REVIEW ARTICLE

Richard P. Cambria, MD, Section Editor

Risk of intracerebral aneurysm rupture during carotid revascularization

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Objective: **Robust guidelines exist for the treatment of carotid stenosis and intracranial aneurysms independently, however, the management of tandem carotid stenosis and intracranial aneurysms remains uncertain. Although the prevalence of tandem pathologies is small (1.9%-3.2%), treating carotid stenosis can alter intracranial hemodynamics potentially predisposing to aneurysm rupture. In this review, our aim was to assess the safety of intervention in this cohort, by analyzing outcomes from the published literature.**

Methods: **The preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines were used to conduct the review. Articles from 1947 to 2012 were searched using EMBASE Classic and EMBASE (November, 1947 -March, 2012) and Ovid MEDLINE(R) In-Process and other NonIndexed Citations and Ovid MEDLINE(R) on Ovid SP, http://ClinicalTrials.gov, http://controlled-trials.com and the Cochrane review database using a predefined search strategy.**

Results: **One hundred forty-one patients from 27 articles were included. Interventions ranged from single (n = 104, 74%), staged (n = 26, 18%) to simultaneous procedures (n = 11, 8%). The largest cohort of patients was treated by carotid endarterectomy alone (n = 92, 66%). The majority of patients presented with a symptomatic carotid stenosis and an asymptomatic ipsilateral intracranial aneurysm (n = 70, 50%). Five subarachnoid hemorrhages occurred (4% [5/140], three within 30 days of the procedure and two thereafter) of which two were fatal. All five occurred in patients who underwent carotid endarterectomy as a single procedure (5%). Two of the five patients presented with ruptured posterior communicating artery aneurysms.**

Conclusions: **Published reports of perioperative aneurysm rupture are rare in individuals with tandem carotid stenosis and intracranial aneurysms. This is the first analysis of all published cases. However, it is limited by the small number of studies and the possible underreporting due to publication bias and underdiagnosis where angiography was not performed. Although we report a low incidence of subarachnoid hemorrhage, analysis of registry data with a larger cohort is warranted to confirm these findings. (J Vasc Surg 2012;56:1739-47.)**

Approximately 5% of patients who undergo angiography for symptomatic extracranial carotid stenosis are found to have a coincidental intracranial aneurysm.[1-3] More specifically, the incidence of carotid stenosis with an ipsilateral intracranial aneurysm (referred to hereafter as *tandem pathologies*) is 1.9% to 3.2%.[4,5]

At present, level 1 evidence exists for carotid endarterectomy (CEA) in symptomatic and in younger asymptomatic patients with significant carotid stenosis.[6,7] However, CEA, in the presence of an intracranial aneurysm, requires careful consideration because of the possibility of subsequent aneurysm rupture and subarachnoid hemorrhage (SAH). Whether any deviation from standard practice[8] is required when treating tandem pathologies has yet to be determined from the current available evidence.

Intervention options for carotid stenosis include medical therapy alone or in combination with CEA or carotid artery stenting (CAS). Revascularization may lead to a periprocedural increase in cerebral perfusion pressure in the context of impaired cerebral autoregulation[9-12] causing an increase in aneurysm wall shear stress.[9] In the presence of high-risk features, including bleb formation,[13] size and location,[14] this scenario may increase the risk of aneurysm rupture, particularly in the arteries of the carotid territory, namely the ophthalmic, anterior choroidal, anterior (ACA) and middle cerebral (MCA), and posterior communicating (PCOMM) arteries.[9,15] Indeed, common carotid ligation was historically practiced to reduce the size of these aneurysms.[16,17]

Tandem pathology may also be problematic in the endovascular treatment of intracranial aneurysms. Endovascular coiling requires access via the internal carotid

