KJO
pISSN: 1011-8942 eISSN: 2092-9382

Korean J Ophthalmol 2013;27(6):470-473
http://dx.doi.org/10.3341/kjo.2013.27.6.470

Case Report

Ophthalmic Artery Aneurysm: Potential Culprit of Central Retinal Artery Occlusion

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Central retinal artery occlusion (CRAO) is one of the most devastating ophthalmic emergencies, causing acute painless visual loss in the affected eye. We describe the first case of acute non-arteritic CRAO associated with peripheral ophthalmic artery aneurysm and its clinical course after intra-arterial thrombolysis therapy. This case suggests that ophthalmic artery aneurysm can be the cause of CRAO and should be included in the differential diagnosis of CRAO.

Key Words: Ophthalmic artery aneurysm, Retinal artery occlusion

Central retinal artery occlusion (CRAO) is one of the most devastating ophthalmic emergencies, causing acute painless visual loss in the affected eye. Its incidence is known to be 0.85 per 100,000 per year [1]. Numerous systemic diseases such as carotid atherosclerosis, cardiac diseases, coagulopathies, tumors, and systemic vasculitis are notorious for close associations with CRAO. Small emboli from these diseases are believed to occlude the central retinal artery [2]. To the best of our knowledge, ophthalmic artery aneurysms have not been reported as the cause of CRAO. Here, we describe the first case of CRAO associated with ophthalmic artery aneurysm.

Received: May 27, 2012 *Accepted*: November 28, 2012

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Case Report

A 59-year-old woman presented to the emergency room with sudden visual loss in the right eye, which had started 15 hours prior. She denied past systemic or ophthalmic history. Her best corrected visual acuities were light perception OD and 20 / 22 OS. Her intraocular pressures were 13 mmHg OD and 12 mmHg OS by Goldmann applanation tonometry. Anterior segment examination was normal, and fundus examination demonstrated a pale retina with typical "cherry red spot" in the right eye. Her blood pressure, glucose, lipid, and coagulation profiles, as well as her erythrocyte sedimentation rate and C-reactive protein were within normal limits. Under the diagnosis of acute non-arteritic CRAO, she was urgently transferred to the neuro-intervention team for intra-arterial thrombolysis. Although there is some debate regarding the efficacy of intra-arterial thrombolysis for treatment of CRAO, her acute onset of symptoms within 15 hours without any systemic

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risk factors, and her insistence for further intervention encouraged consideration for thrombolysis therapy.

Transfemoral cerebral angiography demonstrated normal choroidal blush with a patent right ophthalmic artery which was stemming from a 3.7 × 4.5 × 5.2 mm sized aneurysm located at the origin of the ophthalmic artery (Fig. 1). External cerebral artery angiography revealed visualization of distal parts of the ophthalmic artery via the middle meningeal artery. Due to the fear of aneurysm rupture, and as the middle meningeal artery collateral communication was confirmed, the right middle meningeal artery was selected for target vessel instead of ophthalmic artery and 200,000 units of urokinase and 100 grams of tirofiban were hand injected. Procedures were finished without any clinical or angiographic complications. The next day, her visual acuity improved to finger count and the right fundus showed macular edema with improved vascularity. Fluorescein angiography showed normal arteriovenous transit time and full peripheral perfusion (Fig. 2A and 2B). Spectral-domain optical coherence tomography (SD-OCT; Spectralis OCT, Heidelberg Engineering, Germany) showed increased thickness and reflectivity of the inner retina (Fig. 2C). Goldmann visual field test demonstrated typical temporal islands, and electroretinography revealed decreased amplitude of the b-wave in the affected eye. To evaluate other possible systemic causes of the CRAO, ancillary studies including brain magnetic resonance imaging, transcranial Doppler, Holter monitoring, carotid Doppler, and echocardiography were performed and no further abnormalities were found aside from an asymptomatic patent foramen ovale. Definite treatment of the ophthalmic artery aneurysm was not recommended considering the risk of the procedure. After four months, her visual acuities were stationary (hand motion), but SD-OCT showed atrophy of the inner retina (Fig. 2D).

