Rapid formation of giant aneurysm: case report

LARRY C. FRIED¹ AND ANUNCIACION YBALLE

From the Departments of Neurosurgery and Pathology, University of California, Medical School at Davis, California, U.S.A.

SUMMARY A case is presented which documents the formation of a giant intracranial aneurysm over a period of less than three months. It had previously been postulated by several authors that giant aneurysms form from haematomas in communication with the aneurysm. The gross appearance and histology of the lesion in our case supports this theory.

It is generally believed that the formation of a giant intracranial aneurysm may take years (Cuatico, Cook, Tyshchenko, and Khatib, 1967; Morley and Barr, 1969). Actually, there is very little documentation regarding the rapidity and mechanics of growth of these aneurysms. Several authors have postulated that these lesions form from haematomas in communication with the aneurysm (du Boulay, 1965; Sadik, Budzilovich, and Shulman, 1965; Crompton, 1966; Raimondi, Yashon, Reyes, and Yarzagaray, 1968). The haematomas undergo organization and then are incorporated into the wall of the aneurysm. If this hypothesis is true, then theoretically these lesions could form in just a few months.

We are reporting the following case because it provides some insight into the formation of these interesting lesions.

CASE REPORT

(Sacramento Medical Center no. 14-53-98) This 46 year old white male was completely well until the morning of 19 July 1970, when, while bending over to put on his trousers, he suddenly developed a severe, dull, throbbing headache originating in the back of the neck and extending over the occiput into the frontal region. The headache became progressively worse during the day and he was admitted to the Sacramento Medical Center that evening. Initially in the hospital he felt nauseated and vomited several times. A spinal tap showed uniformly grossly bloody cerebrospinal fluid, with an opening pressure of 220 mm/H₂O. The past history was negative. The physical examination revealed mild nuchal rigidity; the remainder of the examination was negative.

The next day cerebral angiography was performed

by the femoral route. A 13 × 15 mm aneurysm was noted arising from the left anterior cerebral artery. During that evening the patient became restless, complained of severe headache, and then became deeply comatose. His pupils were widely dilated but still reacted to light. He showed decerebrate responses to painful stimuli. Intermittent spontaneous hyperventilation was noted. The patient remained in this condition for several days. Lumbar puncture showed bloody cerebrospinal fluid with the pressure greater than 500 mm of H₂O. Repeat arteriography one week later revealed enlargement of the aneurysm as well as evidence of an intracerebral haematoma in the left frontal lobe (Fig. 1).

In spite of his poor clinical condition, surgery was performed on 29 July. The haematoma was evacuated from the left frontal lobe. This consisted of 50 ml. of old liquifying blood clot. At the base of the haematoma cavity the dome of the aneurysm was identified. A feeding artery believed to originate from the anterior communicating artery was occluded. The dome of the aneurysm was excised after two Mayfield clips were applied high on the broad neck. Methylacrylate was used to coat the remaining aneurysm and clips.

Postoperatively the patient remained in coma without any signs of improvement. His course was complicated by several bouts of pneumonia.

Two months after surgery a RISA cisternogram suggested communicating hydrocephalus.

On 10 October, during an attempt at lumbar drainage, the cerebrospinal fluid changed from clear to bloody. At that time the patient stopped breathing and could not be resuscitated.

NECROPSY FINDINGS The freshly removed brain weighed 1,700 g. The gyri were flattened and fresh blood was noted in the subarachnoid cisterns. The left frontal lobe was largely replaced by a 'giant aneurysm' arising at the point of junction of the left anterior cerebral artery and anterior communicating artery (Fig. 2). This lesion resembled a large

¹ Present address: Division of Neurological Surgery, Medical College of Virginia, Richmond, Virginia 23219, U.S.A.

FIG. 1. Left carotid angiogram on 28 July. The aneurysm had enlarged dramatically. Note the severe spasm of the intracranial internal carotid artery and proximal anterior and middle cerebral arteries.

FIG. 2. Basal view of the brain showing location of aneurysm.

FIG. 3. Hemisection of aneurysm.

FIG. 4. Section through wall of the giant aneurysm. Recently formed granulation tissue and organizing blood clot are evident.

organizing haematoma except that it incorporated the previous aneurysm and was so well encapsulated that it easily shelled out of the brain. The aneurysm measured 5 cm in transverse diameter, 7 cm in anteroposterior diameter, and 7 cm in superior-inferior diameter. The anterosuperior portion had a thin layer of fresh blood. Cross-sections of the brain showed a moderate amount of fresh blood in dilated lateral and third ventricles. There was a clip on the left anterior cerebral artery only across one-half the diameter of the vessel, the residual uncompressed lumen seemed to feed the aneurysm directly.

