

## Carotid-ophthalmic aneurysms – protective features making them a rare cause of subarachnoid hemorrhage

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**Abstract:** *Objective:* To review the rate of carotid-ophthalmic aneurysm (COA) rupture and to identify protective features that may contribute to their low rupture rate. *Methods:* We reviewed the records of 790 patients with 773 aneurysms greater than 2 mm treated by endovascular routes between 2002 and 2012 at our institution. Seventy five carotid-ophthalmic aneurysms were identified in 72 patients. Three injected human cadaver heads were studied to evaluate the perianeurysmal environment of the carotid-ophthalmic region. *Results:* Only 2 (2.8%) of these 72 patients presented with acute SAH due to a ruptured carotid-ophthalmic aneurysm. The average size of ruptured COA was 11.3 mm versus 7 mm for unruptured aneurysms. Most of the aneurysms were discovered in patients who were asymptomatic. The most common presenting symptom was headache. In this study, we also provide cadaveric anatomic illustrations of the perianeurysmal environment in order to investigate the low rate of COA rupture. Additionally, we highlight the existence of a double arachnoid layer consisting of the arachnoid on the inferior aspect of the optic nerve and surrounding the internal carotid artery (ICA), which could further contribute to the low rupture rate of these aneurysms. *Conclusions:* Carotid-ophthalmic aneurysms are uncommon sources of subarachnoid hemorrhage. The perianeurysmal environment surrounding these aneurysms may provide protection, lending these aneurysms to a relatively benign natural history.

**Key words:** internal carotid artery, intracranial aneurysm, ophthalmic artery, subarachnoid hemorrhage, optic cistern, optic membrane

### Introduction

Asymptomatic, unruptured intracranial aneurysms have been detected in 3 to 6% of the general population. However, aneurysmal subarachnoid hemorrhage (SAH) occurs in 6

to 8 per 100,000 people per year. (8, 23, 24) The prognosis of ruptured aneurysms remains poor, with mortality nearing 50% and severe disability occurring in approximately 20% of patients. With the increase use of computed tomography (CT) and magnetic resonance

imaging (MRI), incidental aneurysms are being identified and treated more frequently. Whether or not to treat incidental aneurysms has been a subject of much debate, as there still remains much conflict in the literature surrounding the natural history, risk of aneurysm rupture, and morbidity of microsurgical and endovascular interventions.

Risk factors for aneurysm rupture and subsequent SAH include smoking, hypertension, atherosclerosis, and alcohol consumption. (12) Some authors also have proposed other independent risk factors, including aneurysm size, irregularity in the shape of the aneurysm, and location. (9, 12) Overall, aneurysms of the posterior communicating artery (PCoA) and anterior communicating artery (ACoA) more often present with SAH compared to those located at other regions, such as the paraclinoid region of the internal carotid artery (ICA). (5, 7, 14, 25) This disparity may be due to differences in the perianeurysmal environment (PAE), including certain anatomical constraints. Surrounding bone, dura, nerves, and brain parenchyma may be encountered by aneurysms as they grow, affecting their propensity to rupture. (19, 20) Such constraints may also alter wall shear stress (WSS), a concept has been shown to play a prominent role in aneurysm formation, propagation, and rupture. (1, 9, 21) Indeed, Seshaiyoor et al demonstrated that contact of the aneurysm with surrounding structures may decrease the stress at the fundus of a saccular aneurysm, thereby providing a protective effect and hindering aneurysm rupture. (20)

A limited number of studies have investigated the potential constraints of the perianeurysmal environment and the effect they may have on the risk of aneurysm rupture. (Need references for these studies) These studies suggest that the anatomical constraints of the PAE may prevent aneurysm rupture. It is also conceivable that, in these cases, the components of the PAE may provide a protective barrier when aneurysm rupture occurs, resulting in a more contained hemorrhage and benign clinical course. In this study, we retrospectively reviewed the rupture rate, presenting symptoms, and perioperative morbidity of 75 endovascularly treated carotid-ophthalmic aneurysms (COA's) treated in 72 patients at our institution. We also evaluated the PAE of COA's to investigate possible explanations for the low rate of rupture of these aneurysms.

