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Research Article

Carotid Endarterectomy in Patients with Cerebral Aneurysms

Piffaretti G[1]*, Tarallo A[1], Franchin M[1], Bacuzzi A[2], Rivolta N[1], Ferrario M[1], Castelli P[1] and Tozzi M[1]

[1]Department of Surgery and Morphological Sciences, University of Insubria School of Medicine, Circolo University Hospital, Varese, Italy

[2]Anesthesia and Palliative Care, Circolo University Hospital, Varese, Italy

***Corresponding author:** Gabriele Piffaretti, Vascular Surgery, Department of Surgery and Morphological Sciences, Circolo University Teaching Hospital, University of Insubria School of Medicine, Varese, Italy

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Abstract

Objectives: To present the results of carotid endarterectomy (CEA) in patients with intracranial aneurysm (IA), and evaluate the risk of postoperative hemorrhage due to IA rupture.

Materials and Methods: Between January 2008 and December 2015, all patients treated with primary single sided CEA for extracranial asymptomatic severe carotid artery stenosis or symptomatic lesions were identified. Preoperatively, all patients underwent computed tomography angiography (CT-A). Immediate neurologic assessment of the patient was accomplished upon completion of the intervention. In these patients, CT-A was performed at the 1 year follow-up to assess the characteristics of the IA.

Results: Out of 526 patients who underwent CEA during the study period, 13 (2.2%) were identified to have an extracranial carotid artery stenosis with an IA. There were 7 (54%) males; mean age was 68 ± 9 years (range, 52-85; IQR, 61-73). Urgent CEA was performed in 4 (31%) cases. Postoperative cerebrovascular complication rate was 15% (n = 2): non disabling minor stroke (n = 1), and intracranial hemorrhage (n = 1). Rupture of the IA never occurred. In-hospital mortality rate was 8% (n = 1, the abovementioned hemorrhage). All but 12 (92%) patients were discharge alive and independent. At 1 year follow-up, no patient with tandem lesion died: all of them underwent CT-A control, and IA rupture was never detected.

Conclusions: Authors experience is consistent with the most recent Literature analysis which suggests that CEA does not significantly increases the risk of IA rupture.

Keywords: Carotid Endarterectomy; Cerebral Aneurysms

Introduction

Regardless of the different vascular segments involved, the management of tandem lesions of the supra-aortic vessels requires careful consideration because of the potential mutual cerebrovascular complications. One of the most frequent combination of tandem lesions is represented by the presence of an extracranial carotid artery obstructive disease and an intracranial aneurysm (IA). Up to date, different management strategies have been advocated but optimal management strategy and operative repair is actually controversial [1]. The aim of this paper is to present Authors' experience of carotid endarterectomy (CEA) in presence of an IA, and evaluate the risk of intracranial hemorrhage due to IA rupture.

Materials and Methods

Study population

All patients treated with primary, single sided CEA for extracranial asymptomatic severe carotid artery stenosis or symptomatic lesions were identified over a 7-year period, starting from January 2008; the end of study for the final analysis was December 31st, 2015. It is a single center, observational descriptive study; retrospective analysis of the anonymized data did not require approval of the Institutional Review Board. Indication for intervention followed the indication of the updated guidelines for the management of extracranial carotid disease of the Society for Vascular Surgery (SVS) [2]. Preoperatively, all patients underwent computed tomography angiography (CT-A), and were seen by a stroke neurologist and a neurosurgeon, to confirm and grading the eventual presenting neurological symptoms, and assess the operative risk of the IA. The Willis Circle (WC) morphology and IA assessment for each individual patient was assessed by neuroradiologists specialized in vascular radiology (> 10 years of experience).

