Long term follow-up of patients with coiled intracranial aneurysms
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Long term 3T-MRA follow-up after therapeutic occlusion of the internal carotid artery to detect possible de novo aneurysm formation

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ABSTRACT

Background and purpose: the purpose of this study was to assess the incidence of de novo aneurysm formation, the incidence of subarachnoid hemorrhage (SAH) and growth of existing untreated aneurysms in 52 patients after therapeutic carotid artery balloon occlusion for carotid aneurysms.

Patients and Methods: Between January 1996 and August 2004, 52 patients were treated with carotid artery balloon occlusion for carotid aneurysms. In June 2005, all patients, their next of kin or family physicians were contacted and asked for episodes of headaches or hospital admissions that could be attributed to subarachnoid hemorrhage (SAH). In addition, MRI and MRA at 3.0 Tesla were performed in 26 of 44 surviving patients after a mean follow-up period of 50.2 months (median 43.5, range 14-107 months). MRI and MRA studies were compared with the digital subtraction angiograms at the time of carotid artery occlusion.

Results: During clinical follow up of 52 patients of mean 50.3 months (median 42.5, range 0-107 months), no episodes of SAH were reported (0%, 97.5 % CI 0-8.2%). In the 26 patients with follow up MR, no de novo aneurysms were detected (0%, 97.5 CI 0-13.2%). Five existing untreated small aneurysms in 5 patients had not enlarged after a mean follow up of 40 months.

Conclusion: in this study, therapeutic carotid artery occlusion was not associated with development of new aneurysms or enlargement of existing untreated aneurysms over time.
INTRODUCTION

Therapeutic carotid artery balloon occlusion is a simple, safe and effective method in the treatment of carotid aneurysms that are not suitable for surgical clipping or selective occlusion with coils. After carotid occlusion, hemodynamic alterations will occur in the circle of Willis with increased flow over the contra lateral carotid artery and anterior and/or posterior communicating arteries. There is some concern, expressed in anecdotal reports, that these hemodynamic changes predispose for the formation of de novo aneurysms or enlargement of existing untreated aneurysms, exposing the patient to a risk of subarachnoid hemorrhage (SAH).

In this study, MRI and MR angiography (MRA) at 3.0 Tesla was used to assess the incidence of de novo aneurysm formation and the occurrence of enlargement of existing untreated aneurysms, long term after therapeutic carotid artery balloon occlusion. In addition, we assessed the incidence of subarachnoid hemorrhage (SAH) during an average follow up period of 50.3 months.

MATERIAL AND METHODS

Patients
Between January 1996 and August 2004, 52 patients were treated with carotid artery balloon occlusion for carotid aneurysms. In June 2005, a follow up survey was performed. During the follow up, 8 patients had died: one patient died shortly after carotid occlusion of initial SAH, one patient died of trauma, two elderly patients died in hospital of pneumonia and multiple organ failure, one patient died in hospital of chronic obstructive pulmonary disease, one patient died of lung cancer, one patient died in hospital of cardiac infarction and one patient died at home of “old age”. None of these patients died of SAH. Of the remaining 44 surviving patients, 37 were contacted and asked for episodes of headaches or hospital admissions that could be attributed to SAH. Seven patients could not be traced, but previous clinical and MRI follow up was available in medical records.

Follow up MRA was offered to these 37 patients and 28 (76%) agreed to participate in this study. Between January and June 2005, MRI and MRA were performed in 26 of 28 patients. In two patients, MR imaging could not be performed because of claustrophobia in one and severe cervical kyphosis in the other patient. There were 21 women and 5 men with a mean age of 60.6 years (range 28-81 years). The mean follow-up period of 26 patients after carotid occlusion was 50.2 months (median 43.5, range 14-107 months).

Clinical follow up of all 52 patients, including the deceased patients and patients that could not be traced but who had previous follow up, was mean 50.3 months (median 42.5, range 0-107 months). This study was approved by the ethical committees of both participating hospitals.