Discussion

Peripheral ophthalmic artery aneurysms, which are confined to aneurysms stemming from ophthalmic artery branches distal to the internal carotid artery (ICA), make up a very small portion of intracranial aneurysms, so that only 35 cases have been reported [3]. Among them, 15 cases presented with visual loss, which were mostly due to the compressing effect of the aneurysm itself on the optic nerve, and among those cases, only five had visual improvement after neurosurgery. However, compared to pre-

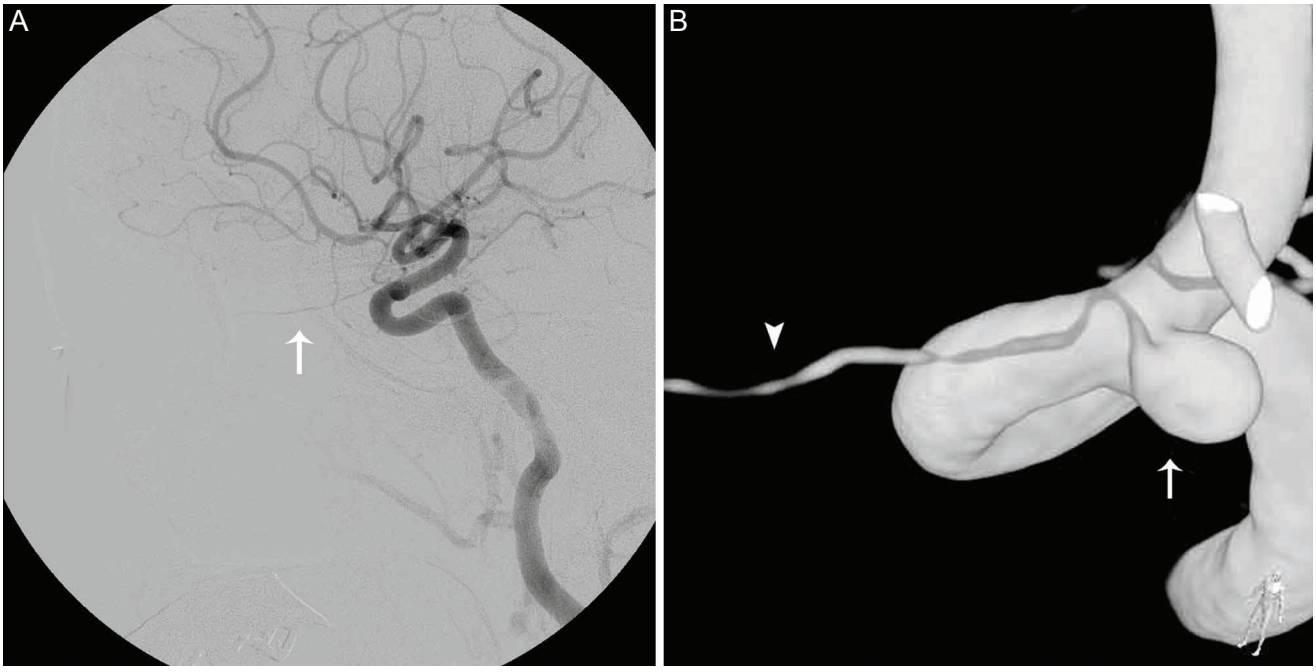

Fig. 1. Internal carotid artery angiogram and three dimensional reconstructed image of the ophthalmic artery. (A) The internal carotid artery angiogram shows patent ophthalmic artery (arrow). (B) Three dimensional reconstructed view of the ophthalmic artery (arrow head) identifies its origin from the 3.7 × 4.5 × 5.2 mm sized aneurysm (arrow).

