atrophic; microvascular decompression was equally applied to these patients if vessel compression was confirmed.

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Vision Loss due to Central Retinal Artery Occlusion Following Embolization in a Case of a Giant Juvenile Nasopharyngeal Angiofibroma

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Abstract: Juvenile nasopharyngeal angiofibroma (JNA) is a benign, vascular, and locally aggressive tumor that arises in the nasal cavity, extending into the nasopharynx and often in to the orbit. It may rarely present to the ophthalmologist with proptosis and optic neuropathy. Preoperative embolization of JNA is done before surgical resection. In this communication, the authors report a rare occurrence of ipsilateral central retinal artery occlusion (CRAO) following embolization with polyvinyl alcohol in a 13-year-old boy with right-sided JNA. Retrospective review of the angiograms pointed out to a suspicious communication between the external carotid artery and the ophthalmic vessels. Pre-embolization detailed study of the angiograms is necessary to avoid such devastating complications. Although rare, vision loss is a possible complication arising from embolization of nasopharyngeal and intracranial tumors, and all patients undergoing these procedures should be informed of the risk of visual loss because it has a lasting impact on the quality of life.

Key Words: Central retinal artery occlusion, embolization, juvenile nasopharyngeal angiofibroma, polyvinyl alcohol, proptosis

Juvenile nasopharyngeal angiofibroma (JNA) is an uncommon, benign, vascular, and locally aggressive tumor that predominantly occurs in adolescent boy. Although the presenting symptom is usually painless nasal obstruction or epistaxis, other symptoms may develop depending on the size and extent of the tumor mass.[1] Stern et al[2] in their review of JNA reported that proptosis was noted in 14% of cases of JNA, whereas decreased visual acuity and partial ophthalmoplegia occurred in 5% and 2% of the cases, respectively. Surgery is the mainstay of treatment of JNA. Preoperative embolization and newer surgical approaches result in less hemorrhage and complete resection of the tumor.[1] Although CRAO has been reported as a result of embolization of nasal vessels for intractable epistaxis[3,4], there have been only 3 previously documented cases of CRAO resulting in visual loss as a result of embolization for a nasopharyngeal angiofibroma.[5–7]

CLINICAL REPORT

A 13-year-old boy presented to our clinic with a history of outward protrusion of the right eye and diplopia since 2 months. He also had a history of repeated right-sided nasal bleeds since 1 year. His other complaints included nasal stuffiness and snoring. His history was significant as he had undergone a transmaxillary excision of a nasal mass through a lateral rhinotomy, 10 months before presentation. There was no history of toothache and the patient's dental history was noncontributory. On examination, his visual acuity was 6/9, N6 OD and 6/6, N6 OS. Color vision was normal in both eyes, when recorded with Ishihara pseudoisochromatic plates. Grade I RAPD was present in the right eye. Right-sided axial proptosis was noted in the right eye with minimal upward dystopia (Fig. 1A). Hertel exophthalmometry measurements were 28 mm in the right eye and 18 mm in the left eye. Vertical diplopia present in all gazes except downgaze suggestive of a mechanical compression inferiorly. On dilated fundus examination, there was disc hyperemia with elevated margins seen suggestive of compressive optic neuropathy in the right eye.

No visual field defects were seen on 30-2 automated visual fields. A computed tomography (CT) scan showed a large

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Received March 25, 2015.
Accepted for publication May 3, 2015.

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The authors report no conflicts of interest.

ISSN: 1049-2275
DOI: 10.1097/SCS.0000000000001936

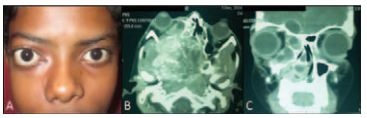

FIGURE 1. A, External photograph depicting the right-sided proptosis. Note the external scar from the previous surgery. B and C, CT scan depicting the extent and aggressive nature of the tumor with its epicenter in the postoperative bed in the right pterygopalatine fossa and the right sphenopalatine foramen.

