Neurosurg Rev (2011) 34:29–38
DOI 10.1007/s10143-010-0290-5

REVIEW

Peripheral ophthalmic artery aneurysm

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Received: 10 April 2009 / Revised: 27 May 2010 / Accepted: 29 August 2010 / Published online: 15 October 2010

Abstract Generally speaking, the term "ophthalmic aneurysms" refers to carotid-ophthalmic aneurysms, which arise from the internal carotid artery (ICA) wall at or around the origin of the ophthalmic artery (OA). In contrast, aneurysms arising from the OA stem or its branches, separate from the ICA are called peripheral OA aneurysms (POAAs). POAAs are a rare entity, which clinical features and natural course are not fully understood. A comprehensive literature review of reported aneurysms involving each segment of the OA was undertaken. The demographics, aetiology, clinical manifestations and treatment of reported POAAs are discussed. Of 35 retrieved cases, ten involved the intracranial segment, two were fusiform aneurysms in the optic canal, 17 arose from the intraorbital segment, and 6 involved either the lacrimal or the anterior ethmoidal branches. In 34 cases, clinical details were available; 18 patients experienced moderate to severe visual impairment including blindness, while seven patients had improvement in visual acuity as a result of surgical treatment. The present clinical review reveals that aneurysms of the OA stem and lacrimal branch are potentially threatening to visual acuity, while intracranial segment and anterior ethmoidal aneurysms can rupture and cause subarachnoid or intraparenchymal haemorrhage. Surgical intervention is mandatory in symptomatic cases to prevent visual deterioration or treat aneurismal rupture; alternatively, for small incidental POAAs "watchful waiting" may be indicated.

Keywords Aneurysm · Ophthalmic artery · Optic nerve · Vision · Subarachnoid haemorrhage

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Introduction

The ophthalmic artery (OA) is the first major branch of the internal carotid artery (ICA) after its emergence from the cavernous sinus (rarely within the sinus); it consists of intracranial, intracanalicular and intraorbital segments and gives off the central retinal, ciliary, lacrimal, posterior and anterior ethmoidal artery branches. A detailed literature review revealed that aneurysms may involve each segment and some branches of the OA, these aneurysms have no direct relationship with the ICA or the circle of Willis. In this review, aneurysms arising from the OA stem or branches are termed peripheral OA aneurysms (POAAs).

Semantically, the commonly used term "ophthalmic aneurysms" is the abbreviation of "carotid-ophthalmic aneurysms"; Drake et al. [16] and Sengupta et al. [52] defined this entity as those which arise from the wall of ICA in the region of the origin of OA. However, other aneurysms were also found in this segment, such as superior hypophyseal aneurysms [10], dorsal ophthalmic aneurysms [2, 15] and blood-blister-like aneurysms [33] at the dural ring. So, "aneurysms of the ophthalmic segment of the carotid artery" is more accurate rather than "ophthalmic aneurysms" in this circumstance. In contrast, POAAs located on the OA "itself" [53, 58] are "true" [24, 41] OA aneurysms. However, "true aneurysm" is an expression occasionally used in differentiation with pseudoaneurysm (also known as false aneurysm); it is better

known as a histological conception rather than an anatomical one. In this sense, "trunk" [50, 58] instead of "true" is more proper; nevertheless, ophthalmic trunk aneurysm cannot be included in its terminal branch aneurysm. Vice versa, "distal OA aneurysm" is not suitable for aneurysm on the intracranial segment (proximal portion [7]). So, POAAs [6, 25, 37, 58] is a most appropriate term to express the anatomical origin of this entity. In the present review, POAAs have extended connotation to cover aneurysms of all portions of OA: the intracranial, intracanalicular, intraorbital segments and terminal ophthalmic branches aneurysms. Aneurysms of the ophthalmic segment account for 11% of all intracranial aneurysms [10]. By comparison, POAAs are much rarer and poorly understood. We herein review the incidence, aetiology, manifestation and clinical course of these aneurysms.

