

Atypical Ocular Sign of Chronic Renal Failure: Bilateral Massive Serous Retinal Detachment

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Abstract

We present a case of bilateral massive serous retinal detachment in hypertensive choroidoretinopathy associated with chronic renal failure (CRF). A 20-year-old male with bilateral blurred vision was referred to our clinic from the department of nephrology. His first examination revealed best corrected visual acuity (BCVA) of hand movements, and weakness of pupillary reactions in both eyes. Fundoscopic examination revealed optic nerve and macular edema, macular star formation, peripapillary retinal hemorrhages, sub-retinal hard exudates, and widespread serous retinal detachment (SRD) in inferior quadrants of retina with yellow-white pre retinal flecks in mid-periphery of the fundus in both eyes. There was extensive subretinal fluid from macula to optic nerve head in spectral domain optic coherence tomography. The patient underwent hemodialysis (HD) treatments 3 times per week. Systemic arterial hypertension was controlled with three different medications. At final visit eight months after admission his BCVA improved to 160/200 in both eyes. This case report suggests that HD treatments and adequate blood pressure control in CRF patients complicated with SRD possibly could improve the retinal appearance and BCVA rapidly.

Keywords: Chronic Renal Failure; Serous Retinal Detachment; Hypertensive Retinopathy

Introduction

Sudden elevations in blood pressure can cause permanent vision complaints by choroidal ischemia. Choroidal ischemia affects retina pigment epithelium (RPE) pumping function negatively and results with serous retinal detachment (SRD) [1]. We present a case with bilateral massive serous retinal detachment (SRD) due to chronic renal failure (CRF), and uncontrolled systemic hypertension. There was not any clues of systemic disorder that have been found to explain subretinal serous detachment except for hypertensive emergency secondary to CRF.

Case Report

A 20-year-old male with bilateral blurred vision was referred to our clinic from the department of nephrology. His first examination revealed best corrected visual acuity (BCVA) of hand movements, and weakness of pupillary reactions in both eyes. Intraocular pressures were within normal limits. Anterior segment examination was unremarkable. Fundoscopic examination revealed optic nerve and macular edema, macular star formation, peripapillary retinal hemorrhages, sub-retinal hard exudates, and widespread serous retinal detachment in inferior quadrants of retina with yellow-white pre retinal flecks in mid-periphery of the fundus in both eyes (Figure 1A-1B). Obvious arterial narrowing and increased venous tortuosity was remarkable bilaterally. There was serous macular detachment extending to the inferior major vascular arcade in spectral domain optic coherence tomography (SD-OCT), (Heidelberg Engineering, Heidelberg, Germany) in both eyes (Figure 1C-1D). He stated that he was feeling good until 2 weeks before. He had the complaints of similar blurred vision, difficulty in walking and consciousness two weeks ago. A review of his medical records revealed that he had uremic encephalopathy and underwent hemodialysis (HD) treatments several times because of CRF.

