CASE REPORT

The clinical dilemma of treating transient ischaemic attack-like symptoms in patients with coexisting arteriovenous malformation

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Accepted 11 February 2014

SUMMARY

Antiplatelet agents are established, evidence-based treatments to reduce the risk of subsequent stroke in patients diagnosed with transient ischaemic attack (TIA). This case discusses the clinical dilemma clinicians are faced with when treating TIA-like symptoms in patients with coexisting arteriovenous malformation (AVM). A 65-year-old Caucasian woman reported a sudden onset of right hand weakness and impaired coordination. The episode lasted for 1 min and then resolved completely. She was subsequently diagnosed with a TIA and started on aspirin 75 mg. Her coexisting medical history included a large left-sided parietal-occipital AVM. The case highlights the clinician's need to balance the risk posed by antiplatelet agents increasing the likelihood of AVM rupture between the risk of untreated TIA progressing to stroke. Factors worth considering include number of vascular risk factors, AVM-related steal phenomena, size of AVM and whether or not there has been previous AVM-related haemorrhage.

BACKGROUND

Transient ischaemic attack (TIA) can be defined as a transient episode of neurological dysfunction due to temporary impairment of blood supply to the brain. Although transient in nature, TIA poses a significant risk for subsequent progression to stroke. The ABCD2 score, based on the patient's age, comorbidities and clinical features of the episode, has become an established clinical score to determine the risk of stroke at 2 and 7 days following a TIA. A risk of stroke of up to 11.7% at 7 days post-TIA has been reported for high ABCD2 scores. In addition, studies have looked at the longer term risk of progression to stroke with approximately 10.5% of patients returning to the emergency department with a stroke at 90 days post-TIA, with half of the cases occurring within the first 2 days.¹

In contrast, stroke can be defined as the permanent loss of brain function due to the impairment of blood supply to the brain. Approximately 85% of strokes are ischaemic in origin while the remainders are haemorrhagic. Rarer causes also occur such as vasculitides. As the second most frequent cause of death worldwide in 2008,² a great deal of emphasis is placed on effectively treating TIA and other risk factors associated with ischaemic and haemorrhagic strokes such as hypertension, diabetes and atrial fibrillation. The treatment of TIA with antiplatelet

agents such as aspirin and dipyridamole has been shown to reduce the risk of subsequent stroke by 20–30%,³ although this comes with a 0.3–0.6% yearly risk of intracranial haemorrhage⁴ as one of its most serious treatment complications.

An arteriovenous malformation (AVM) is a rare condition characterised by a collection of friable blood vessels containing abnormal connections between arteries and veins. It has a <0.001% yearly incidence rate in the USA.⁵ Although discussed here in the context of occurring in the brain, it can also occur in other locations in the body. It is largely congenital in origin with a large majority of patients being asymptomatic. When symptomatic, its most common presentations are headache, seizure and rupture leading to intracranial haemorrhage. Spontaneous intracranial haemorrhage rates of 4–7% have been previously quoted although more modern studies have shown rates to be slightly lower at around 2–4%.⁶ In addition, AVMs have been shown to cause TIA-like symptoms due to steal phenomena that cause focal neurological deficit by altering perfusion in the tissue in the region of AVM.⁷ Management of AVM involves treating symptoms such as headaches and seizures. The Spetzler-Martin grading system is used by neurosurgeons to determine non-operative observation versus operative treatment of AVM itself.

Although there are no large published trials looking at the effect of antiplatelet agents on the risk of intracranial haemorrhage in the context of AVM, the following case highlights the various factors clinicians may take into account when considering the treatment of TIA-like symptoms in a patient with coexisting AVM.

CASE PRESENTATION

In May 2013, a 65-year-old Caucasian woman was referred by her general practitioner to the rapid access TIA clinic of a district general hospital with a sudden onset of right hand weakness and impaired coordination. The episode lasted for 1 min and then resolved completely. She denied any history of associated right leg or facial weakness, speech or swallowing problems, visual disturbance, fits or loss of consciousness and no preceding symptoms such as headache, nausea and vomiting.

She was a retired book keeper who lived with her husband and was fully independent in her personal and domestic activities of daily living including driving. She was an ex-smoker, who stopped smoking in 1979, and rarely drank alcohol.

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To cite: Jabbari E, Sethuraman S, Sekaran L. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-201315

At the age of 12 years (1960), she suffered right-sided hemiparesis due to a left-sided intracerebral haemorrhage. She was discharged from Queen Square Hospital, London, after a 2-month inpatient stay and was left with minimal residual right-sided weakness. In 1995, she suffered seizures and a subsequent MRI revealed a large left-sided parietal-occipital AVM and so she was started on carbamazepine. It was, therefore, assumed that her presentation in 1960 was due to rupture of AVM. She was reviewed at Queen Square in 2005 due to increasing right leg weakness although a repeat MRI found no cause for her symptoms but did highlight her existing AVM (figures 1 and 2). A year later this weakness had stabilised and she was finding benefit from an orthosis. She also suffered from hypertension, hyperlipidaemia and osteoarthritis.

Her drug history revealed that she was taking doxazosin 2 mg once daily, rosuvastatin 10 mg once nocte, carbamazepine prolonged release 800 mg once nocte and paracetamol 1 g as required. She was not known to have any drug allergies.

On examination of this right-handed woman her blood pressure was found to be 180/77 mm Hg with a heart rate of 86 bpm. Heart sounds were normal with no carotid bruits. Chest was clear to auscultation. Neurologically, apart from a right-foot drop, there was no focal, motor or sensory deficit clinically. Visual fields were normal to confrontation and ocular movements were normal. An ECG revealed sinus rhythm and chest X-ray and baseline blood tests revealed no abnormalities. A CT of the head revealed multiple tiny calcific densities in the left parietal-occipital region with cortical atrophy in keeping with a previous AVM. There was no evidence of an acute infarct or haemorrhage (figures 3 and 4). A carotid Doppler scan did not reveal any significant narrowing of the internal carotid arteries. A subsequent echocardiogram and 24 h ECG revealed no abnormalities.