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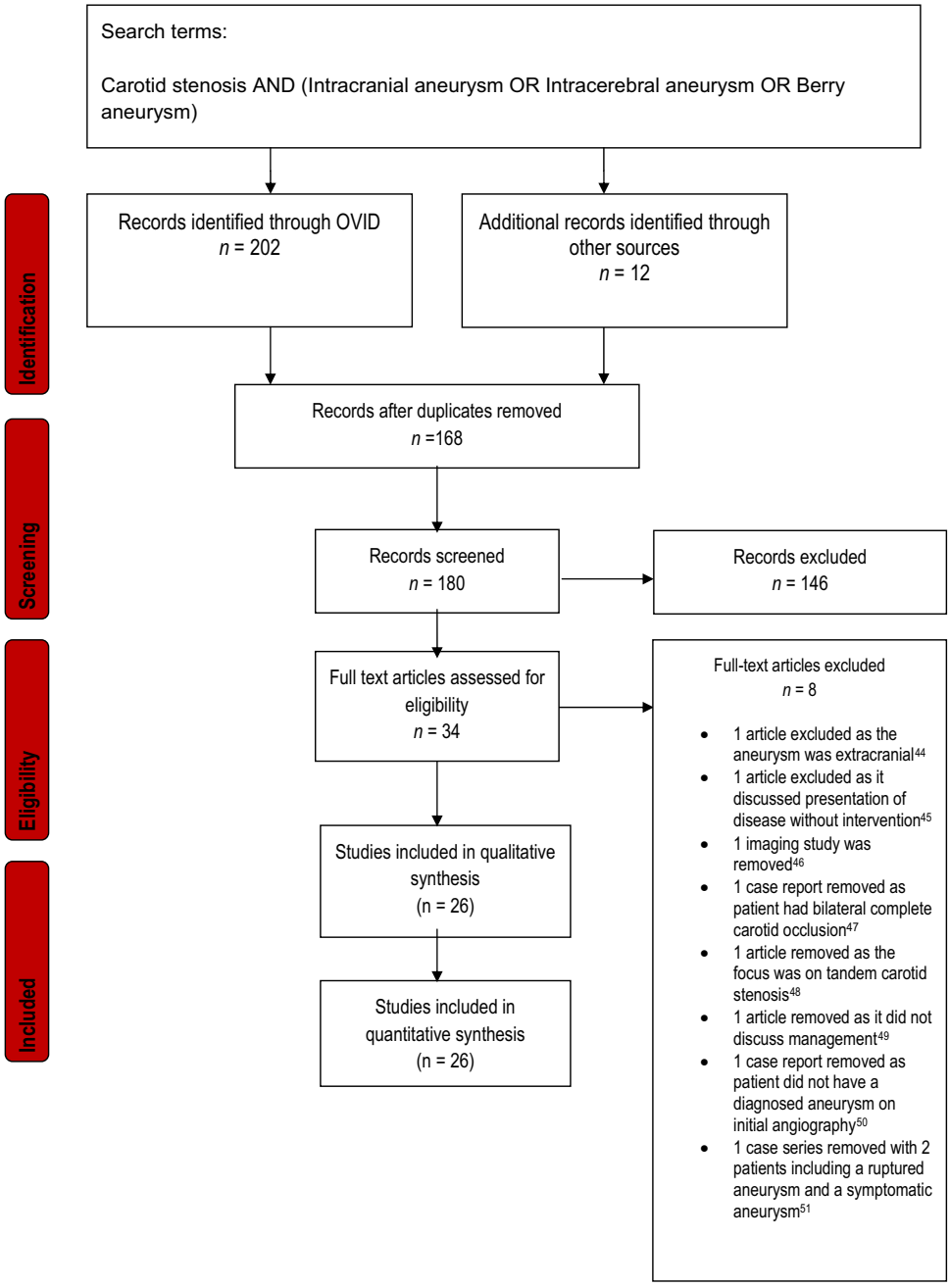

Fig. Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow chart.[44-51]

artery (ICA), which may be difficult if significant stenosis is present.[18,19]

The aim of this systematic review was to analyze the stroke or death rates in interventions for extracranial ICA stenosis with a tandem intracranial aneurysm.

METHODS

A systematic review was undertaken adhering to PRISMA guidelines in March 2012.[20] Databases searched included EMBASE Classic and EMBASE (1947-2012 and Ovid MEDLINE(R) In-Process and Other NonIndexed Citations and Ovid MEDLINE(R) on Ovid SP (1948 to date), http://ClinicalTrials.gov, http://controlled-trials.com and the Cochrane review database (Fig). The search string "carotid stenosis" and ("intracranial aneurysm" or "intracerebral aneurysm" or "berry aneurysm") was used.

No limits, filters, or restrictions were applied. Reference lists within articles were hand searched to include any additional relevant articles. Title and abstract screening was performed independently by two authors (U.K., A.T.), and

further evaluation was undertaken of full-text publications employing a priori eligibility criteria. Authors were contacted in several cases for further information.