Operative detail

General or loco-regional anesthesia was performed, in consultation with the patient, accordingly to the discretion of a multidisciplinary team including a vascular surgeon, an anesthesiologist and a stroke neurologist [3]. All patients were heparinized with 50IU/kg. Intraoperative cerebral monitoring was performed using transcranial Doppler (Multi-Dop® T digital – Compumedics Germany GmbH; Singen – Germany). If cross-clamp intolerance (CCI), shunt (Pruitt-Inahara® – Le Maitre Vascular; Burlington – MA; USA) was inserted through the same arteriotomy used for the CEA. Carotid endarterectomy was accomplished using primary suture: carotid patching (Fluoropassiv® – Vascutek Terumo; Inchinnan – Scotland, UK) was used selectively [3]. Immediate neurologic assessment of the patient was accomplished upon completion of the intervention; soon after surgery, the patient remained in the post-anesthesia care

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Table 1: Demographics, comorbidities and risk factors of the cohort.

	N
Demographics	
M:F	7:6
Age, *(mean ± SD)*	68 ± 9 (IQR, 61-73)
Comorbidities, (%)	
Hypertension	10 (77)
Dyslipidemia	9 (69)
IHD	5 (38)
Diabetes	4 (31)
COPD	2 (15)
Risk factors, (%)	
Stenosis, *(± SD)*	78 ± 11 (IQR, 70-90)
Controlateral occlusion, *(%)*	0 (0)
WC anomalies, *(%)*	1 (8)
PreCVA, *(%)*	7 (54)
TIA	4 (31)
stroke	3 (23)

N: Number; SD: Standard Deviation; IQR: Interquartile Range; IHD: Ischemic Heart Disease; COPD: Chronic Obstructive Pulmonary Disease; WC: Willis Circle; PreCVA: Previous Cerebrovascular Accidents; TIA: Transient Ischemic Attack.

unit for continuous invasive and neurologic monitoring. Any suspect neurologic deficits were promptly evaluated with CT-A to help determine the etiologic mechanism and guide further therapy.

Follow-up

Generally, postoperative medical treatment consisted of 100mg-acetylsalicylic acid (Cardioaspirin® – Bayer; Rome – IT) ad infinitum. Follow-up protocol consisted of an interdisciplinary clinical visit (vascular surgeon plus stroke neurologist), echo-color-Doppler performed at 1 and 12 months after intervention, and on an annually basis thereafter. In these patients, CT-A was performed at the 1 year follow-up to assess the characteristics of the IA.

Definition and outcomes

Clinical and morphologic features, categorization and grading of comorbidities as well as outcomes measures were classified accordingly to the reporting standards for carotid interventions appointed by the SVS [2]. Primary outcome was prevention of death and all early (<30 days) perioperative cerebrovascular events. Composite end-point at 30-day included freedom from stroke/death/myocardial infarction.

Data analysis

Clinical data were recorded and tabulated in Microsoft Excel (Microsoft Corp, Redmond, Wash) database. Statistical analysis was computed with SPSS, release 23.0 for Windows (IBM® SPSS Inc., Chicago, Ill., USA). Results are presented as mean ± SD for continuous variables, while number (percentage) for the categorical ones.

Results

Out of 526 patients who underwent CEA during the study period, 13 (2.2%) were identified to have an extracranial carotid artery stenosis with an IA. There were 7 (54%) males; mean age was 68 ± 9 years (range, 52-85; IQR, 61-73). Comorbidities and risk factors are presented in Table 1: the IA was asymptomatic and intact in all patients. Urgent CEA was performed in 4 (31%) cases. Loco-regional anesthesia was used in 11 (85%) cases; 2 (15%) was converted to general anesthesia due to rapid worsening of the clinical condition during CCI and CEA was accomplished with shunting. Overall, mean carotid cross-clamp was 20 ± 5 minutes (range, 12-26; IQR, 17-23); primary closure was used in 11 (85%) cases, patching was used in 2 (15%). Postoperative cerebrovascular complication rate was 15% (n = 2): specifically, non disabling minor stroke (n = 1) occurred on postoperative day 1 in a 61 year old lady treated emergently for an acute neurologic syndrome, and intracranial hemorrhage (n = 1) developed on postoperative day 3 in an 85 year old lady due to hyperperfusion syndrome. Rupture of the IA never occurred. In-hospital mortality rate was 8% (n = 1, the abovementioned hemorrhage); composite end-point was 92%. All but 12 (92%) patients were discharge alive and independent. At 1 year follow-up, no patient with tandem lesion died: all of them underwent CT-A control, and IA rupture was never detected.

