Case report

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Headache attributed to ischemic stroke – a new scope for management: a case study

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Abstract

Acute ischemic stroke may present with serious clinical manifestations. Headache attributed to ischemic stroke is one of these clinical manifestations which may be neglected and affect the functional outcome of the patients. Understanding the exact pathophysiology, and recognition of the most clinical and radiological predictors can help to provide good management and open scope for prophylactic approaches. Here, we report a case presented with acute onset of ischemic stroke and developed a new onset headache on the first day of stroke onset. According to the International Classification of Headache Disorders third edition (ICHD-3), the patient has a post-stroke headache. Using transcranial duplex ultrasound, activation of the trigeminovascular pathway could be attributed to the opening of more pain-sensitive collateral channels as the left posterior communicating artery (P Com A). In conclusion, we can predict the development of acute headache at stroke onset based on different clinical and radiological factors. Opening of the collateral channels is strongly implicated in the production of post-stroke headache.

Key words: *post-stroke, acute headache, duplex, collateral.*

Introduction

Acute ischemic stroke may present with serious clinical manifestations. Headache attributed to ischemic stroke is one of these clinical manifestations which may be neglected and affect the functional outcome of the patients [8]. Understanding the exact pathophysiology and recognition of the most clinical and radiological predictors can help to provide good management and open scope for prophylactic approaches [6]. The intracranial stimulation of the pain-sensitive structure as well as acute vasodilation of the potential closed collateral channels was strongly implicated in the development of such headache through the activation of the trigeminovascular system [1].

Case study

A male patient, 56 years old, was known to be diabetic and hypertensive (uncontrolled) with a history of an old stroke and he was a heavy smoker. The patient had a history of ischemic heart disease and underwent a coronary artery bypass graft operation. There were no other special habits of medical importance and the patient is right-handed. The family history was irrelevant. The patient experienced a left hemicranial migraine-like headache 6 months ago, with a frequency of about 3 to 5 times per week. The patient presented to the emergency department with a sudden onset of left-side hemiparesis of pyramidal distribution, unsteadiness of gait during walking more to the left side, repeated vomiting,

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vertigo, and diplopia. The patient did not report any disturbed conscious level, fever, and seizure. On the first day of admission, the patient complained of a right frontoparietal headache, of moderate intensity, that was tightened in character unlike the previous headache he had experienced and sometimes associated with nausea and vomiting. On examination, pulse was 75 beats/min, blood pressure was 110/70 mmHg and respiratory rate was 16/min. He was alert, with mild dysarthria, right abducent and facial nerve affection, bilateral nystagmus, right homonymous hemianopia, left-side hemiparesis (upper limb 4/5, and lower limb 3/5), and extensor planter response bilaterally. There was bilateral ataxia more on the left side, and the rest of the examination was normal. The patient was diagnosed, according to the Oxfordshire Community Stroke Project, with acute ischemic stroke (posterior circulation syndrome). The clinical presentation associated with good clinical status on the National Institutes of Health Stroke Scale (NIHSS) was 9. Magnetic resonance imaging of the brain was done and revealed a right pontine infarction, bilateral cerebellar hemisphere, and occipital infarction more on the left, left thalamic, and high parietal infarction. In addition, there was a picture suggestive of small vessel disease. The patient was admitted to the stroke unit and routine laboratory investigations were taken. The patient received antiplatelet medication and supportive treatment as the patient presented out of the time window. Extracranial and transcranial colour-coded Duplex ultrasonography of the patient was carried out and revealed the following findings:

Carotid arteries:

