CASE REPORT

Persistent unilateral central serous chorioretinopathy in a breastfeeding woman

Christine Elizabeth Lopez-Yang,¹ Charles A Garcia²

¹Department of Ophthalmology, Retinal Vascular Center, Houston, Texas, USA

²Department of Ophthalmology, University of Texas Health Science Center at Houston, Houston, Texas, USA

Correspondence to

Dr Christine Elizabeth Lopez-Yang,
c.lopez.yang@gmail.com

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SUMMARY

A 40-year-old woman presented with blurred vision and floaters in her right eye for the past 7 months. The patient was in the sixth month of pregnancy at onset of the ocular symptoms and had persistent ocular disturbances postpartum. Her medical and ocular history were unremarkable except for LASIK surgery. Examination revealed an uncorrected visual acuity of 20/30 and 20/20 in the right and left eye, respectively. Fundus examination showed a round, subretinal exudate involving the foveal centre. Central serous chorioretinopathy was diagnosed by fluorescein angiogram and optical coherence tomography (OCT) showing foveal neurosensory detachment and treated with intravitreal bevacizumab. At her 4-month follow-up (8 month postpartum), OCT continued to show persistent foveal subretinal fluid. Patient declined further treatment and on follow-up 1 year later, still showed a persistent neurosensory detachment on OCT testing.

BACKGROUND

Idiopathic central serous chorioretinopathy (CSC) is a commonly recognised entity described as a localised macular serous detachment secondary to disruption of the pigment epithelium.¹

There has not been a clear aetiology for the disorder, but it has been associated with psychosocial stress due to transient life events, type A personalities, psychopharmacological medications, corticosteroid use and hypertension.²⁻³ Idiopathic central serous chorioretinopathy is also a recognised complication of pregnancy, possibly due to the elevation of serum catecholamine levels and increased production of corticosteroids, oestrogen and other hormones that occur during this physiological state.⁴

A literature search was conducted on PubMed database in December 2013 with key words 'central serous retinopathy/choroidopathy', 'pregnancy' and 'women'. Articles were all in English. All of the cases reviewed have had resolution of pregnancy associated CSC in their patients on delivery or soon after.

In this case, we report a patient who presented with CSC during her second trimester of pregnancy, but did not have complete anatomical or symptomatic resolution up to her 17-month follow-up postpartum.

CASE PRESENTATION

A 40-year-old woman was referred to our retinal service, presenting with new onset of blurred vision and floaters in her right eye for the past 7 months.

The patient was in the sixth month of her first pregnancy when her symptoms began and had a normal delivery at 9 months with persistent ocular disturbances. The patient's ocular history revealed LASIK surgery performed 10 years previously. Her medical history was unremarkable, with present medications being Nexium and a multivitamin. Social history was relevant for occupation—the patient was a medical resident and was studying for her board examinations at that time. Family history was unremarkable.

INVESTIGATIONS

Examination at the time of presentation revealed uncorrected Snellen visual acuities of 20/30+1 right and 20/20 left. The slit lamp examination was unremarkable and the fundus examination showed a round, subretinal exudate in the right eye involving the foveal centre. Optical coherence tomography (OCT; figure 1) revealed subretinal fluid underneath the fovea.

DIFFERENTIAL DIAGNOSIS

Macular degeneration, ocular histoplasmosis, polypoidal choroidal vasculopathy, optic nerve pit, retinal hole, malignant hypertension and toxemia of pregnancy.⁵

Figure 1 Optical coherence tomography of the macula showing subretinal fluid underneath the fovea with no signs of retinal pigment epithelium disruption.

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TREATMENT

Patient was treated on presentation with a single dose of 0.05 cc intravitreal bevacizumab (25 mg/mL; Avastin) in the right eye.

