Intraorbital Arteriovenous Malformation Treated by Transcatheter Embolization; a Case Report

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Purpose: To report the clinical and radiological findings and management of a patient with intraorbital arteriovenous malformation (AVM) treated by transcatheter embolization of the feeding artery.

Case Report: A 15-year-old female patient was referred with a one year history of left eye proptosis without prior trauma. Orbital CT scan and MRI demonstrated a large intracanal mass lesion extending to the extraconal space in the inferior orbit; angiograms revealed an intraorbital AVM. Superselective catheterization of the feeding artery and embolization with absorbable gelatin particles (Gelfoam) and non-absorbable polyvinyl alcohol particles were performed in two separate sessions followed by surgical debulking. Signs and symptoms were diminished after 14 months.

Conclusion: Intraorbital AVMs can be treated by embolization of the feeding artery followed by surgical removal of the AVM nidus when the lesion is accessible.

INTRODUCTION
Arteriovenous malformations (AVMs) are progressively enlarging communications between arteries and veins that bypass normal capillary beds. In contrast to arteriovenous fistula, AVMs are congenital lesions with multiple large feeding arteries, a central nidus, and numerous dilated draining veins.1-3

Management of intraorbital AVMs may be difficult due to the risk of hemorrhage, vascular occlusion during treatment, and collateral damage to surrounding organs.2 We present the clinical and radiological findings together with outcomes of management by transcatheter embolization in a patient with an intraorbital AVM.

CASE REPORT
A 15-year-old female patient presented with pain, redness and congestive symptoms together with protrusion and upward displacement of her left eye from one year ago. She had no history of orbital trauma or family history of vascular disorders. Visual acuity was 20/20 and 20/60 in right and left eyes, respectively. Mild relative afferent pupillary defect (+1) was noted in the left eye. Exophthalmometric readings were 20 mm in the right and 33 mm in the left eye. Ocular motility was normal in the right eye and limited, especially in vertical gazes, in the left eye. No bruits were noted on auscultation and the proptosis was not accentuated by Valsalva maneuver. Ophthalmologic examination

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of the affected eye revealed marked dilatation of conjunctival vessels especially in the inferior fornices, chemosis, and proptosis with upward globe displacement (Fig. 1). Ophthalmoscopy showed mild disc hyperemia in the left eye and was otherwise unremarkable. Neurologic examination revealed no deficits.

Multiplanar MRI revealed a huge intra- and extraconal non-homogenous mass inferior to the optic nerve within the left orbit with variable enhancement and dilation of the superior and inferior ophthalmic veins (Fig. 2).

Angiography revealed a hypervascular mass with early venous filling. The lesion was mainly supplied by the maxillary branch of left external carotid artery (Fig. 3 A). The feeder artery distal to the middle meningeal artery was embolized with absorbable gelatin particles (Gelfoam) through superselective catheterization. One month after embolization, inferior orbitotomy was performed and the anterior accessible part of the looped vascular malformation was debulked. Histopathologic examination revealed abnormal dysplastic vessels. Because of incomplete response after 4 months, embolization was repeated with nonabsorbable polyvinyl alcohol particles which completely closed the main feeder vessel (Fig. 3 B).

Internal carotid angiography demonstrated another collateral supply to the AVM via the ophthalmic artery (Fig. 4). It was not embolized because of the risk of iatrogenic injury.

After six months (two months after the second embolization) visual acuity in the left eye improved to 20/30 and exophthalmometry was 26 mm (Fig. 5).
A Case of Intraorbital AVM; Etezad Razavi et al

DISCUSSION

AVMs are congenital lesions with numerous large feeding arteries, a central nidus and numerous dilated draining veins. AVMs may pulsate and have associated bruits on auscultation. They have a number of larger arterial feeding vessels on angiography and are histologically composed of abnormal dysplastic vessels.

Intraorbital AVMs are rare lesions. Wright reported only 3 cases of arteriovenous shunts among 627 patients with various orbital diseases. Many cases of reported intraorbital AVMs were high flow shunts located in the anterior orbit resulting in proptosis, arterialized conjunctival vessels, chemosis and orbital swelling, which were amenable to intravascular embolization and/or surgery. Intraorbital AVMs are most often related to an intracranial or maxillofacial arteriovenous shunts.

The sudden onset of symptoms in our case might have been due to a thrombotic process in the venous drainage of the AVM. A case of intraorbital AVM with minimal orbital congestion has been reported in whom paresis of the inferior division of the third nerve occurred without visual loss which was presumed to be due to thrombosis in the ophthalmic venous system.

Management of intraorbital AVMs may be difficult due to the risk of hemorrhage, vascular occlusion during treatment, and collateral damage to surrounding organs. Identification of all arterial feeders from both internal and external carotid systems is critical in developing a therapeutic plan. AVMs may be treated by surgical excision or embolization alone. However, in the hands of an experienced interventional neuroradiologist and surgical team, most AVMs may be treated by a combined approach of preoperative embolization followed by surgical excision of the vascular mass. The goal of therapy is closure of the low-resistance shunt.

Chakrabortty et al reported complete removal of the intraorbital contents after unsuccessful endovascular and surgical treatment of AVM. In our case, the AVM had two major feeder vessels, the larger from the external carotid system via the maxillary artery and the other from the internal carotid system and ophthalmic artery. Embolization of the maxillary feeder vessel was performed, but intervention through the ophthalmic artery was not possible without iatrogenic risks. Kim and Kosmorsky reported a case of arteriovenous communication between branches of the internal and external carotid systems and the ophthalmic veins located within the orbit. Embolization therapy of the lesion resulted in a branch retinal artery occlusion.

The therapeutic options are very limited in cases of AVM in the posterior orbit, particularly when the dura of the optic nerve is involved. In our case, because of the posterior extension of the lesion (Fig. 2), it was not possible to remove the nidus surgically and debulking was limited to the anterior vascular loops.
Significant decrease in symptoms, chemosis and exophthalmos in our patient after embolization of the feeding artery suggests that this method is useful for similar cases and may be considered as an appropriate treatment option for intraorbital AVMs.

REFERENCES