Table 2: Literature summary of CEA in patient with IAs.

Authors	*Patients*	*Stroke*	*IA rupture*
	(n)	*(%)*	*(%)*
Ladowski, *et al. [4]*	19	2	0
Orecchia, *et al. [5]*	10	2	0
Kappelle, *et al. [11]*	90	7	0
Ballotta, *et al. [8]*	13	0	0
Suh, *et al. [9]*	6	0	0
Borkon, *et al. [10]*	11	0	0

CEA: Carotid Endarterectomy; IA: Intracranial Aneurysms.

Discussion

Authors' experience confirms that tandem lesions comprising extracranial carotid artery stenosis and IA is a rare finding; the 2.2% prevalence rate reported in the present cohort is similar to the estimated 1.9% to 3.2% range reported in a recent synthesis of the published data on this topic [1]. Although Authors did not aim to assess the incidence of IAs, this is a robust finding because all patients included in this cohort of CEA underwent preoperative evaluation of the WC anatomy [4-10].

Tandem lesions deserve careful evaluation because we have a sort of triple threat: the risk of the carotid stenosis, the risk of the IA, and also the risk of the mutual complications. An analysis of large data base, Kappelle et al. [11] found that the 5-year risk of ipsilateral stroke after CEA in patients with unrepaired IA was 10% as opposed to 22.7% in medically treated patients with unrepaired IA, and concluded that CEA can usually be performed without repairing any incidental IA first. Authors' experienced similar findings with this cohort of patients: the overall stroke rate was of 15% at 1 year follow-up, which is in agreement with the 6.6% to 20% range reported by the experiences (Table 2) comprising >10 cases [4,5,8,10]. Although rupture prevention was the main end-point in all the experiences and it was never observed, these data shows that patients with tandem lesions are at high-risk for postoperative cerebrovascular events.

There is theoretical concern that CEA might cause sudden changes in intraluminal pressure, thereby increasing the risk of hemorrhage [1,12]. The reported risk of intracranial hemorrhage following CEA ranges between 0.2% and 0.8%, but is documented in only 0.05% of these tandem lesions [1]. The recent review of Kahn et al. [1] showed that hemorrhage can occur independently following CEA where no aneurysm is identified. It is, therefore, unlikely that ruptured aneurysms are the main cause of cerebral hyperperfusion hemorrhage. This occurred also in Authors' series because one of the two strokes was hemorrhagic in origin but was not cause by the IA rupture.

The effects of CEA on IA growth are not well understood or reproducible. The published experience on this topic with varied follow-up showed no significant enlargement of the aneurysm following intervention [1]. However, sudden increase in cerebral blood flow has been shown to persist for at least 1 month following CEA [13]. Accordingly to this data, the present analysis did not aim to report on long-term outcome; however, Authors' follow-up was consistent with all patient surviving at 1 year and receiving CT-A which proved IAs were all unchanged dimensionally.

Limitation

There are limitations to this analysis. First and foremost it is a retrospective analysis even if patients were enrolled consecutively and prospectively. Second, the number of patients is low. Last, preoperative imaging did not provide information on the functional activity of an anatomically complete WC. Nevertheless, our cohort is the most recent one and homogeneous from a perioperative management point of view.

Conclusion

Authors experience is consistent with the Literature analysis: although overall stroke rate was not negligible in these patients, specifically to the aim of the paper, CEA does not significantly increase the risk of IA rupture.

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