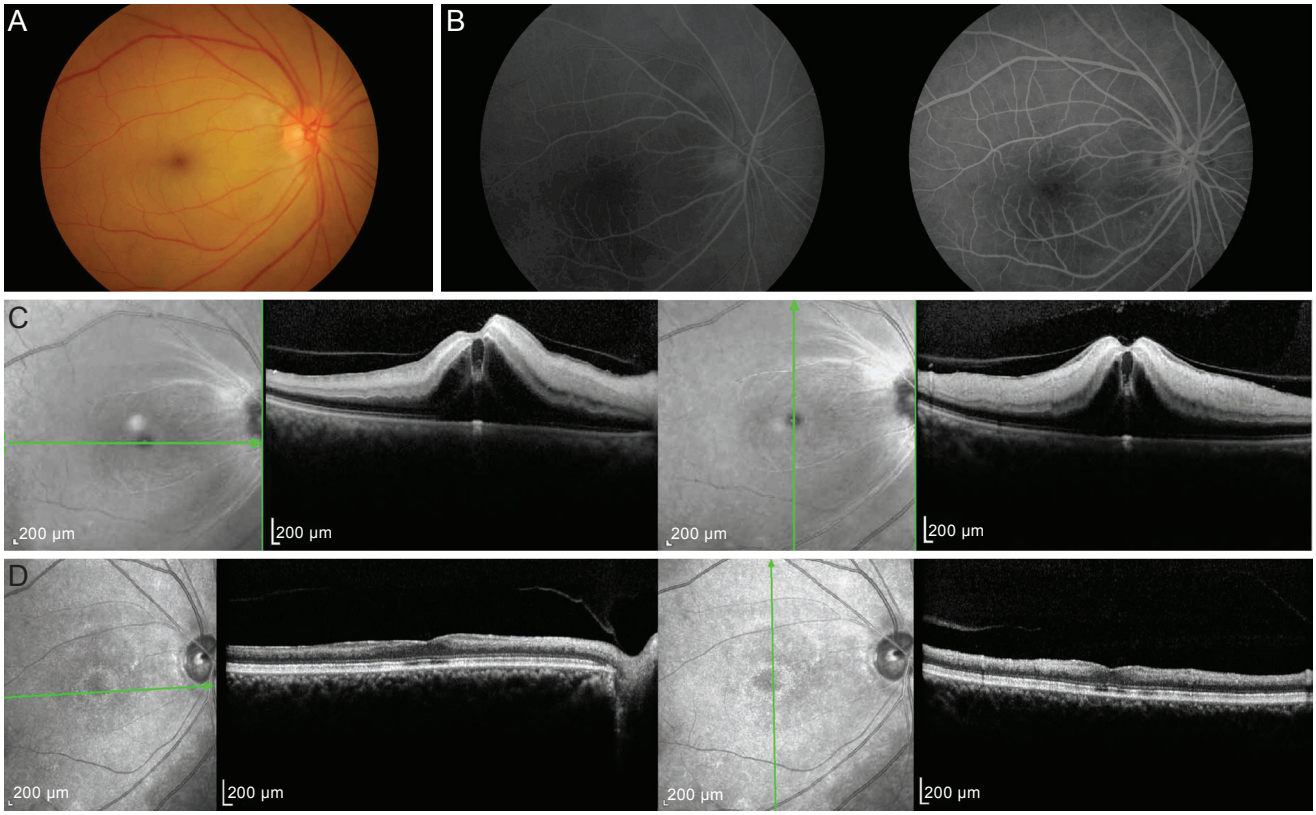

Fig. 2. Fundus photography, fluorescein angiography and spectral-domain optical coherence tomography (SD-OCT) after intra-arterial thrombolysis. (A) One day after thrombolysis, the retina was still edematous with a typical "cherry red spot" appearance but showed improved vascularity. (B) Retinal arterial filling showed normal arterio-venous transit time of 11 seconds (left 20 seconds, right 31 seconds). (C) SD-OCT showed increased thickness and reflectivity of the inner retina typical of central retinal artery occlusion. (D) After 4 months of treatment, the inner retina thickness had markedly decreased.

vious cases, our patient was unique in that her visual loss was acute in onset and her fundus findings were typical for CRAO, which made a compressing effect on optic nerve unlikely. The possibility of the aneurysm itself compressing the ophthalmic artery or the ICA causing visual loss could be excluded as the ophthalmic artery was shown to be patent during cerebral angiography.

Qureshi et al. [4] reported that embolization can occur from aneurysms even in the absence of angiographically demonstrated clots within the aneurymal sac. The prevalence of ischemic events secondary to embolization from unruptured intracranial aneurysms was shown to be 3.3%. CRAO caused by an embolism from an intracranial aneurysm has rarely been reported. Haritoglou et al. [5] reported a case of CRAO associated with an aneurysm of the ICA, and highlighted the importance of differentiating intracranial aneurysm as the cause of CRAO in young patients without risk factors for vascular occlusive disease. Since our patient had no known risk factors for CRAO except an ophthalmic artery aneurysm in the involved eye, emboli derived from the aneurysm might be the most probable explanation.

Opting to treat the ophthalmic artery aneurysm requires prudent consideration as an aneurysm carries the risk of spontaneous rupture. Since the aneurysm was small in size, and was asymptomatic, observation was deemed more beneficial to the patient rather than invasive surgical intervention [6]. Endovascular coil embolization may aggravate embolic risk with increased thrombogenicity, which was another reason to bypass treatment.

In conclusion, we describe the first case of CRAO associated with an ophthalmic artery aneurysm. Embolism from ophthalmic artery aneurysm can be a cause of CRAO and it is meaningful to include ophthalmic artery aneurysm in the differential diagnosis of CRAO without other systemic risk factors.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Acknowledgements

This study was partly supported by a grant from the National Research Foundation of Korea Grant 2012R1A-2A2A02012821.

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