Case Report

Dural arteriovenous fistulas of the cavernous sinus with onset of intracerebral haemorrhage mimicking hypertensive putaminal hemorrhage

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Summary

We describe a patient with dural arteriovenous fistulas of the cavernous sinus (CS-dAVFs) who developed an intracerebral haemorrhage (ICH) mimicking hypertensive putaminal haemorrhage. Drainage into the superior ophthalmic vein (SOV) and inferior petrosal sinus (IPS) was not demonstrated on cerebral angiography, and only cortical venous reflux into the Sylvian vein was observed. In cases of venous drainage concentrated on the Sylvian vein, CS-dAVFs could indicate ICH with radiological appearance resembling putaminal haemorrhage.

Keywords: Dural arteriovenous fistulas; putaminal hemorrhage; Sylvian vein.

Introduction

Dural arteriovenous fistulae of the cavernous sinus (CS-dAVFs) usually present with ocular symptoms or pulsatile tinnitus, intracerebral haemorrhage (ICH) is rare [1–7]. The annual incidence of ICH in CS-dAVFs is believed to be around 1.8% [3]. A higher risk of bleeding if there is an appearance of draining into cortical veins following occlusion of other drainage routes and the existence of venous varices on the route of venous drainage [1, 3]. Most reported cases of ICH due to dAVFs have had subcortical haemorrhage [4–6], we describe a patient with CS-dAVFs who developed an ICH mimicking putaminal haemorrhage.

Case report

A 73-year-old woman with hypertension presented with a sudden onset of consciousness disturbance, right hemiparesis and slight chemo-

sis of the left eye. Initial computed tomography (CT) of the head showed an ICH, 4-cm in maximum diameter, in the outer portion of the left putamen (Fig. 1A), and a diagnosis of hypertensive ICH was considered. The following day, magnetic resonance imaging (MRI) was performed to exclude the possibility of haemorrhagic infarction. T2-weighted imaging (T2WI) revealed an area of relatively extensive hyperintensity around the haematoma in the surrounding white matter (Fig. 1B). However, diffusion-weighted imaging (DWI) did not indicate hyperintensity around the haematoma (Fig. 1C). In addition, magnetic resonance angiography did not show any evidence of steno-occlusive lesions of the main intracranial arteries.

These findings did not indicate haemorrhagic infarction. Neurological status was stable and follow-up CT showed no expansion of the haematoma, so conservative treatment was initiated. However, neurological status deteriorated suddenly on hospital day 5. CT showed marked expansion of the haematoma into the left frontal lobe (Fig. 1D). Emergency evacuation of haematoma was considered and cerebral angiography was performed to exclude the possibility of a cryptic vascular lesion such as an arteriovenous malformation.

The arterial phase of left (ipsilateral to haematoma) internal carotid angiography (ICAG) showed no clear abnormality, with the exception of mass effects from the haematoma, and the venous phase of left ICAG did not opacify cortical veins that are typically anastomotic to the left Sylvian vein, was observed. In cases of venous drainage concentrated on the Sylvian vein, CS-dAVFs could indicate ICH with radiological appearance resembling putaminal haemorrhage.

Contralateral right external carotid angiography (ECAG) revealed dAVFs of the right cavernous sinus, supplied by the right foramen rotundum artery, which drained into the left cavernous sinus with cortical venous reflux to the left Sylvian vein via the posterior intercavernous sinus (Fig. 1B–C). CS-dAVFs were also supplied by the meningeal branches of the right internal carotid artery. Drainage to the right superior ophthalmic vein (SOV) and bilateral inferior petrosal sinus (IPS) were not noted on either right ICAG or right ECAG, but drainage to the left SOV was faintly apparent on right ECAG (Fig. 2C).

Based on the results of cerebral angiographies, left ICH due to venous hypertension with cortical venous reflux via right CS-dAVFs was diagnosed. We therefore decided to perform immediate transvenous embolization (TVE) for this patient. Although the left IPS was not
opacified on angiography, a microcatheter was successfully introduced into the left cavernous sinus via the left IPS. The proximal portion of the left SOV and the orifice between the left Sylvian vein and the left cavernous sinus were packed with interlocking detachable coils (Boston Scientific Corp., Watertown, MA) and fibered platinum coils (Boston Scientific Corp., Watertown, MA). Immediately after TVE, right ECAG demonstrated disappearance of cortical venous drainage into the left Sylvian vein, and altered venous drainage of CS-dAVFs from the left Sylvian vein into the left IPS (Fig. 2D). After the embolisation the patient recovered uneventfully. Chemosis of the left eye disappeared the next day after TVE and level of consciousness gradually improved. The patient was transferred to another hospital for rehabilitation after 6 weeks.

Discussion

The natural course and clinical symptoms of dAVFs are highly variable and depend on the locations of the shunts and the venous drainage patterns. Anterior drainage through the SOV usually causes ocular symptoms such as exophthalmos and chemosis, while inferior drainage through the IPS produces pulsatile tinnitus. Spontaneous regression of arteriovenous shunts is not rare, and patients could have no or only slight symptom for many years. However, a small group of patients suffer subarachnoid haemorrhage, subdural haemorrhage, ICH and venous infarction [1–7].

Leptomeningeal venous drainage, variceal or aneurysmal venous dilatation, galenic drainage [1–3] and the locations of the petrosal sinus and straight sinus [2, 3] have been shown to represent significant factors predisposing to an neurological complications. With a transverse-sigmoid sinus dAVF, posterior drainage into the superior petrosal sinus and IPS may lead to cere-