New developments in imaging and treatment of intracranial aneurysms

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Chapter 6

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 (SAH). In addition, magnetic resonance imaging (MRI) and magnetic resonance angiographic (MRA) at 3 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 mean 50.3 months (median 42.5, range 0-107 months), no episodes of SAH were reported. In the 26 patients with follow up MR, no de novo aneurysms were detected (0%, 97.5 Confidence Interval 0-13.2%). Five existing untreated small aneurysms in 5 patients had not enlarged after a mean follow up of 40 months.

CONCLUSION
Therapeutic carotid artery occlusion is not associated with a higher risk for 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 not suitable for surgical clipping or selective occlusion with coils (1-4). After carotid occlusion, hemodynamic alterations will occur in the circle of Willis with increased flow over the contralateral 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) (5-9). In this study, we used MRI and MRA at 3.0 Tesla (10) 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 (SAH) during a follow up period of mean 50.3 months.

MATERIALS EN 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 and 6 elderly patients died of cardiac or pulmonary disease. None of these patients had 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 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, total 1310 months). Clinical follow up of all 52 patients, including the deceased patients and patients that could not be traced but who had a previous follow up, was 2616 months (mean 50.3 months, median 42.5, range 0-107 months).

This study was approved by the ethical committees of the 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 sensivity 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 MOTSA 3D-TOF 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 chunks. 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.

Fig 1 Follow up MRA in a 71 year old woman 28 months after right carotid artery balloon occlusion for a giant cavernous sinus aneurysm. An additional 1.7 mm anterior communicating artery aneurysm (arrow) was unchanged in size.
Fig 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.
B and C: MRA MIP and source images after 27 months demonstrates unchanged size.

Long term 3T-MRA follow-up after therapeutic occlusion of the internal carotid artery to detect possible de novo aneurysm formation.
RESULTS

In the 26 patients, no de novo aneurysms (0%, 97.5% Confidence Interval 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 small (2-5mm) aneurysm (fig 1 and 2) and none of these 5 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 occurred.

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 (8) of 19 articles published between 1962 and 2000 describing 30 cases with angiographically proven de novo aneurysm formation after carotid occlusion, incidence varied from 0.7 to 3.4% with an average of 2%. On the other hand, in other reviews of therapeutic carotid artery occlusion, no mention is made of similar cases, so cumulative incidence may be lower than 2% (8), 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 prospective studies (11,12,13). 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 (13), the cumulative incidence of recurrent SAH was 3.2% in the first 10 years after initial SAH and was 22 times higher than expected in populations with comparable age and gender.
In view of the relatively high incidence between 2.3 and 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, it is unlikely that the reported estimated incidence of symptomatic or asymptomatic de novo aneurysm formation after carotid artery occlusion of less than 2% can be attributed to carotid artery occlusion alone. In fact, all patients with diagnosed aneurysms have a much higher risk of new aneurysm formation and (recurrent) SAH than the general population and there is no solid evidence that therapeutic artery occlusion increases this risk. Our prospective study, although small and with limited follow up, confirms this assumption. 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 \(^{(1,14)}\). Fear of inducing new aneurysm formation by hemodynamic stress is no argument to refrain from this simple therapy and to proceed to other therapies with much higher complication rates such as direct surgical clipping, bypass surgery or endovascular treatment with coils or liquid embolics with assistance of a supporting balloon or endovascular stent \(^{(15-18)}\). These therapies should be restricted to patients who cannot tolerate carotid artery occlusion. In conclusion, therapeutic carotid artery occlusion is not associated with a higher risk for development of new aneurysms or enlargement of existing untreated aneurysms over time.

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REFERENCES


