

## Bilateral Central Serous Retinal Detachment in Protein-losing Enteropathy

Dear Editor,

Serous retinal detachment (RD) is one of the most common causes of RD, and it has numerous ocular etiologies, such as central serous chorioretinopathy (CSC), intraocular tumors, and posterior scleritis, as well as systemic etiologies, such as Vogt-Koyanagi-Harada disease [1]. However, hypoalbuminemia, observed in conditions like nephrotic syndrome and protein-losing enteropathy (PLE), is a rare etiology of serous RD, and there are few reports to date [2]. We report a case of bilateral central serous RD and severe chemosis due to underlying PLE.

A 32-year-old male was referred to the Farabi Eye Hospital Retina Clinic due to progressive loss of vision over a period of three months, which had worsened 1 week before presentation. His visual acuity was counting fingers at 1 m in the right eye and 2 / 10 in the left eye. His past medical history was unremarkable except for presence of edema of the extremities, diarrhea, and intermittent abdominal pain for the past 6 months. Ophthalmic examination revealed moderate chemosis in both eyes and mild edema of the eyelids. Funduscopic examination demonstrated elevation of the macula in both eyes, which was more prominent in the right eye with exudates at the nasal side of the macula. Other ocular examinations were normal. Optical coherence tomography, fundus auto fluorescence, and fluorescein angiography (FA) were normal and confirmed the presence of sub-retinal fluid and serous detachment at the macula of both eyes (Fig. 1A-1L).

A systemic evaluation was performed, including laboratory tests that revealed serum hypoalbuminemia (2.4 g/dL; normal range, 3.5 to 5.5) with normal kidney function. The patient was referred to a gastroenterologist for further

evaluation. To determine the patient's diagnosis, liver biopsy, endoscopy with multiple biopsies from the small intestine, and colonoscopy were performed, and all examinations were unremarkable. Finally, the patient was diagnosed with PLE and treated with oral prednisolone 30 mg per day in addition to a high protein and low fat diet to reverse the hypoproteinemia. At re-examination 2 months later, the patient's visual acuity had improved to 5 / 10 in the right eye and 8 / 10 in the left eye with a decrease in subretinal fluid on optical coherence tomography in both eyes.

There are only a few reports of systemic hypoalbuminemia causing serous detachment, and the cases have different etiologies. Venkatramani et al. [2] reported a case of PLE with bilateral inferior serous RD and visual acuity of 20 / 40 in both eyes. After treatment with methylprednisolone 30 mg/day, the serous RDs gradually resolved during 5 months of follow up, and the visual acuity improved to 20 / 25 in both eyes.

