(45)

Central serous chorioretinopathy during pregnancy complicated by systemic hypertension – a case report

Centralna surowicza chorioretinopatia u kobiet w ciąży powikłanej nadciśnieniem tętniczym – opis przypadku

Maciej Gawęcki, Ewa Groszewska

Ophthalmology Ward, J.K. Łukowicz Specialist Hospital in Chojnice
Head: Maciej Gawęcki, PhD, MD

Summary: We present a case of a pregnant woman with pregnancy induced systemic hypertension who developed bilateral central serous chorioretinopathy diagnosed after caesarean delivery. Central serous chorioretinopathy can occur in the third trimester of pregnancy and generally has a benign course and good prognosis. Our patient ended up with significant and permanent visual impairment in one eye. Probably in some cases, early ophthalmic diagnostic management, including optical coherence tomography could help identify patients at risk of pemanent visual impairment due to chronic central serous chorioretinopathy and assess their eligibility for subtreshold micropulse laser treatment.

Key words: central serous chorioretinopathy, central serous retinal detachment, optical coherence tomography, systemic hypertension.

Streszczenie: Przedstawiamy przypadek pierwiastki z ciążowym nadciśnieniem tętniczym, u której po porodzie cesarskim stwierdzono obustronną surowiczą chorioretinopatię. Obustronna surowicza chorioretinopatia może wystąpić w trzecim trymestrze ciąży i zwykle ma łagodny przebieg oraz dobre rokowanie. W prezentowanym przypadku u pacjentki doszło do trwałego pogorszenia ostrości wzroku w jednym oku. W niektórych przypadkach wczesna diagnostyka okulistyczna z zastosowaniem tomografii siatkówki pomogłaby wyodrębnić grupę pacjentek, u których przewlekła postać obustronnej surowiczej chorioretinopatii w przebiegu ciąży niesie ryzyko pogorszenia ostrości wzroku, i zakwalifikować je do leczenia za pomocą podprogowej laseroterapii mikropulsowej.

Słowa kluczowe: centralna surowicza chorioretinopatia, centralne surowicze odwarstwienie siatkówki, optyczna koherentna tomografia, nadciśnienie tętnicze.

Introduction

Ocular complications during pregnancy are not a common occurrence. The clinical entities which can manifest as ocular fundus abnormalities include pregnancy induced hypertension, preeclampsia, central serous chorioretinopathy (CSCR), Purtscher-like retinopathy, diffuse intravascular coagulation and emboli originating from amniotic fluid (1,2). CSCR in pregnancy is described as ocular disease occurring in the third trimester of otherwise uncomplicated pregnancy (3). The underlying cause of the disease is the elevated level of endogenous cortisol, which increases the permeability of choriocapillaries and retinal pigment epithelium (RPE) (4). In rare cases, CSCR or central serous retinal detachment (CSRD) can develop during pregnancy secondarily to systemic hypertension, especially preeclampsia (5–8). What differs preeclampsia from eclampsia is that the latter occurs as a sudden event close to the end of pregnancy and does not have a chronic course. Nevertheless, the presentation of both conditions can be very similar and both are associated with good prognosis.

CSCR in general ophthalmology is a relatively common disease usually affecting young men between 20 and 50 years of age. However, its etiopathogenesis has not been fully understood yet. Most frequently ophthalmologists deal with the idiopathic CSCR, with severe stress considered to be a trigger, and type A personality a predisposing factor (9). CSCR can also develop after systemic steroid therapy or be concomitant with other ocular diseases, such a retinitis pigmentosa, chorioretinal folds or episcleritis. The actual mechanism of CSCR is still only hypothesised. For many years two major theories about pathogenesis of the disease have been mentioned. The first one assumes that the RPE pathology, which causes the loss of its properties of a pump to eliminate subretinal fluid (10), leads to serous fluid accumulation beneath the sensory retina. According to the second theory, the initial pathology affects choriocapillaries, significantly increasing their permeability. In the consequence of pressure increase, the serous fluid is pushed into the sensory retina. Both theories may overlap: choriocapillary abnormalities may be accompanied by the RPE pathology. Recently, though, the authors more often stress the key role of endogenous and exogenous corticosteroids in the pathogenesis of the disease (4). It seems that overactive mineralocorticoid pathway induces secondary changes to the choroid (leakage, increased permeability) and in consequence disruption of the RPE cells.

ISSN 0023-2157 Index 362646

The course of CSCR is usually benign. Subretinal fluid usually resorbs spontaneously within 1–3 months. Decrease of visual acuity is insignificant in most cases; however, patients mainly report reduced vision quality. In less frequent cases, chronic CSCR develops, which lasts over 6 months, sometimes even several years. The long-term presence of subretinal fluid causes permanent damage to the photoreceptors and, in turn, significant and permanent vision impairment (11–13). Recurrent CSCR affects $^1/_3$ of all cases. Choroidal neovascularization (CNV), which occurs in about 8% of all cases, is a vision threatening complication of CSCR. It requires prompt and precise diagnosis, as effective treatment is possible with intravitreal injections of vascular endothelial growth factor inhibitors (anti-VEGF) (14, 15).