– diffuse increase in intima-media thickness on both sides,

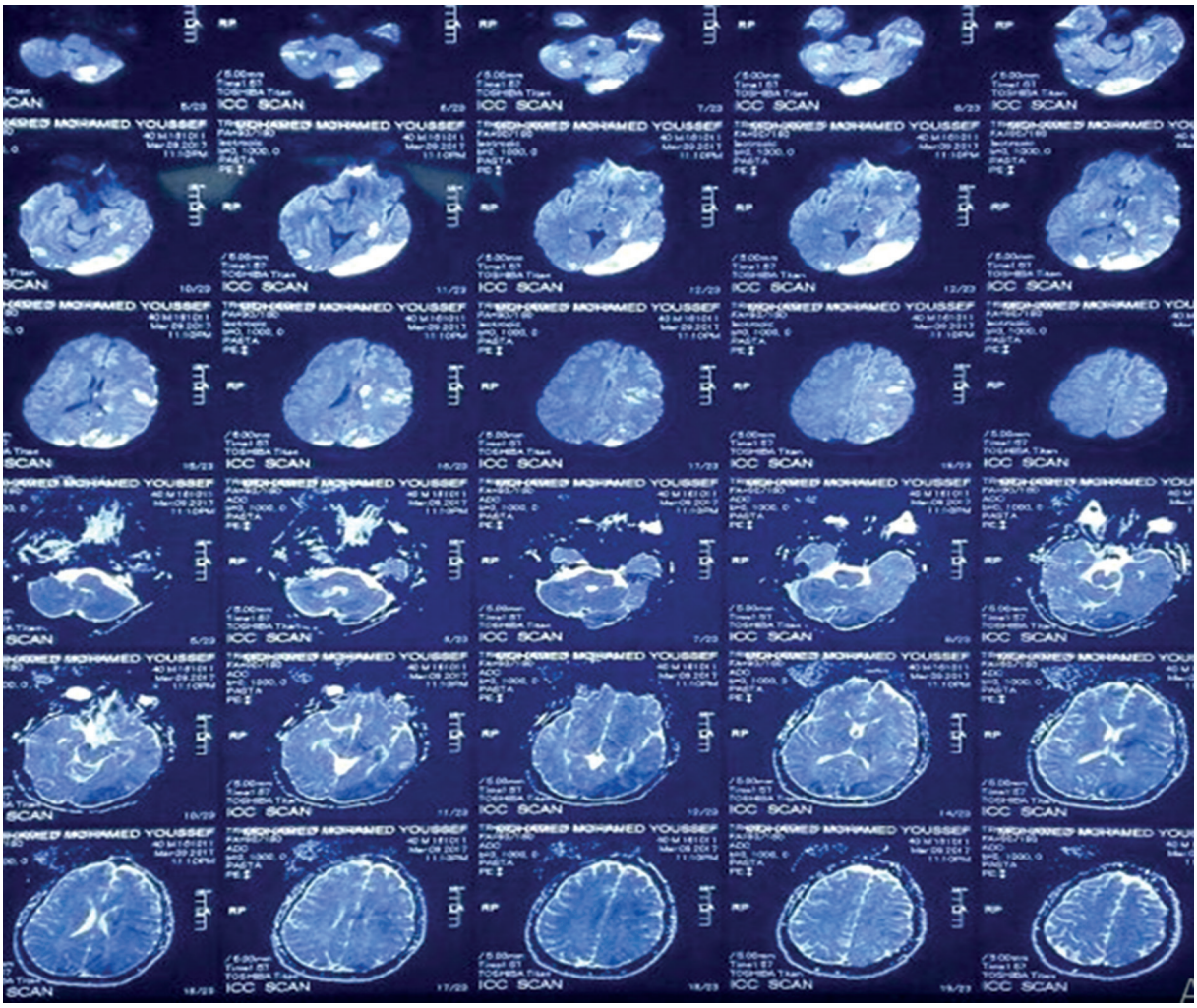

Fig. 1. MRI brain: right pontine infarction, bilateral cerebellar hemisphere and occipital infarction more on the left, left thalamic and high parietal infarction.

– right internal carotid artery (ICA) proximal stenosis > 80% by North American Symptomatic Carotid Endarterectomy Trial (NASCET) and flow analysis,
– bilateral complicated plaques at the level of both bulbs cause no major hemodynamic changes on the left side.

Vertebral arteries:

– complete left subclavian steal indicating severe stenosis vs. occlusion of the proximal left subclavian artery,
– compensatory increase in the right vertebral flow velocity.

Transcranial colour-coded Duplex examination:

– right ICA blunted flow with the good collateral filling of the right middle cerebral artery (MCA) and anterior cerebral artery (ACA) *via* the right posterior communicating artery (P Com A),
– increased velocity in other arteries of the circle of Willis indicating possible moderate intracranial stenosis.

Discussion

Regularly updated data on stroke and its pathological types, including data on its risk factors, incidence, prevalence, mortality, disability, and associated comorbidities, are important for evidence-based stroke care planning and resource allocation [4]. One of the most significant comorbidities accompanying ischemic stroke is the headache attributed to ischemic stroke. Furthermore, this headache is often confusing to clinicians as it is probably masked by serious clinical manifestations of stroke [9]. Therefore, it may be neglected and become severe and persistent and can affect the functional outcome of the patient [3]. Moreover, we must increase our knowledge about the prevalence of acute headache at stroke onset, important clinical and radiological predictors, clinical characteristics of the headache, possibly associated variables that increase the chance of its development and understanding of

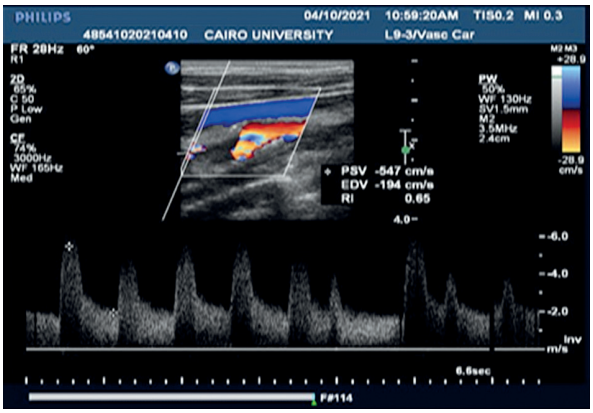

Fig. 2. Right internal carotid artery (ICA) showed increased flow velocity (peak systolic velocity [PSV] 550 cm/s).

