Growth and Spontaneous Regression of a Middle Cerebral Artery Aneurysm after Surgical Clipping

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Summary

We present a case of growth and spontaneous regression of a middle cerebral artery aneurysm after surgical clipping. A 36-year-old woman who presented with grade II subarachnoid haemorrhage was found to have a right middle cerebral artery aneurysm. The aneurysm was surgically clipped in view of the morphology of the aneurysm. Second clipping was performed four months later as there was re-growth of the neck of the aneurysm. Again re-growth of the aneurysm was noted five months after the second clipping.

As further clipping of the aneurysm would be difficult, external and internal carotid arteries bypass was the treatment option but unfortunately, the by-pass operation was unsuccessful. Interestingly, angiogram performed after the by-pass surgery revealed partial thrombosis and decreasing size of the aneurysm. Complete obliteration of the aneurysm was noted without further intervention one month after the failed by-pass surgery.

Introduction

Growth of aneurysm after surgical neck clipping and endovascular coiling has been reported. Various explanations of the growth of aneurysm have been hypothesized, namely haemodynamic stress and biological events such as angiogenesis^{1,2,3}. A case of growth and spontaneous regression of a ruptured middle

cerebral artery aneurysm after surgical clipping is presented. The biological events governing the growth and regression of the aneurysm are discussed.

Case Report

A thirty-six years old woman first presented with headache in December 1999. There was no neurological deficit on examination. CT scan of the brain revealed intracerebral hemorrhage at the right temporal lobe and Hunt and Hess grade II subarachnoid hemorrhage.

Cerebral angiogram demonstrated a wide neck aneurysm at right middle cerebral artery (MCA) bifurcation (figure 1). The lenticulostriate perforators originated from the superior trunk, whereas the anterior temporal artery originated from the inferior trunk of the right MCA. In view of the morphology of the aneurysm, surgical clipping was elected and performed. A small neck remnant of the aneurysm was noted in the post-operative angiogram as well as spasm of the M1 and A1 segment of the right middle and anterior cerebral arteries (figure 2). With conservative treatment, the patient recovered with excellent outcome without any evidence of vasospasm.

Follow-up cerebral angiogram in April 2000 revealed re-growth of the aneurysm (figure 3). The patient was asymptomatic. Second clipping of the aneurysm was performed. Again, a small neck remnant persisted after second surgical

Figure 1 A) Non-contrast CT scan of brain demonstrated haematoma at right temporal lobe with subarachnoid haemorrhage at right sylvian fissure. B) Subsequent cerebral angiogram revealed an aneurysm at right middle cerebral artery bifurcation.

clipping. The neck remnant of the aneurysm slightly enlarged over the following two months and enlarged further to become a bilobed aneurysm subsequently in September 2000 (figure 4). At this stage, the M1 segment of the right middle cerebral artery had decreased in caliber. As further clipping of the aneurysm would be difficult, external carotid and internal carotid arteries by-pass followed by endovascular sacrifice of the MCA was the treatment option, but unfortunately the by-pass operation was unsuccessful. Endovascular coiling was not considered in view of the wide neck of the aneurysm.

Interestingly, angiogram performed after the failed by-pass surgery revealed partial thrombosis and decreasing size of the aneurysm (fig-

ure 5). Partial remodelling of the insular branches of the right middle cerebral artery was also present with sufficient hemispheric circulation. One week later, the patient presented with weakness of the left upper limb, which lasted for two days and recovered subsequently. Cerebral angiogram at that time demonstrated decreased flow in the aneurysmal sac with a clot in the sac of the aneurysm that bulged into the middle cerebral artery (figure 6). There was decreased flow to the distal MCA branches, which were tiny in caliber compared to previous angiogram.

Complete obliteration of the aneurysm and right middle cerebral artery at its bifurcation was demonstrated in the latest angiogram in October 2000 (figure 7). Thrombosed right middle cerebral artery extended to the first insular branch that was remodelled. The hemispheric circulation was not temporalized. Collateral circulation from leptomeningeal supply of right anterior cerebral artery and infratemporal branches of the right posterior cerebral artery was noted. Clinically, mild weakness of the right hand persisted, but was slowly recovering with physiotherapy. No further treatment was proposed at that time.

Discussion

Growth of aneurysm after surgical neck clipping and endovascular coiling has been reported. Various explanations of the growth of aneurysm have been hypothesized, namely haemodynamic stress and biological events such as angiogenesis^{1,2,3}. The growth and regress of an aneurysm is still a myth. The inability to predict the occurrence, recurrence or rupture of an arterial aneurysm leads to frustration¹ and failure of treatment. Angiogenesis and vascular remodelling undoubtedly play a major role in the genesis, growth and rupture of cerebral aneurysms, though little is known about the specific biological mechanisms^{1,2,3,4}. Gross architectural molecular disruption in the walls of intracranial aneurysms has been demonstrated and this illustrates an apparent biological response involving angiogenesis factors². Vascular remodelling, an adaptive phenomenon, occurs throughout life, in response to external stimuli such as haemodynamic changes or injury to vessel wall⁴.