Methods

The PubMed database of the National Library of Medicine and National Institutes of Health and Google Scholar search were used to search for the published cases meeting the definition of POAAs in the first paragraph. The following terms were searched: "ophthalmic artery stem aneurysm", "intracanalicular aneurysm", "intraorbital aneurysm", "ophthalmic artery branch aneurysm", "lacrimal aneurysm" and "ethmoidal aneurysm". Additional cases reported from the reference lists of search results were also gathered. All the reports published in English and Chinese are included.

We comprehensively reviewed all reported cases of POAAs, with special attention to clinical presentation in relation to various anatomic segments of the OA and provide a discussion of clinical features, diagnostic workup and treatment of POAAs.

Results and analysis

Intracranial segment POAAs

There have been ten cases of aneurysms on the intracranial segment of OA reported so far. The patients consisted of seven men and three women ranging in age between 20 and 70 years at presentation. Five of the aneurysms were on the right side and the other five on the left (Table 1).

Six patients had unruptured intracranial POAAs. Four of them had various ophthalmologic symptoms to some degree, including visual loss. The first patient's vision improved with resolution of papilledema which subsided after partially unroofing the optic canal and clipping the OA proximal to the aneurysm [24]. The other two had their ICA ligated [44] or clipped [7]. They experienced transient hemiparesis [44] or blepharoptosis [7] ophthalmologically unchanged [44] or improved [7], respectively. The first patient who presented only with a horizontal visual field defect was observed and had no further symptom, followed up the next year [1]. The other two patients who were free of visual disturbance were comorbid with other vasculopathies. One had a ruptured anterior communicating artery aneurysm [50]; another had moyamoya disease and received bilateral superficial temporal artery to middle cerebral artery bypasses [25]. All aneurysms of these two patients were clipped.

Four patients had ruptured intracranial POAAs. Two of the patients' aneurysms ruptured after trauma. One of them resulted in three episodes of epistaxis weeks after an automobile accident; the right ICA was ligated distal to the carotid bifurcation and clipped intracranially, no recurrent rhinorrhagia occurred [27]. The other patient who lost consciousness after an explosion, angiography, performed to rule out an intracranial haematoma, were unexpectedly found to have saccular aneurysm arising from the OA resulting to the clipping of the aneurismal stalk after clipping the proximal portion of the OA [38]. The two patients remained blind in the affected eyes. The other two patients' aneurysms ruptured spontaneously and caused subarachnoid haemorrhage (SAH), and both patients manifested with consciousness disturbance, one with normal visual acuity and without noted visual field defect [58], another was confirmed with visual loss on the affected eye during hospitalization, improving the general condition for the surgery [53]. The aneurysms were confirmed by three-dimensional rotational digital subtraction angiography (DSA); both were clipped via pterional approach. The two patients recovered without definite neurological deficit.

Intracanalicular segment POAAs

Only two cases of intracanalicular aneurysms have been reported in recent years. The patients were male adults with left unruptured fusiform aneurysms (Table 2). The first patient experienced painless visual loss in the left eye decreased to finger counting at 10 cm, underwent unroofing of the optic canal from the anterior clinoid process to the posterior aspect of the orbit, but the aneurysm could not be clipped due to inferior adherence to the optic nerve, which then resulted to the application of endovascular approach with intravenous anticoagulation [41]. The second patient's vision dropped to hand motion status 4 days after admission. He accepted endovascular parent artery occlusion rather than surgical clipping [5]. These two patients were treated similarly by endovascular means, with transarterial coil obliteration of the proximal OA, and confirmation by external carotid angiography of an adequate