OUTCOME AND FOLLOW-UP

On follow-up with the patient 4 weeks later, she still reported blurred vision in the right eye and fluctuating, blurred vision in her left eye. Snellen uncorrected visual acuities were 20/30–1 right and 20/20 left eye, respectively. Fundus examination was normal in the left eye; right eye showed retinal pigment epithelium (RPE) mottling around the fovea and temporally. Fundus fluorescein angiography (FA; [figure 2](#)) was conducted at this time and showed minimal leakage near the macula and the same findings described above. It was decided to defer treatment and to see if spontaneous resolution occurred and considering additional treatment if she had worsening symptoms.

Patient returned 3 months later and reported having occasional blurred vision, especially at night time, as well as ‘a darkness’ in vision in the right eye. Snellen uncorrected visual acuities were 20/30–1 right and 20/20–2 left. Fundus examination was normal in the left eye; the right eye showed persistent subretinal subfoveal exudate. OCT was performed showing subretinal fluid involving the foveal centre, cerebrospinal fluid: 273 μ m ([figure 3](#)). Patient deferred further treatment at this time and was advised to return in 6 weeks for follow-up.

She returned almost a year later with the same symptoms of floaters and blurred night vision in her right eye. Snellen uncorrected visual acuities were the same but on fundus photography with autofluorescence, the right eye now showed diffuse RPE changes in the macula ([figure 4A, B](#)). OCT ([figure 5](#)) demonstrated persistent neurosensory detachment underneath the fovea in the right eye; left eye was unremarkable. The patient was advised to monitor changes in her vision and to return in 6 months to perform an FA and rule out a possible progression to choroidal neovascular membrane.

DISCUSSION

Cortisol may be an important causative factor in CSC by increasing the permeability of choroidal vessels, altering the ion transport and delaying wound healing, all which contribute to the accumulation of subretinal fluid.⁶ Pregnancy is associated

Figure 2 Fluorescein angiogram of the right eye showing delayed choroidal filling with retinal pigment epithelium atrophic changes in the macula, seen as focal areas of hyperfluorescence surrounded by an area of hypofluorescence.

Figure 3 Optical coherence tomography of the macula showing persistent subretinal fluid underneath the fovea.

Figure 4 (A) Colour fundus photograph of the right eye showing the macular lesion with some faint retinal pigment epithelium changes adjacent inferior temporal. (B) Autofluorescence fundus photograph showing more clearly an oval area of mottled, increased fluorescence adjacent to the macular lesion towards the inferior temporal arcade.

Figure 5 Optical coherence tomography of the macula showing subfoveal neurosensory detachment underneath the fovea.

with a state of increased hypothalamic-pituitary-adrenal axis function, as shown by elevations in urine free cortisol, plasma 17-hydroxysteroids, total and free plasma cortisol, and corticosteroid-binding globulin values, suggesting greater tissue exposure to glucocorticoids during pregnancy.^{7,8} High progesterone concentrations during pregnancy have an antiglucocorticoid action, which may explain the resistance to cortisol action with subsequent increased free cortisol levels. Allolio *et al*⁹ demonstrated significant correlations between serum progesterone and salivary cortisol during late pregnancy.

Our patient presented with her symptoms during her second trimester of pregnancy, which is classically described in various studies.^{2,4,8,10,11} Hirji *et al*¹² have described CSC as a finding more commonly associated with a complicated pregnancy (eg, eclampsia), but most cases reported in the literature deal with uncomplicated pregnancies.

Most cases have spontaneous resolution of the subretinal fluid within 4 months of its onset.

The presence of subretinal precipitates in OCT imaging appears to be associated with an increased probability of complete recovery and may be accumulations of shed photoreceptor outer segments in the subretinal space, accumulations of fibrin or lipid, or macrophages clearing the subretinal space of such debris.^{4,6,13}

Some cases, though, experience permanent visual disturbances or diminished visual acuity consistent with persistent or recurrent detachments. These are associated with more diffuse changes and damage in the RPE, diffuse fluorescein leakage and more severely depressed visual dysfunction.^{6,14} These patients have long-standing subretinal fluid that cannot be reabsorbed efficiently because of choroidal disease and extensive dysfunction and loss of the RPE. Clinical factors associated with this incomplete resolution include absence of subretinal precipitates, recurrence, hormone replacement therapy, duration greater than 5 months and presence of pigment epithelial detachment (PED).⁶ In long-standing CSC, findings may include RPE atrophy that exposes underlying choroidal vascular patterns, areas of RPE pigment clumping and even bone spicules.¹³