MRI protocol
MRI and MRA was performed on a 3.0-Tesla system (Philips Intera R10, Philips Medical Systems, Best, The Netherlands) using the sensitivity encoding (SENSE) phased array head coil (MRI Devices, Gainesville, Florida, USA).

The MR protocol included axial and coronal T2-weighted fast spin echo, coronal T1-weighted spin echo and high resolution multi slabs 3D-TOF (MOTSA) MRA sequences. Imaging parameters for the T1-weighted spin echo sequence were 570/12 (TR/TE), 256x256 matrix (reconstructed to 512x512), 180-mm field of view, 90% rectangular field of view, 3-mm thick sections with 0.3-mm gap. Parameters for the T2-weighted fast spin echo sequence were 3394/80 (TR/TE), 400x400 matrix.
(reconstructed to 512x512) 230-mm field of view, 70% rectangular field of view, 3-mm thick sections with 0.5-mm gap. For the MOTSA 3D-TOF MR sequence the parameters were as follows: 3D fast field echo T1-weighted sequence, 21/4 (TR/TE) flip angle 20º, 512x512 matrix (reconstructed to 1024x1024), 200-mm field of view, 85% rectangular field of view, 1.0-mm thick sections, interpolated to 0.5-mm, 160 slices acquired in 8 slabs. The measured voxel size of the MOTSA 3D-TOF MR sequence was 0.39 x 0.61 x 1 mm and the reconstructed voxel size 0.2 x 0.2 x 0.5 mm. MRI and MRA studies were interpreted by two experienced neuroradiologists in consensus and were compared with the digital subtraction angiograms at the time of carotid artery occlusion to assess the incidence of de novo aneurysm formation and growth of additional aneurysms. All patients and their family physicians were informed on the imaging findings.

Statistical analysis
We recorded the overall number of de novo aneurysms and overall number of episodes of SAH and calculated cumulative incidence rates with corresponding 95% confidence intervals (CI).

RESULTS

In the 26 patients, no de novo aneurysms (cumulative incidence rate 0%, 97.5% CI 0-13.2%) were identified on MRA after a mean follow up of 50.2 months (median 43.5, range 14-107 months, 109 patient years). Five of 26 patients had one untreated additional aneurysm on the following locations: anterior communicating artery 2 mm (figure 1), left ophthalmic artery 3 mm (figure 2), left middle cerebral artery 5 mm, right M2-M3 junction 3 mm and left superior cerebellar artery 2 mm. None of these 5 small additional aneurysms had enlarged in size after a mean follow up of 40 months (range 21-60 months, 16.6 patient years).

During clinical follow up of all 52 patients of mean 50.3 months (median 42.5, range 0-107 months, 218 patient years), no episodes of headache or hospital admissions that could be attributed to SAH were reported (cumulative incidence rate 0%, 97.5% CI 0-8.2%).
Figure 1
A. Additional 1.7-mm anterior communicating artery aneurysm (arrow) in a 71-year-old woman with a giant right cavernous sinus aneurysm treated with carotid artery occlusion.
B. MRA 28 months after carotid artery occlusion demonstrates unchanged size (arrow).

Figure 2
A. Left internal carotid angiogram at the time of right internal carotid artery occlusion for a giant ophthalmic aneurysm in a 43-year-old woman shows additional small mirror ophthalmic aneurysm with a wide neck (arrow).
B. MRA maximum-intensity-projection image after 27 months demonstrates unchanged size (arrow).
C. MRA source image after 27 months demonstrates unchanged size (arrow).
DISCUSSION

There is some concern that increased hemodynamic stress in the circle of Willis after therapeutic balloon occlusion predisposes to the formation of de novo aneurysms or enlargement of existing aneurysms. Since no systematic follow up data are available, frequency is not well established and publications are anecdotal. 5-9. In a review 6 of 19 articles published between 1962 and 2000 describing 30 cases with angiographically proven de novo aneurysm formation after carotid occlusion, incidence in 7 large series (including more than 30 carotid occlusions) varied from 0.7% to 3.4% (average 2%), whereas two other smaller series (with fewer than 30 procedures) reported an incidence of 10% and 11%, respectively. On the other hand, other reviews of carotid ligation (some including more than 100 patients), failed to disclose new aneurysms, so cumulative incidence may be lower than 2% 6, with unknown annual incidence rate. The interval between carotid occlusion and the onset of symptoms due to de novo aneurysms specified in 28 cases varied from 3 to 25 years with an average of 9.6 years with 20 of 28 (74%) between 3 and 10 years. The location of 27 de novo aneurysms was the anterior communicating artery in 11 (41%), the carotid artery in 14 (52%) and the vertebral artery in two (7%).

