Intermittent Broca’s aphasia management in an emergency unit: from theory to practice

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Abstract Although Broca’s aphasia (BA) may mimic different neurological illness, its sudden onset often requires an emergency approach. In this paper, the management of a case of intermittent BA occurred in a young woman without history of neurological, cardiovascular and arrhythmic diseases is discussed. Diffusion-weighted magnetic resonance imaging showed two areas of hypoperfusion in the terminal branches of the left medial cerebral artery not previously diagnosed by computed tomography. Although there were no eligibility criteria for thrombolysis, patient received intravenous treatment with recombinant tissue-type plasminogen activator (rt-PA) over 1 h and at the end of rt-PA infusion aphasia completely disappeared without neurological sequelae. Transesophageal echocardiography revealed a thrombus in the left atrial appendage not previously detected by transthoracic echocardiography. In the month following the cardioembolic stroke, heart rhythm was monitored for 30 days by an external loop recorder and during this test two episodes of silent lone atrial fibrillation were collected.

Keywords Broca’s aphasia · Lone atrial fibrillation · Thrombolysis

Introduction

Broca’s aphasia (BA), also called ataxic, expressive or non-fluent aphasia is commonly observed following a cerebral vascular event within the left hemisphere at the Broca’s area, a territory supplied by the left medial cerebral artery (MCA). Although BA may mimic different neurological illness, its sudden onset often requires an emergency approach. Risk of MCA stroke increases with age and its highest incidence is in the seventh and eighth decade of life. On the contrary, in young people, MCA stroke is far less common and haemorrhagic stroke is the most common aetiology, while dissection atherosclerosis and thromboembolic occlusion are relatively rare causes. In this paper, the management of a case of intermittent BA occurred in a young adult without history of neurological, cardiovascular and arrhythmic diseases is discussed.

Case report

A 41-year-old woman was admitted to our emergency department for BA without aura or visual disturbance 7.40 a.m., 10 min after waking up. She denied arterial hypertension, smoking, drugs or alcohol intake, except for episodes of migraine headaches with visual aura until 6 years earlier. She has taking contraceptive medication by vaginal
administration (NuvaRing) since 4 years ago. At physical examination, cranial nerve motility was normal and there were no focal neurological signs; the patient did not have fever or meningeal syndrome. She speaks haltingly, without intonation and had difficulty in producing spontaneous speech, naming and repeating. Cardiac examination revealed a soft grade 2/6 systolic ejection murmur at the second left intercostal space and cardiac apex; the heart rate was rhythmic. There were no carotid murmurs and blood pressure was 120/82 mmHg. 40 min after the onset of symptoms, aphasia spontaneously resolved but reappeared 2 min later associated with agraphia and acalculia. The patient underwent non-contrast cerebral computed tomography (CT) that was negative for intracranial and subarachnoidal bleeding. Diffusion-weighted magnetic resonance imaging (MRI) showed two areas of hypoperfusion in the terminal branches of the left MCA, respectively in the territory of superior and inferior branches (Fig. 1). At 8.55 a.m., the patient was transferred to our neuroscience intensive care unit. At 9.05 a.m., aphasia disappeared for about 15 min and during this time neurological examination remained unchanged. The electroencephalogram did not detect abnormalities suggestive of epilepsy or migraine. Transthoracic echocardiography (TTE) revealed a mild patent foramen ovale (PFO) and a mild mitral valve insufficiency; atrium size was normal. Transcranial Doppler (TCD) ultrasound investigation performed in basal condition detected many microembolic signals with a “shower-curtain” pattern after 10 s from the saline bubble injection. The TCD did not report intracranial arterial stenoses or occlusions. At the end of this examination, BA recurred with left migraine.

No stenosis or sub-intimal dissection in the carotid arteries or deep venous thrombosis in the legs was observed during colour-Doppler ultrasonography. Global neurological impairment was evaluated applying the National Institutes of Health Stroke Scale (NIHSS) score [1], and according to this scale the patient had a low NIHSS (2–3). The same low neurological impairment was observed applying the ABCD score (score 3) recommended by the Stroke Prevention and Educational Awareness Diffusion (SPREAD) guidelines (http://www.spread.it).

