Unusual association of diseases/symptoms

Central serous chorioretinopathy after blunt trauma

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Correspondence to Dr Claudia Maria Prospero Ponce, cmprosperop@gmail.com**Summary**

The authors report a case of a 46-year-old Hispanic male with central serous chorioretinopathy (CSC) following blunt trauma to the left eye. The patient presented with a complaint of throbbing headache and blurry vision in left eye. The patient was diagnosed with diabetes mellitus 1 year previous to the event. On examination, uncorrected visual acuity was 20/20 OD, 20/200 OS. No anisocoria or afferent pupillary defect was present. Intraocular pressure was normal. Subconjunctival haemorrhage and lid ecchymosis were present in OS and fundus examination showed serous macular detachment and central retinal pigment epithelium detachment, and no evidence of diabetic retinopathy. Optical coherence tomography OS showed subretinal fluid and fluorescein angiography demonstrated the typical 'smokestack' pattern of leakage into the subretinal space. The patient received observational therapy for 4 months and the CSC spontaneously resolved with visual acuity of 20/20 in left eye.

BACKGROUND

Central serous chorioretinopathy (CSC) is a condition characterised by the serous detachment of the neurosensory retina secondary to retinal pigment epithelium dysfunction with or without choriocapillaris involvement. This condition results in decreased visual acuity, metamorphopsia, and decreased contrast sensitivity. To our knowledge, CSC has been previously reported to be idiopathic or in relation to: type A personality, systemic steroid levels, drugs (ecstasy), infection (*Helicobacter pylori*, syphilis, etc), laser-assisted insitu keratomileusis (LASIK) treatment, rhinoplasty, and even after viewing a solar eclipse, but it has not been reported after blunt trauma in the eye.

CASE PRESENTATION

A 46-year-old Hispanic gentleman who presented with blurry vision in the left eye and throbbing headache after he had sustained blunt trauma to his left eye 5 days before the visit. The patient stated that he was diagnosed as a non-insulin dependent diabetic approximately 1 year previous to the visit and started treatment with metformin. The patient denies use of steroids, including systemic, intra-articular, nasal, inhaled, oral, intravenous and topical. He also had a surgery for nose fracture repair 2 years previous to the current event. On examination, the uncorrected visual acuity was 20/20 OD, 20/200 OS not improved with refraction. Pupils were equal and reactive to light and near, with no afferent pupillary defect. Extraocular movements and confrontation visual field showed no restriction in either eye. Intraocular pressure was 19 mm Hg OD and 18 mm Hg OS. Right eye showed normal slit lamp exam (SLE), and fundus examination. On the left side, external examination showed both upper and lower lid ecchymosis, SLE showed temporal subconjunctival hemorrhage in the left eye with no other signs of anterior segment trauma. On fundus examination, no diabetic retinopathy was found, and a serous macular detachment and central retinal pigment epithelium detachment were noted OS

(figure 1). Optic nerves were normal and no evidence of optic disc pit was found. Optical coherence tomography (OCT) in OS showed neurosensory detachment of the left macula (figure 2). Arterio-venous phase (figure 3A) and late

Figure 1 Fundus photograph of the left eye showing neurosensory detachment of most of the macula with retinal pigment epithelium defect superionasal to the fovea (arrow).

Figure 2 Optical coherence tomography of the left eye demonstrating neurosensory detachment of the left macula (arrow).

Figure 3 (A) Arterio-venous phase fluorescein angiography of left eye showing focal leakage superonasal to the fovea. (B) Late phase fluorescein angiography of left eye showing the progressive expansion of hyperfluorescence from the site of leakage (asterisk) and pooling of fluorescein in the area of retinal detachment (arrows). This is the classic 'smokestack' appearance of central serous chorioretinopathy.

phase (figure 3B). Fluorescein angiography (FA) was taken. FA of OS demonstrated the typical 'smokestack' pattern of leakage into the subretinal space, with pooling of fluorescein in the area of detachment on late films (figure 3B). The previous results confirmed the diagnosis of central serous retinopathy following blunt trauma.

DIFFERENTIAL DIAGNOSIS

Vitreomacular traction

- Fundus examination and OCT showed neurosensory detachment of the retina without macular hole or vitreomacular traction.

Polypoidal choroidal vasculopathy

- No nodular hyperfluorescence was found in the early-phase FA typically seen in pattern of polypoidal choroidal vasculopathy.

