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Anatomy of the Ophthalmic Artery's Current Surgical and Therapeutic Applications: A Review

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ABSTRACT

Numerous anatomical and surgical textbooks and studies have extensively investigated and reviewed the ocular artery's anatomy. Its intracranial and extracranial courses, branching, significance for vision, and interactions with diverse intracranial diseases are all interesting points. Reappraising its anatomy from a clinical standpoint is necessary due to advances in our understanding of the pathophysiology of some diseases, such as the development of aneurysms, central retinal artery occlusion, and retinoblastoma, as well as the development of new therapeutic modalities, such as superselective catheterization, intra-arterial fibrinolysis, and intra-arterial chemotherapy. This review's objective is to look at the clinical anatomy of the ocular artery and compare it to fresh diagnostic and therapeutic uses.

Introduction

The first intracranial branch of the internal carotid artery is called the ophthalmic artery (OA) (ICA). It appears shortly after the ICA leaves the cavernous sinus, travels via the optic canal and into the orbit after a brief intracranial trip. The eyeball and periophthalmic tissues become vascularized there as it ramifies in a complicated way. The central retinal artery (CRA), which vascularizes the retina and is the most important branch of the OA [1], is crucial for vision.

OA is the fifth branch of the ICA and is a member of its sixth segment, according to Bouthillier's classification scheme. For clinical purposes, the internal carotid artery (ICA) is divided into its first or cervical segment, second or petrous segment, which gives origin to the caroticotympanic and vidian artery, third or lacerum segment, fourth or cavernous segment, which provides the meningohypophyseal and inferolateral trunk, fifth or clinoid segment, sixth or ophthalmic segment, which gives origin to the internal carotid artery and superior

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Understanding the pathogenesis, diagnostic process, and therapy methods for its many illnesses requires knowledge of the intricate anatomy of OA. Surgery for the therapy of OA aneurysms is defined by the intracranial and intracanalicular course [3,4]. The clinical picture of anterior ischemic neuropathy is explained by microvascularization of the optic nerve (ON) head from OA [5]. For the treatment of central retinal artery occlusion (CRAO) or retinoblastoma chemoembolization, OA origin indicates the right method for selective catheterization of OA or superselective catheterization of CRA [6]. The goal of this review is to examine the anatomy of OA with a focus on its clinical uses.

Material and Methods

For research on OA anatomy, an electronic bibliographic search was carried out in Medline Embase, CINAHL, and Cochrane Library. There were other terms used, including "central retinal artery," "central retinal artery blockage," "ophthalmic artery," "ophthalmic artery aneurysm," and "retinoblastoma chemoembolization." The appropriate results were hand-searched and chosen. Furthermore, relevant papers were hand-searched in the literature of the chosen articles. For this analysis, only English-language articles were used.

Ophthalmic Artery Origin

The first branch of the ICA to emerge from the cavernous sinus and reach the cranial cavity is the OA. Between 0.7 and 1.8 mm are used to describe its diameter [4, 7-9]. According to Hayreh and Dass [4], it emerges above the dura in 83.6% of cases and takes an intradural course, in 6.6% of cases, it does so slightly above the dura, and in 10% of cases, it does so below the dura and takes a wholly or partially extra dural course. Similar findings by other studies [7, 10, 11] have been published. In 40% of cases, the superomedial wall of the ICA is where OA begins, followed by the anteromedial wall in 51% of cases, the medial wall in 6%, and the superior wall in just 3% of cases [4]. Other researches have discovered different percentages, therefore those figures are not generally agreed upon [7,10-11]. A brief study of its embryological development is required to comprehend OA variants. Three arteries, the dorsal OA, ventral OA, and middle meningeal artery, supply the eyeball and its contents in the early embryo (MMA). Through a formed and clinically significant anastomosis between those two systems or as numerous little anastomoses, the connection between MMA and OA endures even after MMA is translocated as an External Carotid Artery (ECA) branch in adult life.

In their examination of 1655 magnetic resonance angiographies in a Japanese population, Uchino et al. [12] discovered that 1.45% of instances of OA and 0.42% of cases of persistent primitive dorsal OA

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originated from MMA, respectively. The former can be explained by the dorsal OA not regressing, and the latter by the dorsal OA and ventral OA both regressing with the anastomosis between the OA stem and MMA improving.

