ORIGINAL ARTICLE

De Novo Aneurysm Formation after Carotid Artery Occlusion for Cerebral Aneurysms

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ABSTRACT

Therapeutic parent artery occlusion has been routinely utilized for management of some intracranial aneurysms. One possible long-term complication of this procedure is "de novo" formation of aneurysms. The purpose of this paper is to estimate the incidence of de novo aneurysm formation, the time period between occlusion and formation, and the most common sites of formation. A PubMed search was performed for all articles between 1970 and 2008 reporting cases of both therapeutic carotid occlusion and de novo cerebral aneurysms. The 20 papers reviewed reported 187 patients having undergone therapeutic carotid occlusion. Of the 163 patients reported in complete-case series, seven developed new aneurysms (4.3%). Thirty-six total new aneurysms were reported, ranging from 1 to 5 per patient. The average time period between occlusion and detection of de novo aneurysm was 9.1 years (range: 2 to 20 years). These aneurysms occurred mostly in the anterior circulation, predominately the anterior communicating artery and posterior communicating artery, and frequently occurred contralateral to the site of occlusion. Therapeutic parent artery occlusion is a likely risk factor for de novo aneurysm formation. Noninvasive follow-up studies should be performed, especially between 2 and 10 years after occlusion.

KEYWORDS: New aneurysms, therapeutic carotid occlusion, risks

Therapeutic carotid artery occlusion is a routinely utilized method of treatment for cerebral aneurysms that are surgically unclippable and unsuitable for endovascular treatment. Formation of de novo aneurysms is a well-recognized late complication, which could lead to late hemorrhage. Hemodynamic changes of the cerebral circulation in the circle of Willis and contralateral carotid artery may predispose patients to the formation of de novo aneurysms at these sites. Risk factors such as gender, age, history of hypertension, cigarette smoking, and multiple aneurysms also may play a role in formation of new aneurysms. The purpose of this literature review is to estimate the incidence of de novo aneurysm formation, the most common sites of formation, and the period of time between vessel occlusion and aneurysm formation.

METHOD

A PubMed search was performed for all articles reporting cases of therapeutic carotid artery occlusion and de novo aneurysm formation between 1970 and 2008.

Exclusion criteria included regrowth of aneurysms at the original site, artery occlusion performed for

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Skull Base 2010;20:405–408.

Received: October 19, 2009. Accepted after revision: March 4, 2010. Published online: April 27, 2010.
DOI: http://dx.doi.org/10.1055/s-0030-1253578.
ISSN 1531-5010.

reasons other than cerebral aneurysms, and omission of the follow-up time period or original site of the aneurysm from the article.

RESULTS

During the time period between 1970 and 2008, 20 papers were identified (Table 1) which reported 187 patients who underwent therapeutic carotid occlusion for cerebral aneurysms and were followed for de novo aneurysm formation. Of these patients, 28 were identified with de novo aneurysms (representing 36 total new aneurysms).

To estimate the incidence of de novo aneurysm formation, only patients reported as part of a case series representing all cases of internal carotid artery (ICA) occlusion at an individual institution over a given period of time were included. Of the four studies reporting such data,[1–4] 163 patients were reported. Seven of these patients developed new aneurysms, representing an incidence of de novo formation of 4.3%.

The initial aneurysm was located in the ICA in 26 of the 28 patients identified (92% of de novo aneurysms). In the remaining two cases, one aneurysm occurred in the middle cerebral artery (MCA) and the other in the anterior communicating artery (ACOM). In only one

