

SEVERE OPTOCHIASMATIC ARACHNOIDITIS AFTER RUPTURE OF AN INTERNAL CAROTID ARTERY ANEURYSM

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SUMMARY — The case of a 24-year-old man with progressive visual loss due to optochiasmatic arachnoiditis is presented. The cause of the arachnoiditis was subarachnoidal bleeding due to rupture of an internal carotid artery aneurysm. The aneurysm was clipped 5 years after the first episode of bleeding. The diagnosis of optochiasmatic arachnoiditis was confirmed during the operation. This case is presented in order to discuss the causes, the symptoms and the therapeutical possibilities of this rare condition.

Aracnoidite optoquiasmática grave após ruptura de aneurisma da artéria carótida interna.

RESUMO — Relato do caso de paciente masculino com 24 anos de idade, com perda progressiva da visão devida a aracnoidite optoquiasmática. A causa da aracnoidite foi ruptura de aneurisma da parede dorsal da artéria carótida interna direita, com hemorragia subaracnóidea. O aneurisma foi clipado 5 anos após o primeiro episódio de sangramento. O diagnóstico de aracnoidite optoquiasmática foi confirmado durante cirurgia. A finalidade do relato é rever e discutir as causas, o quadro clínico e as possibilidades terapêuticas dessa condição rara.

Optochiasmatic arachnoiditis is an unusual inflammatory disease at the base of the skull^{4,18}. The diagnosis is difficult in the majority of the cases and it is based on the history of the patient and the surgical findings. If a correct diagnosis is not made soon the patient may become blind.

We describe the case of a man with progressive bilateral visual loss due to an optochiasmatic arachnoiditis caused by the rupture of an internal carotid aneurysm.

CASE REPORT

JRM, a 24-year-old man presented severe headaches and epileptic fits 5 years prior to admission to our clinic. He remained unconscious during some hours. This was his first epileptic episode and he started to complain of headaches and progressive visual loss since then. Seven months later he was admitted to another hospital for investigation of the headaches and the visual disturbances. At that admission a severe bilateral visual loss with papiledema was observed. Angiographic studies of both carotid arteries were performed and disclosed bilateral subdural hematoma over both fronto-parietal hemispheres. The hematomas were evacuated through frontal burr holes. After this operation the headaches improved, but the visual acuity showed progressive impairment. The patient became blind

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two months after the operation. On May 22, 1986 the patient was admitted to our Department complaining of headaches. His parents reported that he presented since some months ago behavioural disturbances, with confusion and hypersexuality. At examination we found a demented patient, blind, with no cranial nerve palsies nor extremities palsies. The optic nerve discs were pale and atrophic. A CT scan (Fig. 1) showed a hypodense zone in the right temporal pole as well as in the suprasellar region. Angiographic studies revealed an aneurysm of the dorsal wall of the right internal carotid artery, after the origin of the

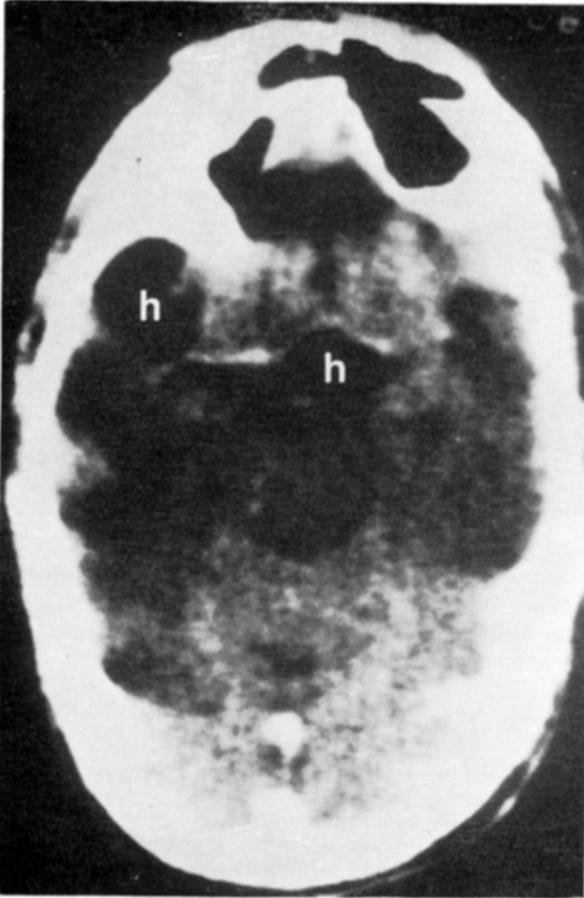


Fig. 1 -- Case JRM. CT-scan examination showing hypodensity (h) in the suprasellar and anterior temporal region.