Since levels of progesterone drop substantially postpartum, the persistence of the symptoms in this particular patient cannot be explained through the mechanism of progesterone-cortisol associated levels alone. We suggest instead that the absence of early resolution of the subretinal fluid and secondary dysfunction of the RPE are findings consistent with RPE mottling

around the fovea and infero/temporally, and an absence of subretinal precipitates could have made this a chronic, persistent case of CSC.

The management of acute cases of CSC is usually conservative. Since most of these cases resolve spontaneously in a matter of 6–12 weeks, it is advisable to observe the eye closely. In recurrent or persistent cases, the treatment depends on the location of the lesion. If the lesion is focal and extrafoveal, focal photocoagulation is acceptable but in cases in which the leak is subfoveal or the leakage pattern is diffuse, photodynamic therapy has been shown to be beneficial.^{2,3,15} Small case series have shown potential benefit of treating CSC with bevacizumab.¹⁶ However, another case series showed no beneficial effect of bevacizumab on CSC.¹⁵

Breast feeding also complicated the selection of diagnostic procedures and treatment for this patient. The excretion of drugs into milk depends on several factors which include lipid solubility, molecular size, ionisation, concentration and half-life in maternal plasma, protein binding and breastfeeding phase. The most important factor determining the concentration of the medication present in breast milk is maternal serum levels.¹⁷

Fluorescein can be detected in human milk after administering it intravenously in women who are lactating. The levels measured are minimal and are unlikely to cause phototoxic reactions in breastfeeding infants. Nevertheless, since it can still be found in breast milk up to 62 h after administration, there may be a cumulative effect. It is recommendable to avoid exposing the neonate to intense light a few days after the maternal procedure.¹⁸

Since verteporfin (category C drug) is excreted in human milk, photodynamic therapy (PDT) was not recommended as a form of treatment. Instead, intravitreal bevacizumab single dose treatment was offered. Plasma to milk transfer is highest during the first week of breast feeding due to the presence of larger gaps between the alveolar breast cells and the same might be true during the last part of breast feeding, when glandular involution occurs and milk amount decreases. Large molecules that have a high molecular weight, such as monoclonal antibodies (eg, IgG), hardly penetrate into human milk.¹⁷ Unlike many animal species, human IgG secreted in breast milk is not absorbed across the intestinal epithelium. The potential for adverse reactions in nursing infants from intravitreal bevacizumab is, therefore, unknown and the decision to treat and/or continue nursing should be made taking into account these findings.¹⁹

Since the patient did not have a significant improvement in either her symptoms or in OCT imaging after bevacizumab treatment, it was decided to continue with observation only.

Newer advances in diagnostic imaging, such as indocyanine green angiography and fundus autofluorescence, may also help identify changes in the RPE and choroid of which we were previously unaware of or improve their visualisation.^{1 2} Roisman *et al*²⁰ recently conducted a study in which macular microperimetry was performed in patients at the onset of CSC. Those with a macular sensitivity of less than 20 dB had a 4.5 times higher risk of developing non-resolution after a 3-month follow-up. The results of this study suggest that microperimetry may be a valuable test to assess the prognosis of acute CSC. It may help decide to provide early treatment, reducing the chance of permanent vision impairment and CSC chronicity. These imaging modalities may help guide the treatment and response to treatment in patients for whom visual acuity may not be sufficient to quantify improvement.

Learning points

- ▶ Central serous chorioretinopathy (CSC) is a recognised complication of uncomplicated pregnancies.
- ▶ Pregnancy related CSC may not always spontaneously resolve.
- ▶ Early recognition of findings associated with poor prognosis and microperimetry may aid in timely treatment and resolution of this disease.
- ▶ Special considerations for pregnant/nursing women poses a challenge to finding an appropriate treatment that will not harm the fetus/child or deferring treatment until it is a viable option.

