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CASE REPORT

Bilateral sclerochoroidal calcification in a case with asymptomatic primary hyperparathyroidism

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Abstract

Sclerochoroidal calcification is an uncommon degenerative ocular disease that is characterized with calcium deposits at the level of choroid and sclera. This condition could be related to calcium pyrophosphate metabolism disorders such as primary hyperparathyroidism. We presented a case who received the diagnosis of the primary hyperparathyroidism after the detection of asymptomatic fundus lesions on a routine eye examination.

Keywords: Optical coherence tomography; optical coherence tomography angiography; primary hyperparathyroidism; retinal imaging; sclerochoroidal calcification.

Parathyroid gland regulates calcium-phosphate metabolism.[1] Hyperparathyroidism often occurs due to autonomous excessive parathormone secretion of the chief cells in the parathyroid gland. However, it may also occur as a result of a compensation mechanism secondary to the increased renal calcium loss. Metastatic deposition of calcium can often be observed at the vascular network, kidneys, lungs, and gastric mucosa as the result of hypercalcemia.[2]

Sclerochoroidal calcification (SCC) is an uncommon degenerative ocular disease that is characterized with calcium deposits at the level of choroid and sclera and is first described by Goldstein and Miller[3] in a patient with hyperparathyroidism in 1982. SCC is a benign disorder with a good visual prognosis. Although sclerochoroidal calcification is commonly reported to be an idiopathic condition,[4] it has been linked to the abnormal calcium-phosphorus metabolism (hyperparathyroidism, pseudohypoparathyroidism, vitamin D intoxication, sarcoidosis, hypophosphatemia, and chronic renal failure),[5] Bartter,[6] and Gitelman syndromes.[7] Shields et al.[8] could not identify any systemic cause in 42 of 53 patients (79%) with SCC who had sufficient laboratory test results in their records. In the remaining cases, hyperparathyroidism was found out in 11 of 53 cases (20%). Other detected secondary causes were included parathyroid adenoma, diuretic use, Gitelman syndrome, and Bartter syndrome.[8]

We present a case of SCC with asymptomatic primary hyperparathyroidism that was detected following a routine eye examination.

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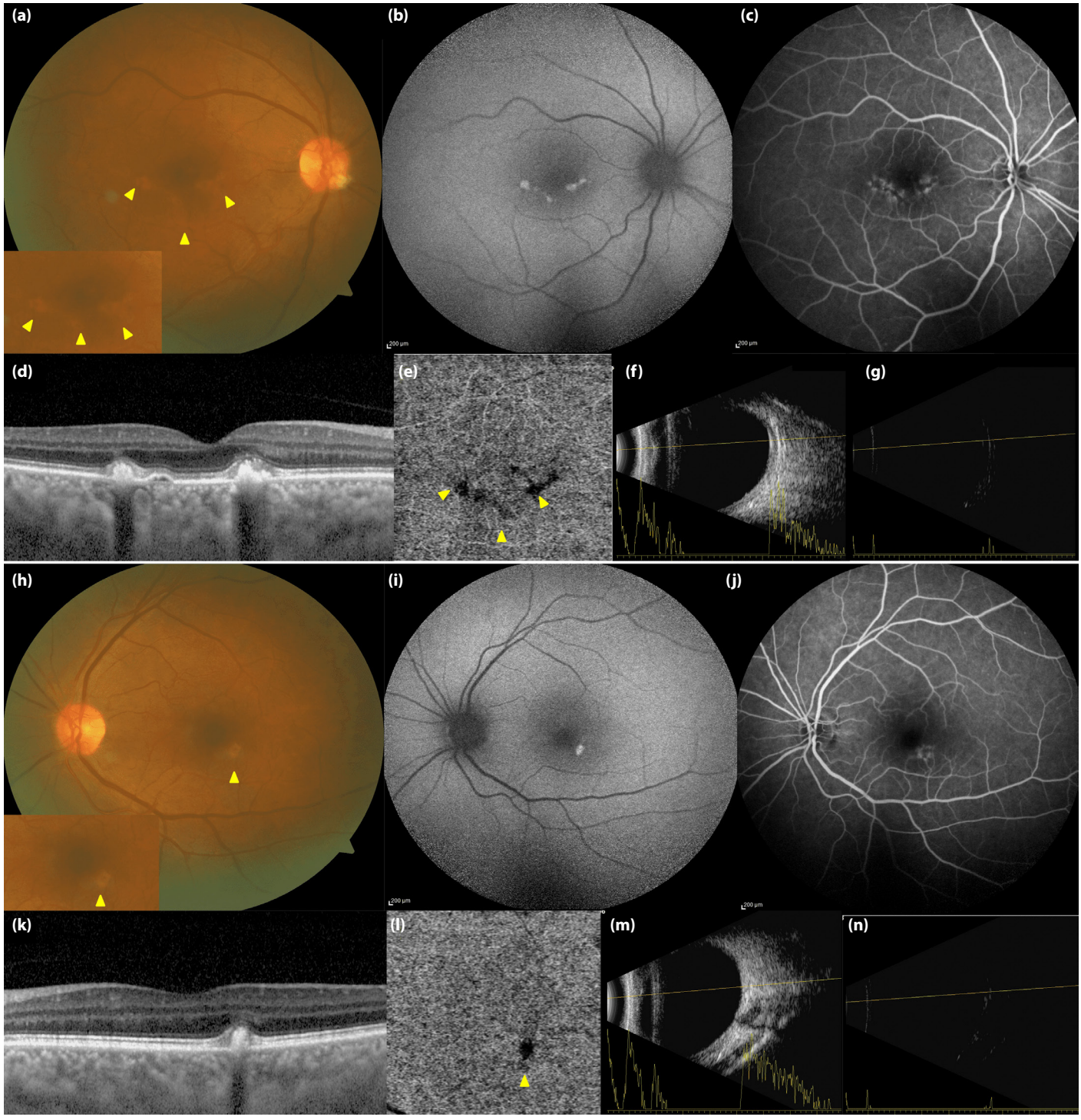