There are three possible outcomes with vas-

Figure 2 A) Small neck remnant of the aneurysm in the post-operative angiogram. Note the spasm of M1 segment of right middle cerebral artery and A1 segment of the right anterior cerebral artery. B) The neck remnant of the aneurysm was more clearly visualized in the 3-D rotational angiography.

Figure 3 Re-growth of the aneurysm in the subsequent cerebral angiogram (A) frontal and (B) lateral views.

cular remodelling⁵, the aneurysm may: i) regress without intervention due to repairs of endothelial cells after a vascular injury, ii) be stable without increasing in size over the years due to failure of repairs, iii) increase in size and eventually rupture secondarily to failure of repair and healing of the vessel wall.

Unfortunately, we are unable to predict the growth rate or re-growth of the aneurysm based on its morphological appearance. In our illustrated case, the rapid growth of the aneurysm after second clipping needed further intervention in view of the postulated instability of the angioarchitecture and the supposed risk of re-bleeding. The sudden regression

could be explained by the fact that endothelial repair exceeded endothelial destruction. However, the underlying triggering factors, which could be intrinsic or extrinsic, remained unanswered. It is a pity that the angiogenetic mechanism is not fully understood at this stage. With better understanding of the biological event of the pathogenesis of cerebral aneurysm, the management of cerebral aneurysm will have a new horizon.

Mortality from re-bleeding of a ruptured cerebral aneurysm is high. The re-bleeding rate of an incompletely obliterated cerebral aneurysm after surgical clipping or endovascular coiling is the other important and interesting

Figure 4 A) Small neck remnant persisted after second surgical clipping. B) The neck of the aneurysm enlarged further to become a bilobed aneurysm five months after the second clipping.

Figure 5 A) Frontal and (B) lateral internal carotid angiogram revealed partial thrombosis and decreasing size of the aneurysm after the failed external and internal carotid arteries by-pass surgery.

Figure 6 The size of the aneurysm was smaller with clot within the sac of the aneurysm. There was extension of the clot into the middle cerebral artery as well. Flow to the distal MCA branches especially the superior trunk was decreased and the caliber of the MCA branches was tiny.

phenomenon worth discussing. This will invariably affect the management of the patient and thus the mortality and morbidity. After two to nine years' angiographic follow-up of surgically treated cerebral aneurysms, David et Al⁶ reported 2.5% enlargement of small residue neck and 1.9% re-bleeding per year. Similarly, Feuerbeg et Al⁷ reported 3.8% re-growth and 3.8% re-bleeding rate of incompletely clip aneurysms. The majority of the neck remnants remained stable or spontaneously obliterated.

In comparison to the group treated by endovascular coiling, Cognard et Al⁸ reported 14% re-growth of aneurysm with no re-bleeding. One re-bleeding (0.8%) from re-growth of aneurysm after endovascular treatment was reported by Fuse et Al⁹. On the other hand,

Byrne et Al¹⁰ reported 14.7% re-growth and 7.9% re-bleeding. Aneurysm re-growth is related to the morphology of the aneurysm as illustrated by Hayakawa et Al¹¹. 17% of small aneurysms with small neck recanalised after incomplete coiling. A higher rate of recanalization was noted in large and giant aneurysms with neck remnant of was 87% and 90% respectively¹¹. The reported rate of re-growth and re-bleeding of an incompletely occluded aneurysm is variable. Again, this points to the intrinsic biological event of the genesis and growth of an aneurysm that is perhaps, variable in individual aneurysms.

Therefore, the decision for further treatment of a re-growth aneurysm is multifactorial and needs to be individualized.

Conclusions

Vessel wall biological behavior is certainly influenced by shear stress changes. However, the simple rheologic interpretation of growth and repair of arterial aneurysm does not provide an acceptable explanation to anticipate outcome. Caution should be placed on the indications of high morbidity intervention in some instances.

The growth and regression of an aneurysm is unpredictable and at the moment beyond our control. We may stabilize the disease by mechanical means. However, specific causative mechanisms associated with the biological behavior of the lesions have remained elusive. Continuing research is mandatory before myth becomes evidence.

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Figure 7 A) Frontal and (B) lateral cerebral angiogram showed complete obliteration of the aneurysm and the right middle cerebral artery at its bifurcation. Thrombosed right middle cerebral artery extended to the first insular branch that was remodelled.