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Choroidal calcification revealing a primary hyperparathyroidism: A case report.

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Abstract

Choroidal calcification is an uncommon benign lesion occurring in predominantly elderly. This condition is ordinarily idiopathic. However, it can be caused by phosphocalcic metabolism disorders.

We report the case of a 72-year-old woman followed-up in the ophthalmology department for proliferative diabetic retinopathy who was found to have a bilateral yellowish lesion in the fundus during a routine eye examination corresponding to a choroidal calcification. Metabolic analyses led to the diagnosis of primary hyperparathyroidism.

Choroidal calcifications warrant a systemic evaluation to identify a potential underlying disease.

Introduction

Choroidal calcification is an uncommon benign condition characterized by multifocal yellowish placoid lesions typically located in the temporal regions of the fundus, which can be unilateral or more often bilateral [1,2].

These calcifications are often discovered incidentally in older adults and can simulate intraocular tumors such as choroidal metastasis, choroidal osteoma, choroidal melanoma or lymphoma [3–5]. Choroidal calcifications are most often idiopathic. However, they can be associated with calcium-phosphorus metabolism disorders and tubular hypokalemic metabolic alkalosis syndromes [3,6–8].

We report a particular observation of a 72-year-old woman in whom choroidal calcifications led to the diagnosis of primary hyperparathyroidism.

Case presentation

We present the case of a 72-year-old female, non-smoker, with a medical history of hypertension treated with dual combination therapy (Valsartan and Amlodipine) and type 2

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diabetes mellitus diagnosed 20 years earlier and managed with Metformin and two times daily injections of basal insulin. The patient is followed-up in the ophthalmology department for proliferative diabetic retinopathy. She was effectively treated with panretinal laser photocoagulation and intravitreal injection of vascular endothelial growth factor inhibitors (Avastin). During last routine eye examination, she had a visual acuity of 5/10 in both eyes. Fundus showed a yellow plaque-like irregular lesion at the posterior pole of the right eye corresponding to a choroidal calcification (figure 1.A). Both right (figure 1.A) and left (figure 1.B) eyes display signs of diabetic macular edema.

Etiological investigation was carried out to screen for an underlying metabolic disorder. Biological evaluation showed normal renal function (creatinine clearance 65.8 ml/mn), normal serum sodium and potassium levels, normal arterial blood gas parameters, an HbA1C level of 7.8% and an elevated serum calcium level of 2.8 mmol/L with elevated parathormone level of 160 pg/ml and normal vitamin D level of 32 ng/ml. The diagnosis of primary hyperparathyroidism was thus confirmed. Since there are neither severe hypercalcemia nor bone or renal complications, surgical treatment was not recommended.

