New developments in imaging and treatment of intracranial aneurysms

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Chapter 9

Summary
CHAPTER 1

In Chapter 1 we describe the background and aim of the study.

CHAPTER 2

In Chapter 2, we compared coils coated with transforming growth factor-β (TGF-β) to standard platinum coils in an experimental setting. TGF-β coil modifications are aimed primarily at increasing the “biological activity” of the coil, because standard platinum coils are biologically inert and fail to induce sufficient scar formation. TGF-β is a ubiquitous growth factor that has been demonstrated to increase collagen synthesis, and endothelialization in vivo. These biological functions are considered beneficial in the setting of coil embolization of aneurysms. In 12 New Zealand White rabbits elastase-induced saccular aneurysms were embolized with standard platinum coils or with TGF-β coated coils. The aneurysms embolized with TGF-β coated coils underwent earlier cellular aneurysm neck coverage than those embolized with standard coils, but differences in coverage between coated and control coils were no longer present at later time points. We concluded that these data suggest that improvements in intra-aneurysmal cellular proliferation resulting from coil modifications, although significant in the early postembolization phase, dissipate over time.

CHAPTER 3

In Chapter 3, we compared clinical and angiographic results of embolization of 101 intracranial aneurysms with Nexus coils to historical results of coiling of 120 aneurysms with GDC 10 coils and 115 with Cordis TruFill coils (both standard platinum coils) using an identical methodology. Nexus coils are standard complex platinum coils with interwoven PGLA microfilament threads. Polyglycolic/polylactic acid (PGLA) addition to bare platinum coils is intended to reduce the reopening rate of coiled intracranial aneurysms. Initial occlusion in aneurysms treated with Nexus coils was (near) complete in 97 aneurysms and incomplete in 4 aneurysms. There were no permanent procedural complications (0 in 95 patients, 0%, 97.5% CI 0.0-3.3%). Mean aneurysm volume was 180.2 mm³ (median 71, range 5-1624 mm³). Mean packing was 19.4% (median 18.3%, range 7.5-38.9%). Six months angiographic follow up in 87 of 101 aneurysms showed incomplete occlusion in 14 (16%) and 12 of those (14%) were additionally coiled. The mean packing of 19.4% of Nexus coils was significantly lower than 22.9% for GDC 10 and 29.7% for Cordis TruFill coils. Other clinical results, including reopening and retreatment rates, were not statistically different. We concluded that there was no reduction in the reopening rate in aneurysms treated with Nexus coils.
CHAPTER 4

In Chapter 4, we evaluated the morbidity, mortality, and angiographic results of coiling of asymptomatic incidental aneurysms and compared the characteristics of these patients and aneurysms with those of patients with incidental aneurysms that were not treated. During a 10-year period, 97 patients without a previous subarachnoid hemorrhage, presented with incidentally found intracranial aneurysms. In 48 of these patients, 58 aneurysms were coiled. Permanent morbidity of coiling was 2.1% (1 of 48) and mortality was 0%. Of 46 aneurysms with angiographic follow up, 45 were completely or near completely occluded. To obtain these results, 3 aneurysms were coiled more than once. Coiled incidental aneurysms did not rupture during the median follow-up period of 28.5 months. Compared to untreated patients with incidental aneurysms, coiled patients were younger and more often had multiple aneurysms. Aneurysms of coiled patients more often had a small neck, were more often located on the carotid artery, and were less often located on the middle cerebral artery. We concluded that coiling of incidental intracranial aneurysms has a low complication rate in selected aneurysms and patients.

CHAPTER 5

In Chapter 5, we used 3.0 Tesla Magnetic Resonance Imaging to evaluate aneurysm size in relation to evolution of clinical symptoms in 39 patients with large or giant carotid artery aneurysms treated with therapeutic carotid artery occlusion. These patients had clinical and magnetic resonance imaging (MRI) follow-up of at least 3 months (mean, 35.9, median 29, range 3–107 months, 117 patient-years). Clinical presentation was mass effect caused by the aneurysm in 32, subarachnoid hemorrhage in 3, and epistaxis in 1 patient. Two aneurysms were an incidental finding and 1 was additional to another ruptured aneurysm. All aneurysms seemed to have thrombosed completely after carotid artery occlusion as observed on early and late MRI and MRA follow-up studies. At the time of the latest MRI follow-up, 29 (74%) of the 39 aneurysms were obliterated totally, two aneurysms decreased to 25% of the original diameter, two aneurysms decreased to 50%, and five aneurysms decreased to 75%. Two aneurysms remained unchanged in size after 49 and 58 months. At the latest clinical follow-up evaluation, symptoms of mass effect were cured in 19 (60%), improved in 10 (31%), and remained unchanged in three (9%) of the 32 patients. We concluded that therapeutic carotid artery occlusion is a simple, safe, and effective treatment for large and giant carotid artery aneurysms. Almost all aneurysms involuted completely or substantially decreased in size over time. Alleviation of symptoms of mass effect was achieved in most patients.
CHAPTER 6