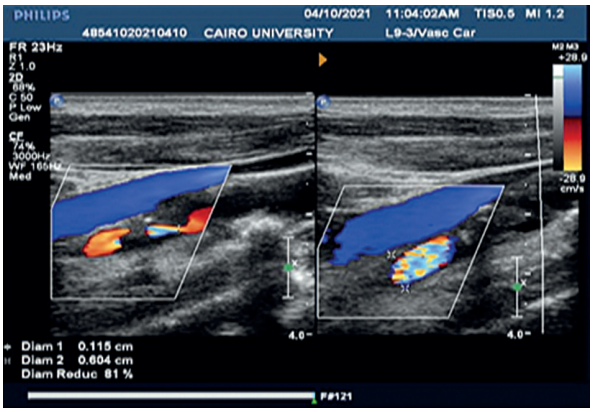

Fig. 3. Right extracranial internal carotid artery (ICA) stenosis > 80%.

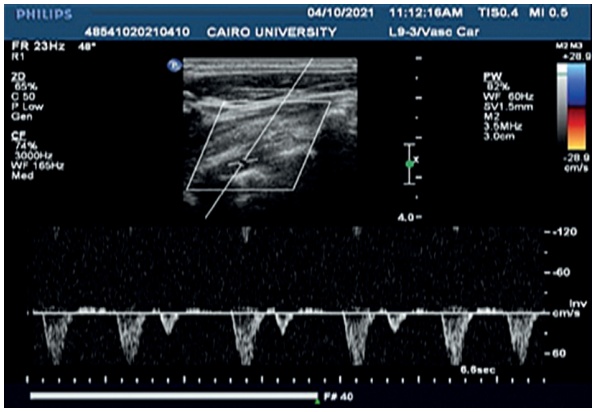

Fig. 4. Complete subclavian steal seen in the left vertebral artery with inverted flow (proximal left subclavian severe stenosis vs. occlusion).

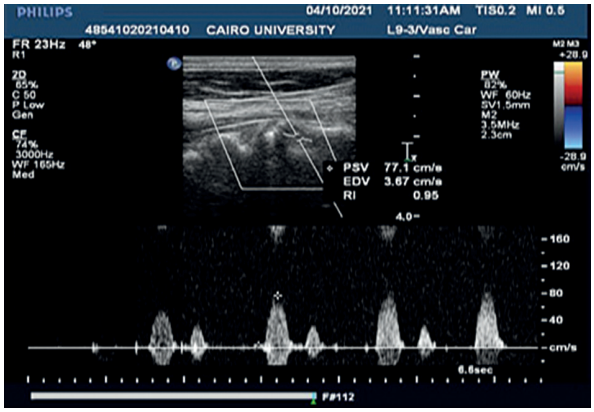

Fig. 5. Right vertebral (compensatory flow) (peak systolic velocity [PSV] 109 cm/s).

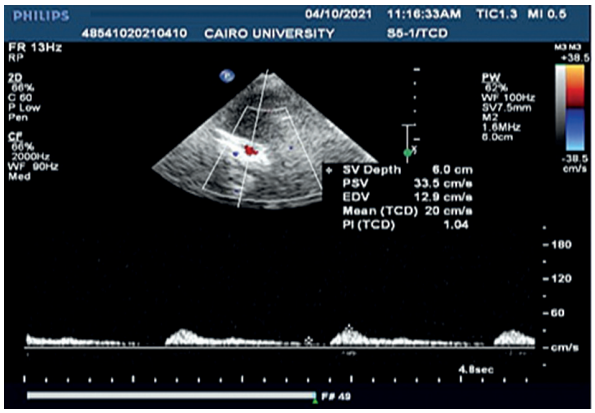

Fig. 6. Right internal carotid artery (ICA; intra petrous part) (Blunted flow peak systolic velocity [PSV] 34 cm/s).

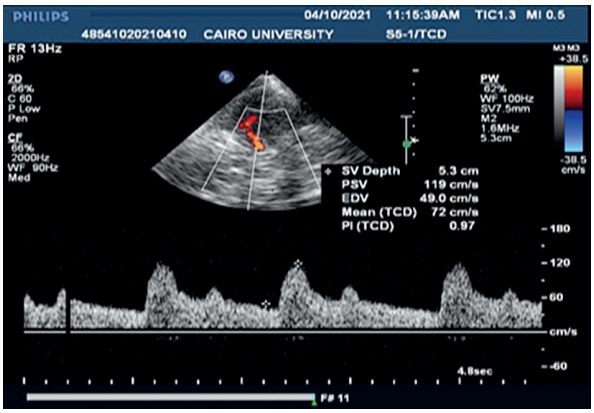

Fig. 7. Right middle cerebral artery (MCA; peak systolic velocity [PSV] 120 cm/s).

