

Cerebellar Arteriovenous Malformation Associated With Multiple PICA Aneurysms : Case Report

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Abstract : A case of cerebellar arteriovenous malformation associated with three aneurysms of the posterior inferior cerebellar artery is reported. Theories concerning the aetiology of the combination of these lesions are reviewed and the problem of which

lesion the priority of surgical management should be directed is also discussed.

Key Words : Intracranial aneurysm, cerebellar arteriovenous malformation, posterior inferior cerebellar artery

INTRODUCTION

Although the coexistence of intracranial aneurysms and arteriovenous malformation (AVM) is not uncommon, it is rarely encountered in the posterior fossa (7, 8, 13). Since Walsh and Kings's first report in 1942, there have been several publications on this association. The incidence of aneurysms coexisting with AVMs ranges from 2.7 % to 9.3 % (7, 11). However, this incidence falls to 0.7 - 0.9 % in the posterior fossa (11, 12, 15).

CASE REPORT

A 44-year-old female was referred to Gazi University Medical School, Department of Neurosurgery on August 25, 1992, with a one-month history of loss of consciousness following a sudden and severe headache. She had been hospitalized with the diagnosis of intraventricular haemorrhage filling the lateral, third and fourth ventricles (Figs. 1 and 2), and remained unconscious for 15 days. She had responded well to conservative treatment, regained consciousness, and was then referred to our clinic.

On admission, she was conscious and alert, complaining of an intense occipital pain. Neurological

examination revealed bilateral papilledema, moderate nuchal rigidity, positive Babinski sign on the left and mild monoparesis of the lower left extremity. She was considered to be a grade 2b on Yaşargil's scale.

On magnetic resonance imaging (MRI) an AVM located posterior to the fourth ventricle was observed (Figs. 3 and 4). Four-vessel digital subtraction angiography revealed an AVM of 3 cm diameter, on the right side of the vermis, being fed by both the vermian and hemispheric branches of the right posterior-inferior cerebellar artery (PICA), and draining into the transverse and sigmoid sinuses and the vein of Galen. Additionally, two aneurysms of the vermian branch, one 0.5 cm in diameter, were also observed (Figs. 5, 6 and 7). On August 31, 1992 a median osteoplastic occipital craniotomy was performed with the patient in sitting position. The vermian branch of the PICA proximal to the AVM was explored and the aneurysm with the greater diameter was eliminated by bipolar coagulation, keeping the lumen of the artery intact. The second aneurysm was then clipped, the distal part of the vermian branch was coagulated and cut just before its entrance to the AVM, and dissection was carried out laterally,

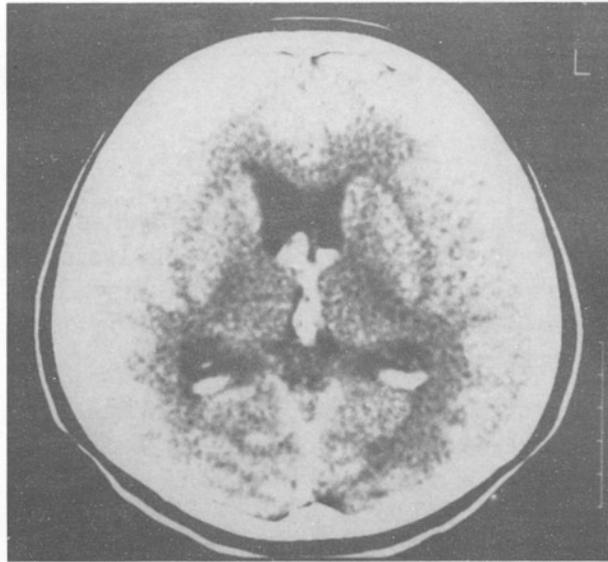
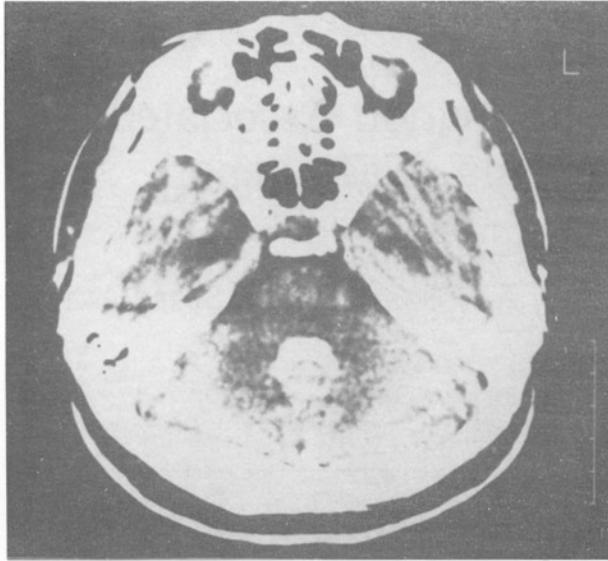