Section through the aneurysm showed a small zone of lamination 1.5 cm from the neck which represented the wall of the pre-existing aneurysm (Fig. 3). Otherwise the aneurysmal sac was filled with old blood clot. Microscopic sections of the wall showed a layer of proliferating granulation and fibrous tissue (Fig. 4).

DISCUSSION

We have presented this case because it documents the rapid growth of an intracranial aneurysm of originally moderate size to a giant aneurysm in the short duration of two months. Since the feeding vessel to this aneurysm was not completely clipped and a significant portion of the aneurysm was not excised, there remained a locus for re-expansion of the sac and further haemorrhage. Although the methylacrylate coat was no guarantee against rupture of the aneurysm, it should have prevented re-expansion of the wall to gigantic proportions.

Drake and Vanderlinden (1967) have shown that repeated haemorrhage often follows incomplete treatment either by clipping or plastic coating. Although both techniques were used in our case they proved to be inadequate. Re-expansion of aneurysms after incomplete (or even adequate) treatment is not rare (McKissock, 1965). Partial ligation may leave a locus minoris resistentiae that can slowly increase in size and rebleed (Björkstén and Troupp, 1962). However, these recurrent aneurysms have been of modest size.

Cuatco *et al.* (1967) believed that the process of enlargement is quite slow and insidious, spanning a period of years, perhaps a decade before signs and symptoms became manifest. In their two cases they performed carotid ligation for the treatment of internal carotid aneurysms. In one case a small aneurysm grew to 4.5×4 cm in $3\frac{1}{2}$ years.

Jane (1961) reported a case of an immense aneurysm of the posterior inferior cerebellar artery in a 1 year old child. The size was $6 \times 5 \times 5$ cm, and it consisted of laminated clot in various stages of organization. Obviously, even if this lesion developed *in utero*, it represents a fairly rapid formation of a giant aneurysm. In our case there was essentially no lamination of the aneurysm wall except for a remnant of the pre-existing aneurysm. The wall was composed of only one layer of granulation and fibrous tissue. Therefore, it is possible that a giant

aneurysm may be initiated by only one limited haemorrhage. Although the location of the aneurysm in the haematoma cavity may be fortuitous, we believe it is also consistent with recurrent haemorrhage and subsequent organization rather than re-expansion of an intact aneurysm.

REFERENCES

- Björkesten, G. af, and Troupp, H. (1962). Changes in the size of intracranial arterial aneurysms. *Journal of Neurosurgery*, **19**, 583–588.
- du Boulay, G. H. (1965). Some observations on the natural history of intracranial aneurysms. *British Journal of Radiology*, **38**, 721–757.
- Crompton, M. R. (1966). Mechanisms of growth and rupture in cerebral berry aneurysms. *British Medical Journal*, **1**, 1138–1142.
- Cuatco, W., Cook, A. W., Tyshchenko, V., and Khatib, R. (1967). Massive enlargement of intracranial aneurysms following carotid ligation. *Archives of Neurology*, **17**, 609–613.
- Drake, C. G., and Vanderlinden, R. G. (1967). The late consequences of incomplete surgical treatment of cerebral aneurysms. *Journal of Neurosurgery*, **27**, 226–238.
- Jane, J. A. (1961). A large aneurysm of the posterior inferior cerebellar artery in a 1-year-old child. *Journal of Neurosurgery*, **18**, 245–267.
- McKissock, W. (1965). Recurrence of an intracranial aneurysm after excision. Report of a case. *Journal of Neurosurgery*, **23**, 547–548.
- Morley, T. P., and Barr, H. W. K. (1969). Giant intracranial aneurysms: diagnosis, course, and management. Proceedings of the Congress of Neurological Surgeons, Toronto, 1968. *Clinical Neurosurgery*, **16**, 73–94.
- Raimondi, A. J., Yashon, D., Reyes, C., and Yarzagaray, L. (1968). Intracranial false aneurysms. *Neurochirurgia*, **11**, 219–233.
- Sadik, A. R., Budzilovich, G. N., and Shulman, K. (1965). Giant aneurysm of middle cerebral artery. A case report. *Journal of Neurosurgery*, **22**, 177–181.