Case report

A 22-year old female in the 33rd week of her first pregnancy was admitted to the Department of Gynaecology and Obstetrics as an emergency due to significant systemic hypertension and proteinuria. As the attempts to treat the condition medically failed, the decision was made to terminate the pregnancy by cesarean section. Directly after the childbirth, the patient complained of significant bilateral visual acuity (VA) decrease. Ocular exam conducted on the same day revealed the distance visual acuity of 0.05 and 0.20 in the right and left eye, respectively. The near VA was 0.125 in both eyes. As the general health status of the patient made it difficult for her to take and remain in an upright position for other diagnostic tests, ultrasound ocular exam was ordered, which revealed bilateral, local serous detachment of the sensory retina (Fig. 1). She was diagnosed with CSRD secondary to preeclampsia and a more detailed exam with additional diagnostic tests was scheduled after her postoperative recovery. Two days later, the patient had a full ophthalmic examination, which showed bilateral visual acuity improvement to 0.50. Spectral domain optical coherence tomography of the retina (SOCT) confirmed the presence of serous retinal detachment in both eyes (Fig. 2), although the amount of subretinal fluid was not significant and the area of CSRD was limited in the right eye. A small area of moderate hyperreflectance was shown within the hyporeflective subretinal fluid space in the RE, most likely representing fibrin accumulation. In the LE, a large amount of subretinal fluid was present, especially in the temporal part of the macular area. We revealed numerous hyperreflective dots along the internal margins

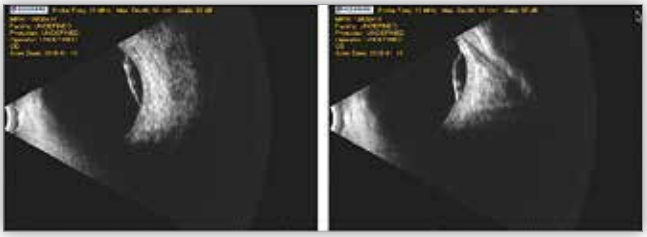

Fig. 1. Ultrasound scan of both eyes performed one day after delivery. A significant detachment of sensory retina is visible bilaterally in the posterior pole.

Ryc. 1. Badanie ultrasonograficzne obu gałek ocznych wykonane dzień po porodzie. Widoczne znaczne surowicze odwarstwienie siatkówki sensorycznej w biegunie tylnym siatkówki w obojgu oczach.

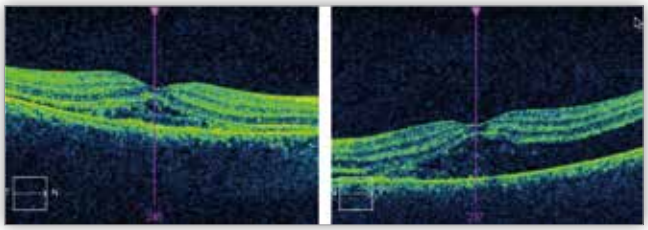

Fig. 2. Retinal SOCT performed two days after childbirth. Serous retinal detachment, more extensive in the LE, is visible in the scans. Visible areas of moderate hyperreflectivity within the fluid space represent the accumulation of fibrin and macrophages.

Ryc. 2. Spektralna optyczna tomografia siatkówki obojga oczu wykonana po 2 dniach od porodu. Na skanach widoczne surowicze odwarstwienie siatkówki sensorycznej, większe w OL. W obrębie przestrzeni płynowej widoczne obszary umiarkowanej hyperrefleksyjności odpowiadające złogom fibryny i kumulacji makrofagów.

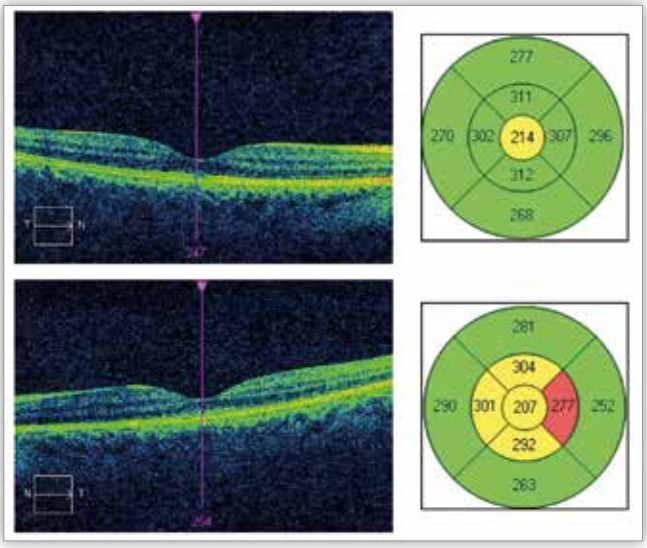

Fig. 3. SOCT of both eyes and retinal thickness maps obtained one month after childbirth. The previously observed subretinal fluid has resolved, but the retinal thinning is present, especially in the LE (red and yellow areas).

