The Ocular Coherence Tomography (OCT) Findings In Sympathetic Ophthalmia

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Background

Sympathetic ophthalmia (SO) is a bilateral granulomatous panuveitis(1,2), which follows ocular contusion, penetrating eye trauma, or previous eye surgeries, e.g., catract surgery, glaucoma interventions, like cyclodestruction or vitreoretinal surgeries.(3).the sympathizing eye presents from 2 weeks to 50 years after the exciting eye insult, but 80% of cases happen during the first 3 months(4, 5). The incidence of SO is estimated about 0.03/ 100,000 (6). In this study we would explain the imaging findings of an acute case of SO. The informed consent was taken from the patient.

CASE presentation

The patient is a 20 year-old man with history of right eye trauma due to mine explosion in the west parts of Iran. One day after the trauma primary repair was done for a full thickness corneoscleral laceration and uveal prolapse in a city hospital. Two weeks later, due to retinal incarceration at the laceration site, pars plana deep vitrectomy with SO tamponade and Penetrating keratoplasty was performed. Six weeks after the vitrectomy, he came back

complaining of the left eye decreased vision since about 3 days ago. At this time in the right eye the vision was no light perception, and there was slight cellular reaction in the anterior chamber and vitreous. In the left sympathizing eye , the best corrected visual acuity was 1.0 LogMAR, there was cellular reaction in anterior chamber and vitreous, with multiple elevated serous retinal detachments in funduscopy. (figure 1)

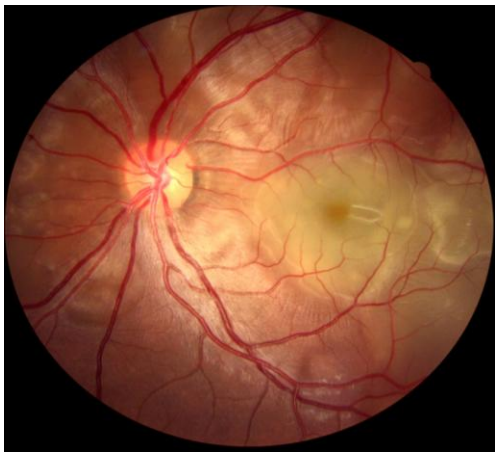


Figure 1

In the infrared and autofluorescence images of the sympathizing eye, there was hyporeflective patches in the posterior pole. In the autoflourescence image , around the hypoflourescent patches, there was hyperreflectivity, compatible with reactional stress-induced retinal pigment epithelium (RPE) hyperplasia.(figure 2)

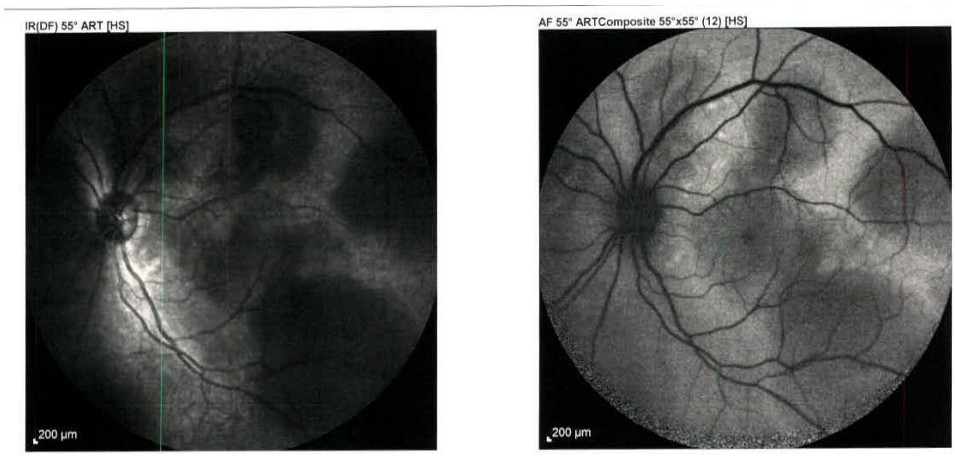


Figure 2

In Fluorescin angiography (FA) imaging, (figure 3) performed with the Spectralis HRA + OCT system (Heidelberg Engineering, Heidelberg, Germany), choroidal fluorescence seemed partially blocked. Multiple progressively fluorescent dots were noted at the level of RPE, resembling pinpointing leakages. Pooling happened gradually in Serous retinal detachments spaces mainly from the periphery of the spaces. Dark rims are noted within the pooling spaces.