Study eligibility criteria. Articles reporting the management of patients with tandem carotid stenosis and intracranial aneurysms were included. Case reports and case series were included because of the rarity of this condition. Exclusion criteria included studies, which solely assessed imaging techniques and did not comment on management, patients presenting with ruptured or symptomatic intracranial aneurysms, review articles, editorials, letters, and reports of patients with extracranial ICA aneurysms.

Data extraction. Raw data were extracted and tabulated; individual patient data were used where available (Table I). Safety end points were 30-day any territory stroke, death, and a composite of stroke or death. Death as a stand alone end point was chosen, as it was the most robust end point across studies without an independent neurologist. The composite end point stroke or death accounted for the competing risks of stroke and death.

RESULTS

A total of 202 studies were identified in the initial search, with 168 after deduplication. Following title and abstract screening, 34 articles remained. Seven articles were excluded (Fig).

Individual cases as follows were excluded if they did not meet the inclusion criteria (ie, were not relevant to safety outcomes for carotid revascularization with a tandem intracranial aneurysm). Two patients were removed from Ballotta et al[5] as they were unfit for any intervention. One patient from Leon et al[19] was removed, as there was no intracranial lesion on initial angiography. Two patients were removed from Kann et al[21]; one refused treatment and one died of a myocardial infarction before intervention. One patient was removed from Iwata et al,[22] as they presented with both a carotid aneurysm and intracranial aneurysm. Four patients were removed from Takolander et al,[23] as they had an intracerebral hemorrhage following intervention, with no proven aneurysm. Two patients from Yeung et al,[24] were removed, as both had no carotid intervention. Forty patients were removed from Kapelle et al[4] and three from Kann et al,[21] as they were treated with medical therapy alone. One patient with subclavian stenosis and no carotid stenosis was removed from Yeung et al.[24] A patient with total occlusion of the left subclavian artery and 10% bilateral internal carotid artery stenosis was removed from Yeung et al.[24] Six patients were excluded from Badruddin et al,[18] as they presented with symptomatic aneurysms, which required carotid stenting for endovascular treatment. Two more patients were removed from Badruddin et al, as they presented with an SAH. One patient from Leon et al[19] was removed, as the patient presented with persistent headaches attributed to an aneurysm with an asymptomatic carotid stenosis requiring stenting for endovascular coiling.

Demographic data. Twenty-six studies were included in the qualitative synthesis, of these 11 were case reports, 13 were case series, one a subgroup analysis from the North American Symptomatic Carotid Endarterectomy Trial (NASCET),[4] and one a book chapter[25] (Table I). A total of 141 patients were included in the analysis between the years of 1965 and 2011. Demographic data from patients in Kapelle et al[4] could not be extracted, as this was pooled with patients who received medical therapy alone.

The mean patient age was approximately 62 years. The sex of 81 patients was reported: 36 (44%) male and 45 (56%) female. The degree of stenosis was reported in 72 patients; 67 (93%) had stenoses of ≥75% and five (7%) had stenoses <75% (range, 33%-70%). The conversion from European Carotid Surgery Trial (ECST) to NASCET measurements was performed using the following formula: ECST % stenosis = 0.6 (NASCET % stenosis) + 40.[26]

The location of 123 aneurysms was reported as follows: 38 (31%) were located in the MCA, 38 (31%) PCOMM, 13 (11%) anterior communicating artery (ACOMM), 14 (11%) cavernous ICA, 11 (9%) distal intracranial ICA, three (2%) ophthalmic artery, two (2%) supraclinoid ICA, two in the anterior choroidal artery (2%), and one basilar artery aneurysm (1%).

The size of 61 aneurysms was reported. Twenty-five (41%) were ≤5 mm, 19 (31%) were 6 to 9 mm, and 17 (28%) were ≥10 mm. Presenting symptoms were stated in 99 patients with 91 (92%) presenting with a symptomatic carotid lesion, and eight (8%) patients were asymptomatic. The most frequently reported scenario was a symptomatic carotid lesion with an asymptomatic ipsilateral intracranial aneurysm (n = 70, 50%; Table II).