Fig. 1 and 2: Computerized tomography (CT) examinations of the patient showing the haemorrhage filling the lateral, third and fourth ventricles.

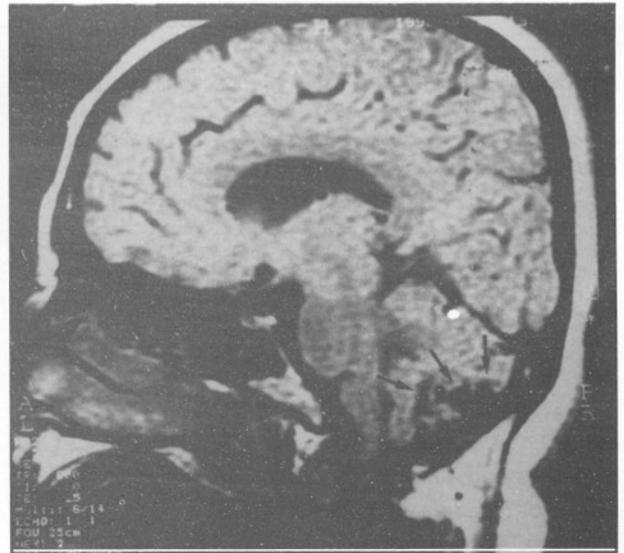
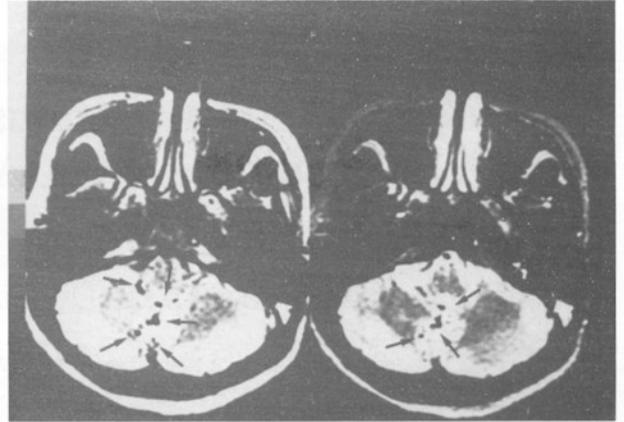


Fig. 3 and 4: Axial (Figure 3) and sagittal (Figure 4) MR images exhibiting an AVM posterior to the fourth ventricle (arrows).



Fig. 5: Early arterial phase of preoperative vertebral angiogram. Two aneurysms (arrows) on the dilated right P.I.C.A. are seen.