Given the above clinical picture and her known vascular risk factors, her presentation was attributed to a left-middle cerebral artery TIA and she was therefore started on aspirin 75 mg once daily. She was well 1 month later at follow-up at Queen Square although the neurologists decided to stop aspirin until further investigations with CT arteriogram and MRI of the brain. Her next follow-up appointment is due in February 2014.

INVESTIGATIONS

MRI of the head (see the Case presentation section)

CT of the head (see the Case presentation section).

Figure 1 Axial section MRI of the head from 2005 showing a large parietal-occipital arteriovenous malformation.

DIFFERENTIAL DIAGNOSIS

- ▶ TIA
- ▶ AVM-related steal phenomena
- ▶ Focal seizure.

TREATMENT

Aspirin 75 mg.

OUTCOME AND FOLLOW-UP

No adverse outcomes at 1-month follow-up. Next due to be followed up in February 2014.

DISCUSSION

AVM is a rare vascular anomaly that poses a small but significant risk of spontaneous intracranial haemorrhage. While there have been no large published trials looking at the effect of antiplatelet agents on the risk of intracranial haemorrhage in the context of AVM, we can hypothesise that the risk is increased due to bleeding being an established adverse effect of these drugs. Therefore, treating TIA-like symptoms in patients with coexisting AVM poses a clinical dilemma for clinicians. Ultimately, the rarity of this situation implies that decision-making should be made on an individual basis and takes into account a number of different factors.

Before deciding on a management plan, the clinician must first take a thorough history in order to decide whether the patient's presentation is actually due to a TIA. This case highlights that in patients with an existing intracranial abnormality, such as an AVM, it can be difficult to correctly attribute transient neurological symptoms to a diagnosis of either TIA or seizure activity. Elements in the history that may be more suggestive of seizure activity include the presence of warning symptoms before the onset of transient neurological symptoms and impaired consciousness during the episode, both of which were not present in the index case. The situation is further complicated by the possibility of TIA-like symptoms being caused by localised AVM-related steal phenomena.⁷ In the index case, this is certainly relevant as a large left-sided parietal-occipital AVM may well explain the presence of transient right arm weakness and impaired coordination.

This case also highlights the importance of the role of MRI in patients with TIA. The decision to initiate antiplatelet medication for this patient could have been made easier if the patient was subjected to an urgent MRI on her presentation. If the scan

Figure 2 Coronal section MRI of the head from 2005 showing a large parietal-occipital arteriovenous malformation.

Figure 3 Axial section CT of the head from 2013 showing calcific densities and cortical atrophy associated with arteriovenous malformation.

had shown diffusion-weighted imaging (DWI) positive areas for hypoperfusion, then the clinician would have been more inclined to diagnose a TIA and initiated antiplatelet therapy. This is supported by evidence which has shown that stroke recurrence risk at 90 days was 32.6% in patients with DWI positivity and large vessel occlusive disease.⁸ This highlights the vital prognostic role that MRI has in risk stratifying patients with TIA. However, the availability of urgent MRI services is still limited in comparison with that of CT, especially in the district general hospital setting, as was in this case. We, therefore, expect the role of MRI to become more prominent as an urgent imaging modality in patients presenting with TIA-like symptoms in the future.

Ultimately, if the clinician concludes that a patient has, indeed, suffered from a TIA while having a coexisting AVM, then the factors involved in deciding appropriate management may be divided into two sections: vascular risk factors and AVM-related factors. In a patient with multiple vascular risk factors such as smoking, hypertension and diabetes and/or previous vascular events such as TIA/stroke and myocardial infarction, the benefits of treating TIA with antiplatelet agents are likely to outweigh the hypothesised increased risk of intracranial haemorrhage associated with AVM. With regard to AVM-related factors, studies have shown that increasing age, deep brain

location,⁹ previous rupture and large size¹⁰ have the highest risk of subsequent haemorrhage so that the risk of using antiplatelet agents may well outweigh potential benefits. Therefore, despite the vascular risk factors that were present in the patient in question, a justified rationale for stopping aspirin would have been the large size of the AVM, the hypothesised previous rupture of the AVM and the possibility of the transient neurological symptoms being due to localised AVM-related steal phenomena.

Regardless of the decision taken, a regular follow-up in a tertiary centre with periodic monitoring of the size of AVM is necessary. In addition, fully informing the patient of the risks versus benefits of treating a TIA with antiplatelet agents and actively involving them in the decision-making process are vital.

Learning points

- ▶ There are no large published trials looking at the effect of antiplatelet agents on the risk of intracranial haemorrhage in the context of arteriovenous malformation (AVM).
- ▶ In patients with coexisting AVM, it can be difficult to correctly attribute transient neurological symptoms to a diagnosis of either seizure activity, transient ischaemic attack (TIA) or AVM-related steal phenomena.
- ▶ MRI has an important prognostic role in patients with TIA, although its availability in the district general hospital setting may be limited.
- ▶ The case highlights the clinician's need to balance the risk posed by antiplatelet agents increasing the likelihood of AVM rupture between the risk of untreated TIA progressing to stroke.
- ▶ Factors worth considering include: number of vascular risk factors, size of AVM and whether or not there has been previous haemorrhage from the AVM.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Figure 4 Axial section CT of the head from 2013 showing no evidence of acute infarct or haemorrhage.

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