Occult choroidal neovascularisation (CNV)

- Late mottled hyperfluorescence rather than early hyperfluorescence with pigment epithelial detachment is typical for occult CNV.

Optic disc pit

- No evidence of optic disc pit at fundus examination or FP.
- The leakage pattern of late-phase FA showed the typical appearance of CSC.
- The absence of retinoschisis temporal to disc margin and fluorescein pooling around the ON makes the optic disc pit very unlikely.

TREATMENT

The patient received observation treatment and consecutive fundus examination.

OUTCOME AND FOLLOW-UP

The clinical and OCT picture improved over a 4 month period with residual symptoms of diplopia, flashes and pain in the left eye. Best corrected visual acuity was 20/20 in both eyes with a refractive error of $+0.25+0.25 \times 150$ in OD and $-0.25+0.25 \times 126$ in OS. Fundus examination showed almost complete resolution of the previously noted subretinal fluid in the left eye. FA and OCT in the following visits are shown in figures 4 and 5.

Figure 4 Fluorescein angiography of left eye showing absence of leakage, and the residual window defects in the retinal pigment epithelium layer in the macula, at the 4 months follow-up visit.

Figure 5 Optical coherence tomography of the left eye 4 months after the acute event, showing minimal subretinal fluid (arrow).

DISCUSSION

The first reports of CSC were in 1866, by von Graefe,¹ who thought it was a central relapsing retinitis. After his hypothesis, there were several attempts to describe the pathophysiology of this entity, but despite the effort, little could be done without the understanding of the anatomy of the choriocapillaris, membrane of Bruch and retinal pigment

epithelium (RPE).² It is now known that the pathophysiology involves a disturbance in the permeability of the choriocapillaris leading to dysfunction of the RPE (leakage of electrolytes, proteins and water) that will result in a serous detachment of the neurosensory layer of the retina or the RPE itself.^{2–5} CSC causes visual impairment in all of the cases, and can lead to visual loss if the photoreceptors in the foveal zone are damaged⁶ or if choroidal neovascularisation occurs.⁷ However, the majority of the patients with CSC resolve without treatment, with recovery of their visual acuity.^{2–3} In patients in whom the cause is steroid therapy, the dosage of the medication must be reduced or stopped if possible, in order to improve CSC.^{2–3–8}

CSC has a higher prevalence in male gender and between 20 and 40 years of age.^{2–3} It has been related to type A personality,⁹ high steroid levels,^{2–3–8} recreational drugs such as ecstasy,¹⁰ infection,^{11–12} adrenocorticotrophic hormone treatment,¹³ LASIK procedure,¹⁴ rhinoplasty,¹⁵ and after viewing a solar eclipse.¹⁶ No previous reports of CSC after eye trauma were found in the literature.

The gold standard therapy of CSC has not yet been established, however, previous reports showed benefit in the observational therapy for the first 3 months if no foveal leakage, choroidal neovascularisation or other condition demanding invasive therapy is identified.

Laser treatment may be considered in several scenarios, such as: persistent serous detachment beyond 3–4 months, recurrent disease in an eye with visual deficit due to previous serous detachment, contralateral permanent visual deficit from previous CSC, presence of signs of chronic CSC (cystic changes in neurosensory retina, widespread RPE abnormality, etc) and when prompt restoration of vision is needed for occupational matters.^{2–3–17} Early treatment with laser has been suggested if subretinal granular deposits are found, due to their high association with occult new vessel formation,¹⁸ if the patient has a recurrence, less than 20/40 best corrected visual acuity and aggravated symptoms.^{17–19} Other treatment options are: intravitreal injection of bevacizumab^{2–3–20} and verteporfin photodynamic monotherapy, used when the leakage is near the centre to the macula.¹⁷

In this case, the patient had spontaneous resolution of the neurosensory retinal detachment and improvement of visual acuity. However, regular follow-up visits were needed in his case to rule out foveal atrophy² and chronic CSC²¹ primarily seen 4 months after the acute event.

Learning points

- ▶ In the general practice of ophthalmology it is important to be familiar with the causes of visual loss in a patient that undergoes eye trauma.
- ▶ Although CSC is not commonly the cause, the ophthalmologist needs to exclude it by performing a thorough examination of the anterior and posterior segments of the eye.
- ▶ Observational therapy is the most commonly used therapy if no other condition requiring prompt invasive therapy is present.
- ▶ Understanding the pathophysiology and predisposing risk factors of CSC will help the ophthalmologist finding the best treatment option.