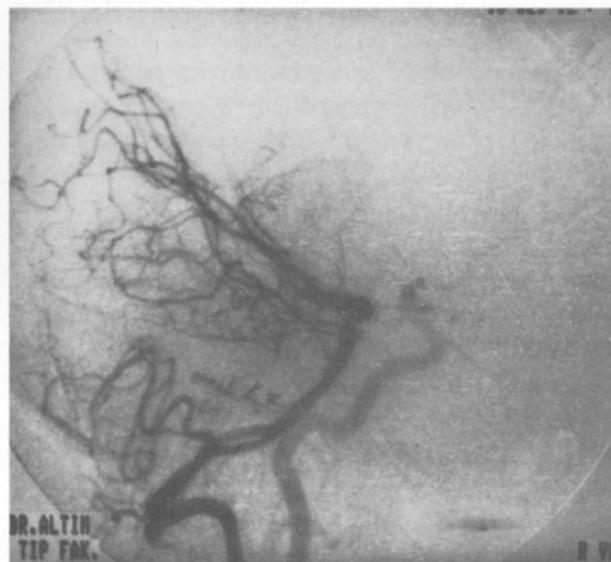
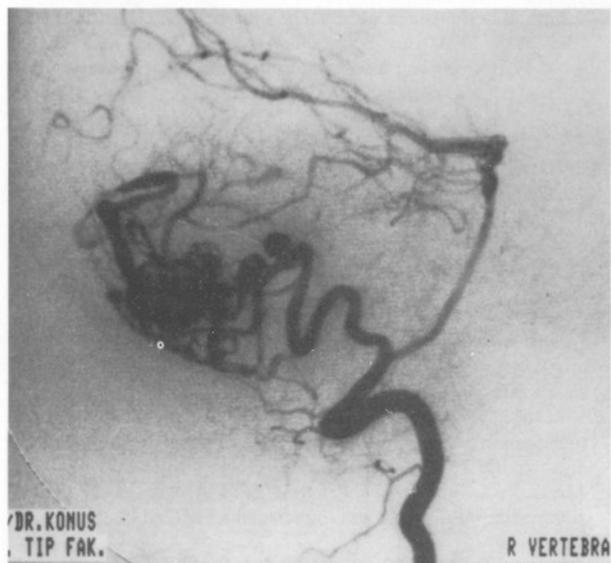
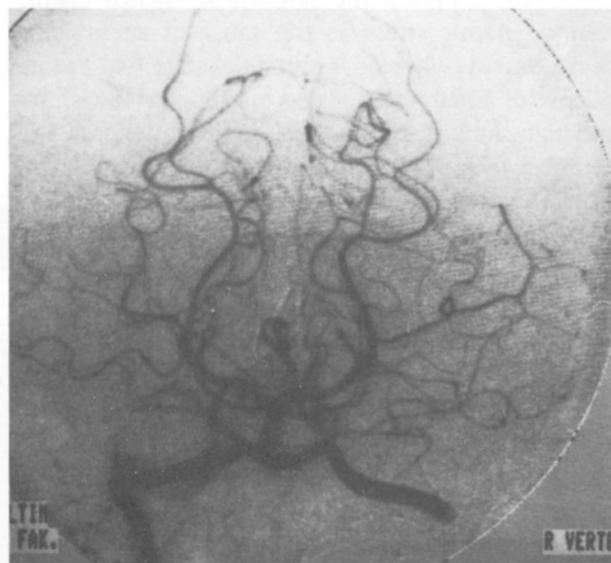
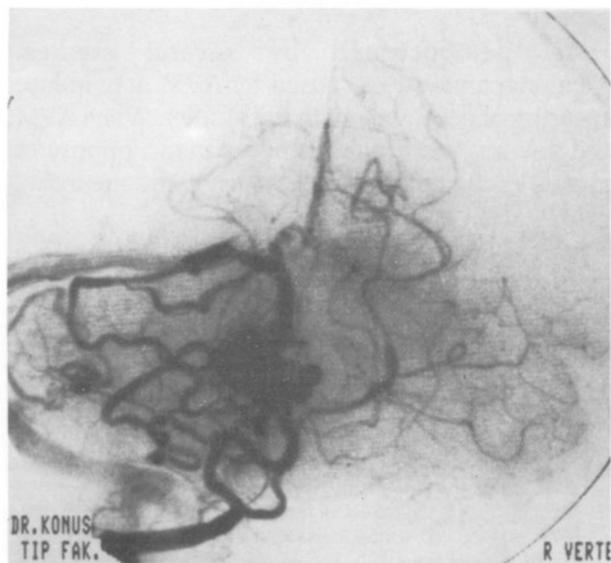


Fig. 6 and 7: Late arterial phase of preoperative vertebral angiogram, anteroposterior and lateral views.