Rarely, the internal maxillary artery (IMA) and the MMA anastomosis may never form or may regress, causing the MMA to originate from the OA [13]. These two variations have a big clinical effect. While OA originating from MMA can cause blindness in the event of accidental or surgical traumatism, persistent primitive dorsal OA hampers dissection for OA aneurysm because it originates intracavernously [10, 11].

According to Picard et al. [12], Hassler et al. [14], Islak et al. [15], Hannequin et al. [16], and Li et al. [17], OA can have a variety of origins from the anterior cerebral artery. This variant is consistent with the persistence of ventral OA, and instead of what is seen below, its main clinical importance is the abnormal course of OA above ON. Rare reports of double OA caused by the persistence of both ventral and dorsal primordial OA exist [18–21]. OA origin from the basilar artery has been recorded twice, but there is no adequate explanation for this severe variance [22].

Course

Intracranial Course and Ophthalmic Aneurysm

OA has a brief intracranial journey after its origin before penetrating the dura and entering the optic canal. Although tiny (0.5-9.5 mm according to Hayreh and Dass [4]), this distance is crucial for surgical intervention since OA aneurysms occur there. The path of OA is not linear between its origin and the optic canal since it makes one or two angles.

Neurologic symptoms from aneurysms of the OA include headaches and a loss of vision acuity. Subarachnoid bleeding, which could be lethal, is how an OA aneurysm ruptures.

Because the hemodynamic changes brought on by the aneurysm are apparent, some researchers think it is both possible and useful to diagnose OA aneurysms using orbital ultrasound [23].

An OA aneurysm may be treated with surgical ligation or embolization. Due to obstruction from the ON, clinoid process, and dural edge of the optic canal, surgical access to the area is challenging. Because to the ophthalmic artery's closure, the manipulation of the optic nerve, or damage to the small OA blood vessels and retinal ischemia brought on by temporary occlusions, visual loss is a serious and frequently avoidable side effect of treatments for OA aneurysms [24].***Intracanalicular Course.***

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Following its intracranial trajectory, OA travels beneath the posterior margin of the falciform ligament, pierces the ON's dura mater, typically inferiorly, and enters the optic canal laterally and next to the ON. OA intracanalicular aneurysms are quite uncommon, and there are few descriptions of them in the literature [8, 38]. The clinical image is perhaps more intense since they are under more pressure at ON.

Intraorbital Course and Branches.

The OA intraorbital route is divided into three sections. OA exits the optic canal inferiorly, laterally, and parallel to ON, running beside and parallel to it (1st part). Then, it travels medially to the ON, crossing above (83%) or below (17%). (2nd part). It is finally divided into its branches and medially to the ON (3rd part). The anterior and posterior ethmoidal arteries, palpebral arteries, supraorbital artery and its terminal branches, dorsal nasal artery, and frontal artery are all branches of the OA. When OA crosses over ON, the first branch is typically a common trunk for the MPCA and CRA, the second branch is LPCA, the third branch is LA, the fourth branch is a common trunk for the superior rectus and levator, the fifth branch is the posterior ethmoidal and supraorbital, the sixth branch is another MPCA, the seventh and eighth branches are muscular branches, the ninth branch is the anterior ethmoidal, the tenth branch is the inferior or media

Intra-arterial chemotherapy for retinoblastoma, a tumor that affects 1 in 50.000 children and can cause visual loss, metastases, and death if left untreated, has been made possible through OA targeted embolization. Systemic chemotherapy and radiotherapy are common treatments that have favorable benefits but also have negative side effects such neutropenia, hearing loss, metachronous new tumors, and eye enucleation [25]. The safety profile is satisfactory, displaying only minor and localized side effects such a hematoma at the site of the artery puncture in the groin, avascular retinopathy, and eye irritation [26]. Embolization or surgical ligation are the preferred treatments. Treatment does not cause further vision loss because they are typically located distally [18, 27].

Branches of Ophthalmic Artery

Central Retinal Artery.