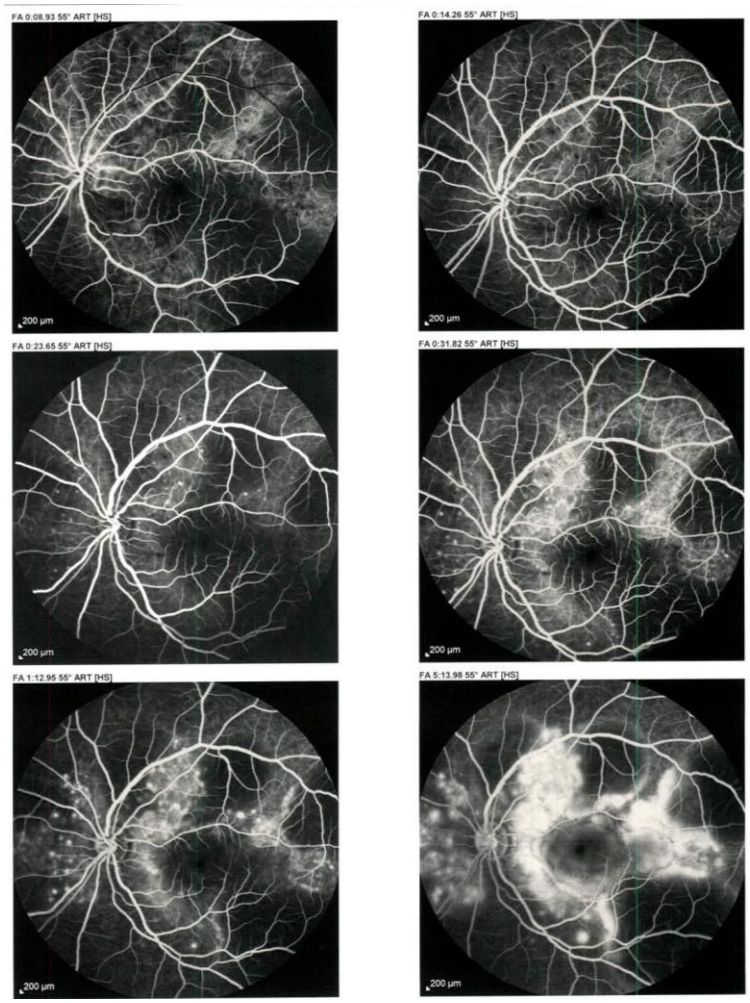
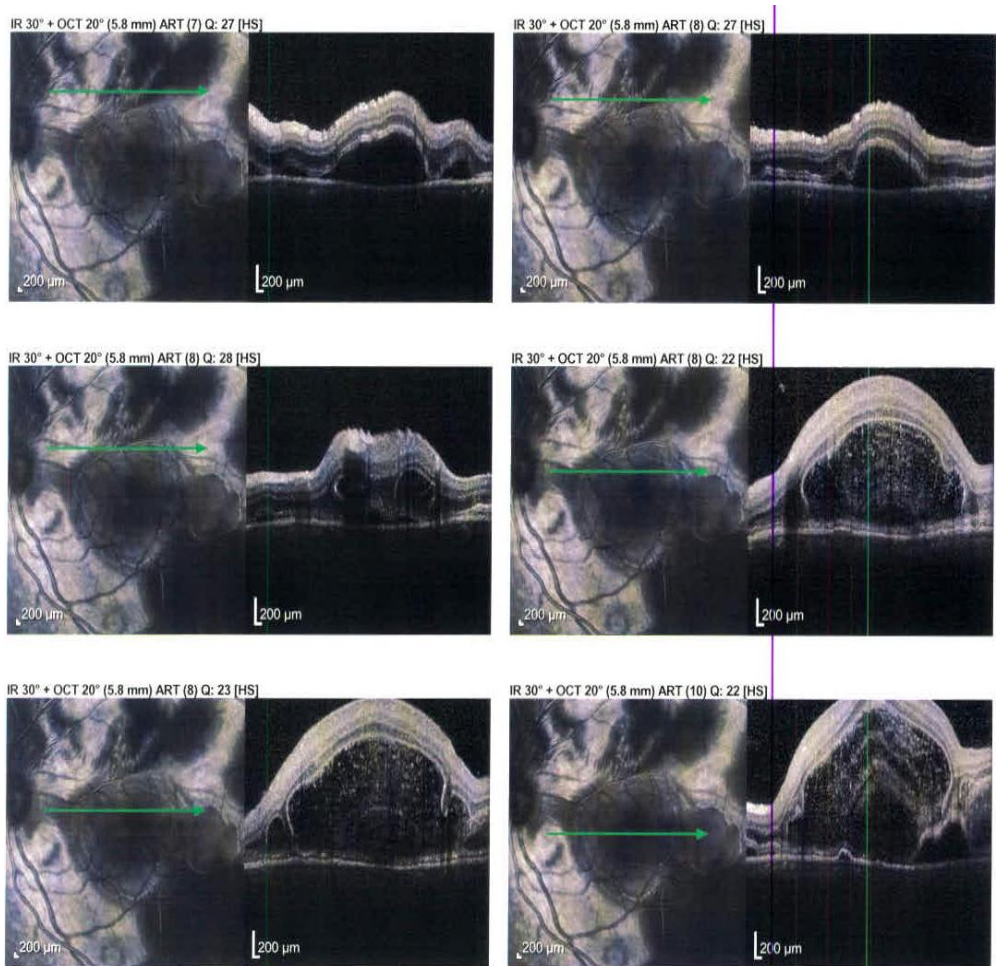