ophthalmic artery (Fig. 2). This aneurysm was not observed at the first angiography performed 5 years before. Considering the age of the patient and the natural history of ruptured intracranial aneurysms, the surgical treatment was indicated. The aneurysm was exposed through a right fronto-temporal craniotomy. The right temporal pole showed a cystic formation, and a severe arachnoiditis of the whole skull base was noted. An aneurysm of the dorsal wall of the ICA distal to the origin of the ophthalmic artery was found. The dome of the aneurysm was very adherent to the right optic nerve, which probably occluded it after its rupture (Fig. 3). The aneurysm was successfully clipped with an Yasargil clip. The optic nerve and the chiasm were very thin discolored, embebed in a fibrosis with cystic formation. The cause of the bilateral amaurosis of the patient was the severe arachnoiditis produced by the subarachnoidal bleeding. Surgical lysis of the

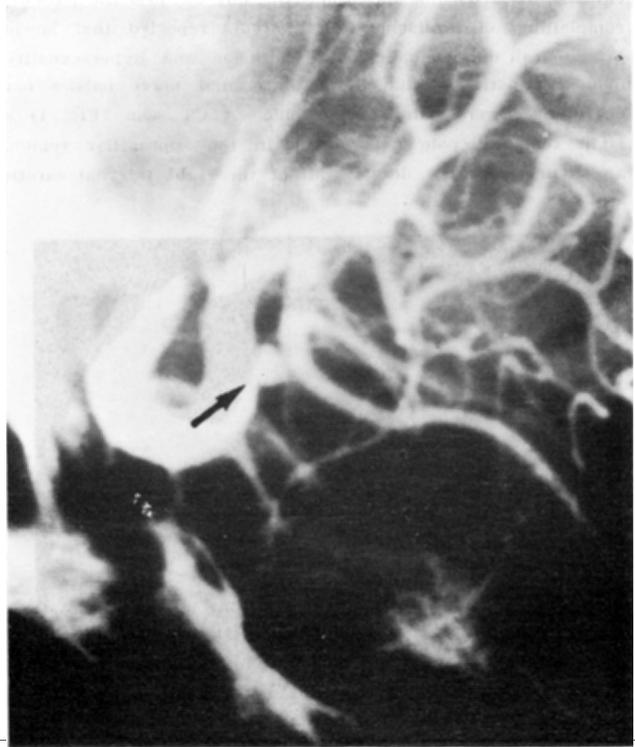


Fig. 2 — Case JRM. Angiographic study of the right internal carotid artery showing the aneurysm in the dorsal wall of this artery, between the ophthalmic and the posterior communicating arteries (arrow).

adhesions was not carried out because the patient was already blind, and it could produce additional damage to other structures at the skull base. The postoperative course was uneventful. The patient could be discharged ten days after the operation without new neurological deficits.

COMMENTS

Optochiasmatic arachnoiditis caused by subarachnoidal haemorrhage due to rupture of intracranial aneurysms has been very seldom reported in the literature⁸. It is an inflammatory process leading to fibrosis and adhesions involving the structures at the base of the skull. Males under 40 years of age are more frequently affected¹⁷. Pathological features are thickening of the arachnoid with adhesions and cystic formation filled with cerebrospinal fluid². The chiasm and the optic nerve may be affected with consequent atrophy of the visual fibers. This condition has multiple causes⁴ including infections such as tuberculous meningitis^{2,10,13,14}, syphilis, acute and chronic meningitis, cysticercosis^{15,16}, infections of the neighbouring structures (paranasal sinuses, teeth)³, trauma¹, polyarteritis nodosa¹², intracranial bleeding⁸, foreign body reaction⁷, after neurosurgical procedures (e.g. exeresis of pituitary tumors). Bleeding from intrachiasmatic arteriovenous malformations usually produces sudden chiasmatic visual field loss⁸. This haemorrhage could eventually lead to a local arachnoiditis. The symptoms are produced by inflammation, direct compression (constriction), or impairment of vascular supply to the optic fibers³ and other neighbouring structures.