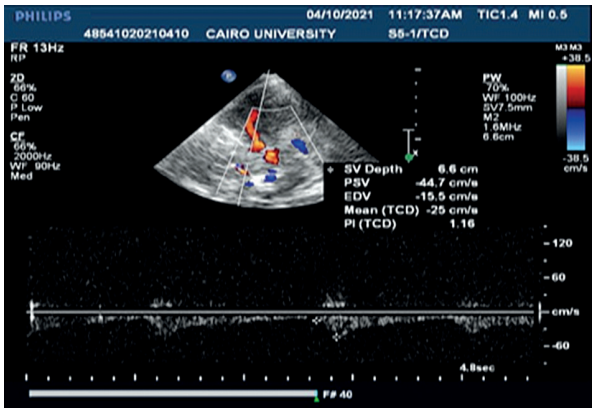

Fig. 8. Right anterior cerebral artery (ACA; peak systolic velocity [PSV] 44 cm/s).

the underlying pathophysiology. Hence, we can provide better prophylactic management and improve the functional outcome. We reported that a male patient complaining from a right frontoparietal headache, of moderate intensity, was tightened in character associated with nausea and vomiting and was of new onset. This could be attributed to the activation of the trigeminovascular pathway [9]. This could be explained by intracranial vasodilation or increased flow through opened collaterals during the acute phase of ischemic stroke as right ICA showed blunted flow with the good collateral filling of the right MCA and ACA *via* the right P Com A. In our case, the onset of headache was in a very close temporal relation with the stroke symptoms; on the first day of stroke onset. This could be explained by the endothelial release of bradykinins, prothrombotic factors, interleukin-6, nitric oxide metabolites, and excitotoxic substances during the acute phase of ischemic stroke. Although cardioembolic stroke is more frequently associated with the production of headache at stroke onset, our case showed carotid artery stenosis > 80%, indicating that ischemic stroke was attributed to large vessel atherosclerosis. This could explain the significant association between thrombotic, large vessel disease, and post-stroke headache rather than cardioembolic stroke. Moreover, the prevalence of headache associated with large vessel disease was reported more frequently than with small vessel disease, and this indicates the important implication of arterial calibre in headache production. Furthermore, enhanced activation of the trigeminovascular pathway, in addition to collaterals opening, occurs mainly ipsilateral to the infarction explaining the development of ipsilateral right hemicranial post-stroke headache in our case [2]. Ischemia of the central structures involved in the pain-modulating system can provoke the development of headache. This could be explained in our case by increased velocity in other arteries of the circle of Willis indicating possible moderate intracranial stenosis which participated in the central ischemia besides the stroke mechanism [5]. Our patient experienced an acute headache at the onset of clinical manifestations of posterior circulation infarction; in the vertebrobasilar territory. This could be explained by dysfunction of the serotoninergic nuclei of the brainstem, ischemia of the trigeminal nucleus, or the dura of the posterior fossa [7]. This could give an acceptable explanation of why the acute headache at stroke onset is strongly associated with posterior circulation infarction in our case. Finally, the literature suggested the headache attributed to ischemic stroke was not related to intracranial vasodilation or increased flow through collateral circulation [9]. This could be assigned to the lack of association between post-stroke headache and angiographically demonstrated collateral circulation [7]. Moreover, through our case, we added valuable data to the understanding of the pathophysiology, clinical and radiolog-

ical predictors for post-stroke headache through the assessment of cerebrovascular structures and hemodynamics using duplex ultrasound.

Conclusions

We can predict the development of acute headache at stroke onset from different clinical and radiological factors. A history of old infarction, pre-stroke headache, posterior circulation infarction, large artery disease, obesity, lack of sleep, and presence of psychological stress were the most prevalent clinical predictors. The most frequent radiological predictors were the presence of a picture suggestive of small vessel disease, and recent infarction, especially in the cerebellum. Opening of the collateral channels is strongly implicated in the production of post-stroke headache. Increased knowledge about the nature of this headache provides better prophylactic approaches and hence improves the outcome of stroke.

Ethics approval and consent to participate

This study was conducted in concordance with the Declaration of Helsinki and the participant signed written informed consent before being enrolled in the study. The institutional review board (IRB) approval was obtained from the ethical committee of the Neurology Department, Faculty of Medicine, Al-Azhar University, Cairo. The ethical code is Near-Med._70. Headache attributed to ischemic stroke; a new scope for management; a case study._000070

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Disclosure

The authors report no conflict of interest.

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