Table 1 Accumulated published cases of intracranial segment POAAs

Number	Author, year	Age/Sex	Trauma history	Clinical presentation	Side of aneurysm	Treatment for aneurysm	Concomitant angiopathy	Outcome
1	Kinley and Leighninger, 1952 [27]	42 years, M	Accident	Visual loss	Rt	ICA occlusion	No	Blind
2	Parkinson et al., 1961 [38]	31 years, M	Explosion	Coma	Lt	Clipping	No	Blind
3	Raitta, 1968 [44]	20 years, M	NM	Visual loss	Rt	ICA ligation	No	VA unchanged
4	Alexander, 1970 [1]	24 years, F	NM	Visual field defect	Lt	Observation	No	VA unchanged
5	Jain, 1970 [24]	44 years, M	NM	Visual loss	Rt	OA clipping	No	VA improved
6	Cunningham and Sewell, 1971 [7]	37 years, F	NM	Visual loss	Lt	ICA clamping	No	VA improved
7	Sato et al., 1999 [50]	50 years, M	No	Headaches	Rt	Clipping	ACoA aneurysm	Good
8	Kawaguchi et al., 2001 [25]	26 years, F	NM	Episodic dizziness	Rt	Clipping	Moyamoya disease	Good
9	Yanaka et al., 2002 [58]	54 years, M	NM	SAH	Lt	Clipping	No	Good
10	Seo et al., 2006 [53]	70 years, M	Fall	SAH, visual loss	Lt	Clipping	No	Good

F female, *M* male, *NM* not mentioned, *Rt* right, *Lt* left, *VA* visual acuity, *SAH* subarachnoid haemorrhage, *OA* ophthalmic artery, *ACoA* anterior communicating artery

choroidal blush. In both these patients, visual function was preserved and improved satisfactorily.

Intraorbital segment POAAs

To date, 17 cases of intraorbital segments POAAs have been reported. Clinical details of the case reported by Wheeler and Baker [57] were not available. The other 16 patients consisted of 12 males and four females, whose ages ranged from 34 to 64 years. One patient had bilateral intraorbital aneurysms [49]. Of the remaining 15 patients, 11 aneurysms were on the right side and four on the left side (Table 3).

Three patients had at least one blind eye related to the aneurysm of this segment or trauma. One of the two patients with unilateral blindness coughed causing the intraorbital aneurysm to rupture spontaneously, which resulted in a disastrous immediate blindness and whole ophthalmoplegia [34]. Another patient experienced sudden blindness with a feeling of fullness, but without pain. Selective left ICA angiography demonstrated an intraorbital aneurysm, while the choroid blush was still evident [9]. Bilateral blindness in the third patient was caused by missile injury. About 2 weeks later, he developed a rather rapidly right-sided pulsating exophthalmos; repeated angiography revealed an enlarging lobulated aneurysm. His OA was clipped at its origin; exophthalmos subsided but his vision did not recover [43].

Significant rapid and severe deterioration of visual acuity was reported in three patients. Two patients' eyesight decreased within 2 weeks, among them, one's vision dropped to 20/200 with central scotoma [36], and another's vision reduced to light perception with merely a feeling of periorbital pressure [18]. Both these patients had operations to occlude the parent artery. Although postoperative angiography showed the choroidal crescent supplied by the external carotid collaterals, neither of them had any visual improvement. The third patient's visual acuity decreased to 20/400 in 2.5 months, but while he refused surgery of any kind his vision was found to have not worsened in the following 3 years [46].

Moderately severe visual impairment was reported in three patients. The first patient had buzzing noises and pain in the left eye with pulsating exophthalmos, eyesight

Table 2 Accumulated published cases of intracanalicular segment POAAs

Number	Author, year	Age/Sex	Trauma history	Clinical presentation	Side of aneurysm	Treatment for aneurysm	Concomitant angiopathy	Outcome
1	Piché et al., 2005 [41]	53 years, M	No	Painless visual loss	Lt	OA occluded	No	VA improved
2	Choi et al., 2008 [5]	35 years, M	2 accidents	Progressive visual loss	Lt	OA occluded	No	VA improved