Fig. 1. Right eye; color fundus picture, multiple, pale, yellow-white, and round-shaped lesions with ill-defined borders at the inferior part of the fovea (yellow arrows) **(a)**. Fundus autofluorescence (FAF) image and hyperautofluorescent foci at the inferior part of the fovea **(b)**. Fundus fluorescein angiography, arteriovenous phase, and hyperfluorescence at the lesion site of the fovea **(c)**. Optical coherence tomographic (OCT) picture, Subretinal drusen-like hyperreflective deposits corresponding to the lesion sites (yellow arrows) **(d)**. Choriocapillaris slab (6×6 mm) of optical coherence tomography angiography (OCTA) and flow deficit areas (yellow arrows) corresponding to the lesion sites at the inferior part of fovea **(e)**. B-scan ultrasonographic picture (10MHz probe) and multiple hyperechoic linear areas at the level of choroid and sclera with a gain of 100 dB and 50 dB. **(f and g)**. Left eye; color fundus picture, a pale, yellowish, nummular lesion (yellow arrow) at the inferotemporal part of fovea **(h)**. FAF image, hyperautofluorescence of the lesion **(i)**. Fundus fluorescein angiography, arteriovenous phase, and hyperfluorescence at the lesion site of the fovea **(j)**. OCT picture and subretinal hyperreflective deposit at the level of retinal pigment epithelium layer (yellow arrow) **(k)**. Choriocapillaris slab (6×6 mm) of OCTA and flow deficit areas (yellow arrow) corresponding to the lesion site **(l)**. B-scan ultrasonographic picture (10 MHz probe) and Multiple hyperechoic linear areas at the level of choroid and sclera with a gain of 100 dB and 50 dB **(m and n)**.

Case Report

A 64-year-old woman had a routine eye examination with the complaint of itchy eyes. Her medical history included well-treated breast cancer (2 years ago) and osteoporosis. She has been on bisphosphonate and vitamin D combination for the osteoporosis for almost 2 years. Her best-corrected visual acuity was 20/30 bilaterally. Slit-lamp examination was unremarkable except for 2+ nuclear sclerosis in both eyes. Intraocular pressure was within normal limits, while dilated fundus examination disclosed multiple pale, yellowish, and round-shaped lesions at the fovea of the right eye (Fig. 1a). There was a nummular yellowish and pale lesion at the inferotemporal quadrant of the left fovea (Fig. 1h). These deposits were hyperautofluorescent (Fig. 1b and i) and exhibited staining on fluorescein angiography (Figb 1c and j). Optical coherence tomography images revealed bilateral subretinal hyperreflective ovoid deposits with backshadowing (Fig. 1d and k). Choriocapillaris slabs of optical coherence tomography angiography demonstrated flow deficit areas corresponding to the location of the deposits (Fig. 1e and l). B-scan ultrasonography depicted multiple linear hyperechoic accumulations inside the choroid at both posterior poles (Fig. 1f-g and 1m-n). Full systemic work-up was carried out with the help of an endocrinologist to look for possible abnormal calcium and phosphorus metabolism, Bartter, and Gitelman syndrome. Abdominal and thyroidal ultrasonography was reported as normal. Laboratory tests revealed high parathormone levels of 115 pg/ml (range; 14–72 pg/ml) with normal serum calcium levels. Other laboratory tests such as count blood cell test, calcitonin levels, Vitamin D level, and kidney function test were within normal limits. A final diagnosis of idiopathic hyperparathyroidism was reached out and the patient was informed about the diagnosis. No additional treatment was recommended.

Discussion

SCC is characterized by a variably elevated often yellow-white, multilobular, or plaqueoid subretinal lesion with irregular borders that are typically located in the superotemporal region of the midperipheral fundus. Lesions can be noted as flat or mass-like structure in many occasions.[9] In the present case, we detected the hyperechoic flat lesions at the choroid and sclera using B-scan ultrasonography on our clinical suspicion.

We presented a case who received the diagnosis of primary hyperparathyroidism after the detection of asymptomatic fundus lesions on a routine eye examination. In an in vitro study, Zhang et al.[10] showed that the increase in calcium intake caused an increase in lipofuscin accumulation in photoreceptor outer segments phagocytosing retinal pigment epithelium cells. In our case, subretinal deposits detected in OCT can be considered in this regard.[10]

Conclusion

This case once again pointed out the importance of attentive examination to catch the subtle clinical signs and only after that appropriate multimodal ophthalmic imaging methods facilitated the diagnosis. Although SCC is often an idiopathic disorder, all patients with SCC should be examined for the underlying calcium-related systemic conditions.

Informed Consent: Written informed consent was obtained from the patients for the publication of the case report and the accompanying images.

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