### **Materials and methods**

This study was approved by the institutional review board of Rush University Medical Center. We conducted a retrospective chart review of 790 patients with 773 intracranial aneurysms larger than 2 mm treated by endovascular embolization between 2002 and 2012. These aneurysms were treated by three physicians (DKL, RM, MC). Of these 773 aneurysms, 75 (9.7%) occurred at the carotid-ophthalmic junction. Admission data, operative reports and imaging studies were reviewed to collect information on patient's age, gender, aneurysm location, size and aneurysm rupture status.

We also performed dissection of the carotid-ophthalmic region in three formaline

fixed human cadaver heads to describe the PAE of the ophthalmic carotid region from which aneurysms arise. On each cadaver head, a standard pterional craniotomy was performed followed by extradural drilling of the lesser wing of the sphenoid. This was followed by opening of the dura and proximal sylvian fissure dissection under 20x microscope magnification. The carotid artery medial to the anterior clinoid was identified in each head. Anterior clinoidectomy was performed and the ophthalmic artery was identified. Subsequently the region lateral, medial, and superior to the ophthalmic artery was identified and examined.

## Results

75 Endovascularly Treated Carotid-ophthalmic Aneurysms:

Of 773 endovascularly treated aneurysms at our institution, 75 were found to be located at the carotid-ophthalmic junction. These 75 COA's occurred in 72 patients. Data surrounding these COA's is presented in Table 1. The presenting symptoms of the 72 patients with COA's is presented in Table 2. Most of the aneurysms were discovered in patients who were asymptomatic. The most common presenting symptom was headache. Other presenting symptoms included visual disturbance, dizziness and syncope. All the aneurysms in this series were treated via endovascular methods. Specific treatment methods are listed in Table 1.

Six patients (8.3%) presented with acute subarachnoid hemorrhage. However, in four of these patients the source of the hemorrhage was very clearly defined to be an aneurysm

other than the one in the carotid-ophthalmic region. Only 2 (2.8%) patients presented with acute SAH due to ruptured COA's. One of these patients had bilateral COA's. One presented as Hunt and Hess grade 1, while the other patient presented as Hunt and Hess grade 3. One patient had a modified Fisher grade 1 SAH and one patient had a modified Fisher grade 2 SAH. No patient presenting with a ruptured COA required permanent cerebrospinal fluid (CSF) diversion. The mean size of unruptured COA's was 7.0 x 6.4 mm, while the mean size of ruptured aneurysms was 11.3 x 9.3 mm.

Complications occurred in 9 of the 72 (12.5%) treatment procedures (Table 3). The most common complication was procedure-related infarction, which occurred in 4 patients (5.5%). Groin-related complications occurred following 2 (2.8%) procedures. One of these was a femoral artery pseudoaneurysm requiring thrombin injection, while the other was a superficial groin hematoma that spontaneously resolved. Acute intra-procedural, in-stent thrombosis occurred in 1 patient. Angioplasty was performed immediately and the patient was placed on a heparin drip post-operatively. There were no clinical sequelae.

Evaluation of the PAE in Cadaveric Models:

We evaluated the carotid-ophthalmic junction in three human cadaver models and found that COA's may be situated in a unique environment (Figures 1-6). The ophthalmic segment of the internal carotid artery (ICA) is located between the origin of the ophthalmic artery (OA) and the origin of the PCoA. (4)

**TABLE 1**  
**Characteristics of 75 endovascularly treated carotid-ophthalmic aneurysms treated in 72 patients**

Mean age, yrs (SD)	55.0 (14.5)
Male, N (%)	9.0 (12.5)
Mean height, mm (SD)	9.1 (5.5)
Mean width, mm (SD)	6.5 (4.6)
Ruptured*, N (%)	2.0 (2.8)
Treatment, N (%):	
Neuroform stent + coiling	34 (45.3)
Pipeline embolization device	17 (22.7)
Enterprise stent + coiling	10 (13.3)
Pipeline embolization device + coiling	4 (5.3)
Coiling alone	5 (7.3)
Other	5 (7.3)

