40. Chadwick D, Reynolds EH, Marsden CD. Anticonvulsant-induced dyskinesias: a comparison with dyskinesias induced by neuroleptics. J Neurol Neurosurg Psychiatry 1976; 39: 1210–1218.
41. Harrison MB, Lyons GR, Landow ER. Phenytoin and dyskinesias: a report of two cases and review of the literature. Mov Disord 1993; 8: 19–27.

Fusiform aneurysm of a persistent trigeminal artery

D Agrawal[1] MCH, AK Mahapatra[1] MCH, NK Mishra[2] MD

[1]Department of Neurosurgery, [2]Department of Neuroradiology, All India Institute of Medical Sciences, New Delhi, India

Summary *Objective*. Fusiform aneurysms of the persistent trigeminal artery are rare and endovascular treatment of these aneurysms has not been attempted previously. We describe a case of persistent trigeminal artery with a fusiform aneurysm in its middle third, managed using Guglielmi detachable coils (GDC). *Clinical Presentation.* A 50-year-old, diabetic and hypertensive patient presented with sudden onset headache and neck stiffness. On examination, she was conscious but disoriented, without cranial nerve or sensorimotor deficits. Four-vessel cerebral digital subtraction angiography revealed a fusiform aneurysm of the middle third of a persistent trigeminal artery on the left side with adult type posterior cerebral arteries. *Intervention*. Guglielmi detachable coils were used for occlusion of the persistent trigeminal artery. *Results.* The procedure was well tolerated but delayed ischemic neurological deficits developed due to vasospasm. *Conclusions*. (1) In spite of angiographically documented independence of the anterior and posterior cerebral circulation, occlusion of a persistent trigeminal artery using endovascular techniques may result in posterior circulation stroke due to a number of factors, including occlusion of brainstem perforators taking origin from the persistent trigeminal artery or vasospasm. (2) The timing for endovascular intervention following aneurysmal rupture remains poorly defined.
© 2005 Elsevier Ltd. All rights reserved.

Journal of Clinical Neuroscience (2005) **12**(4), 500–503
0967-5868/$ - see front matter © 2005 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jocn.2004.06.018

Keywords: persistent trigeminal artery, fusiform aneurysm, endovascular coiling, Guglielmi detachable coil, posterior circulation stroke

Received 12 February 2004
Accepted 18 June 2004

Correspondence to: A.K. Mahapatra MCh, Department of Neurosurgery, All India Institute of Medical Sciences, C.N. Centre, Ansari Nagar, New Delhi 110 029, India. Tel.: +91 11 6594577; Fax: +91 11 6862663;
E-mail: akmahapatra_22000@yahoo.com

INTRODUCTION

Persistent trigeminal artery is one of the most common embryonic vascular communications persisting into adult life. However, aneurysms of a persistent trigeminal artery are very rare and only 29 cases have previously been reported.[1–26] We describe a case of trigeminal artery aneurysm presenting with SAH and managed with endovascular coiling. This is the second case in the world literature describing a fusiform aneurysm of a persistent trigeminal artery, and the first case report describing endovascular coiling of same, using Guglielmi detachable coils (GDC). The role of a persistent trigeminal artery in augmenting the posterior circulation and the importance of timing of endovascular intervention are discussed.

CASE REPORT

A 50-year-old female presented with a one day history of sudden severe headache, followed by repeated vomiting and neck stiffness. She was a known diabetic and hypertensive, on regular medication, for 15 years. She had a history of sudden onset bilateral blindness one year previously that had almost completely recovered within a week. Carotid Doppler ultrasonography done at that time was suggestive of only mild narrowing of both common carotid arteries. MRI brain had shown a right occipital lobe infarct. She had been started on aspirin and ticlopidine, which she had been taking regularly and had been asymptomatic until the present episode.

On examination, she was an obese lady (108 kg) conscious but disorientated. Cranial nerve, motor and sensory examinations were essentially normal. Marked neck rigidity was present and Kernig's sign was positive. A non-contrast CT scan of the head showed diffuse SAH in the basal cisterns. Four-vessel cerebral digital subtraction angiography revealed a persistent trigeminal artery on the left side with a fusiform dilatation in its mid-portion, which was in severe spasm without any demonstrable distal flow (Fig. 1). The superior cerebellar artery was seen arising distal to the insertion of the persistent trigeminal artery into the basilar artery, and the basilar itself appeared stenosed proximal to this junction. The ipsilateral posterior communicating artery was also hypoplastic and the posterior cerebral arteries were filling from the basilar artery (Fig. 2).