The foundation of vision is CRA. In 77.5% of instances, it is the initial OA branch. It develops from the first portion of intraorbital OA in 22.1% of cases, the second part in 58.7% of cases, and the third part in 18.3% of cases. It occurs independently in 37.5% of cases, in a common trunk with MPCA, with LPCA in 11.5% of cases, and in a common trunk with MPCA and LPCA in 1.9% of cases. Following a difficult path, it penetrates the ON dura, typically in its lower and medial section, and reaches the retina.

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Experiments on monkeys have shown that ischemia of the retina results in its necrosis after around 4 hours [28,29]. In a clinical environment, inadequate CRA occlusion or the emergence of a collateral intrachoroid network may prolong retinal survival time [30]. The cilioretinal artery, an arterial branch that arises from the LPCA or MPCA and supplies part of the retina, is an anatomical variant that is advantageous in CRAO. In 6-32% of people, cilioretinal arteries are present, and in 14–18% of those, they are bilateral. Typically, they provide the retina's temporal half [31]. Additionally, cilioretinal arteries have the uncommon ability to supply the entire retina [33] and to form an anastomotic network with typical CRA branches [32].

Clinical history and fluorescein fundus angiography are used to diagnose CRAO. Emboli are visible, and sometimes their nature can be inferred from their angiographic appearance. Small, refractile, yellow plaques indicate cholesterol emboli, a single white plaque indicates calcific emboli, and small, pale bodies are indicative of fibrinoplatelet emboli [34]. There is no conclusive cure for CRAO. Sublingual isosorbide dinitrate, systemic pentoxifylline, or carbogen hyperbaric oxygen inhalation are examples of common treatment techniques.

Posterior Ciliary Arteries.

The OA that supplies the choroid has branches called posterior ciliary arteries. They are classified as either medial or lateral depending on where they reside. They range in number from 1 to 5, but are typically 2 or 3 (80% of the time). In 3% of cases, there is just one posterior ciliary artery (always the medial), in 39%, in 48%, in 8%, and in 2%, there are two, three, four, and five. There are inconsistent reports of superior or inferior posterior ciliary arteries [10, 36].

The function of the posterior ciliary arteries is essential for vision. The choroid and outer layers of the retina receive direct nutrition from them. Additionally, they take part in the cilioretinal arteries' role in supplying the inner retina layers, and they occasionally anastomose with the CRA via the Zinn circle [35]. Additionally, they take part in ON hematosis at the lamina cribrosa and collaborate with the CRA and OA branches in the prelaminar and retrolaminar regions, respectively. Ciliary circulation dysfunction has a close relationship to anterior ischemic optic neuropathy [36].

Lacrimal Artery.

The LA nourishes the periorbital and nearby muscular and lacrimal branches. In terms of embryology, it comes from MMA. There is no connection or only weak anastomotic branches between LA and OA since this prototype continues in about 28% of instances during adulthood. The lateral palpebral artery, a short recurrent meningeal branch, a muscle branch, and the glandular artery (for the lacrimal gland) are the LA branches [6, 18]. The LA has a diameter of around 0.7 mm [11].

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Ophthalmic Artery Anastomoses

Rich anastomotic networks in OA function protectively in the event of blockage. In 90% of patients of acute proximal OA occlusion without systemic vascular dysfunction, vision is preserved [10]. There are two types of anastomotic networks: deep and superficial.

Due to the risk of blindness in embolization instances, communication between the ECA and OA systems is clinically important. In situations of epistaxis with ICA obstruction, reverse embolization through ECA has been used, or chemotherapeutic administration through MMA in cases with inaccessible OA [37,38].

CONCLUSIONS

The anatomy of OA is intricate. It presents a surgical and anatomical challenge due to its dual intracranial and extracranial route, tiny size, closeness to numerous major anatomical elements, particularly ON, and critical role in vision. In particular for conditions like retinoblastoma and CRAO, advancements in neurosurgery, ophthalmology, and interventional radiology have boosted diagnostic and therapy options. Epistaxis and intravascular lesions are also treated with OA. For these reasons, it is essential to have a thorough understanding of its anatomy from a clinical standpoint in order to protect the patients' interests.

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