The incidence of de novo aneurysm formation and recurrent SAH in patients with aneurysms treated by clipping was recently established in large studies. 11-13 In these studies, CT angiography was used to detect de novo aneurysms and enlargement of existing aneurysms long term after surgical clipping of a ruptured aneurysm. In 610 patients, 19 definitive de novo aneurysms were found in 14 patients (after a mean interval of 9.1 years) and 42 probable de novo aneurysms were found in 34 patients. This corresponds to an overall incidence of de novo aneurysm formation between 2.3 and 10% and an annual incidence between 0.37 and 1.20%. Of 19 definitive de novo aneurysms, 12 (63%) were located on the middle cerebral artery, 2 (11%) on the anterior communicating artery, and 5 (26%) on the carotid artery. Of these 19 aneurysms, 18 (95%) were smaller than 5 mm. In the same study, 4 of 18 (22%) existing aneurysms had enlarged in the first 5 years of follow up and 13 of 53 (25%) in the first 10 years. In a study of 752 patients with previously clipped ruptured aneurysms, the cumulative incidence of recurrent SAH was 3.2% in the first 10 years after initial SAH 13 and was 22 times higher than expected in populations with comparable age and gender.

The relatively high cumulative incidence of 2.3-10% of de novo aneurysm formation in patients with clipped aneurysms and the cumulative incidence of recurrent SAH of 3.2% in the first 10 years seem higher than the estimated incidence of less than 2% of symptomatic or asymptomatic de novo aneurysm formation after carotid artery occlusion, although patients with carotid artery occlusion have not been studied systematically. Therapeutic carotid occlusion probably does not increase the incidence of new aneurysm formation over time compared to patients with clipped aneurysms, but may influence the location of these new aneurysms. After carotid occlusion, new aneurysms occur almost exclusively on the contra lateral internal carotid artery and anterior communicating artery; these vessels are the site of main hemodynamic changes with increased blood flow to supply the contra lateral carotid circulation. In patients with clipped aneurysms, new aneurysms most frequently develop on the middle cerebral artery, a location seldom encountered after carotid occlusion.

Our prospective study, although small and with limited follow up, confirms the assumption that carotid artery occlusion by itself does not seem to induce new aneurysm formation. However, longer term follow up will be needed for definitive conclusions, since new aneurysms may develop after as long as 25 years. Our MRA protocol on a 3.0 Tesla system 10 is a suitable screening tool for intracranial aneurysms. High resolution images of cerebral vasculature without administration of
contrast material are obtained and aneurysms of 2 mm or even smaller (figure 1) can be depicted with confidence.

Balloon or coil occlusion of the carotid artery is generally used in symptomatic large and giant carotid aneurysms and is a simple, safe and effective treatment. Tolerance to carotid occlusion can be tested reliably by clinical and angiographic test occlusion protocols, even in patients under general anaesthesia. Fear of inducing new aneurysm formation by hemodynamic stress is probably unfounded and should not be an argument to proceed to alternative therapies that preserve patency of the carotid artery but are technically more challenging. These alternative therapies, such as direct surgical clipping, bypass surgery or endovascular treatment with coils or liquid embolics with assistance of a supporting balloon or endovascular stent, are associated with higher complication rates. In our practice, these therapies are restricted to patients who cannot tolerate carotid artery occlusion.

CONCLUSION

In this study therapeutic carotid artery occlusion was not associated with development of new aneurysms or enlargement of existing untreated aneurysms over time. Longer-term follow-up will be needed to draw more definitive conclusions.

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