CASE REPORT

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Ocular manifestation in a patient with chronic kidney disease: A rare case report

Kronik böbrek hastalığı olan bir hastada oküler belirtiler: Nadir bir vaka raporu

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ABSTRACT

Central serous chorioretinopathy (CSCR) is a maculopathy affecting young individuals and presenting with painless, gradual loss of vision. It has multifactorial etiology, and various systemic diseases are associated with it. CSCR can result in permanent loss of vision, and a number of treatment modalities have been suggested. Here we aimed to report a rare case of a patient with chronic kidney disease (CKD) with CSCR as an ocular manifestation presenting to a secondary care institute in a hilly terrain.

Keywords: *Central serous chorioretinopathy, kidney, ocular*

ÖZ

Santral seröz koryoretinopati (SSKR) genç bireyleri etkileyen ve yavaş ve ağrısız ilerleyen görme kaybıyla ortaya çıkan bir makülopatidir. Multifaktöriyel bir etiyolojisi vardır ve çeşitli sistemik hastalıklarla ilişkilidir. Kalıcı görme kaybıyla sonuçlanabilen santral seröz koryoretinopati için birkaç tedavi yaklaşımı önerilmektedir. Bu çalışmada, oküler bir belirti olarak SSKR ile birlikte kronik böbrek hastalığı olan ve tepelik bir arazide bulunan ikinci basamak bir sağlık merkezine başvuran nadir bir vaka sunulmaktadır.

Anahtar Kelimeler: *Santral seröz koryoretinopati, böbrek, oküler*

INTRODUCTION

Subretinal serous fluid accumulation and retinal detachment are seen in patients with central serous chorioretinopathy (CSCR) (1). Von Graefe was the first to describe this disease entity in 1866 (2). CSCR predominantly affects young and middle-aged adults, with predominance in males and Asians (3). Our case was a middle-aged female with CKD.

CASE PRESENTATION

A 46-year-old female (Figure 1) presented to a secondary care institute of a hilly state with a history of painless loss of vision in the left eye along with metamorphosia for a week. There was no history of delayed dark adaptation and color vision abnormalities. Her developmental, prenatal, perinatal, postnatal, menstrual, and family histories were all unremarkable. Old records brought by the patient revealed a history of being treated for chronic kidney disease (CKD) from a tertiary care institute for the past 2 years, and she was on injections of erythropoietin, calcium, folic acid, telmisartan, atorvastatin, and multivitamins. There was no history of any other systemic illness, steroid intake, or recent or old surgery.

On examination, her speech, intelligence, general physical examination findings, and systemic examination findings were normal. Her visual acuity was 6/9 in the right eye and 6/60 in the left eye and improved to 6/6 in the right eye with +0.5 D sphere and to 6/24 in the left eye with +1.0 D sphere. The pupillary reactions, ocular movements, slit lamp examination findings, intraocular pressure, gonioscopy findings, and color vision were normal. Fundus examination with direct ophthalmoscopy revealed a characteristic "ring reflex" at the macula (Figure 2). On the Amsler Grid chart, the lines were described as "distorted" by her. Fundus fluorescein angiography (FFA) revealed the characterized "ink blot" pattern of CSCR (Figure 3). Fresh complete blood examinations revealed a blood urea level of 30 mg/dl, creatinine level of 2 mg/dl, raised triglyceride levels, and positivity for hepatitis B surface antigen as the only significant findings.

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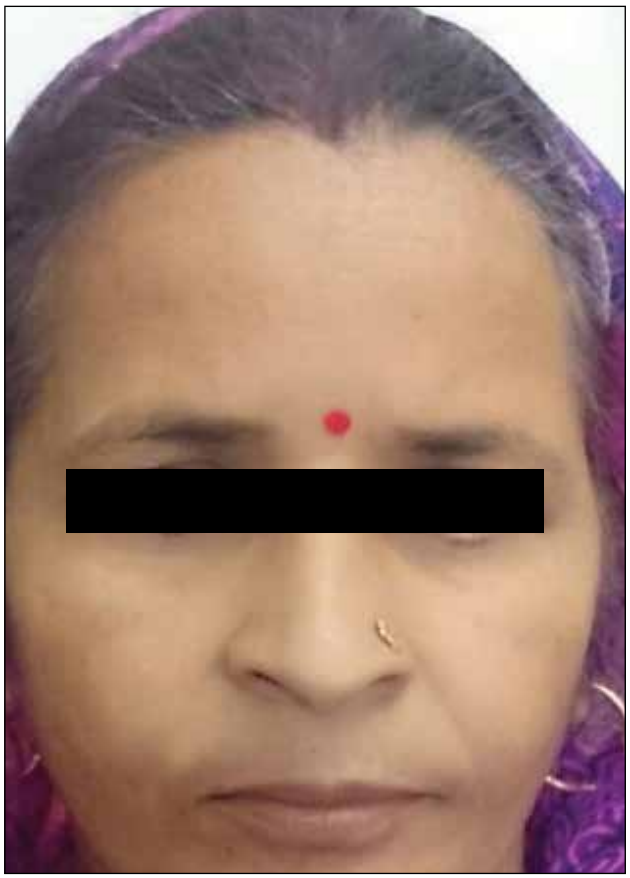

