Traumatic intracranial aneurysm due to arterial injury at surgery

Case report

G. REES COSGROVE, M.D., JEAN-GUY VILLEMURE, M.D., AND DENIS MELANÇON, M.D.

Departments of Neurosurgery and Neuroradiology, McGill University, Montreal Neurological Hospital, Montreal, Quebec, Canada

A case is presented of false intracranial aneurysm of traumatic origin which developed following direct surgical attack on a congenital anterior communicating artery aneurysm. The secondary aneurysm was discovered on the 25th postoperative day and was clipped at a subsequent procedure. The possible pathophysiology is discussed with respect to other direct vessel injuries.

KEY WORDS □9 traumatic aneurysm □9 false saccular aneurysm □9 intracranial aneurysm □9 subarachnoid hemorrhage □9 arterial injury

TRAUMATIC intracranial aneurysms are rare. Burton, et al., 4 classified trauma sustained by the intracranial arterial vasculature to be of either a direct or indirect nature. Indirect trauma occurs during blunt head injury, whereas direct trauma usually occurs during penetrating head injury (such as by depressed skull fragments or projectiles). Direct injury to the cerebral vessel may also occur during surgery.

The following case report describes the development of a secondary false aneurysm near the operative site following a direct surgical attack on a congenital aneurysm of the anterior communicating artery (ACoA). The case is fully documented with preoperative and postoperative arteriograms and confirmed by operative findings on two occasions.

Case Report

This 36-year-old woman was perfectly well until 1 month prior to admission, when she suddenly developed a severe headache associated with nausea. This headache lasted approximately 12 hours but resolved spontaneously, and she did not seek medical advice. On the day of admission, she again suffered a severe bifrontal headache of sudden onset, followed by a brief period of unconsciousness.

Examination. The patient was drowsy and somewhat confused. Her vital signs were stable and she had a normal neurological examination except for a small flame-shaped hemorrhage on left funduscopy.

Computerized tomography (CT) scanning revealed a moderate amount of subarachnoid blood in the interhemispheric and right Sylvian fissures. Four- vessel cerebral angiography demonstrated a 6-mm saccular aneurysm of the ACoA and a hypoplastic left A1 segment (Fig. 1). Repeat CT scanning on the 4th day after subarachnoid hemorrhage (SAH) revealed ischemia of the right frontal pole surrounding an intrahemispheric clot. Clinically, the patient was stable and a repeat angiogram 10 days after admission showed no change in the aneurysm and a moderate amount of spasm of the anterior cerebral vessels.

First Operation. On the 11th day after SAH, a right pterional approach was used to locate the aneurysm. The lesion was approximately 4 × 6 mm in size and pointed anteriorly and inferiorly. The neck was dissected and clipped without difficulty. There was no obvious bleeding and no other aneurysm was present.

The patient was alert postoperatively, without neurological deficit. Her only complaints consisted of mild persistent frontal headaches. A CT scan performed on the 12th postoperative day revealed that the previous right frontal hematoma was resolving, but that a significant mass effect remained.

Three weeks postoperatively, she suddenly experi-
G. R. Cosgrove, J. G. Villemure and D. Melançon

FIG. 1. Preoperative right internal carotid angiogram, oblique view, revealing a small saccular aneurysm of the anterior communicating artery.

enced a severe bifrontal headache with loss of consciousness. A CT scan revealed recent SAH with extension of a frontal hematoma across the midline (Fig. 2). Repeat angiography revealed a large (15 × 10 mm) saccular aneurysm with a narrow neck arising from the region of the ACoA. There was no clip occluding the neck of this aneurysm (Fig. 3 left).

Second Operation. Twenty-five days after her first operation and 4 days after her second SAH, the patient underwent surgical reexploration. The previously applied aneurysm clip was found to be completely occluding the neck of the original aneurysm. Only 2 mm from this aneurysm, a second aneurysm with a narrow neck was observed originating from the superior aspect of the ACoA. This aneurysm had not been present at the first procedure. During dissection of this secondary aneurysm, profuse hemorrhage was encountered. This was managed with local pressure, packing, and controlled hypotension. The neck of the aneurysm was then occluded with a small Heifetz clip.

The patient made an excellent recovery without neurological deficit. A postoperative angiogram confirmed obliteration of both aneurysms (Fig. 3 right).

Discussion

Although over 100 cases have been reported of intracranial aneurysm developing after closed or penetrating head injury, only a handful have been described due to direct arterial injury at surgery. Finkemeyer was the first to describe such a case in 1955. In that case, an aneurysm of the right middle cerebral artery developed after removal of a meningioma of the right orbital roof. Other authors have since reported cases of aneurysm formation following obvious direct arterial injury during surgery for brain tumors. Other reports include aneurysm formation following repeated subdural taps, evacuation of subdural hematoma, removal of nasal polyps, ventricular tap, mastoidectomy, and transphenoidal surgery. More recently, cases have been reported of false aneurysm formation at the suture line of an extracranial-intracranial vascular anastomosis.

The pathophysiology of traumatic aneurysm formation is not completely understood. In the majority of cases, direct injury to the artery at time of surgery was obvious, as witnessed by profuse and brisk hemorrhage. A through-and-through laceration or the complete disruption of the full thickness of the arterial wall results in a false aneurysm with organized hematoma or brain tissue forming the aneurysm wall. Most reported cases of direct traumatic aneurysms are of this type.

True aneurysms arise when the arterial wall is only partially disrupted. In most cases of this type, the internal elastic lamina and media are damaged but the adventitia remains intact, forming the outer wall of the aneurysm. This has been demonstrated experimentally by White. Two cases have been reported of aneurysm development at the site of temporary Mayfield clip placement. There was no obvious injury at surgery and it was therefore postulated that the clip had somehow damaged the arterial wall; the resultant weakness produced subsequent dilatation and aneurysm formation. Drake has suggested an additional hypothesis; that the tearing of a small arterial branch from the parent artery could leave a complete full-thickness defect in
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the vessel wall. With confined hemorrhage, a false aneurysm would develop.

In our case, it is unlikely that adventitial stripping played any significant role, as no dissection was carried out on the superior aspect of the ACoA where the secondary aneurysm arose. Indirect injury to the vessel wall is doubtful as manipulation at surgery was minimal. There was no obvious hemorrhage at surgery and therefore it is unlikely that the aneurysm developed at the site of any significant laceration. In addition, most aneurysms that develop from a full-thickness rent in the arterial wall have a wide base. The secondary aneurysm we discovered had a narrow neck.

It is possible, especially during sustained hypotension at surgery, that a small perforation could seal rapidly with a hemostatic plug; as arterial blood pressure returned to normal levels postoperatively, this weak point could rupture, leading to false aneurysm formation. In the same way, if a small perforating arterial branch of the ACoA was torn at its origin, an aneurysm could form at this defect in the vessel wall.

References