Figure 3

In the ocular coherence tomography (OCT) imaging (figure 4) In areas with attached retina there was diffuse intraretinal edema mainly in the nuclear layers and ganglion cell layer, and the IS-OS junction appeared as a distinct hyperreflective layer. Multiple exudative dots was seen within the retina. In Serous retinal detachments areas there was subretinal pigment clumping and subretinal fluid accumulation with multiple layerings, mimicking retinal layers. Hyperreflective layers were precipitated over the retinal pigment epithelium (RPE) too. This subretinal layering showed a septation at the borders of the Serous retinal detachment areas. The IS OS junction was disrupted especially at the junction of the attached and detached retina. The photoreceptor layer seems intact but the RPE layer seemed undulated beneath which there

was hyperreflective precipitations. The RPE layer got irregularity with thickening in some points and discontinuity in other points.



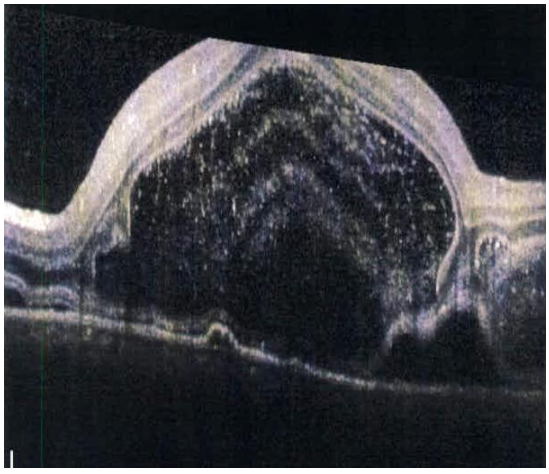
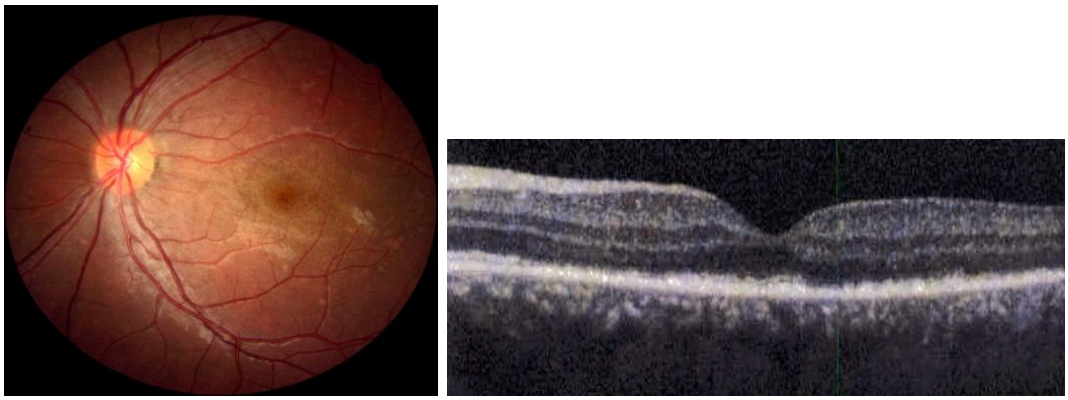


