Anterior Communicating Artery Aneurysm Associated with Tuberculum Sellae Meningioma

—Case Report—

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Abstract

A 50-year-old male presented with a very unusual case of a calcified anterior communicating artery (AComA) aneurysm associated with a tuberculum sellae meningioma. Until 10 years previously, the patient had been a professional soccer player for 15 years. He noticed a slight decrease in visual acuity in the right eye 7 years before. The patient was in the care of an oculist throughout this period. Two months before admission, a significant and rapid decrease of vision in the right eye occurred. Computed tomography and magnetic resonance imaging showed a round-shaped, partially calcified tumorous lesion. Four-vessel angiography revealed a large AComA aneurysm. During surgery, a tuberculum sellae meningioma was found in combination with an AComA aneurysm with a completely calcified wall. The meningioma was resected totally. The AComA aneurysm with a calcified wall could not be clipped or resected and was left alone. His visual deficit improved postoperatively.

Key words: anterior communicating artery aneurysm, tuberculum sellae meningioma, visual apparatus

Introduction

Intracranial aneurysms may be associated with tumorous lesions, but association of intracranial meningioma with arteriovenous malformation or aneurysm is rare. Intracranial aneurysms with meningiomas have been reported. Enlargement of tuberculum sellae meningioma or anterior communicating artery (AComA) aneurysm may cause disturbances of the visual function. The combination of these lesions will cause disturbances of the visual function earlier than either pathology alone and the loss of vision will progress more rapidly.

We describe a case of AComA aneurysm associated with tuberculum sellae meningioma.

Case Report

A 50-year-old male had been a professional soccer player for 15 years, and was still in excellent health when he retired from the sport. At the age of 43 years he first noticed less clear vision in his right eye than his left eye while watching TV. Examination by an oculist found no visual field deficits, so the patient’s subjective complaint was considered to be the result of his slightly elevated blood pressure. The patient had regular check-ups and since visual acuity in his right eye did not decrease and he adapted to the situation, no special additional diagnostic work-up nor treatment was carried out. He noticed no significant deterioration, until 2 months before admission, when he became convinced and there was progressive and rather rapid loss of vision in his right eye. Ophthalmological examination found a significant loss of vision in the right eye with a slightly degenerated optic disc. Computed tomography (CT) and magnetic resonance (MR) imaging revealed a large (2 cm in diameter), round-shaped lesion at the midline close to the skull base, at the site of the AComA, the chiasm, and between the optic nerves. Neuroimaging indicated a giant, partially thrombosed AComA aneurysm with a partially calcified wall.
wall (Figs. 1 and 2). Four-vessel angiography found an AComA aneurysm (Fig. 3). The diagnosis was a giant, partially thrombosed and calcified AComA aneurysm. He was transferred to our neurosurgical department for treatment.

The frontotemporal approach was used. The sylvian fissure was split along the entire length of the middle cerebral artery (MCA). The tuberculum sellae meningioma was encountered in the typical location. At that point the aneurysm was not visible. The right internal carotid artery (ICA) and the proximal MCA were white due to the homogeneously calcified artery wall. The first segment of the anterior cerebral artery (ACA1) on the right side was rather large and of normal appearance. The right optic nerve, the right optic tract, and the right half of the chiasm had been pushed by the tumor from the anterior and inferior side in the posterior and lateral directions, so that the structures of the visual apparatus were squeezed between the tumor anteriorly and the AComA aneurysm and the ACA1 on the right side posteriorly. The meningioma was separated from the dura at the tuberculum sellae and removed using the piecemeal technique. The right half of the chiasm was paper thin and pressed against the large calcified AComA aneurysm. The right ACA1 was not sclerotic and was double the size of the left ACA1, which was calcified and grayish-white in appearance. The aneurysm mass was oval in shape and hard, and grayish-white in color like the right ICA, the right MCA, and the left ACA1. The second segments of the bilateral anterior cerebral arteries (ACA2s) emerged from the aneurysm, and both were of normal appearance without sclerotic changes of the wall. The aneurysm was very hard and could not be clipped, so was left untreated. The resection of the tuberculum sellae meningioma was expected to allow the chiasm and both optic nerves and optic tracts to move in the anteroinferior direction. There was no more contact between the AComA aneurysm and the visual apparatus.

The patient, who tolerated the procedure well, reported visual improvement on the 1st postoperative day. The patient was discharged from the hospital on the 7th postoperative day. Follow-up CT and MR imaging after surgery showed only the small “tumorous” lesion representing the AComA aneurysm with typical calcifications of the wall (Fig.
Follow-up angiography was not performed since the aneurysm was undisturbed during surgery.

Discussion

Several questions should be addressed and answered in connection with the presentation of symptoms, diagnostic work-up, and surgical treatment of this case. Were the patient’s symptoms characteristic enough to call for a more active approach and earlier imaging? Was there any clue in the clinical picture which could point toward the pathology or even toward dual pathology? Could an exact diagnosis of dual pathology be made on the basis of the available findings from the CT scans, MR images, and angiograms? Was the incomplete surgical treatment correct? Could endovascular treatment be instituted for this fusiform, calcified AComA aneurysm? Should bypassing of the AComA aneurysm have been performed? What should be the next step?

The clinical symptoms had lasted for several years and were subjective (loss of visual acuity in the right eye). The patient was not aware of this disturbance until he closed his left eye while watching TV and noticed that he did not see as well with the right eye as with the left one. The slight arterial hypertension found prior to the objective visual disturbances was considered the probable cause of the subjective, slight visual deterioration in the right eye. No further diagnostic work-up was considered necessary as the fundi were normal at that time. The patient did not come for treatment at his first visit to the oculist but rather for a consultation; later, the patient adapted to the slight deficit and simply ignored it. However, a unilateral decrease in visual acuity should most probably be checked more frequently and more extensively. Since his visual function did not change much in 7 years, it is difficult to say that the clinical picture should have pointed to the pathologies. However, the steady and more rapid loss of vision during the last 2 months did indicate a pathology of the suprasellar region. However, the patient was slightly hypertonic and in good physical condition (never pale), so that a pituitary tumor was unlikely. Finally, the further loss of vision did call for CT, MR imaging, and angiography. The calcifications at the dorsal side of the “tumorous” mass suggested that the whole mass corresponded to an aneurysm. However, careful comparison of the CT scans and MR images with the angiograms showed that neither size nor shape of the aneurysm and the lesion on the CT scans and MR images were not in agreement. In our opinion, the surgical procedure could have been disastrous, if carried out differently from the way it was actually performed. Any attempt to exclude the aneurysm by clipping could crack the calcifications in the wall and result in the occlusion of one or both ACA2s. Performing a bypass from the right ACA1 to both ACA2s was not necessary because there was no further compression on the visual apparatus. This aneurysm could not grow nor rupture, since the wall of the aneurysm was circumferentially calcified.

In our opinion, an AComA aneurysm with a circumferentially calcified wall, with one single functioning ACA1 from the right side entering this calcified aneurysm, and with the origin of the two ACA2s set wide apart, is not amenable to endovascular treatment. Any endovascular procedure in our case would have resulted in the occlusion of both ACA2s. Follow-up CT and MR imaging was performed during the 1st postoperative week and during the 2nd postoperative month, respectively, and the next MR imaging will be performed after one year. We believe that this aneurysm will never rupture, so no treatment will be necessary and future examinations will check for recurrence of the meningioma.

References

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