As the patient had no new neurological deficits despite no flow through the persistent trigeminal artery on DSA, and as there was good filling of both posterior cerebral arteries and the superior cerebellar arteries from the vertebral artery (adult type of persistent trigeminal artery), parent artery occlusion using endovascular coiling was planned. A two day delay from the time of initial imaging to the time of endovascular intervention occurred due to technical reasons. The repeat angiogram done two days later, at the time of coiling, showed an increase in the spasm of the persistent trigeminal artery, with no demonstrable flow through it. Endovascular coil packing of the aneurysm was undertaken via the left vertebral artery. GDC detachable coils were placed inside

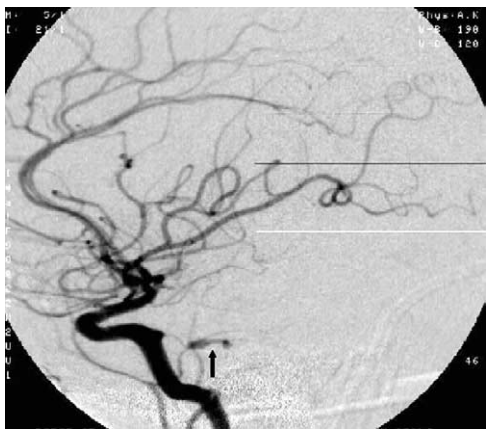

Fig. 1 Left carotid angiogram (lateral view) showing left persistent trigeminal artery with a fusiform dilatation in its middle portion (black arrow). There is severe spasm of the persistent trigeminal artery with no demonstrable flow distally.

 © 2005 Elsevier Ltd. All rights reserved.

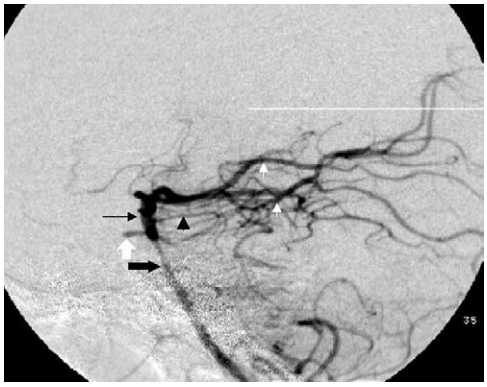

Fig. 2 Left vertebral angiogram (lateral view). The superior cerebellar arteries (black arrow head) are seen arising distal to the insertion of the persistent trigeminal artery into the basilar (fine black arrow), and the basilar artery appears stenosed proximal to this junction (thick black arrow). The posterior cerebral arteries (fine white arrows) are supplied by the basilar artery. The distal end of persistent trigeminal artery fills by retrograde flow (thick white arrow).

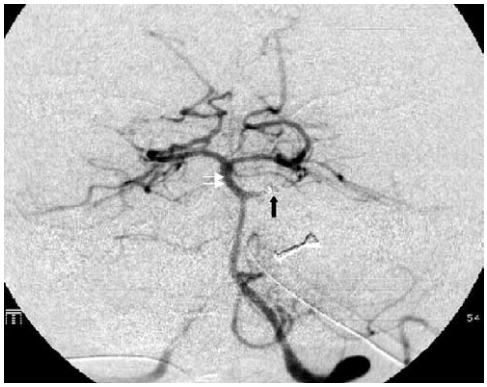

Fig. 3 Left vertebral angiogram after coiling (anteroposterior view). GDC are seen within the aneurysmal dilatation (black arrow). Flow through the basilar artery is well maintained (white arrows).