M male, *Lt* left, *OA* ophthalmic artery, *VA* visual acuity

Table 3 Accumulated published cases of intraorbital segment POAAs

Number	Author, year	Age/Sex	Trauma history	Clinical presentation	Side of aneurysm	Treatment for aneurysm	Concomitant angiopathy	Outcome
1	Mortada, 1961 [35]	51 years, F	No	Pulsating exophthalmos, visual loss	Lt	CCA ligation	No	Exophthalmos cured
2	Rubinstein et al., 1968 [48]	36 years, M	Two concussions	Loss of central vision	Rt	No	No	Unchanged
3	Meyerson and Lazar, 1971 [34]	54 years, M	Head injury	Haemorrhage, proptosis and blind	Rt	OA obliteration	No	Blind
4	Danziger and Bloch, 1974 [9]	38 years, M	NM	Sudden blind	Lt	No	No	Blind
5	Rengachary and Kishore, 1978 [46]	63 years, M	NM	Swelling, burning and visual loss	Rt	No	AVF	Unchanged
6	Rahmat et al., 1984 [43]	34 years, M	Missile injury	Pulsating exopthalmos, blind	Rt	OA clipping	No	Blind
7	Ogawa et al., 1992 [36]	63 years, F	NM	Exophthalmos, visual loss	Rt	Trap and resection	No	VA recovered
8	Kikuchi and Kowada, 1994 [26]	54 years, M	NM	Disorientation and memory disturbances	Rt	No	AVM	Unchanged
9	Kawaguchi et al., 2001 [25]	51 years, M	NM	Frontal headaches	Lt	No	AVF	Good
10	Dehdashti et al., 2002 [13]	34 years, M	NM	Minimal visual field defect	Rt	No	BA aneurysm	Unchanged
11	Ernemann et al., 2002 [18]	64 years, F	NM	Periorbital pressure, visual loss	Rt	OA sacrifice	No	VA not recover
12	Kleinschmidt et al., 2004 [28]	47 years, M	NM	Unconsciousness	Rt	No	AVM	Symptomless
13		44 years, M	Head kicked	Chronic ache, visual loss	Lt	OA and aneurysm embolization	AVM	VA improved
14	Wang et al., 2007 [56]	45 years, M	No	Visual loss	Rt	Neck clipping	CM	VA preserved
15	Sabatino et al., 2009 [49]	52 years, M	NM	Neck pain, nausea and vomiting	Bilateral	No	Multiple aneurysms	Unchanged
16	Pandey et al., 2010 [37]	57 years, F	NM	Headache and ptosis	Rt	No	Multiple aneurysms	Unchanged

The intraorbital segment ophthalmic artery aneurysm in Wheeler's series had no description on clinical details [57]

F female, *M* male, *NM* not mentioned, *Rt* right, *Lt* left, *VA* visual acuity, *CCA* common carotid artery, *OA* ophthalmic artery, *AVF* arteriovenous fistula, *AVM* arteriovenous malformation, *BA* basilar artery, *CM* cavernous malformation

decreased to 6/18, arteriogram showed a tortuous, delayed emptying aneurismal dilatation. After ligating the left common carotid artery, pulsating exophthalmos subsided, and the aneurysm was no longer palpable [35]. The second patient developed a large, vascular lesion of the left orbit following a kick to the head, and decades later his visual acuity decreased to 6/36, then corrected to 6/9. The patient received embolization of the distal OA and largest aneurysm, and then the aneurysm and associated AVM were resected. The corrected visual acuity increased to 6/6 [28]. Wang et al. [56] reported a patient with a vision of 0.3 in the eye harbouring aneurysm; after removal of the concomitant haemorrhagic intracranial cavernous malformation, the aneurysm neck was clipped. Postoperative angiography demonstrated patency of the OA, and preoperative visual acuity was restored. Another patient with impaired and considerably fluctuating eyesight whose exact visual acuity was not available refused surgical treatment but was found to be clinically stable in the following 18 months [48].

Some patients with intraorbital POAAs have no noticeable ophthalmologic symptoms and were identified fortuitously by imaging, performed for unrelated neurological problems [13, 25, 26, 28]. Nine of the 16 patients with intraorbital aneurysms were comorbid with other vascular lesions, i.e., arteriovenous fistula [25, 46], arteriovenous malformation [22, 24], intracranial aneurysm(s) [13, 37, 49] and cavernous malformation [56].

Terminal POAAs

To date, there have been six terminal POAAs reported arising from the lacrimal or anterior ethmoidal arteries, equally distributed between both sides and genders (Table 4). The only lacrimal aneurysm reported by Heimburger et al. [21] was the earliest POAA retrieved in this review. The patient presented with left eye proptosis, oculomotor palsy, blurred optic disk and visual acuity dropping to 20/70. The aneurysm was found and removed by transcranial exploration to the orbit, and her visual acuity improved to 20/40, with unimpaired ocular movement. This case has detailed intraoperative data, but no pre- or postoperative angiography.

