

Circumscribed Ciliochoroidal Effusion Presenting as an Acute Angle Closure Attack

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Abstract

Purpose: To report a case of choroidal effusion probably caused by angiotensin receptor II blocker.

Case Report: A 52-year-old man with aplastic anemia and high blood pressure who developed unilateral acute angle closure glaucoma after receiving oral cyclosporine and angiotensin II receptor blocker (losartan). Ophthalmic examination revealed visual acuity of 20/30 in the left eye, mild mydriasis, iris bombe, no anterior chamber reaction, mild conjunctival hyperemia, and the intraocular pressure of 30 mmHg. After laser YAG iridotomy, funduscopy was performed showing a choroidal circumscribed lesion at the inferotemporal quadrant. Diagnostic tests ruled out any inflammatory or malignancy process, and the choroidal lesion spontaneously disappeared. After 20 months of follow-up, patient's ocular remained stable.

Conclusion: This is the third case of choroidal effusion associated with angiotensin II receptor blockers. Since idiosyncratic ciliochoroidal effusion is a diagnosis of exclusion, it is mandatory to rule out more frequent causes, such as inflammatory or malignant processes.

Keywords: Angiotensin II Receptor Blocker; Angle Closure Attack; Ciliochoroidal Effusion

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INTRODUCTION

Ciliochoroidal effusion is the accumulation of serous transudate in the suprachoroidal space producing choroidal elevation. Most common etiologies are inflammation or hypotony; however malignancy, drug reaction, infectious, and idiopathic forms have also been reported.^[1-3]

We describe a case of choroidal effusion presenting as an acute angle closure attack in a patient with aplastic

anemia treated with oral cyclosporine and angiotensin receptor blocker (losartan) and discuss about the diagnosis.

CASE REPORT

A 52-year-old male patient recently diagnosed with idiopathic aplastic anemia and high blood pressure was treated with cyclosporine and angiotensin II receptor blocker (ARB) (losartan), respectively. He presented with sudden loss of vision in the left eye (OS) and photopsia. In ophthalmic examination, best corrected visual acuity (BCVA) was 20/20 in the right eye (OD) and 20/32 in the left eye. Biomicroscopy showed a deep anterior chamber OD and iris bombe, no anterior chamber reaction, and mild conjunctival hyperemia OS; gonioscopy

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represented Shaffer III and Shaffer 0 in the right and left eyes, respectively. The ocular tension was 14 mmHg OD and 30 mmHg OS. After laser iridotomy, treatment with topical dexamethasone, 0.5% topical timolol, and topical tropicamide eye drops were initiated. Angle closure attack resolved. Funduscopy was unremarkable in the right eye but showed an amelanotic choroidal well-circumscribed dome-shaped lesion in inferotemporal quadrant of the left eye [Figure 1]. A short course of oral steroids was initiated, the patient was referred to a specialist to rule out any inflammatory process, lymphoma, melanoma, or metastasis. Retinography exhibited mass in the inferotemporal quadrant, and ocular fluorescein angiogram showed hyperfluorescent spots in the lesion area lasting until later phases; no-double circulation, no vasculitis, and no leopard spot were noticed. Ocular magnetic resonance imaging (MRI) showed choroidal lesion (10 mm × 2 mm) with hypercaptation to gadolinium [Figure 2]. Body scanner tomography was unremarkable. Two months later, all tests ruled out inflammatory process, systemic lymphoma, melanoma, or metastasis and ophthalmic exploration showed

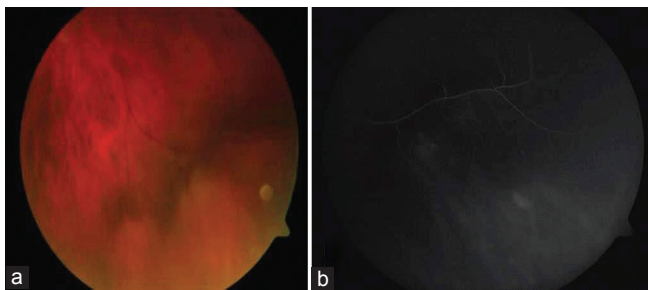


Figure 1. (a) Left eye retinography shows amelanotic choroidal well-circumscribed tumor in inferotemporal quadrant. (b) Left eye fluorescein angiography shows hyperfluorescence spot that maintains until late phase.

BCVA of 20/20 and gonioscopy of Shaffer III for both eyes. Funduscopy OS revealed choroidal detachment and choroidal lesion disappeared evincing only vortex vein in that area [Figure 3]. No lesion was seen in ocular MRI. Twenty months later, the patient's ocular condition remained stable.

