coagulation disorders (such as activated protein C resistance or factor V Leiden mutation), immobilization, pregnancy, smoking, medication with corticoids, family disposition, and COCs.[3] Underlying conditions that may cause CVST vary, and the etiology is unknown in such cases. Several studies have indicated that COCs and thrombophilia greatly increased the risk of CVST.[7] Combined estrogen-progestin oral contraceptives have been associated with an increased risk of venous thromboembolic events. This thrombotic risk was attributed to the estrogen content, which prompted the development of oral contraceptives containing less estrogen. Use of formulations containing lower-dose estrogen still confer about 2-fold to 4-fold increased risk of venous thromboembolic events compared with non-use.[8] As the most common causes of HH are infarctions, meningiomas, gliomas, and metastatic lesions, these must be kept in mind. However, only a few patients with HH presenting with neurosyphilis, migraine, demyelinating disease, arteriovenous malformation, or neurosarcoidosis have been reported.[9]

MRI and MRV are the best tools for both the diagnosis and the follow-up of CVST.[10] In our case, MRI found right acute occipital lobe infarction (see Fig. 2A-D). MRV showed thrombosis of the right transverse sinus and left internal jugular vein (see Fig. 3A, B).

Venous thrombosis has traditionally involved the superficial cerebral venous system,[11] as described in our case. To our knowledge, this is the first published case with HH due to CVST associated with COC usage.

Available treatment data from the literature offer the use of anticoagulation in patients with CVST.[12] Oral anticoagulation is recommended for 3 months in patients with idiopathic CVST and for 3 to 6 months if it is related to pregnancy or oral contraceptives. In patients with hereditary thrombophilia, it is used for 6 to 12 months or longer.[4] Anticoagulant therapy was started after the diagnosis of CVST in our patient and continued. Her symptoms improved rapidly, and the visual field defect was much improved in 1 week.

Conclusion

Homonymous hemianopia is an uncommon vision problem, and our case highlights the importance of imaging techniques to explain unexpected clinical findings. All females must be informed about the increased risk of venous thromboembolism during COC usage. The other possible diseases should be excluded with detailed clinical history, laboratory findings, and imaging techniques. The most important aspect of this case is the early diagnosis and treatment. Therefore, an accurate and timely diagnosis is important in such cases, and accurate techniques are required for appropriate treatment.

Gökçen Çoban, Aylin Karalezli, Bahriye Horasanlı, Nilüfer Yeşilırmak
Başkent University Faculty of Medicine, Konya, Turkey

Correspondence to:
Gökçen Çoban, MD: drgokcencoban@gmail.com

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Proton beam radiotherapy of progressive pediatric choroidal osteoma: First experience

Choroidal osteoma is a rare intraocular tumour predominantly appearing in healthy females in the second or third decade of life.[1] Usually, the tumour emerges unilaterally; only in 20% to 25% of cases is choroidal osteoma bilateral.[1] It is typically located in the juxtapapillary or peripapillary region.[1] Funduscopically, choroidal osteoma presents as a yellow-white to orange-red lesion.[1] Ultrasonography reveals an elevated choroidal lesion with high reflectivity and a typical acoustic shadowing.[1] Visual

prognosis is unpredictable, but 56% to 58% of patients end up with poor visual acuity (VA) at 10 years (20/200 or worse).[2,3] Complications occurring with choroidal osteoma resulting in VA deterioration include tumour growth, decalcification, and choroidal neovascularization (CNV). Despite its benign nature, choroidal osteoma may grow in about 51% of cases by 10 years.[2] Decalcification is seen in 46%, and CNV may develop in 31% by 10 years.[2] No standard therapy for choroidal osteoma has yet been established. The lack of such a standard especially affects the management of pediatric osteoma in young children.