The other five terminal POAAs all arose from the anterior ethmoidal branch. Two occurred in females, and three in males, whose ages ranged from 44 to 65 years old at presentation. Four patients' aneurysms ruptured, causing SAH [17, 55] or frontal intracerebral haematoma [8, 45]. Of the two SAH patients, one has both ICAs occluded in the neck and the right vertebral artery stenosed at origin. The aneurysm was obliterated by cautery [55]. The other patient's aneurysm was at the anastomosis of the left orbitofrontal artery and the left anterior ethmoidal artery; the aneurysm was neck clipped and excised [17]. The two patients with intraparenchymal haemorrhage have almost identical histories. Both are adult females, their frontobasal haematomas were evacuated, the aneurysms were excised and parent vessels were coagulated [8, 45]. The non-ruptured aneurysm was on the anterior ethmoidal artery, which fed an olfactory groove meningioma. The flow-related aneurysm disappeared early after removal of the tumour [54].

Discussion

Demographics of POAAs

POAAs are a small entity, only a small number of cases have been reported, involving each segment of the OA. Especially the intracanalicular segment POAAs are ex-

Table 4 Accumulated published cases of terminal POAAs

Number	Author, year	Age/Sex	Trauma history	Clinical presentation	Side of aneurysm	Treatment for aneurysm	Concomitant disease	Outcome
1	Heimburger et al., 1949 [21]	58 years, F	NM	Exophthalmos, visual loss	Lt	Excision	No	VA improved
2	Tasker, 1983 [55]	65 years, M	NM	SAH	Rt	Cautery	ICAs occlusion, rVA stenosis	Good
3	Enomoto et al., 1985 [17]	44 years, M	No	SAH, intracerebral haemorrhage	Lt	Clipping and excision	No	Good
4	Ranjan and Joseph, 1994 [45]	45 years, F	No	Intracranial haemorrhage	Lt	Excision	No	Good
5	Tachikawa et al., 2002 [54]	51 years, M	NM	Generalized seizure	Rt	No	Olfactory groove and convexity meningiomas	Good
6	da Costa et al., 2006 [8]	55 years, F	NM	Intracerebral haemorrhage	Rt	Excision	Rt ICA occlusion	Good

F female, *M* male, *NM* not mentioned, *Rt* right, *Lt* left, *VA* visual acuity, *SAH* subarachnoid haemorrhage, *ICA* internal carotid artery, *rVA* right vertebral artery

tremely rare, with merely two cases reported in 2005 and 2008. The intraorbital aneurysms have been reported long ago before application of angiography, and these diagnoses were mainly based on proptosis symptom. Pulsating exophthalmos was considered to be an attribute of intraorbital aneurysm, yet most of the cases were carotid cavernous fistulas or intracranial lesions [21, 22, 46]. Heimburger et al. reviewed the literature and found only six patients with an intraorbital aneurysm visualized either at operation or at autopsy; however, all the diagnoses were debatable [21].

Of the 34 patients with clinical details, 24 were male and ten were female. The male predominance of POAAs contrasts sharply with the striking female incidence of carotid-ophthalmic aneurysms [16]. Their ages ranged from 20 to 70 years, averaging to 46.79 years old. Although intraorbital segment POAAs seems to have preference on the right side in male adults, we cannot conclude yet the predominance of POAAs with such small numbers.

Aetiology of POAAs

Developmental arterial wall medial layer reduction is one aetiology of aneurysmal occurrence at arterial bifurcations around the circle of Willis, also thought to apply to POAAs. Rubinstein et al. [48] presumed their patient to be congenital in origin, the patient reported by Pandey et al. had a strong family history of intracranial aneurysms [37], and Heimburger et al. [21] confirmed the pathological alteration of the lacrimal aneurysm to have an absent elastic layer. Haemodynamic stress in the parent vessel may be another factor for the pathogenesis of POAAs [4, 25, 26]. Kawaguchi et al. [25] demonstrated significant high flow velocity and low-resistance index by color Doppler flow velocitometry imaging on the OA harboring the aneurysm in two patients. Therefore, the dome of aneurysm may expand in the direction of the blood flow thrust in the parent vessel. Yet, in the intracranial POAAs, dilation of the aneurysms could be different from that of haemodynamic stress, due to restrain from the anterior clinoid process or distal dural ring [53, 58]. The haemodynamic stress theory is verified by aneurismal disappearance after removal of the parent artery-feeding tumour. Repeated Doppler flow velocimetry imaging showed increased blood flow markedly reduced in the OA postoperatively [54].

