

# Abducens nerve palsy in subarachnoid hemorrhage – a clinical sign suggestive of ruptured posterior inferior cerebellar artery aneurysms

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## Introduction

Compressive cranial nerve syndromes can be useful bedside clues to the diagnosis of an enlarging intracranial aneurysm and also guide subsequent evaluation, as with an acute oculomotor nerve (CN III) palsy that is presumed to be a posterior communicating artery aneurysm and a surgical emergency until proven otherwise. Herein, we describe a cranial nerve syndrome for posterior inferior cerebellar artery (PICA) aneurysms that associates subarachnoid hemorrhage and an isolated abducens nerve (CN VI) palsy. The CN VI has a short cisternal segment from the pontomedullary sulcus to Dorello's canal, remote from most PICA aneurysms but in the hemodynamic pathway of a rupturing PICA aneurysm that projects towards Dorello's canal.

## Methods

Clinical and radiological data from 106 surgical patients with PICA aneurysms (66 ruptured, and 40 unruptured) were retrospectively reviewed. A group of 174 patients with other aneurysmal subarachnoid hemorrhage (aSAH) underwent similar analysis to control for non-specific effects of SAH. Univariate statistical analysis compared incidence and risk factors associated with CN VI palsy in subarachnoid hemorrhage.

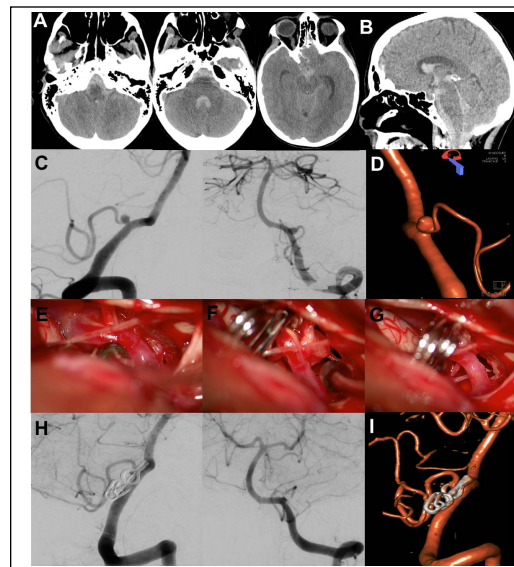
## Results

Overall, 13 of 280 patients (4.6%) had CN VI palsy at presentation, and all of them had ruptured aneurysms (13/240, 5.4%). CN VI palsies were observed in 12 patients with ruptured PICA aneurysms (12/66, 18.1%) and 1 patient with other aSAH (1/174, 0.1%;  $p < 0.0001$ ). PICA aneurysm location in ruptured aneurysms was an independent predictor for CN VI palsy on multivariate analysis ( $p = 0.001$ ). PICA aneurysm size was no different in those patients with or without CN VI palsy (average size 4.4 mm and 5.2 mm, respectively). Within the PICA aneurysm cohort, modified Fisher grade ( $p = 0.011$ ) and presence of a thick cisternal SAH (modified Fisher 3 and 4) ( $p = 0.003$ ) were predictors of CN VI palsy. In all patients with ruptured PICA aneurysms and CN VI palsy, dome projection and presumed direction of rupture were directed toward the ipsilateral and/or contralateral Dorello's canal, in agreement with laterality of the CN palsy. In cases with bilateral CN VI palsies, a medial projection with extensive subarachnoid blood was observed near bilateral canals.

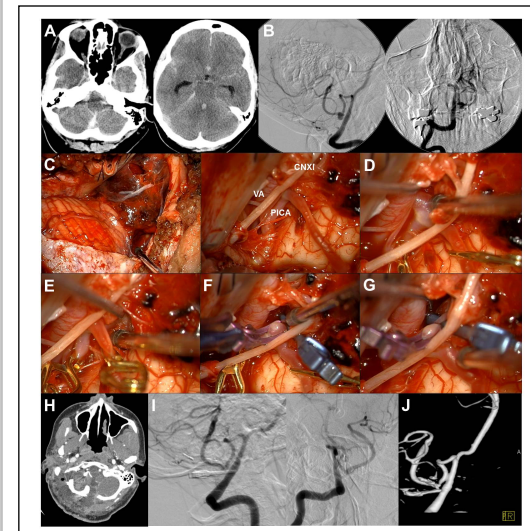
## Conclusions

We establish a localizing connection between an isolated CN VI palsy, SAH, and an underlying ruptured PICA aneurysm. CN VI palsy is an important clinical sign in aSAH and when present on initial clinical presentation may be assumed to be due ruptured PICA aneurysms until proven otherwise.

The deficit may be ipsilateral, contralateral or bilateral, and is determined by the direction of the aneurysmal dome projection and extent of subarachnoid bleeding towards Dorello's canal, rather than by direct compression.



This 65 year-old woman presented with severe headache, diplopia, nausea, and vomiting, and a complete left CN VI palsy was observed on neurological examination (Hunt and Hess 2). SAH was diagnosed on CT scan (modified Fisher grade 4) (Figure 1 A-B), and ruptured 4 mm left PICA aneurysm was seen angiographically, with its dome projecting superolaterally towards the ipsilateral Dorello's canal (Figure 1 C-D). Through a left mini far-lateral craniotomy, the aneurysm was clipped using a straight fenestrated clip over the PICA origin and a second straight clip through a small tag in the fenestration to complete the closure (Figure 1 E-G). Indocyanine green video angiography demonstrated PICA patency and no further filling of the aneurysm. Postoperative angiography confirmed complete occlusion of the aneurysm (Figure 1 H-I). The patient recovered well from surgery, was discharged home 10 days after admission, and had complete recovery of the left CN VI palsy at 6-week follow-up evaluation (mRS of 2).



This 49-year-old woman presented with severe headaches for 2 days, gait disturbances, and a double vision with left lateral gaze, and a left CN IV palsy was observed (Hunt and Hess grade 2). CT showed SAH (modified Fisher grade 4, Figure 2A) and angiography revealed a 6 mm right bilobulated PICA aneurysm (Figure 2 B). The larger lobule projected inferiorly and the smaller lobule projected medially to the contralateral side. Through a right far lateral approach, clot within the cisterna magna was evacuated, the vertebral artery was exposed for proximal control of the aneurysm, PICA was dissected from distal to proximal in the cerebellomedullary fissure to the aneurysm (Figure 2 C-D). With temporary clips placed on PICA proximal and distal to the aneurysm (Figure 2 E), the aneurysm's larger lobule was clipped with a straight and the smaller lobule was clipped with a curved clip. ICG angiography confirmed PICA patency with no residual filling of the aneurysm. Postoperative angiography confirmed complete occlusion of the aneurysm (Figure 2 H-J). Patient recovered well from surgery, was discharged 17 days after admission, and partially recovered her contralateral CN VI function at six-week follow-up evaluation (mRS of 2).