Mode of intervention. Thirty-seven (26.2%) patients underwent staged or simultaneous procedures for both carotid stenosis and intracranial aneurysms, and 104 (73.7%) patients only had carotid intervention. Ninety-three (65.9%) patients underwent CEA alone, nine (6.3%) had CAS and simultaneous aneurysm coiling, 13 (9.2%) had CEA followed by aneurysm clipping as a second procedure, eight (5.6%) had aneurysm clipping alone, seven (4.9%) had clipping followed by CEA as a second procedure, two (1.4%) had CAS and then coiling as a second procedure, two had CAS only (1.4%), one (0.7%) had clipping followed by CEA as a second procedure, one (0.7%) had CEA and simultaneous aneurysm clipping, one (0.7%) had CEA followed by plastic encasement of aneurysm, one (0.7%) had carotid angioplasty and simultaneous aneurysm clipping, one (0.7%) had wrapping of aneurysm with fascia, one (0.7%) was treated by wrapping of aneurysm in fascia followed by CEA as a second procedure, and one (0.7%) patient had percutaneous transcarotid angioplasty followed by clipping of one aneurysm and coiling of another.

Symptomatic carotid stenosis and ipsilateral anterior circulation aneurysm. Within the largest and most clinically relevant subgroup of 70 patients, sex was reported in 53 patients (22 [42%] male, 31 [58%] female). The mean age was 62 years (SD ±9). Degree of stenosis was measured by varying methods and reported in 49 patients where 47 (96%) had a >70% stenosis and two (4%) had <70%. Aneurysm

Table I. Studies included for the management for unruptured intracranial aneurysms with concomitant carotid stenosis

First author	*Publication year*	*Number of cases included*	*Type of study*	*Prospective/ retrospective*	*Symptomatic lesion*
Pool et al[25]	1965	1	Book chapter	P	Carotid
Denton et al[52]	1973	1	Case report	R	Carotid
Shoumaker et al[53]	1974	1	Case report	R	Carotid
Adams et al[15]	1977	1	Case report	R	Carotid
Stern et al[32]	1979	15	Case series	R	Carotid
Ladowski et al[27]	1983	16	Case series	R	Carotid
		2			Carotid
Takolander et al[23]	1983	1	Case series	R	Carotid
Kajiwara et al[54]	1984	1	Case report	R	Carotid
Orecchia et al[55]	1985	6	Case series	P	Carotid
		2			Carotid
Pappada et al[56]	1996	7	Case series	R	Carotid
		2			
Detry et al[57]	1997	2	Case series	P	Carotid
Kann et al[21]	1997	4	Case series	R	Carotid
		1			Carotid
Temiz et al[58]	1999	1	Case series	R	Carotid
		1			Carotid
Kappelle et al[4]	2000	32	RCT subgroup analysis	R	Carotid
		7			Carotid
		1			Carotid
Yeung et al[24]	2000	1	Case series	P	Carotid
		1			Carotid
		1			Carotid
McConkey et al[59]	2002	1	Case report	R	Carotid
Nievas et al[60]	2003	2	Case series	P	Carotid
		3			
		1			
Ballotta et al[5]	2005	6	Case series	P	Carotid
		2			Asymptomatic
Navaneethan et al[61]	2006	1	Case report	R	Carotid
Thyrion et al[62]	2007	1	Case report	R	Asymptomatic
Iwata et al[22]	2008	1	Case report	R	Carotid
Espinosa et al[63]	2009	1	Case report	R	Carotid
Gallego Leon et al[19]	2009	5	Case series	R	Carotid
Badruddin et al[18]	2010	2	Case series	R	Carotid
Siddiqui et al[28]	2011	1	Case report	R	Carotid
Suh et al[64]	2011	4	Case series	R	[a]
		2			
Total		141			

CAS, Carotid artery stenting; *CEA*, carotid endarterectomy; *DSA*, digital subtraction angiography; *CTA*, computed tomography angiogram; *MRA*, magnetic resonance angiogram.
The study designs, number of patients, symptomatic lesions, outcome and management are shown.
[a]Not stated.

location was reported in all patients and included 28 (40%) in the MCA, 15 (21.4%) PCOMM, eight (11.4%) ACOMM, eight (11.4%) cavernous ICA, four (5.7%) supraclinoid ICA, three (4.3%) ophthalmic ICA, three (4.3%) in the distal intracranial ICA, and one in the anterior choroidal artery (1.4%).

Forty (58.8%) patients presented with a transient ischemic attack (TIA), symptomatic carotid disease was not further specified in 17 (25.1%), ischemic stroke in eight (11.8%), and amaurosis fugax in three (4.4%).