The clinical features are multiple, usually with loss of visual acuity, which presents a variable course. It may be progressive, with either monocular or binocular involvement, with progression to partial or total blindness, occurring slowly or rapidly. Variable changes in the visual fields have been observed^{5,8}. Additional features are: headache, retro-ocular pain, paralysis of the extraocular muscles, nystagmus, pupillary inequality, absent corneal reflex, polydipsia, polyuria, obesity, amenorrhea, impotence, uni-

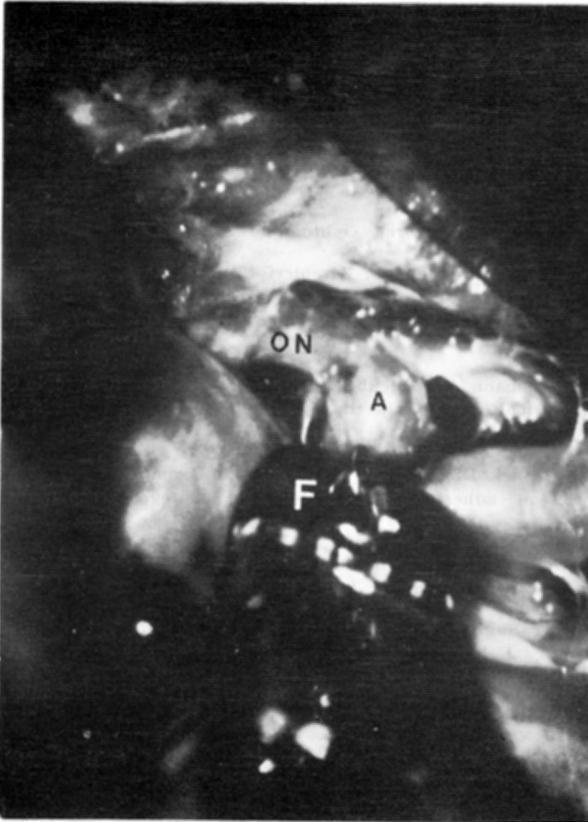


Fig. 3 — Case JRM. Operative picture: the relationship of the aneurysm (A) to the optic nerve (ON) can be seen. F, forceps.

lateral or bilateral anosmia, facial paralysis, and auditory and visual disturbances¹⁸. The correct diagnosis is usually difficult and it is based upon the anamnesis and the findings at the time of surgery. Computed tomography examination, cerebral angiography, RIHSA (radioactive human serum albumin) cisternography, pneumocisternoencephalography may be helpful, but they often fail to indicate the diagnosis¹¹. The majority of intracranial saccular aneurysms are located at arterial divisions. Ohara et al.¹¹ described a 1% incidence for those aneurysms unrelated to arterial junctions. Aneurysms arising from the dorsal wall of the internal carotid artery are uncommon^{9,20}. The proximity of this kind of aneurysm to the optic nerve and chiasm may produce direct compression of these structures or, as in our case, delayed effects from the bleeding. The cause of the arachnoiditis in our case was the subarachnoidal bleeding due to rupture of this aneurysm. This could be clearly demonstrated at the operation. It is surprising that so few cases of optochiasmatic arachnoiditis caused by rupture of intracranial aneurysms have been reported in the English literature^{6,8}. A reason for this is not presently known. The localization of the aneurysm could be an additional factor in the etiology of this inflammatory process. The operation of aneurysms in the acute stage, with removal of clots and blood around the optic structures could eventually avoid this condition.

The treatment of optochiasmatic arachnoiditis remains controversial. Surgical lysis of the adhesions and removal of cysts in the cystic form of the disease is related to improve the symptoms only in minority of cases⁸. Dexamethasone has been used in the majority of the cases but a complete relief of the symptoms can not always be obtained. Recently, cyclophosphamide was successfully used after failure of surgical lysis of the adhesions and dexamethasone therapy⁶. This diagnosis should be suspected when a patient presents progressive visual loss after subarachnoidal bleeding. Treatment should be started as soon as possible, for it can eventually avoid visual disturbances or even blindness, as it occurred in our case.

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