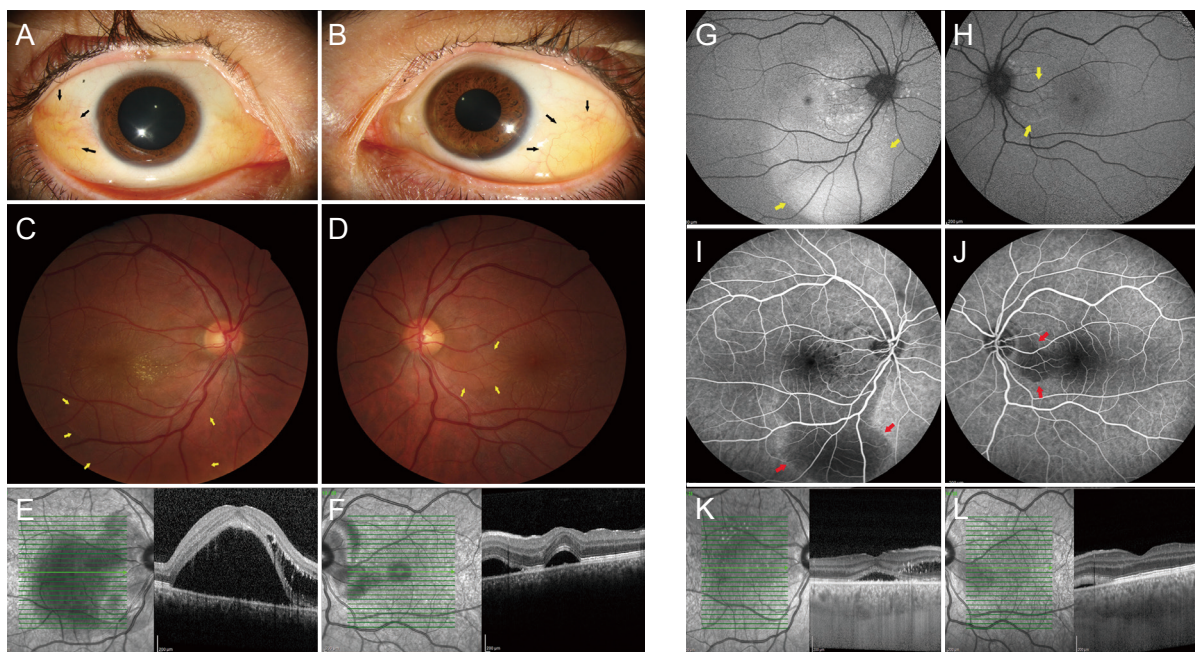
One of the major contributing mechanisms in maintaining adhesion of the neurosensory retina to the retinal pigment epithelium (RPE) is outward flow of fluid from the vitreous across the retina and RPE. It has been postulated that the osmotic pressure of the choroid plays an important role in maintaining this flowability in addition to two other important factors: the active ion-fluid transport pump of the RPE cells and the intraocular pressure [3,4]. Serum albumin is the major factor determining the osmotic pressure of the circulating system, such as the choroidal vessels. In conditions like PLE, decreased serum albumin leads to a reduction in choroidal vessel oncotic pressure, transudation, and accumulation of fluid in the subretinal space.

In contrast to cases of CSC, in which the pathophysiology is mainly due to pachychoroid and defective RPE (in pumping the fluid from the subretinal space toward the choroid), we did not observe any leakage, which is characteristic of CSC, on the FA images in the present case. Instead, in areas with subretinal fluid accumulation, there was blockage with no pinpoint leakage or other signs of hyperfluorescence, such as smoke stack sign or expansile dot. We believe that this finding supports the hypothesis of reduced oncotic pressure as the etiology of sub-RPE fluid

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**Fig. 1.** (A,B) Slit lamp photographs. Note the moderate chemosis prominent at the temporal conjunctiva (arrows) (anisocoria is pharmacologic). (C-H) Retinal elevation was observed in both eyes (arrows) with macular exudate in the right eye. In autofluorescence imaging, hyper autofluorescence in the area corresponding to the retinal elevation was detected due to the presence of subretinal fluid. (I,J) In fluorescein angiography, hypofluorescent areas were seen in both eyes, compatible with the clinical examination and optical coherence tomography (arrows). The hypofluorescence occurred because of accumulation of subretinal fluid and the resultant blockage, without any signs of vascular leakage (hyperfluorescence) such as smoke stack sign or expansile dot in late phases (not shown here). (K,L) Repeat macular optical coherence tomography 2 months later revealed significant resolution of the subretinal fluid. Informed consent was obtained from the patient for publication of this case report and relevant images.

accumulation in this patient. Moreover, any other pathophysiology would have caused at least some foci of hyperfluorescence in FA.

It should be noted that PLE can be an unusual manifestation of systemic lupus erythematosus (SLE) [5]. Because serous RD, like CSC, may occur in SLE, physicians should consider SLE in the differential diagnosis of patients with serous RD. Our patient did not meet the SLE diagnostic criteria. We suggest that the appearance of bilateral central serous detachment should raise suspicion for a diagnosis of hypoalbuminemia, especially if it is accompanied with other signs such as chemosis and no leakage on FA images. Prompt recognition of PLE is essential for restoration of visual function and early treatment of both ocular and systemic manifestations of the disease.

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## Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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