In Chapter 6, we used 3.0 Tesla Magnetic Resonance Angiography to assess the incidence of the novo aneurysm formation and growth of existing untreated aneurysms in patients after therapeutic carotid artery occlusion for carotid aneurysms. 3.0 Tesla MRA and MRI was 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 digital subtraction angiograms at the time of carotid artery occlusion. During a clinical follow-up of 50.3 months (median 42.5, range 0-107 months), no episodes of SAH were reported. In the 26 patients with follow-up MRA, 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.

We concluded that therapeutic carotid artery occlusion is not associated with a higher risk for development of new aneurysms or enlargement of existing untreated aneurysms over time.

CHAPTER 7

In Chapter 7, we assessed the incidence of visible fenestrations of the anterior communicating artery (AcomA) on 3D rotational angiography (3DRA) and evaluated the relationship between fenestrations of the AcomA with aneurysms at this location. With conventional digital subtraction angiography, demonstration of fenestrations of the AcomA is very uncommon. On a dedicated workstation, we systematically reviewed 305 3DRA datasets of the internal carotid artery in 305 patients with aneurysms of the anterior circulation for the presence of fenestrations on the AcomA. In 78 of 305 3DRAs, only the ipsilateral A2 segment was visible; thus, the AcomA could not be evaluated. Of the remaining 227 3DRAs, a fenestration of the AcomA was present in 12 (5.3%; 95% CI 3.0%-9.1%). Of 12 fenestrations of the AcomA,10 (83%) were associated with one or more aneurysms of the AcomA. Of 305 patients, 133 had an aneurysm on the AcomA, and in 127 of these, the AcomA was visible. Of 127 AcomA aneurysms with a visible AcomA, 10 were associated with fenestration, which accounted for an incidence of AcomA fenestrations with AcomA aneurysms of 7.9% (95% CI 4.2%-14.0%). The proportion of fenestrations of the AcomA with aneurysms of the AcomA was 4.4% (10/227), and the proportion of AcomA fenestration with an aneurysm at another location was 0.9% (2/227). This difference was statistically significant (P=.040). Even in retrospect, 11 of 12 fenestrations were not visible on 2D DSA images.

We concluded that in selected patients with aneurysms of the anterior circulation, fenestrations in the AcomA are found with 3DRA in 5.3% of datasets. Most fenestrations were associated with one or more aneurysms of the AcomA.
CHAPTER 8

In Chapter 8, we assessed the procedural time for the coiling of 642 aneurysms and tried to identify predictors of a long procedural time. In particular, we were interested whether implementation of 3DRA in October 2001 had resulted in a decrease of procedural time. The time needed for coiling an intracranial aneurysm is variable and depends on many factors. In this study, the procedural time for coiling was defined as the number of minutes between the first diagnostic angiographic run and the last angiographic run after embolization. The mean procedural time was 57.3 min (median 52 min, range 15-158 min). More than half of the coiling procedures lasted between 30 and 60 min. Multiple logistic regression analysis identified the use of a supportive device (OR 5.4), procedural morbidity (OR 4.5) and large aneurysm size (OR 3.0) as independent predictors of a long procedural time. A poor clinical condition of the patient, the rupture status of the aneurysm, gender, the occurrence of procedural rupture, and aneurysm location were not related to a long procedural time. The mean time for the first 321 coiling procedures was not statistically significantly different from mean time for the last 321 procedures. Implementation of 3DRA had no influence on procedural time.

We concluded that with optimal logistics, coiling of most intracranial aneurysms can be performed in one to two hours, including patient handling before and after the actual coiling procedure.