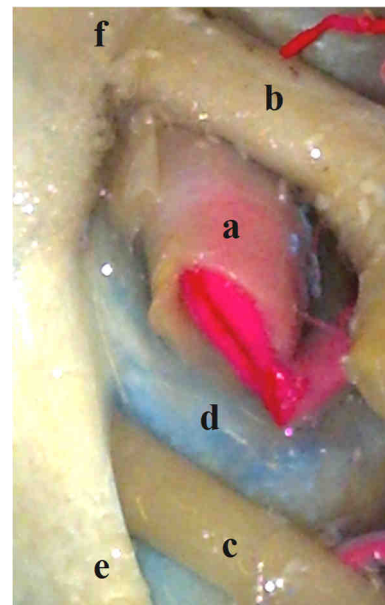
\*Six patients presented with acute subarachnoid hemorrhage. In 4 of these patients the source of subarachnoid hemorrhage was determined not to be the carotid-ophthalmic aneurysm.

**TABLE 2**  
**Presenting symptoms of 72 patients with CO aneurysms**

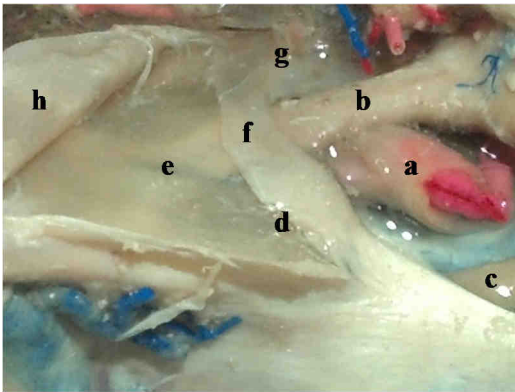
Symptom	N	%
Asymptomatic	35	48.6
Headache	16	22.2
Acute SAH	6	8.3
Visual disturbance	7	9.7
Dizziness	3	4.2
Syncope	2	2.8
Other	2	2.8

**TABLE 3**  
**Complications occurring in 72 endovascular procedures**

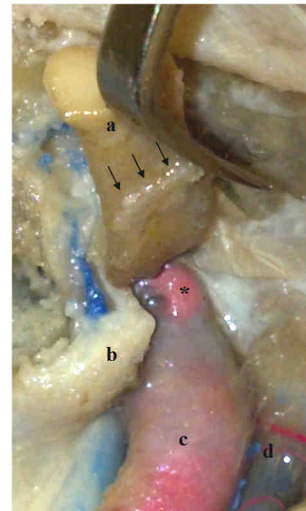
Complication	N	%
Infarct causing hemiparesis	4	5.6
Retroperitoneal hematoma	2	2.8
Groin complications:		
Femoral artery pseudoaneurysm	1	1.4
Groin hematoma	1	1.4
Acute in-stent thrombosis	1	1.4



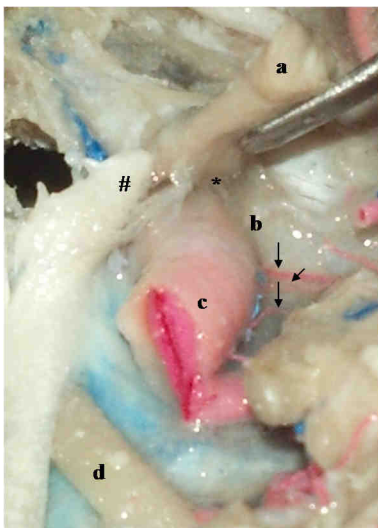
**Figure 1** - View of the supraclinoid internal carotid artery (ICA) from a left sided approach. a) ophthalmic segment of internal carotid artery; b) optic nerve; c) oculomotor nerve; d) posterior clinoid; f) falciform ligament