Considering that a significant number of these aneurysms were associated with other vascular lesions, genetic as well as haemodynamic causes cannot be ruled out. Kikuchi and Kowada [26] reported an intraorbital aneurysm which did not reduced significantly in size after removal of the arteriovenous malformation supplied by the ethmoidal artery of the OA through interethmoidal anastomoses. So, evolution of POAAs may be a result of combined effects of the two mechanisms.

Trauma may be one cause of POAAs development [53]; eight of the 32 patients had a history of trauma. In the patient reported by Rahmat et al. who had a missile injury presented as delayed pulsating unilateral exophthalmos, CT scan showed small, in-driven bone chips close to the aneurysm wall. The bone chips might have initially injured the arterial wall, or repeated pulsations of the artery against the hard bone led to arterial wall injury and formation of the aneurysm [43]. In addition, two intracanlicular fusiform aneurysms may be related to atheromatosis [11, 30].

As shown above, POAAs is as heterogenous as that of commonly seen aneurysms, aetiology including congenital, flow-disturbing, traumatic and atheromatous factors.

Clinical manifestations of POAAs

Clinical manifestations of POAAs vary according to the involved segment of the artery. Intracranial segment POAAs has a similar clinical course as that of carotid-ophthalmic aneurysm, which may rupture and cause SAH. Because the OA goes intimate with the optic nerve, the aneurysm is more apt to compress the nerve. Six of the ten patients had severe visual impairment.

The intracanalicular segment POAAs are contiguous and adherent to the optic nerve in the narrow optic canal, they are more likely to cause optic nerve conduction disorder [5, 41]. Both the reported aneurysms were fusiform. In this particular region, they produced severe visual deficits before rupture.

Intraorbital segment POAAs manifest as visual loss, exophthalmos and extraocular muscle palsy, papilledema, orbital apex syndrome and intraorbital haemorrhage. Neuroophthalmological findings could be explained by direct compression or perfusion reduction [13]. Visual impairment is the most common and severe symptom, including visual field defect, loss of central vision, decreased visual acuity and blindness. Aneurysms of this specific location were thought to follow rather benign courses with slow growth and relatively minor symptoms [48], but as far as vision function is concerned, it is not at all benign.

Terminal segment POAAs can be in the orbit, such as the lacrimal artery aneurysm; while all the reported anterior ethmoidal aneurysms were in the cranium. The former presents like that of the intraorbital segment POAAs, and the latter manifests as subarachnoid and intraparenchymal haemorrahege, or no overt symptom.

Diagnosis of POAAs

Angiography is considered the gold standard to evaluate intracranial aneurysm, including POAAs. Selective internal carotid angiography with subtraction is needed for demonstrating the aneurysm, but common carotid angiography

may not demonstrate the OA and miss the aneurysm [9]. Although CT and CTA [47, 59] or MRI and MRA [20, 23] are effective in determining site and size of intracranial aneurysm, DSA is still recommended to confirm the presence of POAA. Three-dimensional rotational angiography has improved the visibility of intracranial POAAs, which clearly demonstrate its relationship apart from the ICA, avoiding false-negative surgical exploration [58]. Cross-compression manoeuvres including Allcock manoeuvre (carotid compression during vertebral angiography) are easy and considered a safe way to estimate the presence of collateral blood supply from the circle of Willis to the ophthalmic segment of the ICA, while carotid occlusion tolerance test adds significant potential morbidity [32] which may not be necessary to the pre-surgical preparation of these patients. External carotid artery (ECA) angiography should not be a routine examination to evaluate development of ECA circulation to the intraorbital part of OA too, because not all active anastomoses are demonstrated on the ECA angiogram, and a rich collateral supply network exists between the orbital external carotid branches and OA branches [36], so that when OA needs to occlude proximal to the retinal and ciliary artery origins will be well tolerated in 90% of patients expected to remain asymptomatic [19]. To evaluate collateral blood supply, however, balloon occlusion at the origin of the OA combined with ECA angiography [58] is useful when the parent artery needs to be sacrificed, while intraoperative angiography [39] with high resolution X-ray machine or intraoperative computerized tomographic angiography (CTA) [14] is valuable in confirming OA patency in cases when the artery has to be spared. In addition, patient awakening in order to assess immediate vision influence [3] during temporary clamping of the OA may be performed to ensure visual outcome.

