Spontaneous healing of intracranial aneurysms after subarachnoid hemorrhage

Case report

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A case of spontaneous intra-aneurysmal thrombosis, verified angiographically, is reported in a patient with subarachnoid hemorrhage and without surgical intervention. The frequency of such an occurrence and the factors involved are reviewed and discussed.

KEY WORDS • intracranial aneurysm • subarachnoid hemorrhage • cerebral angiography • spontaneous thrombosis • antifibrinolytic drugs

Few cases of complete spontaneous thrombosis verified during life have been reported in the medical literature in patients with subarachnoid hemorrhage (SAH). Recently, in this clinic, we managed a patient whose anterior communicating artery (ACoA) aneurysm failed to fill with contrast material at a second preoperative angiogram 17 days after SAH. The case is presented here, and the literature is reviewed. The question of the frequency of such an occurrence, the factors that might favor such an event, and the permanence of such a thrombosis are discussed.

Case Report

This 26-year-old woman was admitted 3 days after SAH, at the onset of which she had briefly lost consciousness.

On admission, there was neck stiffness but no focal neurological signs; she was normotensive and in good general health. The cerebrospinal fluid was bloody, and the supernatant xanthochromic. There was no significant family history, but for several years she had been taking an oral contraceptive regularly (at the time of admission, Neovlar 21*).

Four days after SAH, a single saccular aneurysm, 10 × 8 × 8 mm in size, arising from the ACoA was demonstrated at four-vessel cerebral angiography (Fig. 1 upper left) using rapid serial angiography and Isopaque (meglumine and calcium metrizoates). There was no evidence of vasoospasm or hematoma. In addition to bed rest, she was given epsilon-aminocaproic acid (EACA), 30 gm intravenously per day for the first 4 days, then 36 gm daily by mouth.

During the next few days the patient became drowsier and developed papilledema, with 2 diopters of protrusion. Repeat spinal
Spontaneous healing of intracranial aneurysms

FIG. 1. Right internal carotid angiography with compression (subtraction) 4 days (upper left), 17 days (upper right), 3 months (lower left), and 14 months (lower right) after SAH. The aneurysm (arrow) is clearly seen on Day 4 but is not visible on the subsequent angiograms.

tap showed no sign of rebleeding and the deterioration was attributed to vasospasm. She was given dexamethasone (Decadron), 16 mg daily, and her condition gradually improved. Right carotid angiography was repeated 17 days after SAH (Fig. 1 upper right). Unexpectedly there was no filling of the aneurysm, despite adequate communication across the ACoA on cross-compression of the carotid artery. Severe arterial spasm was widespread. There was no evidence of progressive ventricular dilatation, with a ventriculocranial index unchanged at 0.31. The patient's condition continued to improve slowly; dexamethasone and EACA were gradually reduced in dose and discontinued by the 24th day. The papilledema gradually subsided after the introduction of acetazolamide (Diamox), 20 mg/kg/day, and furosemide (Lasix), 40 mg daily, on the 24th day. The patient was discharged home 6 weeks after the hemorrhage. She remained well, and after 5 months returned to her previous occupation as a social worker.

To ensure that there was still no filling of the aneurysm, right carotid angiography with cross-compression was repeated twice more, 3 months and 14 months after SAH (Fig. 1 lower). On both occasions, contrast filling of the ACoA was adequate, but on neither occasion was there any filling of the aneurysm. No ventricular dilatation developed. The patient remains well 3 years after SAH.

Discussion

It was suggested by Dandy, in 1944, that perhaps as many as 15% of patients with SAH due to ruptured intracranial aneurysms may be healed by spontaneous thrombosis of the aneurysms. This estimate has been supported by some autopsy studies. For example, Crompton reported complete thrombosis in 21 of 166 patients (13%) who died without surgical interference, and Husepian and Pool reported 10 similar instances among 113 patients (9%). In both series the incidence was smaller in patients whose aneurysms had not bled. On the other hand, Krayenbühl found only one totally thrombosed aneurysm in a series of 7452 autopsies.

The high incidence of complete thrombosis that was found at autopsy has not been encountered during life. At the Karolinska Hospital between 1964 and 1973, rapid serial
cerebral angiography was repeated, without intervening surgery, in 78 patients (84 aneurysms) with SAH, at intervals varying from 2 days to 8 years. Twelve aneurysms had increased in size, six were smaller although there was no vasospasm, the one reported in this article had disappeared, and the remaining were unchanged. From our experience, it seems unlikely that more than 1% or 2% of ruptured intracranial arterial aneurysms will spontaneously thrombose completely. Furthermore, such a thrombosis must be complete to provide a cure, and the fact that partially thrombosed aneurysms may rebleed is well documented. A review of the literature has revealed only six cases in which complete thrombosis of an intracranial arterial aneurysm has been established during life following SAH, without surgical interference with the aneurysm or its parent artery, locally or in the neck. In these six cases, an aneurysm was demonstrated radiologically following SAH, but failed to fill with contrast medium when angiography of the appropriate vessel was repeated after intervals ranging from a few weeks to several years. We have omitted cases in which the artery feeding the ruptured aneurysm did not opacify during the repeat angiography because of gross spasm obliterating the feeding artery or a failing circulation in a moribund patient. The spontaneous disappearance of a number of aneurysms of mycotic origin has been satisfactorily established by a few authors. It is easy to imagine the process by which inflammation of the aneurysm wall might lead to thrombosis and r. pair. A similar process combined with partial narrowing of the aneurysmal neck may account for the complete occlusion encountered in cases where craniotomy has been performed and the aneurysm dissected but not completely ligated or clipped. Similarly, the gradual obliteration of an aneurysm treated by stereotaxic radiosurgery may occur by the same process. It is also well documented that an aneurysm may diminish in size or completely fail to fill with contrast material after partial or complete temporary or permanent intracranial or extracranial occlusion of the principal feeding artery.

Logically, attempts have been made to promote thrombosis in berry aneurysms by combining antifibrinolytic therapy with regionally retarded circulation and controlled hypotension. Khil'ko and Shkliarova reported successful thrombosis in six of nine cases with saccular aneurysms arising from the internal carotid, but no case of complete thrombosis with an ACoA aneurysm. Mullan reported the combined use of partial carotid ligation, hypotensive medication, and antifibrinolytic therapy in 15 patients with bleeding berry aneurysms, while awaiting the optimum time for operation. No information was given regarding the radiological appearance of the aneurysms at the time of intracranial surgery.

In the present case, when angiography on the 17th day after SAH showed no filling of the aneurysm, it was not considered justifiable to expose the patient to the risks of intracranial surgery. The factors leading to the occlusion remain uncertain. Antifibrinolytic therapy and the previous use of oral contraception may have had an influence, although the effect of the latter on coagulation and fibrinolysis in vitro are conflicting. If the failure of the aneurysm to fill with contrast material was due to blood clot, such a clot might recanalize, and rebleeding occur. As Mullan and his coworkers have pointed out, the blood clot that seals the initial aneurysmal hemorrhage seems clinically to have a very short life, as does the clot initiated artificially with an electric current. Only time will indicate the degree of permanence of the thrombosis in the present case and the correctness of the decision to defer surgery, which was contrary to the decision to operate in the case reported by Höök and Norlén.

It is clear from this review that the spontaneous thrombosis of ruptured intracranial arterial aneurysms without intervention is rare and too infrequent for the outcome in the cases in which it does occur to be predicted with confidence.

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Spontaneous healing of intracranial aneurysms

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