Contributors CEL-Y conducted all patient data review, wrote the first draft of the manuscript and rewrote new drafts based on input from co-authors. CAG gave the initial manuscript idea and gave input on manuscript drafts. All authors read and approved the final manuscript.

Competing interests None.

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REFERENCES

- 1 Eandi CM, Ober M, Iranmanesh R, *et al*. Acute central serous chorioretinopathy and fundus autofluorescence. *Retina* 2005;25:989–93.
- 2 Wang M, Munch IC, Hasler PW, *et al*. Central serous chorioretinopathy. *Acta Ophthalmol* 2008;86:126–45.
- 3 Ergun E, Tittl M, Stur M. Photodynamic therapy with verteporfin in subfoveal choroidal neovascularization secondary to central serous chorioretinopathy. *Arch Ophthalmol* 2004;122:37–41.
- 4 Gass JD. Central serous chorioretinopathy and white subretinal exudation during pregnancy. *Arch Ophthalmol* 1991;109:677–81.
- 5 Shuler RK, Mruthyunjaya P. Diagnosing and managing central serous chorioretinopathy. *AAO EyeNet* 2006 (cited 2014 May 20). <http://www.aao.org/publications/eyenet/200602/pearls.cfm>
- 6 Perkins S, Kim J, Pollack J, *et al*. Clinical characteristics of central serous chorioretinopathy in women. *AAO* 2002;109:262–6.
- 7 Lindsay JR, Nieman LK. The hypothalamic-pituitary-adrenal axis in pregnancy: challenges in disease detection and treatment. *Endocr Rev* 2005;26:775–99.
- 8 Sunness J, Haller J, Fine S. Central serous chorioretinopathy and pregnancy. *Arch Ophthalmol* 1993;111:360–4.
- 9 Allolio B, Hoffmann J, Linton EA, *et al*. Diurnal salivary cortisol patterns during pregnancy and after delivery: relationship to plasma corticotrophin-releasing-hormone. *J Clin Transl Endocrinol* 1990;33:279–89.
- 10 Bouzas EA, Scott MH, Mastorakos G, *et al*. Central serous chorioretinopathy in endogenous hypercortisolism. *Arch Ophthalmol* 1993;111:1229–33.
- 11 Quillen DA, Gass DM, Brod RD, *et al*. Central serous chorioretinopathy in women. *Ophthalmology* 1996;103:72–9.
- 12 Hirji N, Watt L, Richardson E. Central serous chorioretinopathy secondary to childbirth. *BMJ Case Rep* 2010;2010:pil: bcr1020103387.
- 13 Nicholson B, Noble J, Forooghian F, *et al*. Central serous chorioretinopathy: update on pathophysiology and treatment. *Surv Ophthalmol* 2013;58:103–26.
- 14 Wang MS, Sander B, Larsen M. Retinal atrophy in idiopathic central serous chorioretinopathy. *Am J Ophthalmol* 2002;133:787–93.
- 15 Colucciello M. Update on central serous chorioretinopathy. *Retin Physician* 2012;9:42–7, 75.
- 16 Lim JW, Kim MU. The efficacy of intravitreal bevacizumab for idiopathic central serous chorioretinopathy. *Arch Clin Exp Ophthalmol* 2011;249:969–74.
- 17 Pistilli B, Belletini G, Giovannetti E, *et al*. Chemotherapy, targeted agents, antiemetics and growth-factors in human milk: how should we counsel cancer patients about breastfeeding? *Cancer Treat Rev* 2013;39:207–11.
- 18 Maguire AM, Bennett J. Fluorescein elimination in human breast milk. *Arch Ophthalmol* 1988;106:718–19.
- 19 Tarantola RM, Folk JC, Boldt HC, *et al*. Intravitreal bevacizumab during pregnancy. *Retina* 2010;30:1405–11.
- 20 Roisman L, Ribeiro J, Fechine F, *et al*. Does microperimetry have a prognostic value in central serous chorioretinopathy? *Retina* 2014;34:713–18.

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