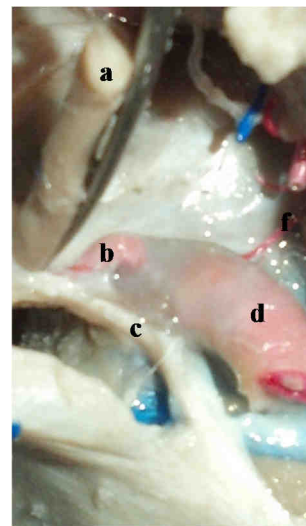
**Figure 2** - View of the supraclinoid ICA from the left side. The left temporal lobe has been resected to better view the region of interest. a) Ophthalmic segment of ICA; b) optic nerve; c) oculomotor nerve; d) anterior clinoid process; e) roof of optic canal (anterior root of lesser wing of the sphenoid bone); f) falciform ligament; g) planum sphenoidale; h) reflected dura



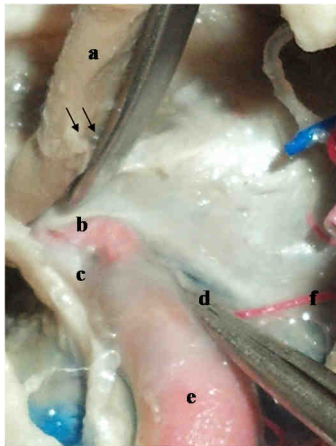
**Figure 4** - a) optic nerve; →) double arachnoid layer consisting of the arachnoid layer on the inferior aspect of the optic nerve and surrounding the ICA; b) lateral edge of falciform ligament after sectioning; \*) ophthalmic artery origin; c) ICA; d) superior hypophyseal arteries



**Figure 3** -View of the supraclinoid ICA from the left side through a trans-sylvian approach. a) optic nerve (cut and reflected superiorly); b) entrance to carotid cave; c) ICA; d) oculomotor nerve; \*) double arachnoid layer of the carotid artery and the inferior aspect of the optic nerve ; →) superior hypophyseal arteries



**Figure 5** - Left sided approach after anterior clinoidectomy with the left temporal lobe removed. a) optic nerve; b) ophthalmic artery; c) distal dural fold; d) ICA; f) superior hypophyseal arteries



**Figure 6** - Left sided view of the Carotid ophthalmic region through a trans-sylvian approach. a) optic nerve; →) The double arachnoid layer of the carotid artery and optic nerve in the area of ophthalmic artery was identified in every caver head; b) ophthalmic artery; c) distal dural fold; d) carotid cave; e) ICA; f) superior hypophyseal artery

The origin of the OA, and thus the proximal limit of the ophthalmic segment, is typically (85%) located distal to the distal carotid dural ring and, therefore, within the subarachnoid space. (6) The ophthalmic artery arises from the dorsal or dorsomedial portion of the ICA underneath the optic nerve. (4) Aneurysms of the carotid-ophthalmic junction arise from the superior wall of the ICA, just distal to the OA origin, and extend superiorly towards the overlying optic nerve.

The falciform ligament, connecting the dura of the anterior clinoid process (ACP) and the planum sphenoidale, covers the optic nerve in the region of the OA origin. In its ophthalmic segment, the ICA is surrounded by the ACP laterally, the optic nerve, falciform ligament and roof of the optic canal superiorly, the tuberculum sella medially and the middle

clinoid and interclinoid dural folds inferiorly. These bony and dural structures provide biomechanical rigidity to the ophthalmic segment of the ICA. Moreover, in our cadaveric dissection, we consistently identified a double arachnoid layer formed by the arachnoid of the inferior aspect of the optic nerve and the arachnoid surrounding the ICA. This double arachnoid layer potentially could give the dome of the formed COA more protection and contribute to a decreased risk of rupture. Furthermore, if the rupture takes place, this double arachnoid layer could limit the degree of dissemination of the subarachnoid hemorrhage.