Treatment of POAAs

In patients with ophthalmological symptoms, related to OA stem or lacrimal POAAs, radical surgical intervention is mandatory to eliminate threats to visual function. Likewise, sentinel headache [12, 42] from intracranial segment or anterior ethmoidal POAAs should be treated to avoid massive haemorrhage. Treatment options for POAAs including trapping, clipping and endovascular embolization are available. The case reports of Jain [24], Cunningham and Sewell [7], Seo et al. [53], Piché et al. [41], Choi et al. [5], Kleinschmidt et al. [28] and Heimburger et al. [21] each described a patient having improvement in visual acuity as a result of surgery or coiling.

Incomplete clamping of the ICA failed to achieve total thrombosis of the aneurysm, and ligating the artery was complicated with transient ischemic syndrome [44]. Instead, if the deconstructive strategy is chosen, clipping the OA at its origin [24] is preferred. Direct surgery was thought to be very dangerous to the function of the optic or oculomotor nerves by inadvertent injury or as a consequence of the surgical interference with the blood supply of the eye or the orbit [22]. With the development of microsurgical technique, OA branches and nerves can be well identified and preserved. There is a rich anastomotic network between OA system and the ECA branches, such as the nasal artery anastomoses with the angular branch of the facial artery, supraorbital artery, distal ophthalmic branches anastomose with the superficial temporal artery etc. [40]. So, after trapping and resecting the POAA on the first intraorbital segment of OA, the patient's vision gradually improved to preoperative level [36]. Despite the presence of abundant collateral blood supply, in most cases, appropriate surgical techniques should be considered to preserve the normal ocular circulation [58]. Neck clipping of the saccular aneurysm while keeping the parent artery patent is the optimal choice. Because of the intimity of OA and the optic nerve, it was believed that neck clipping of the aneurysm is difficult [24, 34, 36, 41, 44]. Nevertheless, Sato et al. [50], Kawaguchi et al. [25], Yanaka et al. [58] and Seo et al. [53] each managed a neck clipping of an intracranial segment POAA; removal of the anterior clinoid process and unroofing the optic canal facilitates exposing and completely clipping the aneurysm [53, 58]. After unroofing the orbit via subfrontal approach, Wang et al. [56] successfully clipped an intraorbital segment POAA by the neck; this is the only neck-clipped intraorbital aneurysm. Enomoto et al. [17] clipped a ruptured aneurysm at the anastomotic site between the frontoorbital artery and the anterior ethmoidal artery by frontotemporoparietal craniectomy.

The interventional treatment of POAAs is a relatively less invasive approach, which does not require manipulation of the optic nerve [5]. When adequate collateralization from ECA branches to the central retinal artery (CRA) is ensured, under sufficient anticoagulation, superselective catheterization of the OA and its branches with microcatheters and steerable microguidewires under digital road mapping, and transarterial obliteration of the OA at its origin or at first intraorbital segment proximal to the origins of the central retinal and ciliary arteries can be safely performed [16, 29], and sacrificing of the OA distal to the "safety point" beyond the origins of the central retinal and ciliary arteries is usually well tolerated [40]. Embolic agents including detachable microcoils [5, 29, 41] and liquid embolic agents [28, 31] may be used. Choroidal blush must be seen in order to make sure to spare the CRA from the anticipated embolization region prior to deployment of embolic agents, lidocaine and amytal provocation tests are recommended to ascertain any equivocal relationship between the catheter tip and critical vessel, the CRA [29, 40]. Pseudoaneurysm of peripheral OA, like ruptured

POAA, manifests as epistaxis or intracranial haemorrhage, and can also be cured by endovascular treatment [6, 51].