Taking into account the patient’s age, her low cardiovascular risk and the two cerebral lesions on MRI we decided to start a thrombolysis treatment with recombinant tissue-type plasminogen activator (rt-PA). At 10.00 a.m., precisely 2 h and 20 min after the onset of the symptoms, the patient received intravenous (IV) alteplase at the recommended dosage of the 0.9 mg kg$^{-1}$; the first 10% of the calculated dose was given as IV bolus and the remaining 90% of dose was given in infusion after 1 h. At the end of rt-PA infusion, aphasia completely disappeared; no complications were observed during thrombolysis and in the following 24 h.

The day after the patient underwent transesophageal (TEE) echocardiography that revealed a thrombus in the left atrial appendage (Fig. 2), the PFO previously during TTE was excluded by TEE. During the hospitalisation, the patient performed an electrocardiogram Holter monitoring for 72 h that did not find any arrhythmia. Laboratory, thyroid and inherited thrombophilia tests were normal. In order to exclude paraneoplastic syndrome procoagulant state, the patient underwent a total body contrast-enhanced CT that showed two perfusion defects in the upper part of left kidney. No pulmonary arteriovenous malformations were detected during CT. Anticoagulation treatment with warfarin was started and patient was discharged without neurological sequelae after 10 days from the cardioembolic stroke.

In the month following, heart rhythm was monitored for 30 days by an external loop recorder (3300 BT device, Vithapone, Mannheim, Germany). During this test two
episodes of lone atrial fibrillation (AF) were recorded. The first AF episode lasted from 6.04 to 7.12 a.m. and the second for six consecutive hours (from 12.30 to 18.30 p.m.). In particular, AF appeared 45 and 52 days after the cerebrovascular event.

Discussion

The management of our case raises important questions. Was thrombolytic treatment indicated in this patient? According to the American Stroke Association guidelines for early management of adult with ischemic stroke, in subjects with low NIHSS score IV thrombolysis is not recommended [1]. In minor strokes, the decisions to perform thrombolysis should not be influenced by the risk of intracerebral haemorrhage (ICH) and systemic bleeding rather than its clinical benefit. In fact, in the National Institute of Neurological Disorders and Stroke (NINDS) study [2], the rate of ICH was very low in ischemic stroke with a lower NIHSS score. Similar results were found by Vaishnav et al. [3] in patients with mild ischemic stroke and by Baumann et al. [4] in patient with an initial NIHSS score of five.

In clinical practice MRI should be required for all new stroke patients? In subjects with acute neurological impairment, non-contrast CT imaging is used to evaluate for infarct and to exclude haemorrhage and other structural lesions that may mimic stroke. The evidence from our experience confirms that MRI is accurate and superior to CT for the diagnosis of acute ischemic stroke and remains the investigation of choice in patients wherever there is diagnostic uncertainty. However, a common belief in clinical practice is that it is logistically impossible and very impractical to perform an MRI for all new stroke patients. This opinion derives mostly from the availability, cost and time required to perform an MRI than other imaging techniques. Furthermore, it would be illogical not to use all available neurological imaging to diagnose an acute cerebral ischemic event particularly in a young subject. In young stroke there may have devastating consequences with respect to quality of life and work ability and often have neuropsychological or social sequelae.

In conclusion, the main issues of this paper are:

1. MCA strokes are complex pathologies which need a mixed team of highly skilled cardiovascular professionals to increase the global effectiveness and safety of different diagnostic and therapeutic decisions.
2. Before excluding AF as a cause of cardioembolic stroke, a longer heart rate monitoring using telemedicine should be performed.
3. Thrombolytic therapy should be based on rational protocols and individual circumstances, rather than blind adherence to the guidelines. However, clinical trials are needed to evaluate the effectiveness and long-term outcomes of thrombolysis in young subjects with a low NIHSS or ABCD score.

Conflict of interest The authors declare no competing interest.

References