8.69 \times 3.62 \text{ mm}. \text{ She was treated with 95\% ethanol irrigation. For the ethanol irrigation procedure, we used the technique described by Behrouzi and Khodadoust.}^1 \text{ The procedure was performed under a retrobulbar block. A 25-gauge needle was used to enter the cyst 1.5 mm posterior to the limbus. Clear fluid (0.15 mL) was aspirated, and the same volume of 95\% ethanol was injected into the cyst and removed after 1 minute. The patient was treated with prednisolone acetate and atropine sulfate eyedrops postoperatively.}

The patient did well, with best-corrected visual acuity of 20/20 and intraocular pressure of 19 mm Hg at 2 months. However, the cyst recurred 6 months after the ethanol irrigation. The size was similar to the initial epithelial inclusion cyst. Therefore, she underwent a second treatment with ethanol.

We have followed the patient for 16 months after her re-treatment. Her visual acuity is 20/25 and her intraocular pressure is 21 mm Hg. No cyst is visible on slitlamp examination; however, B-scan ultrasonography shows a residual $1.70 \times 2.86 \text{ mm}$ cyst that has not grown based on findings from serial echography (Figure 2).

Comment. An epithelial inclusion cyst is a rare and serious complication of ocular penetration. Reported treatment options were reviewed by Behrouzi and Khodadoust.\(^1\) They include excision,\(^2,3\) endodiathermy,\(^4\) photocoagulation, aspiration,\(^3\) and the injection of various agents including trichloroacetic acid, iodine, carbonic acid, and ethanol.\(^1\) Behrouzi and Khodadoust reported a 94\% rate of clinical resolution of cysts with intralesional alcohol, but their follow-up was very short (mean follow-up, 3.85 months) and serial B-scan ultrasonography was not performed. Surgical excision is an option, although potential adverse effects include infection, bleeding, cataract formation, and complications from cyst rupture.

The large size and location of our patient’s cyst precluded surgical excision. We believe this case demonstrates that irrigation with 95\% ethanol can be a safe and effective procedure, even for very large cysts for which other therapies are not possible. To our knowledge, there has been no report following these patients for an extended period with serial clinical examinations and imaging. It has been 22 months since our patient’s initial treatment. She had 1 recurrence at 6 months. This was re-treated, and although there is a very small residual cyst visible only on ultrasonography, its size has remained stable for 16 months.

**Noncompaction Cardiomyopathy Manifesting as Retinal Artery Occlusion**

Noncompaction cardiomyopathy is a rare cardiomyopathy that affects both children and adults.\(^1\) It commonly manifests with heart failure, systemic embolism, and arrhythmia.\(^1,2\) We describe an adult patient with bilateral retinal embolism as the manifesting sign of noncompaction cardiomyopathy.

**Report of a Case.** A 62-year-old nonsmoking man had bilateral sudden painless loss of vision 13 hours prior...
to his visit. He had no ophthalmic history and reported no significant medical illness. Visual acuity was hand motions OD and counting fingers OS. A relative afferent pupillary defect was present in the right eye. Dilated funduscopic examination showed the presence of a cherry-red spot with generalized pale edematous retina in the right eye (Figure 1A) and evidence of retinal edema involving the upper hemisphere in the left eye (Figure 1B). Visual field testing revealed a total field loss of the right eye and hemi-inferior field defect in the left eye (Figure 1C and D). Ocular massage, breathing in a mixture of carbon dioxide and oxygen, and an intravenous bolus injection of acetazolamide, 500 mg, were instituted in the emergency department. He was admitted on that day for systemic workup to assess the primary cause.

His blood pressure was 131/70 mm Hg and his heart rate was 78 beats/min. Systemic examination findings were significant for a pansystolic murmur at the mitral region that radiated to the axilla, displacement of the apex beat to the sixth intercostal space of the anterior axillary line, and presence of thrills.

The results of full blood cell count, urea, electrolytes, blood glucose level, serum fasting cholesterol level, clotting factors, thrombophilia screening, and cardiac enzyme levels were within the normal range. Electrocardiography showed high uptake in the ST segment in V1. Transthoracic echocardiography revealed a dilated left ventricle of 60 mm with an ejection fraction of 60%. There was a concentric hypertrophy with prominent trabeculae seen on the left ventricular wall. The trabeculation thickness was more than twice the thickness of the underlying
ventricular wall (Figure 2A). Color flow imaging demonstrated blood flow within the deep recess between the trabeculations (Figure 2B). Severe mitral regurgitation due to grade 1 anterior mitral valve prolapse was also present. Findings on the carotid Doppler study were normal, and no signs of carotid stenosis or plaques were observed.

A provisional diagnosis of noncompaction cardiomyopathy was made. Cardiac magnetic resonance imaging was recommended to further evaluate the cardiomyopathy, but the patient declined. Long-term oral warfarin sodium treatment was commenced to reduce the risk of systemic embolization. At 1 month after the attack, his visual acuity remained similar in the right eye and improved to 20/200 OS.

Comment. The embryonic arrest of compaction of myocardial fibers seen in noncompaction cardiomyopathy is most frequently observed in the left ventricle.3 The cardiomyopathy is diagnosed by echocardiography or magnetic resonance imaging.

Echocardiography shows trabeculations and deep intertrabecular recesses. Blood flow can be observed within the deep intertrabecular recesses, and the flow is in continuity with the left ventricular cavity. Noncompaction cardiomyopathy is diagnosed echocardiographically when the ratio of trabeculations to the thickness of the underlying ventricular wall is more than 2.

Magnetic resonance imaging shows a 2-layered wall structure comprising a thin compacted epicardium and a thick noncompacted myocardium. Our patient’s echocardiograms are consistent with the diagnosis of noncompaction cardiomyopathy.

Strokes have been reported as a systemic thromboembolism that occurs in patients with noncompaction cardiomyopathy.4,5 The bilateral retinal artery occlusion seen in our patient is likely of a thromboembolic nature. We postulated that the microembolus observed in the left retinal arteriole originated from the heart. The noncompaction cardiomyopathy with relative blood stasis in the intertrabecular recess explains the most probable cause of this phenomenon.4 Thus, it is extremely important to highlight this rare cause of retinal artery occlusion that has resulted in devastating vision loss.

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