CASE REPORT

Central serous chorioretinopathy secondary to tuberculosis: cause or coincidence

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SUMMARY

32-year-old male with diagnosis of central serous chorioretinopathy (CSCR) in both eyes and negative history of administration of any medicine was treated with bilateral focal laser at leakage point, with visual recovery. Recurrence occurred after 2 years in right eye and was treated successfully with acetazolamide. Second recurrence occurred after 1 year. Acetazolamide was restarted, but no improvement was noted. Thorough re-evaluation of the patient revealed a family history of tuberculosis. Ancillary investigations rendered the presumptive diagnosis of tuberculosis, and antituberculosis treatment led to visual recovery with no recurrences. Present case poses two dilemmas: whether CSCR was secondary to tuberculosis or was it an incidental association in tuberculosis endemic population and second whether resolution occurred due to the treatment of tuberculosis or due to mineralocorticoid antagonism action of rifampicin. Significance of patient's history, clinical observation and angiographic studies in the diagnosis of idiopathic and recurrent CSCR is reiterated.

BACKGROUND

The pathogenesis of idiopathic central serous chorioretinopathy (CSCR) remains obscure. Recent studies implicate multifactorial aetiology and complex pathogenesis. It has been described in patients with high levels of either endogenous or exogenous steroids, type A personality and stress.^{1–3} Present case report of CSCR secondary to tuberculosis is the second case reported so far with the first case reported by Vayalambone *et al.*⁴

CSCR is characterised by an idiopathic serous neurosensory detachment in macular region. It typically affects individuals in the age group of 20–50 years. Male preponderance is noted with an M:F ratio of 6:1.^{5–7} The age-adjusted and sex-adjusted incidence of CSCR in a recent population-based study was found to be 5–6 per 100 000 persons.⁸ CSCR presentation has been associated with the use of vaso-constrictive agent such as epinephrine. Several classes of drugs other than corticosteroids have also been reported to be significantly associated with CSCR. Antacid and antireflux agents, over-the-counter sympatho-mimetics, antibiotics (oral), antihistamines, sildenafil citrate, in addition tobacco and alcohol use have also been reported to be risk factor for CSCR. Understanding of pathogenic accumulation of subneural retinal fluid in the macular region is limited. Increasing

evidence implicates an abnormal choroidal circulation as the cause of CSCR as seen on indocyanine green angiography.^{9 10}

CASE PRESENTATION

Thirty-two-year-old male presented with painless diminution of vision in both eyes with metamorphopsia. Best corrected visual acuity (BCVA) was 6/24 oculus dexter (OD): 6/12 oculus sinister (OS). There was no history of administration of any medication or allergy to any drug. Intraocular pressure, ocular motility and slit lamp examination were unremarkable. Dilated fundus examination revealed typical serous elevation of retina in macular area, central macular thickness (figure 1) 357 micron OD and 542 micron OS with retinal pigment epithelial detachment was observed on ocular coherence tomography (OCT). Focal area of hyperfluorescence that increased in intensity on late frames was observed on fundus fluorescein angiography (FFA) (figure 2). Diagnosis of CSCR was rendered. Focal laser at leakage point was done in both eyes. Visual acuity recovered back to normal in both eyes.

First recurrence of symptoms in the right eye was observed after 2 years with BCVA of 6/24 OD:6/6 OS. Left eye was unremarkable. Serous elevation of retina was seen on fundus examination. Right eye had 382 micron of macular thickness (figure 3) with subretinal fluid on OCT and FFA revealed focal leakage from site other than area lasered for the previous leakage. Oral acetazolamide 250 mg thrice a day for 15 days followed by 125 mg thrice a day for 15 days was administered. Gradual recovery was noted after 10 days with full recovery at 3 weeks.

Second recurrence in the right eye occurred after 1 year of second episode. Macular thickness of 359 micron with subretinal fluid was noted (figure 4). FFA showed focal area of leakage, which was from different site than preceding ones. Patient was again put on oral acetazolamide on same dosage, but no improvement was noted even after 4 weeks of medication. Patient was re-evaluated and extensive family history was taken. Family history of tuberculosis was detected. Ancillary investigations for tuberculosis were performed. Mantoux test was positive (25×25 mm), and erythrocyte sedimentation rate found to be raised (35 mm in the first hour). Chest X-ray postero-anterior (PA) view was suggestive of tuberculosis. Antitubercular treatment (ATT) was started in consultation with pulmonary medicine physician. Visual recovery of 6/6 was noted within 2 weeks after commencement of ATT.

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Figure 1 Ocular coherence tomography of both eyes: subretinal fluid in the macular area with increased central retinal thickness of 357 micron in the right eye and 542 micron in the left eye.

Since then, the patient has completed full course of ATT for 9 months and is under regular follow-up with no fresh complaints to date.

DISCUSSION

Tuberculosis causes a myriad of ophthalmic pathology. Primary infection in the eye is rare and typically presents as corneal,

conjunctival and scleral disease. Secondary ocular infection results from hematogenous spread of the organism or from a hypersensitivity reaction to extraocular infection. Reports of CSCR secondary to tuberculosis are sparse.

CSCR is known to resolve spontaneously in 3 months in most of the cases. However, the loss of productive hours due to visual disability is of great concern. Hence various treatment modalities

Figure 2 Fundus fluorescein angiography (both eyes): focal area of leakage in the macular area.

Figure 3 Ocular coherence tomography of the right eye at first recurrence: reappearance of subretinal fluid in macular area with central retinal thickness of 382 micron.

have been propagated for the early resolution of CSCR. These include photodynamic therapy, micro-pulse diode laser photocoagulation and focal laser by argon laser. Mineralo-corticoid antagonists, such as acetazolamide, spironolactone, rifampicin, eplorone and others, are an interesting alternative treatment. Rifampicin is an antituberculous medication that is thought to facilitate catabolism of endogenous steroids. It causes a proliferation of the smooth endoplasmic reticulum and an increase in the cytochrome P-450 content in the liver, thus affecting the metabolism and bioavailability of endogenous corticosteroids, consequently aiding in resolution of CSCR and improving its symptomatology.^{11 12}

Estimation of endogenous mineralo-corticosteroids levels in patients of CSSR may further validate the use of mineralo-corticosteroid antagonist; however, it was not available in the present case due to non-affordability by the patient.

This case and the previous reported case pose two dilemmas: first, whether CSCR was secondary to tuberculosis or was it an incidental association in tuberculosis endemic population and second whether the resolution occurred due to the treatment of tuberculosis or due to the steroid antagonist action of rifampicin. However, the absence of recurrence after ATT points towards first possibility. Large case-control studies are required to validate the association.

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Figure 4 Ocular coherence tomography (right eye) at second recurrence: macular thickness of 359 micron with reappearance of subretinal fluid.

Learning points

- This case demonstrates the role of patient's history, clinical observation and angiographic studies in the diagnosis of idiopathic central serous chorioretinopathy.
- Repeated disease recurrences warrant the physician to find out aetiology other than the established ones.
- Loss of productive hours warrants the need for early treatment of central serous chorioretinopathy.

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