Case report

Compression of the visual pathway by anterior cerebral artery aneurysm

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Visual failure is an uncommon presenting symptom of an intracranial aneurysm. It is even more uncommon in aneurysms arising from the anterior cerebral artery (ACA). We presented 2 patients with an aneurysm of the A_1 segment of the anterior cerebral artery causing visual field defects. One patient presented with a complete homonymous hemianopia due to compression of the optic tract by a giant aneurysm of the proximal left A_1 segment. The second patient had an almost complete unilateral anopia caused by compression of the optic nerve and chiasm by an aneurysm of the distal part of the A_1 segment with a small chiasmatic hemorrhage and ventricular rupture.

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Visual failure is an uncommon presenting symptom of intracranial aneurysm. When visual disturbances do occur, the aneurysm is usually located at the internal carotid, carotid-ophthalmic, anterior communicating, or posterior communicating artery, resulting in unilateral visual field defects, amaurosis, or bitemporal hemianopia, depending on the location and size of the aneurysm (1-8). Visual failure in subarachnoid hemorrhage is commonly accompanied by pain and ophthalmoplegia (3, 4). The onset of visual loss may be acute or gradual, acuity may sometimes be fluctuating, and the extent of visual field defects may be variable (1, 3, 4, 9). Although several cases have been reported (3, 4, 6–13), visual field defects are not often caused by an aneurysm originating from the anterior cerebral artery (ACA). We describe 2 patients with visual loss due to aneurysms of the ACA.

Case reports

Case 1

During the past 2 months, a 29-year-old woman complained of slowly progressive right-sided hemianopia with intermittent periods of left-sided headache. Her medical history included a radical excision of a malignant melanoma on her back 1 year previously. Physical examination revealed normal visual acuity and a right-sided homonymous hemianopia, confirmed by the Goldmann test. Afferent pupillary disturbance was not present.

CT scanning demonstrated a high-density spaceoccupying lesion in the left suprasellar region. A part of this lesion enhances after intravenous contrast was administered. In the assumption that this lesion might harbor a metastasis of malignant melanoma, lumbar puncture was performed, but no malignant cells were found. Magnetic resonance imaging (Fig. 1) and conventional four vessel angiography (Fig. 2) showed that the lesion seen on CT is a giant aneurysm (1.8 by 2 cm) of the proximal part of the A₁ segment of the left ACA that compresses the optic tract. The lumen of the aneurysm is partially obliterated by a thrombus mass. A second angiogram was performed to ensure the presence of collateral circulation: there is a slight overflow from the right carotid artery to the left ACA during compression of the left carotid artery.

The aneurysm was clipped through a left-sided pterional craniotomy. Because of the size of the aneurysm, the clip slided from the neck of the aneurysm towards the ACA during surgery and it was not possible to guarantee an adequate flow of the left ACA. Despite this, the post-operative course was uncomplicated. Postoperative angiogram showed no contrast filling of the aneurysm. The left ACA was not visualized on the angiogram of the left carotid, but there was

Compression of the visual pathway by ACA aneurysm

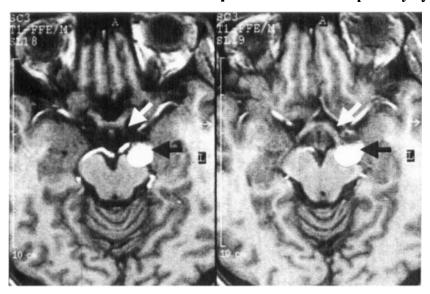


Fig. 1. Preoperative T_1 weighted MRI in case 1 showing a partially thrombosed giant aneurysm of the proximal part of the left A_1 segment (black arrow) which compresses and displaces the left optic tract (white arrow).

overflow from the right carotid artery to the left ACA. The patient's visual fields improved significantly.

Case 2

A 64-year-old man was admitted, because of severe stabbing pain behind the right eye and on the right side of the head. In the 5 days preceding the admission the patient experienced 4 other periods of severe pain in the same area. The third period was accompanied by acute visual field loss of the lower part of the right eye, which was still present

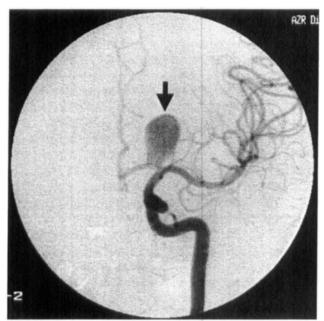


Fig. 2. Case 1. Conventional angiogram reveals the giant aneurysm (black arrow).

on admission. Medical history revealed, besides several general surgical procedures, a period of diplopia 4 years before. This was not confirmed by physical examination at that time. Meningism was not present at physical examination on admission. Apart from a visual field defect in the lower part of the right eye, neurological examination was normal. Cerebrospinal fluid pressure was normal at lumbar puncture and spectrophotometric analysis of the cerebrospinal fluid showed no bilirubin. Goldmann perimetric test demonstrated an almost complete anopia that was mainly localized in the lower half and temporal part of the visual field of the right eye. CT scan showed a small right-sided suprasellar chiasmatic hemorrhage with ventricular

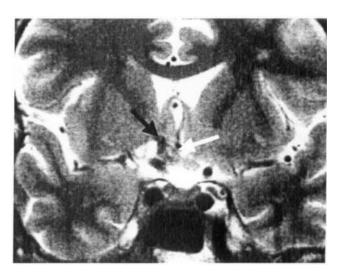


Fig. 3. Preoperative T_2 weighted MRI in case 2 showing an aneurysm of the distal A_1 segment of the right ACA, which points downward (black arrow) and compresses and displaces the optic nerve and chiasm (white arrow).