Figure 4

With the diagnosis of sympathetic ophthalmia (SO) the patient was treated with systemic steroids, 1 g intravenous methyl prednisolone acetate for the first 3 days , then 1 mg/kg oral prednisolone, and 1 month after the treatment , the vision improved to 0.045 LogMAR. After the start of steroid therapy, the serous retinal detachments areas had resolved (figure 5). In OCT the fibrinous subretinal precipitates had largely subsided, (figure 6) The external limiting membrane (ELM), which was not evident during the acute stage of the disease, was visible now, but the IS – OS junction line seemed thinned and disrupted. The sub-RPE undulations had improved, but there was RPE hyperplasia.



Discussion

Some reversible and irreversible OCT findings have been decribed in sympathic ophthalmia(7 – 9). Gallagher (8) reported disorganization and thinning of inner retina with severly disintegrated RPE and choriocapillaries. Gupta et al. (7) reported serous retinal detachment and elongation of photoreceptors which improved with intravenous methylprednisolone treatment. They reported IS – OS junction restoration in all treated eyes. Mahendradas et al. (9) found irreversible RPE

rip secondary to choroidal swelling induced mechanical stretching of RPE layer. They suggested that this RPE tear may aggravate the exudative retinal detachment.

In our case of SO, 2 types of fluid accumulation was noted in OCT. the first type was minimally reflective intraretinal edema , mainly in the nuclear layers, and the second type was the true serous retinal detachment appeared as a nonreflective fluid with multiple layerings. Noticing the diffuse choroidal inflammation in SO, it seems that the RPE blood retinal barrier function has broken down, allowing the influx of fibrinous exudation beneath the retina. It seems that the inflammatory products, mainly fibrin, gradually precipitate, forming a hyperreflective membrane over the RPE layer. Due to the ongoing inflammation, more fluid would egress beneath the retina, which pushes the fibrinous septa away from the retina, forming these layered appearance in OCT. Another explanation for this layering is the possible wax and wane nature of the inflammation due to the chronicity of the disease or incompleted on- and –off treatment , although there was not such a history in our case. This layering could explain for an inflammatory reaction causing these fibrinous precipitation, different from central serous choriretinopathy (CSR) pathogenesis. In CSR due to hypermeable choriodal vessels and pinpoint RPE breakdown , and no inflammation, a transudate subretinal fluid would accumulate beneath the retina, which would not reply to steroid therapy. The fibrinous nature of these septation could explain that how these septa act like a barrier against fluorescin dye pooling in subretinal spaces beneath the retina, which is responsible for the delayed gradual pooling of dye , mainly from the peripheral parts of the subretinal spaces, and the multilobulated nature of this pooling in FA. Meanwhile the dark rims seen in the pooling stages of FA, may correspond to the fibrinous septa seen in in the OCT. On the other hand this case shows the importance of timely diagnosis and treatment, which would not only resolve the subretinal fluid and fibrin septa completely and quickly, but also could prevent oxidative-stress -induced retinal damage and irreversible subretinal fibrosis. With 1 month treatment the layered exudation subsided

completely but the photoreceptor layer seemed disrupted and thinned which may explain for the finally decreased vision.

To our knowledge this is the first report of the layered subretinal fluid accumulation, which shows not only the possible fibrinous exudative nature of the disease, but the importance of early treatment to resolve the exudation , and preventing persistent retinal damages and subretinal fibrosis.

Conclusion

OCT is an useful imaging technique to follow up the reversible changes in SO.

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