Aneurysm size was reported in 40 patients and included 12 (30%) ≤5 mm, 19 (48%) 6 to 9 mm, and nine (22%) ≥10 mm. Two (2.9%) nonfatal strokes occurred within 30 days of procedure in this subgroup of patients; one (1.4%) ischemic and one (1.4%) SAH.

Forty-six patients underwent CEA alone (65.7%), nine had aneurysm clipping followed by CEA as a staged procedure (12.9%), seven had CAS followed by aneurysm coiling simultaneously (10%), two had CEA followed by aneurysm clipping as a staged procedure (2.9%), two had CAS only (2.9%), one had CAS followed by aneurysm coiling as a staged procedure (1.4%), one had CEA followed by aneurysm wrapping in fascia as a simultaneous procedure (1.4%), one had percutaneous angioplasty followed by aneurysm clipping as a staged procedure (1.4%), and one had CEA followed by a plastic encasement of the aneurysm as a staged procedure (1.4%).

Safety end points. Three strokes occurred within 30 days of intervention (2%), two deaths occurred within 30 days of intervention (1.45%), and two fatal strokes occurred beyond 30 days from intervention (1.45%). There were no reports of SAH in patients who underwent intervention for both pathologies. However, two pa-

Table I. Continued.

Intervention(s)	*30-day stroke*	*30-day mortality*	*Post-30-day stroke*	*Post-30-day mortality*	*Imaging modality*
CEA	1	1	0	0	Angiography
CEA and subsequent plastic encasement of aneurysm	0	0	0	0	Angiography
CEA	0	0	0	0	Angiography
CEA	0	0	1	1	Angiography
CEA	0	0	0	0	Angiography
CEA	0	0	0	0	Angiography
CEA with subsequent clipping	0	0	1	0	Angiography
CEA	0	0	1	1	Angiography
Clipping followed by subsequent CEA	0	0	0	0	
CEA	0	0	0	1	Angiography
CEA with subsequent clipping	1	0	0	0	Angiography
Clipping followed by subsequent CEA	0	0	0	0	Angiography
CEA	0	0	0	0	Angiography
Clipping followed by subsequent CEA	0	0	0	0	Angiography
CEA	0	0	0	0	Angiography
Clipping followed by subsequent CEA	0	0	0	0	Angiography
Simultaneous clipping and CEA	0	0	0	0	DSA
Aneurysm wrapped with simultaneous CEA	0	0	0	0	DSA
CEA	1	1	1	1	Angiography
Clipping of aneurysm	2	0	0	0	Angiography
Clipping with subsequent or simultaneous CEA	0	0	0	0	Angiography
Wrapping of aneurysm only	0	0	0	0	Angiography
CEA	0	0	0	0	Angiography
CEA with subsequent clipping	0	0	0	0	Angiography
Aneurysm clipping and simultaneous CEA	0	0	0	0	MRA
Clipping	0	0	0	0	DSA
Clipping followed by subsequent CEA	0	0	0	0	DSA
CEA followed by subsequent CEA	0	0	0	0	DSA
CEA	0	0	0	0	DSA, MRA and CTA
CEA	0	0	0	0	DSA, MRA and CTA
Simultaneous CAS and coiling	0	0	0	0	Angiography
CAS plus subsequent aneurysm	0	0	0	0	DSA
Aneurysm coiling and subsequent CAS	0	0	0	0	MRA
CAS followed by subsequent coiling	0	0	0	0	Angiography
Simultaneous CAS and coiling	0	0	0	0	Angiography
Simultaneous CAS and coiling	0	0	0	0	DSA
CEA	1	0	0	0	Angiography
CEA	0	0	0	0	Angiography
CAS	0	0	0	0	Angiography
	6	2	4	4	

tients did have an intraoperative hemorrhage during aneurysm clipping (not from the aneurysm itself) with a good functional recovery.[27]

Hemorrhagic strokes following carotid intervention. In total, there were five reported aneurysm-related SAHs. Three SAHs occurred within 30 days of the procedure, resulting in two deaths. Two further fatal SAHs occurred after 30 days (Table III).