Table 1. Results of literature search on visual impairment caused by an ACA aneurysm

First author	Year of publication	Number of patients with ACA aneurysms
Henderson (11)	1955	5
Walsh (9)	1964	6 combined ACA and ACoA
Peiris et al. (4)	1980	1
Senter et al. (12)	1982	1
Norwood et al. (3)	1986	1
Versavel et al. (8)	1988	1 (Giant)
Takeshita et al. (13)	1991	1
Tajima et al. (15)	1993	1
Chan et al. (10)	1997	2 combined ACA-ACoA

Fig. 4. Case 2. Conventional angiogram reveals the aneurysm (black arrow).

rupture, without hydrocephalus. Compression of the optic nerve and chiasm was seen with magnetic resonance imaging (Fig. 3). Conventional angiography showed an aneurysm of the distal A₁ segment of the right ACA, pointing downward and slightly forward (Fig. 4) and another aneurysm of the bifurcation of the contralateral middle cerebral artery. During angiography the patient experienced pain in the same area. A repeat CT showed a new lacunar infarct on the right side. A short period of psychosis occurred before aneurysm surgery.

In order to clip both aneurysms, a left pterional approach was used. During surgery it appeared that a part of the dome of the aneurysm was enclaved in the left optic nerve explaining the fact that the hemorrhage was limited to the optic system. Both aneurysms were clipped successfully. The post-operative course was complicated by vasospasm with multiple lacunar infarcts on both sides. Despite this, no neurological deficit was present on discharge besides an organic psychosyndrome, which lasted for 3 weeks. Angiographic follow-up showed that both aneurysms were taken out of the circulation. Visual field examination 5 months postoperatively showed an improvement of the anopia of the right eye.

Discussion

We reported 2 patients with an intracranial aneurysm of the anterior cerebral artery (ACA) causing visual field defects. The first patient had a history of malignant melanoma and was presented with a complete homonymous hemianopia caused by a giant aneurysm of the proximal A₁ segment, without subarachnoid hemorrhage. The visual field defect in this patient corresponded well with the

anatomic lesion of the optic tract. The second patient complained of repetitive stabbing pain behind the right eye and was admitted with an almost complete unilateral anopia. In this patient a small chiasmatic hemorrhage and ventricular rupture was caused by a ruptured aneurysm of the right distal A₁ segment of the ACA. This aneurysm compressed the optic nerve and optic tract. This lesion corresponded with the visual field defect. The aneurysms of both patients were successfully clipped. The visual field of the first patient improved significantly. The anopia of the second patient improved only slightly and he developed a temporary organic psychosyndrome.

The usual clinical presentation of a saccular aneurysm is that of subarachnoid hemorrhage with a severe headache of sudden onset exceeding any headache experienced before, sometimes accompanied with blurring vision for a few seconds or loss of consciousness (6, 14, 15). Compression of intracranial anatomical structures may occur at the time of the bleeding or by a sudden increase in the size of the aneurysm (3). The most common affected site is the third cranial nerve compressed by an aneurysm of the posterior communicating artery or by an aneurysm of the basilar artery between the origin of the posterior cerebral artery and superior cerebellar artery (14). Likewise, but far less common, a distal aneurysm of the anterior inferior cerebellar artery may compress the seventh and eighth cranial nerves as they emerge from the internal auditory meatus (16). Giant aneurysms of the middle cerebral artery may cause epilepsy and contralateral hemiparesis due to compression on the cerebral cortex around the Sylvian fissure (14). Compression of the visual pathway by an intracranial aneurysm leading to visual field defects or decreased visual acuity, is uncommon (1, 3, 5, 6, 8). In a series of 117 patients with SAH, visual field defects caused by an intracranial aneurysm were recorded in 15 cases (13%) (9). In 6 of these 15 cases the aneurysm was located on the anterior

cerebral or anterior communicating (ACoA). Through literature search we found 9 reports (3, 4, 8-13, 15) on visual impairment due to compression of ACA aneurysms involving 19 patients (Table 1). Visual field defects caused by aneurysms from the ACA may be unilateral through optic nerve compression or bilateral (homonymous - and bitemporal hemianopia) if the optic chiasm or optic tract is involved (8, 9, 13). Visual field defects are mostly accompanied by several other ocular signs, such as impaired acuity, pain or scotomas (2, 3, 4, 6). One may be misled by the onset of symptoms: if visual failure due to an aneurysm progresses slowly, the symptoms may resemble that of a tumor (3, 4, 9, 15-18).

The pathogenetic mechanism behind the visual field defects are: direct damage to the nerve fibres, ischemia and/or interruption of axoplasmic flow (14, 19). Spontaneous fluctuation is probably explained by changes in the size of the aneurysm or adjacent hematoma, by variation in intracranial pressure, or by arterial spasm in case of subarachnoid hemorrhage (3, 6, 9). Little is known about the course of visual impairment due to intracranial aneurysms in untreated patients, but worsening of functional loss is to be expected in the majority (4). Sometimes spontaneous improvement has been documented (3). Aneurysm clipping and endovascular treatment may improve the patient's signs and symptoms (3, 5, 7, 8, 15). However, prolonged compression with concurrent papilloedema, and a combination of intra-ocular hemorrhage with ophthalmoplegia is thought to carry a bad prognosis (6, 15).

In conclusion, one should consider an aneurysm of the ACA as a cause of visual impairment.

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