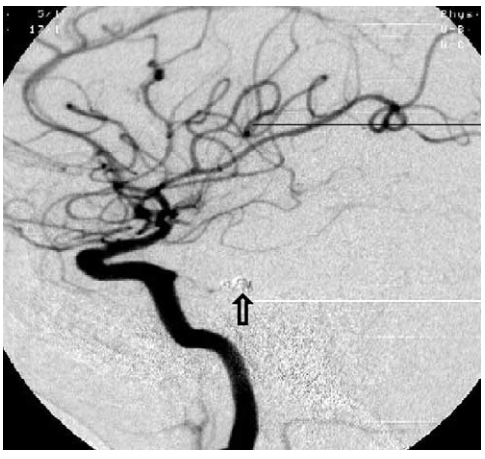

Fig. 4 Left carotid angiogram after coiling (lateral view). GDC are seen within the aneurysmal dilatation (arrow).

the fusiform dilatation, which was successfully occluded (Figs. 3 and 4).

Initially, the patient tolerated the procedure well, however, on post-embolization day four, she deteriorated neurologically and became quadriparetic. A CT scan showed brainstem and bilateral occipital lobe hypodensities. She continued to deteriorate and died two weeks later from gram-negative septicaemia.

DISCUSSION

The trigeminal artery is one of the many embryological anastomoses between the dorsal aorta (the future internal carotid artery) and the bilateral longitudinal neural arteries (the future basilar artery).[1,27,28] Normally appearing in the 3 mm embryo and disappearing by the 14 mm embryonic stage, its persistence is the most common of the primitive carotid-basilar anastomoses in adult life, seen in 0.1–1% of all cerebral angiograms.[1,8,27–31] The clinical significance of a persistent trigeminal artery lies in its association with other vascular disorders, such as absence or hypoplasia of cerebral arteries,[5,12] aneurysms,[1–26,32] arteriovenous malformations,[5] carotid-cavernous fistulas,[5,6] Moyamoya disease,[33] cranial nerve palsies,[34] SAH,[1,5,8] and tic doulourex.[35]

Despite a 14% incidence of aneurysms in patients with persistent trigeminal artery (which is three times more than that of the general population), aneurysms arising from the persistent trigeminal artery are very rare and less than thirty cases have been described previously.[1–26] Most of these are saccular and fusiform aneurysms of a persistent trigeminal artery and are exceedingly rare. Only one case has been described previously in 1974,[3] in which direct surgical clipping of the aneurysmal dilatation was attempted. Though the authors tried to maintain perfusion through the persistent trigeminal artery while applying the clip, post-operative angiogram demonstrated complete occlusion the artery. The patient, however, tolerated the occlusion well.

Guglielmi et al.[36] described endovascular coiling in 1991, which revolutionized the treatment of saccular aneurysms. Though endovascular occlusion of fusiform aneurysms has been reported,[37] the use of GDC in fusiform aneurysms of the persistent trigeminal artery has not been described previously and to our knowledge, this is the first time it has been attempted. GDC embolization offers the advantage of predictability and controllability of the coils prior to detachment, and they are available in various sizes, providing greater flexibility in coiling.

Our patient tolerated the endovascular procedure well but developed a delayed onset brain stem infarct four days later, resulting in a 'locked in syndrome'. Delayed vasospasm probably contributed to the deterioration in the patient's condition. As the embolization was done three days after the onset of SAH, it is possible that vasospasm was at its peak around four days following the procedure (seven days after the onset of SAH), correlating with the onset of clinical deterioration. Serial trans-cranial Doppler ultrasound (TCD), showed significantly increased flow velocities in the basilar artery after the clinical deterioration, compared to TCD done at admission, corroborating the clinical evidence of vasospasm. Delayed ischemic deficits due to cerebral ischemia results in the clinical syndrome of symptomatic vasospasm, and is an inconsistent consequence of angiographic vasospasm.[38] Almost half the patients with angiographic vasospasm develop neurological symptoms.[39] A further 65% of the patients with delayed neurological deficits will have a fatal outcome or permanent neurological deficits.[40] The timing of endovascular intervention in patients with angiographic evidence of vasospasm remains controversial. Guglielmi and Byrne[41] proposed early (within 24 h) treatment, and were of the opinion that the clinical grade or angiographic vasospasm are not contraindications for intervention. However, if parent artery occlusion is contemplated for saccular aneurysms, endosaccular packing with coils immediately after aneurysmal rupture, followed by parent artery occlusion 2–3

© 2005 Elsevier Ltd. All rights reserved.

Download English Version:

https://daneshyari.com/en/article/9193463

Download Persian Version:

https://daneshyari.com/article/9193463

Daneshyari.com