure, such as right ventricular infarction,9 primary pulmonary hypertension,10,11 and pulmonary lesions such as contemporary pulmonary embolism.11,12 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.5,6,13,14 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,8,9 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.

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Stroke triggered by an asthma attack.
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