Figure 1. *Patients photograph*

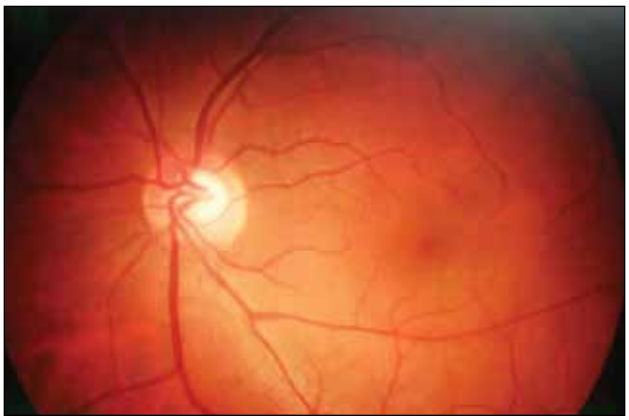

Figure 2. *Fundus photograph of the patients*

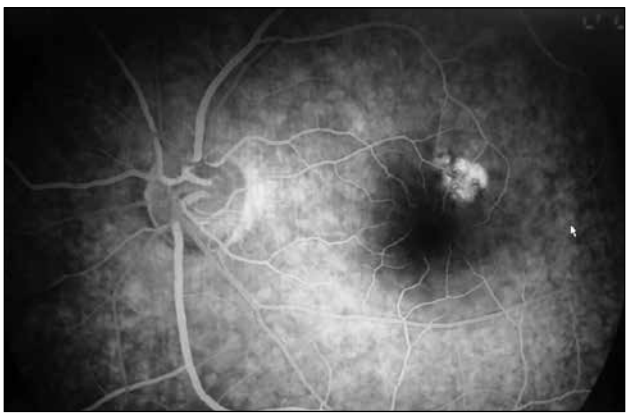

Figure 3. *Fundus fluorescein angiography of the patient*

On the basis of the aforementioned signs and symptoms, a diagnosis of central serous chorioretinopathy (CSCR) was made. The prognosis of the disease was explained to the patient, and we are awaiting a follow-up from her end.

DISCUSSION

Risk factors for developing CSCR are Type A personality, use of steroids, Cushing's syndrome, pregnancy, collagen vascular diseases, sarcoidosis, obstructive sleep apnea, alcohol consumption, allergic respiratory disease, hypertension, use of psychopharmacological medications, *Helicobacter pylori* infection, family history, and organ transplantation (4). Our patient had CSCR with CKD, which is a rare association. None of the aforementioned risk factors was present in our patient. Although the association of hypertension with CKD is known, her blood pressure at the time of examination was normal and old records of her blood pressure during the past few months were within normal limits. Subsequent serial examination of her blood pressure revealed normal levels.

The pathophysiology of CSCR is increased choroidal vascular permeability, which causes increased tissue hydrostatic pressure beneath the retinal pigment epithelium (RPE), leading to the loss of integrity of RPE and fluid leakage (5).

Patients with CSCR present with a history of sudden loss/diminution of vision, micropsia, metamorphopsia, scotoma, or decreased color vision. Steroid therapy and kidney transplantation have again been found to be independent risk factors in developing CSCR (6). Ocular findings in a CKD patient include lid edema, conjunctival pallor, xanthalasma, conjuctivitis, episcleritis, corneal and conjunctival calcification, pinguecula, subconjuctival hemorrhage, rubeosis iridis, neovascular glaucoma, and cataract (7). CKD has rarely been reported. Our case did not have any of the aforementioned ocular findings.

Fundus fluorescein angiography fundus autofluorescence imaging (FAF), indocyanine green angiography (ICGA), and optical coherence tomography (OCT) are the common investigations for CSCR. FFA is characterized by focal fluorescein leaks at the level of RPE and is referred to as a "smokestack" and "inkblot" pattern. In ICGA, inner choroidal staining can appear in the midphase of ICGA and fades in the late phase. FAF patterns are seen is increased autofluorescence at the site of leakage (5).

Most acute CSCR cases resolve spontaneously within 2–3 months (4). When indicated, treatment options include argon laser photocoagulation, transpupillary thermotherapy, photodynamic treatment with verteporfin, use of intravitreal bevacizumab, micropulse diode laser photocoagulation, use of corticosteroid antagonists, and use of aspirin. Any steroid intake should be discontinued (8). A high degree of suspicion on part of the ophthalmologist is required in confirming the diagnosis.

CONCLUSION

Hence in any case of chronic kidney disease with sudden diminition of vision, CSCR should be kept as one of the entities though it is still a rare association with the renal problem.

Informed Consent: Written informed consent was obtained from patients' parents who participated in this study.

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