Competing interests In the general practice of ophthalmology it is important to be familiar with the causes of visual loss in a patient that undergoes eye trauma. Although CSC is not commonly the cause, the ophthalmologist needs to discard it by performing a thorough examination of the anterior and posterior segments of the eye.

Patient consent Obtained.

REFERENCES

1. Von Graefe A, Kurzere A. Notizen und casaistische Mittheilungen vermischten Inhalts: VI. Ueber zentrale recidivirende retinitis. *Arch Klin Exp Ophthalmol* 1866;**12**:211–15.
2. Gemenetzi M, De Salvo G, Lotery AJ. Central serous chorioretinopathy: an update on pathogenesis and treatment. *Eye (Lond)* 2010;**24**:1743–56.
3. Wang M, Munch IC, Hasler PW, et al. Central serous chorioretinopathy. *Acta Ophthalmol* 2008;**86**:126–45.
4. Ryan SJ. Central serous chorioretinopathy. *Retina* 2001;**2**:1153–181.
5. Gass JD. Specific diseases causing disciform macular detachment. *Stereoscopic Atlas of Macular Diseases* 1997;**1**:52–70.
6. Shukla D, Kolluru C, Vignesh TP, et al. Transpupillary thermotherapy for subfoveal leaks in central serous chorioretinopathy. *Eye (Lond)* 2008;**22**:100–6.
7. Teschner S, Noack J, Birngruber R, et al. Characterization of leakage activity in exudative chorioretinal disease with three-dimensional confocal angiography. *Ophthalmology* 2003;**110**:687–97.
8. Marcuson J, Riley T. Central serous chorioretinopathy. *Optometry* 2008;**79**:241–51.
9. Yannuzzi LA. Type-A behavior and central serous chorioretinopathy. *Retina (Philadelphia, Pa)* 1987;**7**:111–31.
10. Hassan L, Carvalho C, Yannuzzi LA, et al. Central serous chorioretinopathy in a patient using methylenedioxymethamphetamine (MDMA) or 'ecstasy'. *Retina* 2001;**21**:559–61.
11. Rahbani-Nobar MB, Javadzadeh A, Ghojatzadeh L, et al. The effect of *Helicobacter pylori* treatment on remission of idiopathic central serous chorioretinopathy. *Mol Vis* 2011;**17**:99–103.
12. Fuchs E. Ein Fall von zentraler residivirender syphilitischer Netzhaut Entzündung. *Zentralbl Prakt Augenheilkd* 1916;**40**:105–8.
13. Zamir E. Central serous retinopathy associated with adrenocorticotrophic hormone therapy. A case report and a hypothesis. *Graefes Arch Clin Exp Ophthalmol* 1997;**235**:339–44.
14. Vasileios GP, Syridon EC, Spyridon DN, et al. Bilateral central serous retinopathy following laser in situ keratomileusis for myopia. *J Cataract Refract Surg* 2011;**37**:778–80.
15. Moschos MM, Droutsas K, Margetis I. Central Serous Chorioretinopathy after Rhinoplasty. *Case Report Ophthalmol* 2010;**1**:90–3.
16. Lee A, Lai TY. Central serous chorioretinopathy after solar eclipse viewing. *J Ophthalmic Vision Res* 2010;**5**:193–5.
17. Basic and Clinical Science Course, Section 12. *Retina and Vitreous*. San Francisco: American Academy of Ophthalmology. 2010;55–9.
18. Wang M, Sander B, la Cour M, et al. Clinical characteristics of subretinal deposits in central serous chorioretinopathy. *Acta Ophthalmol Scand* 2005;**83**:691–6.
19. Robertson DM. Argon laser photocoagulation treatment in central serous chorioretinopathy. *Ophthalmology* 1986;**93**:972–4.
20. Lim JW, Kim MU. The efficacy of intravitreal bevacizumab for idiopathic central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol* 2011;**249**:969–74.
21. Polak BC, Baarsma GS, Snyers B. Diffuse retinal pigment epitheliopathy complicating systemic corticosteroid treatment. *Br J Ophthalmol* 1995;**79**:922–5.

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