Case Report

In April 2008, a 4-year-old female presented with a subretinal tumour of unknown origin in the left eye on the occasion of retinoscopy. At the first examination, the VA in both eyes was 20/20. An amelanotic prominent juxtapapillary tumour, nasal and inferior to the fovea, 2.5 disc diameters in dimension, and orange-red, was seen (Fig. 1A). On optical coherence tomography, no accompanying exudation was detectable. However, a subretinal tumour mass with a bulging elevation of the retina was visible. Ultrasonography found a hyperreflective lesion with an acoustic shadow consistent with a calcified tumour (Fig. 2). The patient's brother had died at age 13 years because of an extraosseous Ewing sarcoma (primitive neuroectodermal tumour [PNET]), a fact that prompted further work-up of the child to exclude choroidal metastasis of Ewing sarcoma. After PNET was excluded using whole-body magnetic resonance imaging, the diagnosis of a choroidal osteoma was made.[4] Sclerochoroidal calcification as a differential diagnosis could be ruled out, given

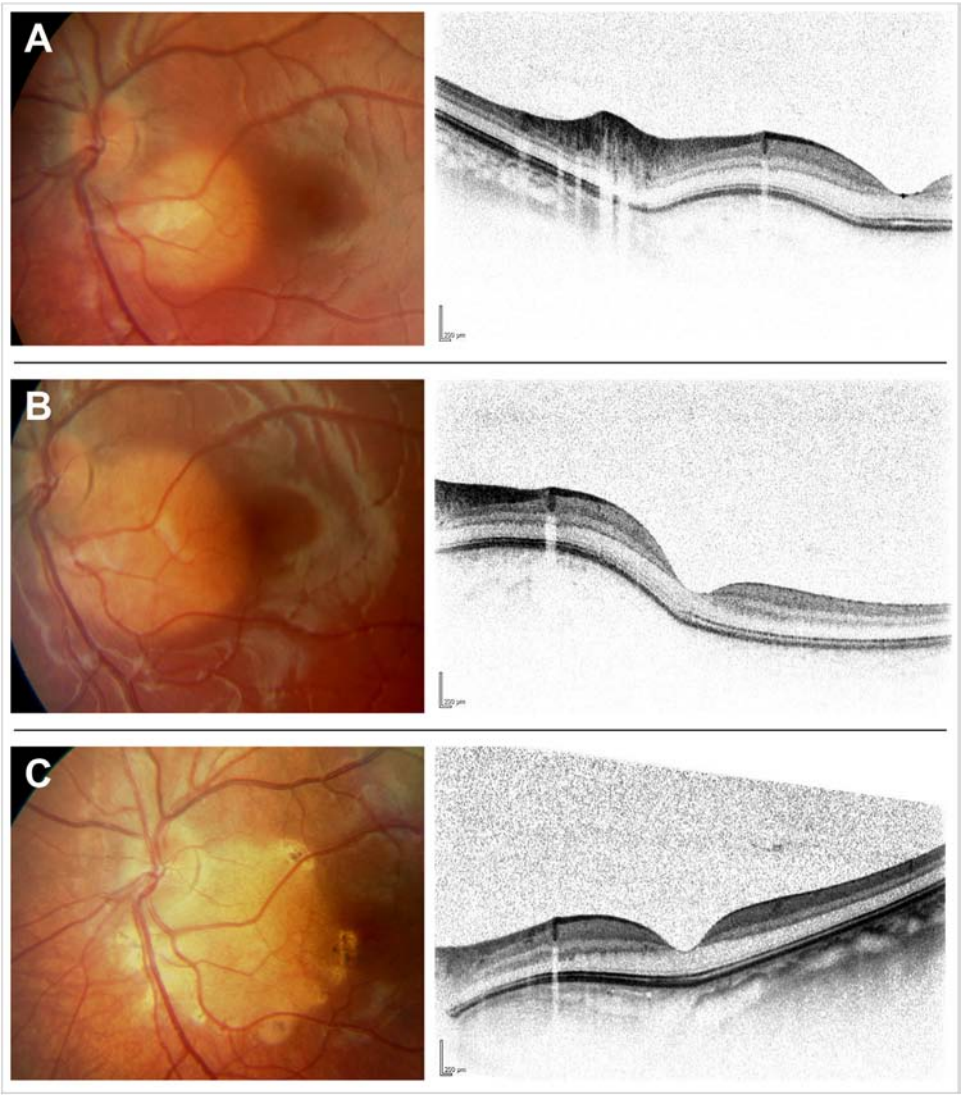

Fig. 1—Funduscopic photograph and optical coherence tomography scan of the choroidal tumour nasal and inferior to the optic disc in April 2008 (A). Six months later, in October 2008, the osteoma had progressed clearly (B). Nineteen months after proton beam radiotherapy, the tumour had regressed, showing discrete retinal pigment epithelium alterations at the edges. The foveal architecture was intact, with visual acuity of 20/22 (C).