DISCUSSION

Ciliochoroidal effusion is characterized by collection of fluid in the suprachoroidal space.^[1] The most frequent causes are secondary to inflammation or ocular hypotony but sometimes could be idiopathic, and its etiology is not totally understood. It is suggested that there may be a congenital scleral anomaly which induced reduced scleral permeability.^[1,2]

Ocular or orbital surgery was not reported in this case; inflammatory, infectious, and malignancy processes were ruled out with diagnostic tests. Therefore, an idiosyncratic drug reaction was suspected as etiology. It is known that angiotensin II has an effect as a vasoconstrictor and in angiogenesis of choroidal vessels and retinal and pigment epithelial membranes;^[4] thus, angiotensin II receptor blocker increases choroidal vasculature permeability that could trigger the clinical presentation in this case.^[2-4]

To our knowledge, there are only two cases reported in the literature of choroidal effusion secondary to ARB, both of them presented after an uncomplicated cataract surgery and one reported by Fromberg et al was associated with angle closure glaucoma at presentation.^[2,3]

Angle closure attack related to choroidal effusion has been frequently reported in patients with primary angle closure glaucoma or nanophthalmos (58%); however it is rare in open angle glaucoma.^[5,6]

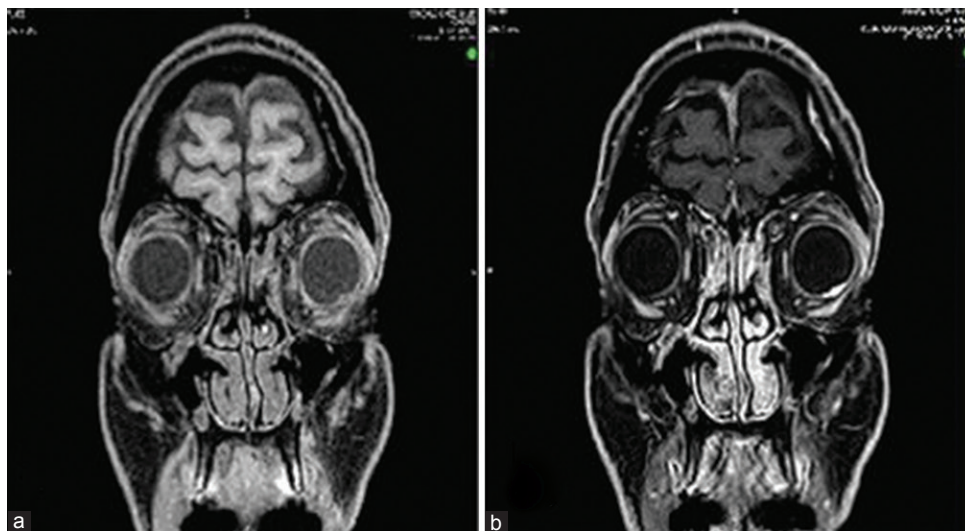


Figure 2. Orbital magnetic resonance imaging in the left eye shows a circumscribed lesion in suprachoroidal space (a). High hypercaptation of lesion under gadolinium contrast (b).

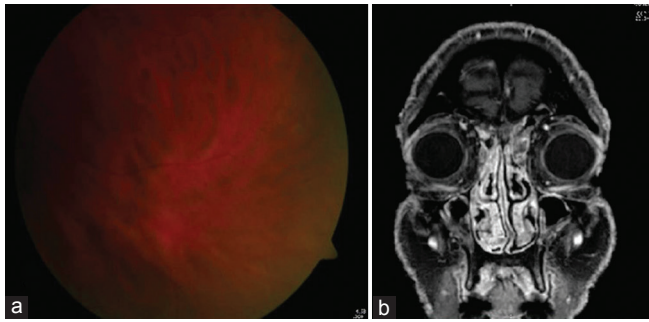


Figure 3. (a) Left eye retinography shows that lesion disappears. (b) Magnetic resonance imaging reveals no choroidal lesion.

There are three recognized factors in this case: first, the condition of aplastic anemia decreases blood viscosity; second, ARB (losartan) increases choroidal vascular permeability; and third, a patient with presumed inherent susceptibility. These factors could have induced accumulation of fluid in the suprachoroidal space, choroidal detachment with anterior displacement of the lens-iris diaphragm, and pupillary block.

The treatment of ciliochoroidal effusion is with sclerectomies/sclerotomies; systemic steroids have been used with low success.^[1-7] The present case had very good response to a short course of systemic steroids with full clinical resolution and stabilization after 20 months of follow-up.

In summary, ciliochoroidal effusion is a rare occurrence, and its presentation as an acute angle closure attack is even less uncommon. This clinical case is the third case reported on choroidal effusion presenting as an acute angle closure attack and the second one associated with an idiosyncratic reaction

to ARB (losartan). Exhaustive diagnostic examinations must be done according to each case. Idiosyncratic drug reaction is a possible etiology which must be kept in mind. Total clinical resolution could be achieved in these cases with a short course of systemic steroids without need to discontinue the drug.

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Conflicts of Interest

There are no conflicts of interest.

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