Fig. 8 and 9: Vertebral angiograms performed in the fourth postoperative month, anteroposterior and lateral views.

reaching the feeding arteries of the hemispheric branch. One more aneurysm, which had been obscured by the AVM on angiograms, was seen and clipped and another baby aneurysm was coagulated on the main trunk of the hemispheric branch of the PICA, before its entrance to the AVM. Then dissection and total removal of the AVM was accomplished.

No postoperative complication was observed. The patient was discharged on the 9th postoperative day, with no additional deficit. On control examination in the third postoperative month, she was symptom-free without deficit, and control angiograms exhibited no remnant of the AVM (Figs. 8 and 9).

DISCUSSION

It is well known that patients with intracranial AVM exhibit a tendency to have one or more intracranial aneurysms (4, 11, 15) and these tend to be multiple (6, 12). Three hypotheses have been advanced for the pathogenesis of this association: a) a congenital developmental pathology causing both conditions to occur (1), b) coincidence (4), and c) haemodynamic stress caused by the hypercirculatory state of the AVM, stimulating development of the aneurysm. The fine statistical analysis of Okomata

et al. (10) supports the hypothesis which claims haemodynamic stress as the cause of aneurysmal development, whereas Brown et al state that the incidence of aneurysms located proximal to low-shunt and high-shunt malformations is nearly identical, and the mechanism is not simply based upon high blood flow or high arteriovenous shunt in these systems (3).

Aneurysms are frequently located on the feeding vessels of the AVM. In a series of 39 patients derived from a total of 400 patients with AVM, Cunha e SA et al. divided the aneurysms into IV categories, according to location I— proximal on ipsilateral major artery feeding the AVM, IA— proximal on major artery related but contralateral to the AVM, II— distal on superficial artery feeding the AVM, III— proximal or distal on deep artery feeding the AVM (bizarre), and IV— on artery unrelated to the AVM (5). The authors stated that nearly all the aneurysms that had bled were of type I or II. In another study, 34% of aneurysms were found to be located on a major feeding artery to the malformation and 25% more were on the proximal portion of the feeding system (16). Moreover, atypical localization of aneurysms is more common when they are associated with AVM (10).

Brown et al. estimated the risk of intracranial haemorrhage among patients with coexisting AVM and aneurysm to be 7% at 1 year compared with 3% among those with AVM alone (3). At 5 years, the risk persisted at 7% per year, while it decreased to 1.7% per year in those with an AVM unassociated with aneurysm. Thus, the length of survival free of intracranial haemorrhage was much longer in patients with AVM alone.

When dealing with aneurysms coexisting with AVM, another controversial point is determination of the lesion that has a higher risk of bleeding and has to be treated first. There are some reports about spontaneous disappearance of aneurysms following AVM removal (7, 14). It is generally suggested that the lesion that bled has to be treated first. Cunha e SA et al. defended the same recommendation and stated that an aneurysm could be treated first, only when the cause of the haemorrhage was indefinite (5). As Batjer et al (2), we also believe that priority should be given to aneurysms, especially when they are seated on the feeding system of the AVM, since an increase in proximal intraluminal pressure may lead to rupture of the aneurysm during dissection of the AVM. We also believe that there is no good reason for leaving aneurysms untouched when clip-page is feasible.

CONCLUSION

As demonstrated by several studies, haemodynamic stress caused by AVM may induce aneurysmal development in the vicinity. When AVM and aneurysm are present in a patient, priority of surgical elimination should be given to the aneurysm.

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