As for aymptomatic patients or those with minor symptoms or signs, such as the incidental POAAs described by Alexander [1], Kikuchi and Kowada [26], Kawaguchi et al. [25] (the second case), Kleinschmidt et al. [28] (the first case), Dehdashti et al. [13] Tachikawa et al. [54], Sabatino et al. [49] and Pandey et al. [37], we agree with cautious observation. These POAAs can be closely observed by regular ophthalmologic examination and angiographic follow up. Surgical attack or endovascular intervention should be indicated without hesitation for any documented enlargement or mass effect of the aneurysm [13] to obviate further visual impairment and aneurismal rupture.

Conclusion

POAAs are a rare entity arising from intracranial, intracanalicular and intraorbital segments or terminal branches of OA. Contrary to aneurysms of ophthalmic segment of the carotid artery which have a marked female predominance, POAAs appear in more male patients. These aneurysms are heterogeneous in nature as other aneurysms, possible mechanisms including arterial developmental abnormity, haemodynamic stress, trauma and atherosclerosis. Ophthalmic stem and lacrimal POAAs are vision threatening, while intracranial segment and anterior ethmoidal POAAs can rupture and cause subarachnoid or intraparenchymal haemorrhage. Detecting POAAs in suspected cases with DSA and early surgical management in symptomatic cases are essential for vision preservation, and preventing and treating aneurismal rupture. Neck clipping and preservation of the artery patent is the optimal surgical choice, and endovascular approach is less invasive without direct optic nerve disturbance. Asymptomatic small unruptured POAAs can be closely observed.

Acknowledgement The authors thank the libraries of Nanjing University and Jinling Hospital for retrieving the literature.

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Comments

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In this issue of Neurosurgical Review, Qiao et al. present a comprehensive review of "peripheral ophthalmic artery aneurysms", which are aneurysms of the ophthalmic artery proper. For a number of anatomical sites like the posterior communicating artery, the anterior-inferior cerebellar artery or the posterior-inferior cerebellar artery, the semantics conventionally used may cause some confusion as to the exact origin of the aneurysm. For instance, "carotid-ophthalmic" aneurysms, which are relatively common, arise from the internal carotid artery at or around the origin of the ophthalmic artery. Indeed, for all these arterial sites, aneurysms which arise past the ostium and spare the parent major artery, therefore called aneurysms proper, are very uncommon.

In this review of peripheral ophthalmic artery aneurysms (POAAs), Qiao et al. identify four distinct segments of the ophthalmic artery, the intracranial, intracanalicular, intraorbital and terminal segments. For each of these segments, the authors provide a comprehensive review of the reported literature, including the various clinical manifestations, a reasonable discussion of the pathophysiology, and the diagnostic workup.

The treatment of these lesions is also discussed in detail in this review. Surgical interruption of the ophthalmic artery at its origin is a very reasonable option for proximal lesions since an abundant anastomotic arterial network exists between the ophthalmic artery and external carotid artery branches. Carotid sacrifice should no longer be considered lightly, in the face of the risks of acute or delayed ischemia, distal embolization, or optic nerve injury. Although new endovascular techniques may allow obliterating the ophthalmic artery with relative ease, one must remain cautious with the choice of embolic agent used, if visual function is to be spared.

Overall, the authors present an interesting and relatively comprehensive review of this rare group of aneurysms.

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The authors present an interesting and rare cause of vision loss and SAH. Qiao et al. have compiled cases of POAA from intracranial, intracanalicular and intraorbital locations. In compiling these case reports, the authors have given us insight into the potential natural history, presentation and aetiology of POAA. Qiao et al. reiterate the importance of conventional digital subtraction angiography and three-dimensional rotation angiography in the diagnosis of rare causes of SAH. The rarity of these lesions makes it difficult to compile a large patient series, but the authors have done a good job of compiling reported cases and by doing so providing insights into these rare vascular lesions.