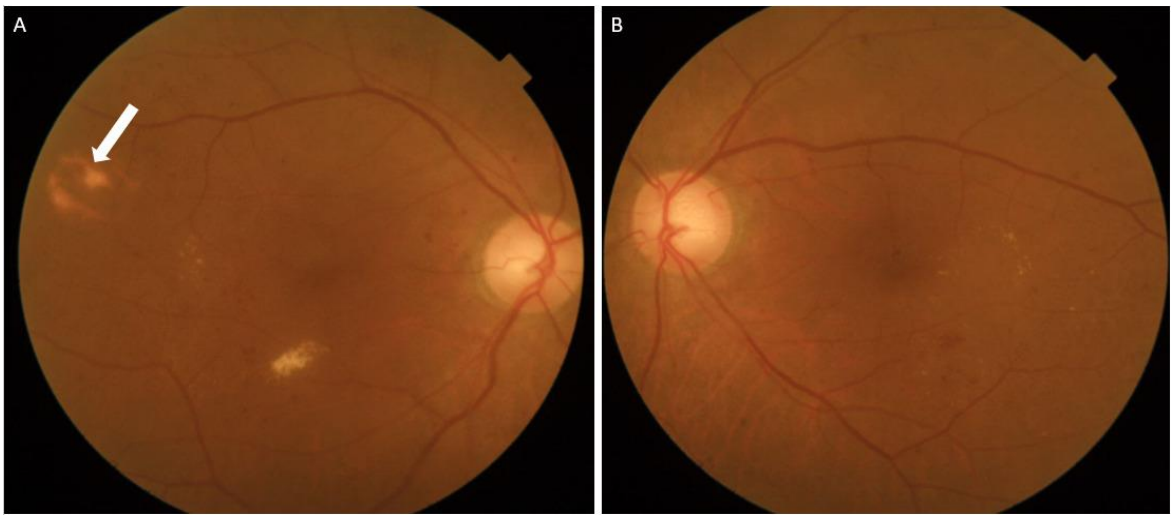

Figure 1: Color fundus photographs. The right eye (A) photograph shows at the posterior pole yellow plaque-like irregular lesion (white arrow) corresponding to choroidal calcification. Both right eye (A) and the left eye (B) display macular thickening, hard exudates within 500 µm of the center of the fovea, microaneurysms and intraretinal hemorrhages corresponding to bilateral diabetic macular edema.

Discussion

Classically, choroidal calcifications are asymptomatic, discovered as un incident finding in older adults [9]. They present as bilateral yellow lesions located in the superotemporal quadrant [10]. It was long believed that these lesions do not grow even through several years of monitoring [3]. However, Boutboul

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et al. reported a case of a patient with sclerochoroidal calcifications involving progressively the macular area suggesting growth of the calcifications after a 24-year follow-up [11].

The pathogenesis of SCC may be either dystrophic, idiopathic, or metastatic [12]. Dystrophic calcifications occur after chronic tissue necrosis or inflammation [4]. Idiopathic calcifications result from chronic oblique muscles movements near the temporal arcades. Metastatic calcification development is due to an abnormal calcium-phosphorus metabolism [4].

While most of choroidal calcifications are idiopathic, phosphocalcic metabolism disorders such as primary hyperparathyroidism, pseudohypoparathyroidism, vitamin D intoxication and chronic renal failure can be found in patients with choroidal calcifications [2]. Similarly, these lesions can also be detected in patients with tubular hypokalemic metabolic alkalosis syndromes such as Bartter and Gitelman syndromes [7].

The prevalence of choroidal calcifications in patients with primary hyperparathyroidism is unknown.

Data regarding the association between choroidal calcifications and hyperparathyroidism are limited. Shield et al. reported that among 33 patients with choroidal calcifications, nine had primary hyperparathyroidism and five had parathyroid adenoma [2]. To the best of our knowledge, choroidal calcifications were the first manifestation leading to a diagnosis of primary hyperparathyroidism in only one case report [13].

Choroidal calcifications remain a misdiagnosed condition, referred out as a choroidal tumor [14,15]. Epidemiologic characteristics and ophthalmological findings facilitate the differentiation and allow to rectify the diagnosis and prevent unnecessary intervention. In choroidal metastasis, the yellow lesion is found in the macula, exposing to a higher risk of visual loss [4]. Ultrasonography doesn't show calcifications. Choroidal melanoma presents as a large pigmented lesion. Choroidal osteoma is commonly diagnosed in young females and presents as an unilateral juxtapapillary lesion [16].

Generally, choroidal calcifications do not require therapeutic intervention. The treatment is conservative with the management of a possible underlying disease [12]. A regular follow-up with multimodal imaging is recommended by some authors [17]. Active treatment with anti-VEGF intravitreous injections, photodynamic therapy and argon laser photocoagulation is proposed in case of vision-threatening complications such as choroidal neovascularization [18].

Visual prognosis is generally good, as the lesions are often located away from the macula and foveal encroachment is rare. However, choroidal neovascularization and serous retinal detachment may rarely occur [3,19–22]. Unfortunately, the visual prognosis of our patient is threatened by the proliferative diabetic retinopathy.

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Conclusion

Choroidal calcifications are most commonly idiopathic and asymptomatic as they rarely cause visual loss. However, underlying systemic conditions should be screened, especially parathyroid and renal disorders which worsen the general prognosis. The present case serves as a reminder that the identification of choroidal calcifications warrants precocious ophthalmological and systemic evaluation to ensure adequate treatment and monitoring.

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