Subarachnoid hemorrhage within 30 days. The first death occurred 3 days following CEA due to rupture of a contralateral PCOMM aneurysm.[25] No further details were available. The second death occurred on postoperative day 10 in a 77-year-old female who underwent CEA for recurrent TIAs, with a hemodynamically insignificant 33% stenosis.[4] At postmortem, hemorrhage was present in the basal cisterns and two 4-mm diameter aneurysms were identified at the origin of the PCOMM and the MCA; however, the source of hemorrhage was not confirmed.

A nonfatal SAH within 30 days of procedure occurred in a 75-year-old male who was admitted with a TIA.[28] Cerebral angiography demonstrated a 60% carotid stenosis in the proximal left ICA with an ulcerated plaque and a 4-mm ACOMM aneurysm. The patient underwent CEA. Blood pressure in the perioperative period was controlled with a systolic range of 115 to 132 mm Hg. Two days later, the patient was readmitted with weakness in the right leg. Blood pressure was 119/60 mm Hg and a computed tomography (CT) and magnetic resonance imaging (MRI) demonstrated an SAH in the distribution of the left ACA consistent with a ruptured ACOMM

Table II. Laterality of carotid stenosis and anterior circulation aneurysm

Location of aneurysm	*Carotid symptomatic*	*Aneurysm symptomatic*	*n*
Ipsilateral	Yes	No	68
	No	No	7
	No	Yes	4
Contralateral	Yes	No	16
Anterior communicating artery	Yes	No	3
	No	Yes	2
Basilar artery	Yes	No	1

The largest subgroup presented with symptomatic carotid stenosis and asymptomatic ipsilateral anterior circulatory intracranial aneurysm.

aneurysm. Coil embolization was undertaken and the patient made a good functional recovery.

Subarachnoid hemorrhage after 30 days. The third death occurred 7 months following CEA for a symptomatic carotid stenosis in an 84-year-old female.[15] The patient had a 5- × 10-mm left ICA–PCOMM junction aneurysm. Post-mortem demonstrated a ruptured, enlarged, 14- × 10-mm aneurysm at this site.

The fourth death was in a 67-year-old female who was admitted with crescendo TIAs.[23] Angiography demonstrated an ulcerated plaque in the right ICA and a 6-mm-diameter right MCA aneurysm. The patient underwent CEA and 9 months postprocedure developed coma. A large hemorrhage in the right cerebral hemisphere in the area surrounding the aneurysm was found on computed tomography. The patient underwent an emergency craniotomy and died on the second postoperative day.

DISCUSSION

This analysis found only three published reports of ruptured intracranial aneurysms within 30 days of carotid revascularization. Deaths occurred in two out of three patients with early SAH, and thus, it is important to understand why these patients had adverse outcomes. All fatalities occurred following CEA; however, as this was the most frequent intervention, it may simply reflect the number of procedures performed.

Both aneurysm- and patient-specific risk factors have been proposed for aneurysm rupture. Patient factors include female sex (relative risk [RR], 1.6; 95% CI, 1.1-2.4), age >60 years (RR, 2.0; 95% CI, 1.1-3.7) and Japanese or Finnish nationality (RR, 3.4; 95% CI, 2.6-4.4).[29] In the native setting, risk factors for aneurysm rupture include size, location, and architecture. Aneurysms of <10-mm diameter have a 0.05% per year chance of rupture compared with <1% per year in those >10 mm.[14] Four out of five patients who experienced an SAH had an aneurysm of <10 mm in diameter and therefore the rupture risk of smaller aneurysms is not clinically insignificant. This finding may be because they are more common, or due to other unknown risk factors that cannot be elucidated from retrospective data. Two out of five ruptured aneurysms were PCOMM aneurysms. This is similar to the native setting, where PCOMM aneurysms are a significant predictor of rupture (eight times relative risk of rupture; $P = .02$[30]).

The effects of carotid intervention on aneurysm growth are not well understood. Of particular relevance is a rupture that occurred 7 months following CEA. The aneurysm was originally 5 × 10 mm and enlarged to 14 × 10 mm and then ruptured. Treatment of carotid stenosis leads to a sudden increase in cerebral blood flow, which has been shown to persist for at least 1 month following intervention.[31] However, available reports with varied follow-up suggest no enlargement of aneurysms following intervention.[18,32] It may be that certain patients with specific aneurysm growth risk factors are more at risk of aneurysm rupture following revascularization.

