

# Oculomotor palsy and drowsiness due to post-thrombectomy subarachnoid haemorrhage falsely suggesting transtentorial herniation

Logesh Palanikumar , Joshua Mahadevan, Timothy Kleinig 

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## ABSTRACT

**Introduction** Post-thrombectomy subarachnoid haemorrhage (SAH) can result in oculomotor palsy and drowsiness, which may falsely suggest transtentorial herniation.

**Case presentation** We present a case of right oculomotor nerve palsy presenting after endovascular thrombectomy (EVT) for a right middle cerebral artery (MCA) stroke. The patient presented with a significant right MCA syndrome and a National Institutes of Health Stroke Scale (NIHSS) score of 10 with CT perfusion demonstrating a large penumbral lesion and a CT angiogram confirming a right MCA M1 occlusion. After thrombectomy, the patient developed a 9mm dilated non-reactive right pupil, and a new ipsilateral near-complete oculomotor nerve palsy. Repeat code stroke imaging demonstrated perimesencephalic SAH). The patient was managed expectantly and her conscious state and oculomotor palsy gradually resolved with an excellent neurological recovery.

**Conclusion** This case underscores the potential for post-thrombectomy perimesencephalic SAH as a rare mimic of symptomatic intracranial haemorrhage with mass effect manifesting as sudden-onset oculomotor nerve palsy.

## CASE SYNOPSIS

A previously well 80-year-old woman presented to hospital with an onset of left facial droop, hemiparesis and both visual and sensory neglect concerning for a right MCA occlusion. Her medical history included ischaemic heart disease treated with aspirin, heart failure with reduced ejection fraction treated with sacubitril/valsartan, previous pulmonary embolism managed with long-term rivaroxaban and chronic obstructive pulmonary disease from an ex-smoking 10 pack-year history.

On admission, her blood pressure was 100/53 mm Hg, with an NIHSS of 10 and an otherwise unremarkable physical examination. Notably, she had a full range of extraocular movements, was afebrile, had dual heart sounds, no elevation of the jugular venous pressure, a clear chest, and abdomen was soft and non-tender to palpation. Initial blood

results were unremarkable. ECG demonstrated sinus tachycardia with first degree heart block. CT code stroke imaging revealed a long segment right internal carotid artery occlusion and a large penumbra of 267 mL with a core of only 7 mL.

A diagnosis of a right MCA M1 occlusion was made, and the patient proceeded to EVT. She was not thrombolysed given anticoagulation with rivaroxaban. Thrombectomy was partly successful with thrombolysis in cerebral infarction (TICI) 2B–2C reperfusion after three passes and a target blood pressure of 100–140 mm Hg was maintained post-procedure. There was no wire perforation or contrast extravasation noted during the procedure. Three hours post-thrombectomy, the patient appeared drowsy and had a non-reactive 9 mm right pupil with partial ptosis. The eye was exotropic and infraducted at rest (down and out) with difficulty in adduction and supraduction consistent with an ipsilateral oculomotor nerve palsy ([figure 1](#)). Urgent repeat code stroke imaging was performed, which revealed a large amount of subarachnoid blood at the perimesencephalic area (particularly along the course of the right oculomotor nerve) but no evidence of large vessel reocclusion (in particular no basilar occlusion) or large symptomatic intracranial haemorrhage (sICH) with mass effect ([figure 2](#)). The patient was managed expectantly with blood pressure control below 140 mm Hg and her usual antiplatelet and anticoagulant medications were held. A 24-hour follow-up MRI revealed large volume blood products within the supratentorial and infratentorial subarachnoid spaces and the ventricular system, consistent with oedema and haemorrhage from reperfusion therapy ([figure 2](#)). The patient subsequently had fluctuating drowsiness thought to be multifactorial from subarachnoid blood causing

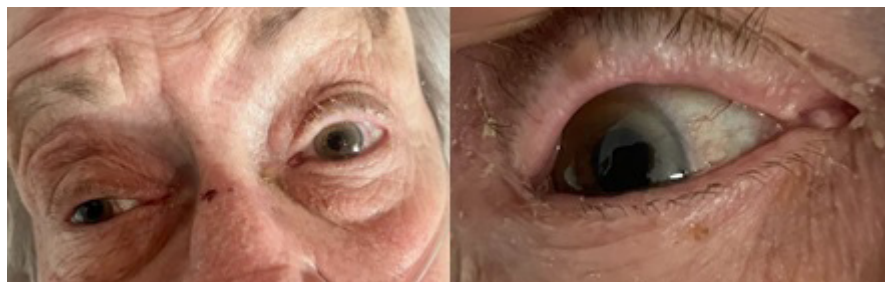


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Neurology, Royal Adelaide Hospital, Adelaide, South Australia, Australia

## Correspondence to

Dr Logesh Palanikumar; logesh.palanikumar@sa.gov.au



**Figure 1** Ipsilateral oculomotor nerve palsy of the right eye post-thrombectomy. The eye was exotropic and infraducted at rest (down and out) with difficulty in adduction and supraduction.

meningeal irritation, a non-infective exacerbation of her chronic obstructive pulmonary disease and delirium. After a period of several days, the patient improved clinically and clopidogrel monotherapy was commenced. Her hemiparesis improved to 4/5 power and her ptosis and ophthalmoparesis gradually resolved. The aetiology of oculomotor nerve palsy and ptosis post-thrombectomy in this case appears to be perimesencephalic subarachnoid blood, due to EVT for large hemispheric stroke with concurrent anticoagulation and antiplatelet medication.