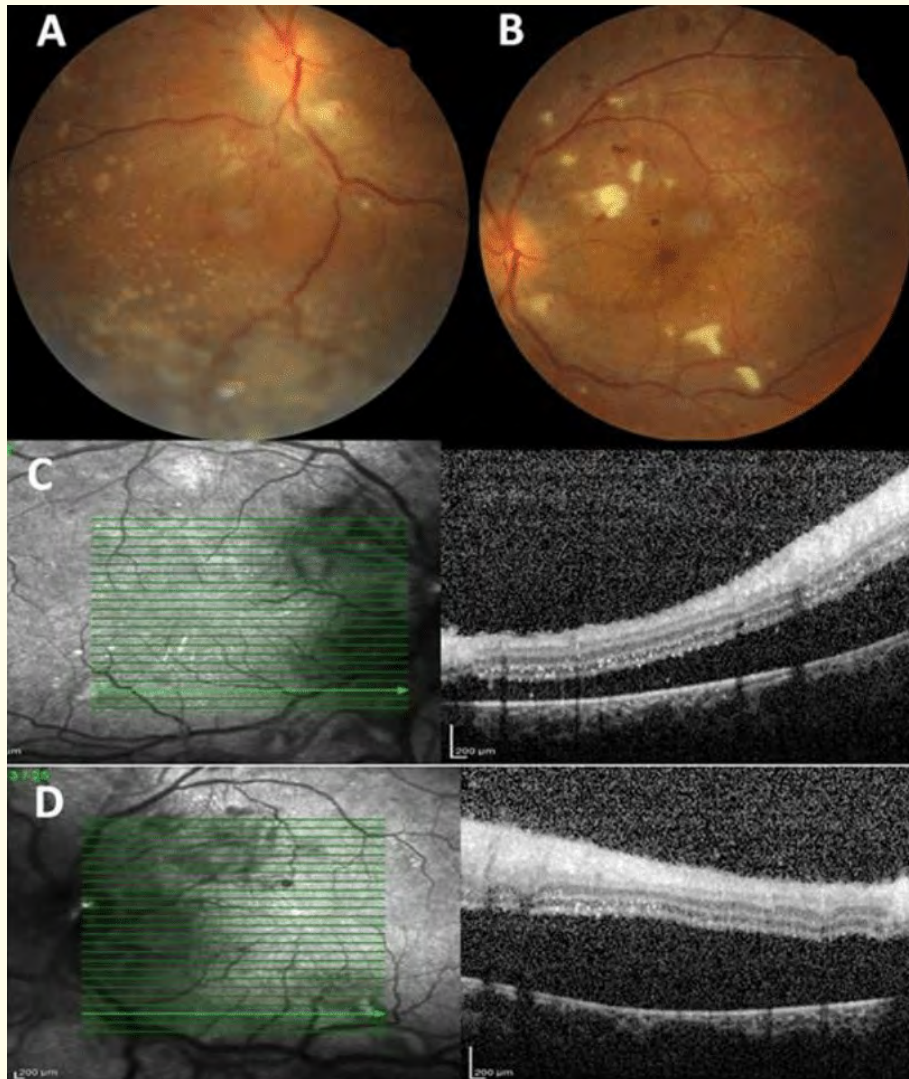


Figure 1: (A-B): Fundus photographs show optic nerve and macular edema, macular star formation, peripapillary retinal hemorrhages, sub-retinal hard exudates, and widespread serous retinal detachment in inferior quadrants of retina with yellow–white pre retinal flecks in mid-periphery of the fundus in both eyes. (C-D): Serous macular detachment extending to the inferior major vascular arcade in spectral domain optical coherence tomography (SD-OCT), (Heidelberg Engineering, Heidelberg, Germany) in both eyes.

On presentation; his metabolic status showed the levels of urea: 148 (20 - 44 mg/dl), creatinine: 9.1 (0.6 - 1.1 mg/dl), glucose: 100 (60 - 110 mg/dl). Systemic blood pressure was 168/98 mmhg. The patient underwent HD treatments 3 times per week. Systemic arterial hypertension was controlled with three different medications (nifedipine 120 mg/day, carvedilol 50 mg/day and valsartan 160 mg/day). We followed the patient weekly. Our initial diagnosis was hypertensive retinopathy due to CRF. We also suspected about Alport

syndrome which is characterized by progressive hereditary nephritis, high-tone sensorineural deafness and and ocular abnormalities. Ocular abnormalities in Alport syndrome include lenticular and retinal changes [2]. Dot-and-fleck retinopathy and anterior lenticonus is usually seen but cataracts, posterior lenticonus, and retinal detachment are uncommon signs in Alport syndrome. Our patient had yellow-white retinal flecks and subretinal fluid, but anterior/posterior lenticonus or hearing problems were not present. To discover the main cause of CRF, we had consultations from heamotology and otolaryngology clinics for hemolytic uremic syndrome and Alport syndrome, respectively. A specific diagnosis was not confirmed for the CRF. Abdominal ultrasonography showed bilateral atrophic kidneys.

After one and a half months his BCVA improved to 80/200 in RE and 100/200 in LE. Pupillary light reflex was weak in RE. We detected relative afferent pupillary defect (RAPD) in LE. Fundoscopic examination showed narrowed and sheathed arterioles, and few exudates bilaterally. Optic nerve swelling was resolved completely. There was not any sign of (SRD) in both eyes neither on fundoscopy nor on SD-OCT. At final visit six months after admission his BCVA improved to 160/200 in both eyes with the same findings (Figure 2A-2B).



Figure 2: (A-B): Fundus photographs of right and left eye showing resolved findings at his final visit.

Discussion

There are variety of conditions which can result in serous retinal detachment. Major causes of serous retinal detachment are idiopathic, congenital, inflammatory, postsurgical, uveitic, hematologic, vascular, and neoplastic [3]. Subretinal fluid accumulation in CRF patients is an uncommon finding. There are a few case reports which presented with SRD in CRF patients. Basu., *et al.* reported a young male patient complicated with SRD and multiple retinal pigment epithelial detachments following HD who had pancreatitis, acute renal failure, and adult respiratory distress syndrome [4]. Yasuzumi K., *et al.* reported another case who had bilateral SRD in the macula in a 14 year-old girl with Alport's syndrome [5]. The mechanism of subretinal serous elevation is not clearly understood. Basu et al claimed that high levels of urea (urea > 60 mg/dl) affects the serum osmolarity and intravascular/extravascular compartments so that the fluid moves into the subpigment epithelial and subsequently into the subretinal space [4]. Secondary to renal failure, repeated edema of the retinal vessel wall may lead to thickening of the vascular wall which can damage to the arterial endothelium [6]. Systemic hypertension is usually accompa-

nied with CRF which is called renovascular hipertension. Systemic blood pressure was also high in our case. Venecia G et al argued that hipertensive choroidal ischemia leads to impairment of outer blood-retinal barrier which causes ischemic retinal pigment epithelium and that results with SRD [1]. Hayreh., *et al.* showed the importance of choroidal ischemia in the genesis of macular edema and SRD in experimental renovascular hipertensive monkeys [7]. Villalba-Pinto., *et al.* recently reported a case with bilateral massive (SRD) in a patient with acute hipertensive attack due to decompensation of CRF [8]. In that report, the patient's BCVA improved rapidly in 2 weeks with the combination of HD treatments and antihypertensive agents. The improvement of BCVA in our patient prolonged to one and half months. The reason for this delay could be the result of inadequate blood pressure control. They also argued that the whitish lesions in mid-periphery of the fundus were secondary to ischemia of the underlying retinal pigment epithelium and the choroid. We think that yellow-white retinal flecks in the mid-periphery of fundus could possibly be the result of the same mechanism. Bilateral optic nerve swelling was the significant difference from the previous report of Pinto. We think that the reason for this is the hipertensive choroidopathy which was more severe in our case. The RAPD was permanent in LE in present case. This might be the sign of hipertensive optic neuropathy. Kovach reported a case of hipertensive optic neuropathy in a young female with renal failure [9]. Although that case was about optic neuropathy, Kovach did not emphasize RAPD in that case.

Conclusion

In conclusion this case report suggests that HD treatments and adequate blood pressure control in CRF patients complicated with (SRD) possibly could improve the retinal appearance and BCVA rapidly.

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