heterodense, destructive, soft tissue lesion with its epicenter in the postoperative bed in the right pterygopalatine fossa and the right sphenopalatine foramen (Fig. 1B-C). The mass measuring $47 \times 42 \times 54$ mm had completely occupied the entire right nasal cavity with destruction of the medial wall of the orbit; the mass was compressing the globe and causing proptosis. Furthermore, there was extension into the right maxillary antrum crossing the midline and displacing the nasal septum. Axial cuts showed extension of the tumor mass into the nasopharynx. An otorhinolaryngology consult was sought and correlating the clinical and imaging findings of previous histopathologic reports, a diagnosis of recurrent JNA was made. The plan was to embolize the tumor using polyvinyl alcohol (PVA) particles to reduce the size of the tumor, which would also relieve the mechanical compression on the optic nerve before excision. Subsequently, digital subtraction angiography and selective endovascular embolization of the right internal maxillary artery with 150 to 250 mm PVA particles was carried out. Following the procedure, the patient complained of loss of vision in the right eye and vision was recorded as no perception of light. On fundus examination, a hyperemic disc with ischemic areas around the optic disc and a cherry red spot were found in the right eye. Therefore, a diagnosis of CRAO was made (Fig. 2). Ocular massage was initiated and continued for 15 minutes and paracentesis was also performed under topical anesthesia. He also received oral acetazolamide 250 mg immediately, which was continued BD for 2 days. The vision recorded on the next day was perception of light with inaccurate projection of rays. Twelve days following the initial embolization, the patient underwent excision of the residual mass. The vision, however, did not improve. Retrospective study of the angiograms showed the presence of a choroidal blush when then external carotid angiogram was performed suggestive of a communication between the external carotid and the ophthalmic artery, which was possibly overlooked before the embolization (Fig. 3). This communication could have led to the flow of PVA into the ophthalmic vasculature leading to CRAO.

DISCUSSION

In a retrospective analysis of 167 cranial base meningiomas that were embolized with PVA, Rosen et al[8] found a 1.8% risk of retinal artery occlusion despite superselective angiography. Casasco et al[7] reported a case of a 18-year-old boy with a left-sided JNA where the technique followed was direct intratumoral injection of permanent liquid polymerizing agent that causes embolization. The mixture injected was a combination of Histoacryl, Lipiodol, and tantalum

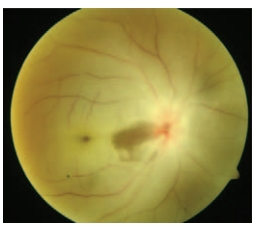

FIGURE 2. Fundus photograph showing the ischemic retina, attenuated vessels, and the area supplied by the cilioretinal artery demarcated separately.

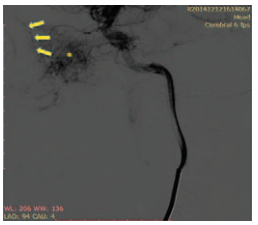

FIGURE 3. Angiogram of the external carotid vascular system showing the vascular angiofibroma (asterisk mark); note the appearance of a carotid blush indicating the presence of a communication between the external carotid vasculature and the ophthalmic artery.

powder. In their case, a small amount of Histoacryl entered the ophthalmic artery, resulting in an acute loss of vision in the left eye.

Ramezani et al[6] reported a case of a 23-year-old man with right-sided JNA who developed CRAO following preoperative embolization with PVA. They reported, however, that a retrospective review of the angiograms revealed the presence of a suspicious collateral artery between the external carotid artery and ophthalmic vessels on the left side, which had not been noticed before embolization. They suggested that the embolus passed mostly via this collateral artery to the left central retinal artery whereas tumor embolization was being carried out through the left-side arteries.[6]

Onerci et al reported a case of a child with JNA who developed CRAO following preoperative embolization. In their case, however, they could not demonstrate any responsible communicating artery and therefore assumed the existence of a branch of the internal maxillary artery supplying the intraorbital contents and the retina in their case.[5]

Considering the long-standing duration of tumor, its vascular nature and the potential for angiogenesis in JNA,[9] it is prudent to expect the presence of collateral vessels in such cases. The different mechanisms in which PVA embolization can cause CRAO can be varied: the presence of congenital variations in vasculature, overforceful injection, which can result in a reflux into the internal carotid system,[10] or collaterals, which may arise because of the tumor's aggressive and vascular nature.[5]

Our case is rare and unusual but potentially avoidable. We, therefore, agree with the recommendations of Ramezani et al[6] that careful evaluation of angiograms for detection of any abnormal collateral vessels or any vascular anomaly before embolization is extremely important. Furthermore, we believe that vision loss is a possible complication arising from embolization of nasopharyngeal and intracranial tumors and all patients undergoing these procedures should be informed of the risk of visual loss because it has long-term consequences and impact on the quality of life. Ophthalmologists and ENT specialists, alike should be sensitive to the possibility of this uncommon rare but devastating complication occurring after PVA embolization for JNA.

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