SHORT REPORT

Orgasmic headache and middle cerebral artery dissection

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A patient with a 20 year history of primary orgasmic headache is described who, after suffering an unusually severe episode of orgasmic headache was found to have a middle cerebral artery dissection. This unusual association of primary and secondary orgasmic headache emphasises the need for a thorough diagnostic examination when the orgasmic headache differs from that of previous episodes or is associated with neurological symptoms.

Orgasmic headache (OH), defined as an explosive headache occurring at orgasm, is most frequently a primary headache disorder.^{1,2} However a number of conditions, mostly vascular, such as subarachnoid haemorrhage, cerebral infarction or haemorrhage, and reversible segmental vasoconstriction occasionally present with OH. We report on a patient with a 20 year history of typical primary OH who had an unusually severe episode of OH showing a middle cerebral artery (MCA) dissection.

CASE REPORT

A 44 year old man was admitted for an unusually severe episode of OH. He had a history of cigarette smoking and mild untreated hypercholesterolaemia. He had no history of migraine but for the past 20 years he experienced typical OHs two to three times a year. Two days before admission, he experienced an excruciating throbbing right frontotemporal headache at orgasm, which was more severe than his usual OHs and was immediately followed by a transient left sided facial palsy, which lasted 90 minutes. The headache remained extremely severe (10/10 on a verbal analogue scale) for five minutes, and then decreased to 2/10 over the next 15 minutes.

On admission, two days later, the patient still had a mild headache. His neurological examination was entirely normal. Blood pressure was 152/86 mm Hg both sides, pulse 80/min regular.

Initial brain computed tomography without contrast showed no sign of subarachnoid haemorrhage (SAH) but showed right basal ganglia and frontal hypodensities. Brain magnetic resonance imaging (MRI) (DWI, FLAIR, T2) showed areas of increased signal with a 35–40% decrease in ADC in the right lentikulostriate and capsular regions extending into the ventricular wall, the frontal cortex, and deep frontal white matter (fig 1A). There was no evidence of SAH on FLAIR and gradient echo weighted (T2) images.

Magnetic resonance angiography (MRA), using time of flight technique without contrast, showed a focal ectasia of the right M1 segment on maximal intensity projection. Native images showed a double channel in the right MCA stem (fig 1C, D). Cerebral four vessel angiography showed a fusiform dilatation measuring 0.5 mm in diameter, and expanding over 3 mm in length at the junction of the

proximal and middle thirds of the right M1 segment (fig 1B). No other vascular anomaly was detected, in particular there was no sign of fibromuscular dysplasia.

Other investigations were normal: electrolytes, blood cell count, prothrombin and coagulation tests, cholesterol level, and cerebrospinal fluid. Blood and urine testing for cocaine, opioids, and tetra-hydrocannabinol were negative. Electrocardiogram as well as transthoracic and transoesophageal echocardiography was normal.

The patient was treated with propranolol and perindopril. His headaches resolved within 10 days. Three month follow up MRA showed disappearance of the arterial wall defects in the right M1 segment with a normalised lumen size. He had no further headaches until 10 months after his stroke, when he experienced a new episode of severe orgasmic headache. Repeated investigations including MRI and conventional angiography were normal (fig 1E, F).

DISCUSSION

After a 20 year history of rare episodes of primary OH, this patient experienced an unusually severe OH episode associated with transient facial weakness suggesting a change in the underlying pathophysiology. Brain imaging studies showed fresh infarction in both the deep and distal right MCA territory suggesting MCA stem disease. MRA and 3D DSA showed a focal dilatation in the right M1 segment with MRA source images showing a true and false lumen, highly suggestive of dissection.^{3,4} This hypothesis is strongly supported by the disappearance of these arterial abnormalities at three month follow up. Furthermore, the arterial segment involved (the MCA stem) is the usual site of MCA dissection.^{3,5} It is therefore probable that MCA dissection represents the underlying mechanism, which led to the unusually severe episode of OH and to an intimal rupture of the MCA stem with formation of a false lumen, occlusion of lenticular striate arteries at their origin, and distal embolism into branches of the anterior MCA division.

MCA dissections are classically rare and severe, presenting mostly with large MCA infarcts,^{6–8} SAH,⁹ or intracerebral haemorrhage.¹⁰ It is however probable that their frequency is underestimated and their severity overestimated because of their notoriously difficult intra vitam diagnosis.³

Sexual activity is one of the numerous triggers of arterial dissection and arterial dissection is a well recognised cause of secondary OH but the arteries most frequently involved are internal carotid or vertebral arteries.³ A case has also been recently reported of basilar artery dissection presenting as OH of recent onset.¹¹ MCA presenting as OH as inaugural and main symptom has so far not been reported.

There is no uniform treatment for MCA dissection because of the double risk of haemorrhage and thrombosis. In this case, although there was a thromboembolic event and no

Abbreviations: OH, orgasmic headache; MRI, magnetic resonance imaging; SAH, subarachnoid haemorrhage; MRA, magnetic resonance angiography; MCA, middle cerebral artery

Figure 1 (A) Initial MRI DWI showing hyperintense signals in the right lenticulostriate arteries and the right anterior MCA division. (B) 3D angiographic reconstruction showing a fusiform dilatation at the junction of the proximal and middle thirds of the right M1 segment, measuring 0.5 mm in diameter, expanding over 3 mm long. (C) Initial MRA MIP showing a focal ectasia on the right MCA stem. (D) Initial MRA TOF showing a double channel of the right MCA stem. (E) 3D angiographic reconstruction control at 10 months. (F) Control angiography at 10 months, right internal carotid artery, left oblique view.

evidence of blood on neuroimaging and CSF, we decided not to give any antithrombotic treatment for fear of bleeding from the ectatic lesion.

Another unique feature of this case is the particularly misleading occurrence of an episode of OH that proved to be secondary in a patient who has a typical history of a primary OH with some 50 episodes over 20 years before the dissection and one episode after the event. Primary OH is one of the two varieties of headaches related to sexual activity identified by the IHS: it is a sudden explosive headache occurring mostly in young male adults just before or at orgasm and lasting a few minutes to a few hours. It is thought to be attributable to the sudden rise in blood pressure that occurs at orgasm and can be as high as 40–100 mm Hg for systolic blood pressure and 20 to 50 for diastolic blood pressure.² This is further supported by the preventive effect of propranolol, which presumably acts by limiting the surge of blood pressure at orgasm.² To qualify as primary, OH should not be attributable to other disorders such as ruptured aneurysm, stroke, reversible vasoconstriction, intracranial hypotension, or indeed dissection, which should all be excluded on first onset of OH.^{1,2}

In our patient the OH attributable to MCA dissection was different from the previous episodes in that it was more severe and for the first time associated with a neurological deficit. It can be hypothesised that a greater and more abrupt change in blood pressure at orgasm triggered the dissection and that the extremely severe OH was attributable to both the rise in blood pressure and the dissection.

This case illustrates the need for investigations not only at the first episode of OH, but also in patients with a typical history of primary OH when the headache differs from the previous ones by its unusual severity or duration or if it is associated with other neurological symptoms, or both. Investigations should include CT, MRI, eventually LP, arterial investigations such as MRA or CT angiography and if all of these investigations are unrevealing, conventional angiography.

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