midazolam is a widely used hypnotic and sedative.

Keywords: torsade de pointes, midazolam, multiple endocrine neoplasia

Final diagnosis

Torsade de pointes, possibly midazolam-induced.

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Cerebrovascular accident in a 77-year-old man

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A 77-year-old man was admitted to our hospital because of sudden left hemiparesis. Neurologic examination showed left hemiparesis (upper extremity 3–4/5 and lower extremity 4/5, MRC scale) and left Babinski's sign without a visual or sensory deficit. The patient improved over the ensuing months with rehabilitation therapy, regaining the ability to walk unassisted, and was left with moderate left spastic hemiparesis.

Brain computed tomography (CT) scan disclosed a subcortical ischaemic infarct in the distribution of the right middle cerebral artery. Magnetic resonance imaging (MRI) was performed because CT scan showed a questionable haemorrhagic component. MRI ruled out parenchymal haemorrhage but showed another abnormality (arrowed in figure 1).

Questions

- 1 What is the most likely pathogenetic mechanism of this patient's stroke?
- 2 What further investigations would be helpful?

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Figure 1 Brain MRI shortly after the stroke

Answers

QUESTION 1

Carotid compression by a pituitary tumour. Brain MRI (figure 1) disclosed a sellar mass extending into the right cavernous sinus, surrounding and encasing the internal carotid artery at the distal juxtaseilar segment. An evident asymmetry was noticed between the siphon and petrous segment of both internal carotid arteries: on the right side, there was intermediate signal and homogenous enhance-

Figure 2 Axial fast spin-echo (5520/128) reveals hyperintense confluent, punctate lesions over the right corona radiata, suggesting ischaemic lesions in the middle cerebral artery territory

Figure 3 MRA two months later. (A) Coronal maximum intensity projection (MIP) angiogram from a 3DFT-PC coronal slab (Venc, 30 cm/s) depicts occlusion of the right internal carotid artery and repermeabilization from the supraclinoid segment. (B) Coronal (top) and axial (bottom) MIPs from an axial 3DFT-TOF set reveal no evidence of flow within the right carotid siphon. The supraclinoid segment is supplied by an enlarged right posterior communicating artery (arrow), giving rise to anterior and middle cerebral arteries with normal branching pattern. Note also the asymmetric flow between both vertebral arteries

A

B

ment within the arterial lumen, indicating extreme slow flow proximal to the stenotic segment. The right corona radiata showed confluent ischaemic lesions (figure 2) involving the distal territory of the right middle cerebral artery, consistent with a near-occlusive stenosis of the right internal carotid artery. Interestingly, no other ischaemic lesions were identified in the rest of the brain, despite the age of the patient. The sellar mass was consistent with meningioma or macroadenoma, but the patient refused surgery.

QUESTION 2

Conventional angiography or magnetic resonance angiography (MRA). Two months after stroke, MRA was performed (figure 3). Both the phase-contrast and the time-of-flight techniques failed to reveal any flow in the right internal carotid artery below the level of arterial encasement. Severe stenosis and slow flow identified during the acute setting had evolved to occlusion, and collateral flow (mainly via an enlarged right posterior communicating artery) was established from the supraclinoid segment, resulting in normal flow through the right anterior and middle cerebral arteries. The left internal carotid artery with its branches and vertebrobasilar system was normal; there was only an asymmetry in flow between both vertebral arteries, the left one being greater.

Aside from directly involving the carotid artery, pituitary tumours may result in stroke due to haemorrhage or infarction of the pituitary gland or to the effects of radiotherapy. This case illustrates that stroke is not always caused by atherothrombotic mechanisms. Some other aetiologies are listed in box 1.

Non-atherothrombotic causes of stroke in the elderly

- giant cell arteritis (other vasculitis less frequently)
- pituitary tumours
- arterial dissections (spontaneous or post-traumatic)
- heightened serum viscosity (polycythaemia, hyperfibrinogenaemia or paraproteinaemia)
- thrombocytosis and thrombocytopenia
- haemoglobinopathies, sickle cell disease
- venous thrombosis
- hypercoagulable states (protein S or C deficiency, antiphospholipid syndrome, antithrombin III deficiency)
- endocarditis (infectious or marantic)

Box 1

Discussion

Occlusion of the internal carotid artery by an intracranial tumour is a rare occurrence, reported mainly as a complication of sellar and parasellar neoplasms. Only occasionally does this situation result in ischaemic stroke.¹ On the other hand, vascular events related to pituitary tumours are usually secondary to haemorrhage or infarct of the pituitary gland² or to the effects of radiotherapy.³

During normal development, the internal carotid artery becomes closely attached to the bone at the level of the base of the anterior clinoid process, which explains why tumours developing in the sellar and parasellar region may compress or obstruct the carotid artery at this level, causing neurologic symptoms of ischaemic origin.⁴

In a review of cases of internal carotid artery occlusion by an intracranial tumour, meningioma and pituitary adenoma were the tumours most often found; the occlusion typically occurred, as in this patient, at the level of the cavernous or supracavernous segment.¹ Arterial encasement and compression of the arterial walls against the surrounding structures are the proposed mechanisms for arterial obstruction. In our case, the first mechanism seems more likely, resulting in slow flow and, finally, thrombosis.

Most cases of stroke are secondary to thromboembolic events and intense efforts are being made to improve its prevention and

therapy. However, even though infrequent, non-atheromatous stenosis of the carotid artery must be considered in the differential diagnosis of cerebrovascular accidents. Medical or surgical prophylaxis to prevent stroke recurrence will be successful only if the aetiology of the initial event has been identified. Pituitary tumours may be amenable to surgery and medical treatment and therefore should be considered among the treatable causes of stroke. Several reasons lead us to think that, in this patient, stroke was probably caused by the pituitary tumour and that this was not an 'incidentaloma'. There was an obstruction of the internal carotid artery ipsilateral to the infarction and the area of infarction corresponded both with the neurological deficit and with the vascular territory of the occluded artery. Furthermore, the rest of the brain vasculature was normal and there was no evidence of infarcts at other levels.

The question arises of how often these tumours should be looked for in the setting of stroke. In fact, in this case the tumour was not initially suspected; it was the brain MRI, performed because of a question of haemorrhage, that revealed it. Medical literature shows that pituitary tumours are identified at autopsy in 1.5–27% of unselected asymptomatic patients,^{5,6} and some 10% of the normal adult population have pituitary abnormalities on MRI that are compatible with adenomas, although most of them do not require treatment.⁷ Further analysis of large series are needed to establish the cost–benefit ratio of these studies.

MRI is the preferred imaging method for detecting pituitary pathology, because of its superior contrast resolution and the facility of imaging in the coronal and sagittal planes.⁸ Since MRA is noninvasive, this technique seems advisable in patients with tumours involving sellar and parasellar regions to rule out carotid artery compromise. Its actual value, however, should be systematically evaluated in prospective studies.

Final diagnosis

Stroke secondary to carotid artery occlusion by a pituitary tumour.

Keywords: pituitary tumour, stroke, carotid artery, occlusion

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