Table 1 Cases of Cerebral De Novo Aneurysms Following Carotid Occlusion

Article	Case Type	No. of Patients who Underwent Carotid Occlusion	No. of Patients with De Novo Aneurysms	Sex	Age (yr)	Original Aneurysm	Time Interval (yr)	De Novo Aneurysm(s)
Winn et al (1977)[4]	CS	37	1	M	19	R/PCOM	3.8	L/PCOM
Klemme (1977)[10]	CR	1	1	F	41	R/PCOM	9	L/PCOM
Roski et al (1981)[3]	CS	57	2	M	52	L/ICA	4	R/ICA
				F	31	R/ICA	2	L/ICA & BA
Salar and Mingrino (1981)[11]	CR	2	2	M	44	R/PCOM	9	L/PCOM
				M	35	R/PCOM	15	L/PCOM
Clark and Ray (1982)[12]	CR	3	3	M	51	R/MCA	16	L/Oph + L/ ACOM + 2MCA + R/Perical
				F	53	L/ICA	14	R/Ophthalmic
				M	47	R/ACOM	18	Jun of L/ACA & L/ MCA + L/ICA
Ostergaard (1985)[13]	CR	1	1	F	18	L/ICA bifurcation	3	R/PCOM
Dyste and Beck (1989)[14]	CR	1	1	F	49	R/Ophthalmic	6	ACOM
Heros (1989)[15]	CR	1	1	N/a	N/a	R/Ophthalmic	4	ACOM
Drapkin and Rose (1992)[16]	CR	1	1	M	56	R/ICA bifurcation	20	L/PCOM + L/ Ophthalmic
Fujiwara et al (1993)[17]	CR	4	1	F	58	R/Ophthalmic	8	ACOM
Yoshimura et al (1994)[18]	CR	1	1	M	35	L/ICA & L/MCA	5	L/CAROTID bifurcation
Ladziński et al (1994)[19]	CR	1	1	F	55	R/ICA	8	ACOM
Timperman et al (1995)[2]	CS	58	2	F	58	L/Cavernous ICA	2	ACOM
				F	49	R/Cavernous ICA	4	ACOM + ACA
Parekh et al (1995)[20]	CR	1	1	M	37	L/ ICA	18	Origin of R/PCOM
Mauri et al (1995)[21]	CR	1	1	M	30	R/Cavernous ICA	9	ACOM
Ogasawara et al (1995)[22]	CR	1	1	F	58	R/ICA	13	R/PCOM
Johnston et al (1998)[23]	CR	3	3	M	10	L/ICA	2	Vertebrobasilar
				M	6	L/ICA	5	Vertebrobasilar
				F	10	L/ICA	11	Vertebrobasilar
Niiro et al (2000)[1]	CS	11	2	M	52	L/Cavernous ICA	17	R/Cavernous ICA
				F	61	L/Cavernous ICA	13	ACOM
Briganti et al (2002)[24]	CR	1	1	M	30	R/Cavernous ICA	9	ACOM
Wolf et al (2002)[25]	CR	1	1	F	58	R/Cavernous ICA	5	L/PCA
Total		187	28					36

patient did multiple aneurysms form, one in the MCA and the other in the ICA.

The location of de novo aneurysms showed a more variable distribution: 10 ACOM (27% of de novo aneurysms), eight posterior communicating artery (PCOM) (22%), four ICA (11%), four basilar artery (11%), three ophthalmic artery (8%), three vertebrobasilar artery (8%), two MCA (5%), one posterior cerebral artery (3%), and one pericallosal artery (3%). Fourteen patients formed new aneurysms on the contralateral side (50% of patients with de novo formation). Five patients formed multiple de novo aneurysms (17%).

The average interval between ICA occlusion and formation of de novo aneurysm was 9.1 years (range: 2 to 20 years). In 18 cases (64%), de novo aneurysm formation was detected between 2 and 10 years following occlusion. In 10 cases (36%), formation was detected between 11 and 20 years.

DISCUSSION

The indications for carotid occlusion include large symptomatic intracranial aneurysms, giant carotid cavernous aneurysms, and traumatic carotid aneurysms or fistula that cannot otherwise be treated with microsurgery or endovascular treatment. Long-term complications of therapeutic carotid occlusion include de novo aneurysm formation, aneurysm regrowth at the site of an improperly secured aneurysm, delayed hemorrhage from a de novo or regrowth aneurysm, and multiple aneurysm formation. Additionally, cerebral ischemia represents both a short-term and a long-term potential complication.[1]

Formation of new aneurysms is a potential complication in the management of patients following therapeutic ICA occlusion. The data reviewed here suggest that de novo aneurysm formation occurs in 4.3% of patients undergoing vessel occlusion at an average of 9 years following the occlusion.

Although the pathogenesis is not clear, one hypothesis relates to hemodynamic stress. Following ICA occlusion, vessels are subject to increased blood flow to supply the contralateral circulation. This may give rise to degeneration of the endothelial basement membranes and subendothelial connective tissue of the arterial wall. Additionally, previous literature suggests that the risk factors predictive of initial aneurysm formation likely correlate with de novo formation. These include history of arterial hypertension, cigarette smoking, female gender, age, history of multiple and familial forms of intracranial aneurysms, and hereditary connective tissue diseases.[5–7] Thus, the increased variability in location of de novo aneurysms observed is likely multifactorial due to an underlying diathesis of the vessel to aneurysm formation as well as changes in flow.

Therapeutic artery occlusion remains an important option in treating aneurysms that are otherwise not amenable to surgical or endovascular treatment. Newer treatment options by flow diversion devices such as the Silk and Pipeline stents can treat giant and fusiform aneurysms by arterial reconstruction rather than arterial occlusion, and may soon become FDA approved.[8,9] The Pipeline stent is undergoing clinical trials in the United States. The first Silk device was placed in the United States by the senior author on July 16, 2009 under humanitarian device exemption. ICA occlusion may be a definitive treatment for a particular aneurysm, but it does not mean that a de novo aneurysm will not appear at some time in the future. Thus, patients undergoing therapeutic ICA occlusion should receive continued radiographic follow-up imaging, such as CTA and MRA. This follow-up is especially important in the first decade following the procedure.

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