## Discussion

Unruptured intracranial aneurysms have been detected in up to 6% of the general population, yet aneurysmal subarachnoid hemorrhage occurs in only 6 to 8 per 100,000 people per year. According to the ISUIA study, the annual risk of rupture of anterior circulation aneurysms less than 7 mm is quite low (0-1.5%) compared to larger ones (6.4-40%). (25) Given the poor prognosis and significant morbidity associated with SAH, treatment of aneurysms less than 7 mm incidentally found has been advocated by many.

Aneurysms of the carotid-ophthalmic junction are rare. Prior studies have reported particularly low rates of rupture among aneurysms occurring at the carotid-ophthalmic junction.<sup>16,17</sup> The rupture rate of COA's in our series (2.8%) is similar to that reported previously in the literature. The ISAT investigators found COA's to be

responsible for only 1.4% of SAH.<sup>14</sup> Similarly, Fukerson et al reported a rupture rate of 1.5%, while Henkes et al reported a rupture rate of 2% for CO aneurysms.<sup>3,5</sup> These studies, as well as ours, demonstrate that the rate of rupture of aneurysms at the CO segment is low. Given the particularly low rate of COA rupture, the decision to treat unruptured, incidental COA's less than 7 mm should be approached with caution. A COA of 7 mm might not behave the same as a 7 mm ACoA or PCoA aneurysm due to its surrounding anatomic environment.

Studies evaluating the endovascular treatment of COA's specifically have reported similar complication rates to ours. Fukerson et al reported a morbidity of 7.1% in COA's treated by endovascular methods.<sup>3</sup> Similarly, Sherif et al and Loumiotis et al reported morbidities of 5.3% and 18% and mortality rates of 2.6% and 3%, respectively.<sup>13,22</sup> In our series, we experienced a morbidity of 12.5% (N=9). It should be noted that all complications except one were self-resolving or of minimal clinical significance. One patient in our series had a long-term dense hemiparesis that significantly affected her quality of life. We experienced no procedure-related mortality.

In our series the ruptured COA's were larger than the unruptured aneurysms. One explanation for this finding could be that once the aneurysm grows to a larger size, it expands beyond the aforementioned protective barriers of the carotid-ophthalmic region. We were unable to identify a particular threshold, above which the aneurysm's size no longer permits protection by the PAE. This may be a function

of differences in this region amongst individuals.

While the exact reason for the low rate of rupture of CO aneurysms is unknown, it may be due to a disparity in the aneurysm size, morphology, or wall shear stress. These features, at least in part, may be determined by the anatomical constraints of the perianeurysmal environment at this location.<sup>4,6</sup> The anatomic features of the ophthalmic segment of the carotid artery, as described above, may have important implications for the risk of aneurysm rupture and the extent of subarachnoid hemorrhage that occurs when CO aneurysms rupture. The rigid surroundings of the OA origin and the superior orientation of the aneurysmal axis results in a physically constraining PAE. Structures such as the falciform ligament, optic nerve, anterior clinoid and the distal dural ring may serve to reduce the transmural pressure of the aneurysm. Furthermore, the double arachnoid layer described in this study also could contribute to decreasing the transmural pressure. This membrane may lead to the stability of these aneurysms, decreasing their rate of rupture and possibly limiting the extent of hemorrhage.

#### Conclusion:

Traditionally, in unruptured aneurysms, size and location have been a main determinant in the decision of whether or not to treat. However, anatomic features associated with aneurysm location may play an important role in their natural history and risk of rupture. Here, we demonstrated that COA's have an exceptionally low risk of rupture. We evaluated the PAE surrounding these

aneurysms and found that COA's may be protected by the imposed anatomical constraints of surrounding structures. We propose that this unique environment may play an important protective role in the low incidence of COA formation and rupture. Treatment of incidentally small COA's should be considered with caution.

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