Communication between vascular and neurovascular specialists in the form of multidisciplinary case discussions may benefit patients with tandem pathologies, particularly those with PCOMM aneurysms. Patients undergoing revascularization for carotid stenosis and a tandem intracranial aneurysm may benefit from periodic follow-up imaging where an enlarging aneurysm should expedite intervention.[29]

Aneurysm architecture, including bleb (diverticulum) formation can also influence risk of rupture. Blebs form at areas of high shear stress, ultimately reducing force on the arterial wall due to a counter-current vortex mechanism.[13] This temporary benefit is lost as a result of wall weakness developed by blebs that are at risk of rupture. An increase in wall shear stress following CEA[33] may predispose to aneurysm rupture in those with this specific architectural fea-

Table III. Subarachnoid hemorrhage postcarotid endarterectomy

Authors	*Intervention*	*Stenosis*	*Aneurysm size (mm)*	*Aneurysm location*	*Laterality of stenosis*	*Time of subarachnoid hemorrhage*	*Outcome*
Siddiqui et al[28]	CEA	60%	4	Anterior communicating	Ipsilateral	Day 2	Survived
Pool and Potts[25]	CEA	[a]	[a]	Posterior communicating	Contralateral	Day 3	Death
Kapelle et al[4]	CEA	33%	4	Posterior communicating and middle cerebral artery	[a]	Day 10	Death
Adams et al[15]	CEA	>70%	5 × 10	Posterior communicating	Ipsilateral	7 months	Death
Takolander et al[23]	CEA	[a]	6	Middle cerebral	Ipsilateral	9 months	Death

[a]Data not available.

ture. An architectural analysis of the aneurysm itself prior to intervention to the carotid can be conducted using three-dimensional rotational angiography[13] or by three-dimensional CT and/or MR angiography, which is less invasive and associated with fewer complications.[34]

A final consideration is whether other intracranial hemorrhages postcarotid intervention are a consequence of undiagnosed ruptured intracranial aneurysms. The reported risk of intracranial hemorrhage following CEA ranges between 0.2% and 0.8%; however, SAH is documented in only 0.05% of cases.[35,36] Reports also demonstrate that SAH can occur independently following CEA where no aneurysm is identified.[37,38] It is, therefore, unlikely that ruptured aneurysms are the main cause of cerebral hyperperfusion hemorrhage. However, if SAH is encountered, CT angiography to look for a ruptured aneurysm is suggested.

This article represents the first synthesis of the published data in this area and highlights the need for further large-volume registry data. It has several limitations because of the rarity of tandem pathologies and a possible reluctance to report complications. A significant proportion of the literature is comprised of retrospective case series and case reports and, therefore, is at risk of negative publication bias.[39] Moreover, U.S. practice guidelines recommend duplex ultrasonography as a first-line investigation for carotid artery disease.[8] This may add to the underreporting of tandem pathologies, however, most of these patients have already had detailed cerebral imaging, as they are symptomatic. Therefore, there is no strong rationale for altering these recommendations. Finally, outside of the symptomatic carotid stenosis and ipsilateral asymptomatic aneurysm group, numbers were too small to draw any conclusions.

In summary, these results suggest that rupture of an intracranial aneurysm postcarotid intervention is rare and has only been reported in five cases, of which, three were within 30 days of endarterectomy. This low rate of SAH is similar to the NASCET trial where one patient out of 90 with tandem lesions experienced an ambiguous SAH following CEA. Therefore, at present, there is no strong evidence that CEA significantly increases the risk of aneurysmal rupture, given the prevalence of tandem pathologies and the large number of CEAs performed each year. For surgeons confronted with a recently symptomatic carotid stenosis and an incidental intracranial aneurysm, we recommend both the patient and a neurosurgeon are informed; however, this finding should not delay intervention. Further relevant data would be gained by incorporating the finding of an incidental intracranial aneurysm into UK and U.S. registries. Ongoing trials of carotid stenting such as ACT-1, SPACE-2, and ACST-2, which incorporate catheter angiography will provide further data on the effect of carotid stenting on the rupture risk of intracranial aneurysms.[40-43]

AUTHOR CONTRIBUTIONS

Conception and design: AD, UK, AT, JS
Analysis and interpretation: UK, AT
Data collection: UK
Writing the article: UK, AT
Critical revision of the article: AD, AT, UK, JS
Final approval of the article: AD
Statistical analysis: UK
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Overall responsibility: AD

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