At the time of discharge, the patient was generally well with a mild NIHSS of 2 and was discharged to inpatient rehabilitation.

### CASE DISCUSSION

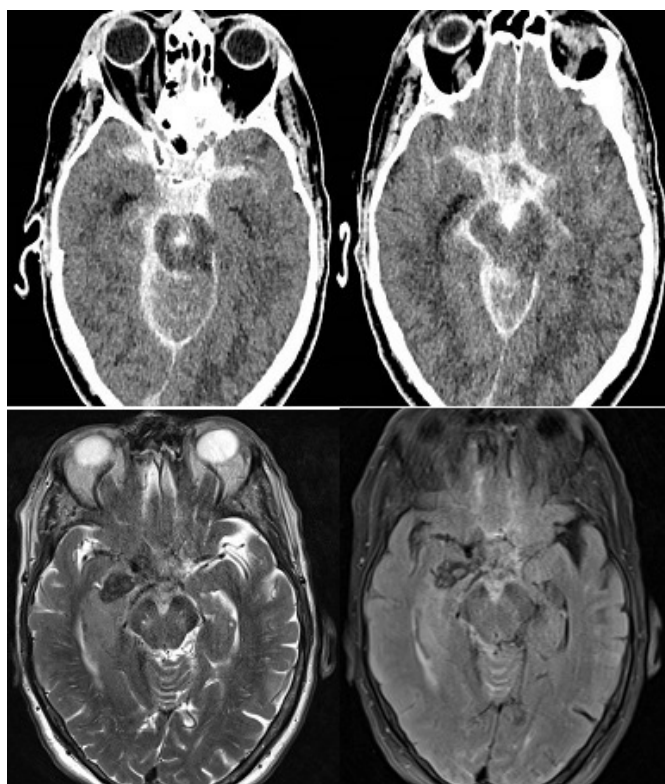
This case of ptosis and oculomotor nerve palsy represents a rare sequela of post-thrombectomy subarachnoid haemorrhage (SAH).

EVT for large vessel occlusion and hemispheric stroke has become standardised practice. Iatrogenic SAH can be a complication of thrombectomy with a systematic review and meta-analysis estimating an incidence of 5.23% with higher correlation relating to distal vessel occlusion and more than three passes with attempted clot retrieval.<sup>1 2</sup> Such cases were associated with higher NIHSS scores and poorer functional independence at 90 days. Severe cerebral vasospasm also appears to be related to higher risk of periprocedural SAH.<sup>3</sup>

Isolated oculomotor nerve palsy has been reported previously as a rare consequence of perimesencephalic SAH (PNSH) with associated ptosis and pupillary involvement.<sup>4</sup> In SAH cases where a cause of bleeding is not readily identified, haemorrhage is either confined to the midbrain in a pattern described as perimesencephalic compared with aneurysmal bleeding confined to the Sylvian and interhemispheric fissures. Cases within the literature support that non-aneurysmal PNSH carries an excellent prognosis with low risk of recurrent bleeding.<sup>5 6</sup>

Focal neurological signs within PNSH appear to be rare with deficits more commonly involving hemiparesis, leg paresis or facial and abducens nerve palsies.<sup>7</sup> Multiple theories have been postulated for the pathophysiology of PNSH including arterial or venous bleeding secondary to thrombosis as in ischaemic stroke, capillary bleeding or arteriovenous malformation. Oculomotor nerve palsy relating to PNSH may be due to haematoma or blood product breakdown, brainstem ischaemia or elevated intracranial pressure causing hypoperfusion of the oculomotor nerve.<sup>8</sup> Traction of small perforator vessels during thrombectomy has also been postulated as another mechanism.<sup>8 9</sup> The absence of headache or specific ocular pain in PNSH also appears to be suggestive of oculomotor ischaemia rather than a compressive pathology.<sup>4 9</sup> The MRI in our case did not reveal brainstem ischaemia or infarction and a follow-up CT angiogram with perfusion did not reveal vasospasm or a new ischaemic penumbra.<sup>10</sup>

In the presented case, the principal differential diagnosis prior to imaging was concern regarding catastrophic intracranial haemorrhage and herniation resulting in an



**Figure 2** CT and 24-hour MRI images. Repeat CT imaging post-thrombectomy at time of deterioration, demonstrating large volume subarachnoid blood at the perimesencephalic area (particularly along the course of the right oculomotor nerve). A 24-hour MRI demonstrating large volume blood products within the supratentorial and infratentorial subarachnoid spaces and the ventricular system.

ipsilateral non-reactive dilated pupil. The initial presentation post-thrombectomy was defined by unilateral ptosis and oculomotor nerve palsy with altered consciousness. Fortunately, imaging demonstrated PNSH rather than sICH and after a period of fluctuating consciousness and headache due to meningeal irritation, our patient made an excellent neurological recovery with complete resolution of her ocular symptoms. It is, therefore, prudent to pursue neuroimaging and expectant management in such cases.

To our knowledge, this is the first published case of isolated ptosis and oculomotor nerve palsy after PNSH following EVT for hemispheric stroke. The case illustrates the risk of SAH in distal vessel occlusion with multiple attempted passes at clot retrieval. The cause of PNSH can be multifactorial and in the case of ischaemic stroke, local nerve ischaemia along with degradation of toxic blood products may be implicated. In these circumstances, repeat neuroimaging is warranted but neurological recovery is often excellent with expectant management.

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#### ORCID iDs

Logesh Palanikumar <http://orcid.org/0000-0001-5812-7491>

Timothy Kleinig <http://orcid.org/0000-0003-4430-3276>

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