Ryc. 3. Spektralna optyczna tomografia siatkówki obojga oczu oraz mapy grubości siatkówki wykonane po miesiącu od porodu. Na skanach widoczna zupełna resorpcja płynu podsiatkówkowego, lecz na mapach grubości siatkówki widoczne wyraźne ich ścieńczenie, zwłaszcza w OL (barwy żółta i czerwona).

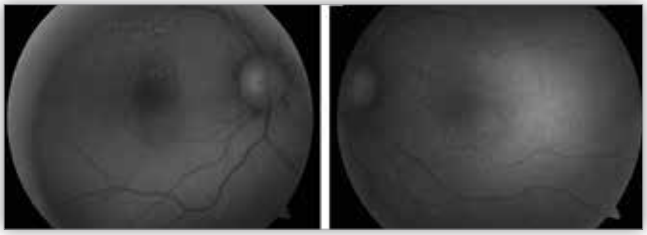

Fig. 4. Autofluorescence images of the fundus taken one month after childbirth. In the posterior pole, the punctuate areas of hyperfluorescence are visible, representing lipofuscin accumulation in the RPE cells secondarily to chronic CSCR.

Ryc. 4. Zdjęcia autofluorescencji dna oka wykonane po miesiącu od porodu. W biegunie tylnym widoczne drobne punktowate ogniska hyperfluorescencji odpowiadajace złogom lipofuscyny w komórkach RPE po przebytej surowiczej chorioretinopatii.

of this hyporeflective fluid space, which depicted the macrophage migration in that area and confirmed the chronic manifestation of CSCR. The outpatient follow up exam performed one month after childbirth showed a complete resolution of subretinal fluid (Fig. 3). The visual acuity returned to normal in the RE (1.0), but in the LE, the BCVA remained at the level of 0.50. The SOCT scans showed sectoral retinal thinning in the macular area. The autofluorescence image of the fundus demonstrated tiny spots of hyperfluorescence in the same areas where subretinal fluid had previously been observed, which represented lipofuscin accumulation in RPE cells (Fig. 4).

Discussion

CSCR or CSRD in women with pregnancy-induced hypertension is not frequently reported. The course of preeclampsia, which may develop during pregnancy, is rather dramatic and requires surgical termination of pregnancy by cesarean section, with ophthalmology consultation being of secondary importance in such circumstances. Any noticeable visual disturbances usually resolve without treatment. Serous chorioretinopathy and central serous retinal detachment are characteristic ocular complications observed in preeclampsia. CSRD develops in less than 1% of women with preeclampsia. Systemic hypertension results in angiospasm, which causes choroidal ischemia leading to the serous detachment of sensory retina. Similarly to idiopathic CSCR, quite common in ophthalmology, both CSCR and CSRD in pregnancy usually have good prognosis. Commonly, after a few days or weeks, subretinal fluid resolves spontaneously and the visual acuity returns to normal (16). In our patient, the normal VA was back just in one eye, with the second eye affected by the permanent vision impairment presenting as retinal thinning. SOCT scans showed the presence of fibrin in the fluid space and the presence of macrophages at the margins of the detachment. These findings are suggestive of a chronic course of the disease (17, 18). Fibrinogen can be present in some cases of CSCR during pregnancy, however the prevalence of this phenomenon is not clear. Sunness reported its presence in 90% of pregnant women with CSCR, but later reports did not confirm such high prevalence (19). Additionally, macrophage migration is quite typical of long-term detachment of neurosensory retina. In our opinion, benign serous chorioretinopathy had most likely been present earlier during pregnancy. Preeclampsia, which developed subsequently, caused an exacerbation of the retinopathy subjectively perceived by the patient as a sudden vision deterioration, which made her seek specialist help. We also cannot exclude the overlap of chronic CSRC and sudden CSRD in preeclampsia. Although patients with pregnancy-induced hypertension are quite often referred to ophthalmologists, the OCT is not a routine procedure in such cases. Optical coherence tomography is a non-invasive and safe procedure, which enables easy diagnosis and monitoring of CSCR. It is widely accessible and can be performed in most eye clinics. However, before we order OCT in a pregnant patient with systemic hypertension, we have to answer the question, whether an early diagnosis of CSCR during pregnancy actually leads to treatment commencement. In most cases of CSCR, especially of sudden onset, the initial recommendation is watchful waiting as the prognosis is typically good. All chronic cases, which they may result in retinal damage, are challenging, though. So far, there is no systemic or topical medication of proven efficacy in CSCR. The best results were achieved with intravitreal injections of vascular endothelial growth factor inhibitors (anti-VEGF therapy). However, their use during pregnancy is generally avoided as they may pose a risk to embryo-fetal development. Just recently, promising results of subthreshold micropulse laser treatment in chronic CSCR have been published (20–22). This treatment is not associated with any systemic risks, so can be safely used in pregnant patients. If it is available, early OCT-based diagnosis of CSCR in pregnant women as well as the proper follow up could protect patients from the devastating consequences of chronic manifestation of this disease.

Conclusion

Patients with pregnancy-induced hypertension should undergo routine ocular exams including optical coherence tomography of the retina. Untreated chronic CSCR in pregnancy poses a risk of visual impairment.

References:

1. Errera MH, Kohly RP, da Cruz L: *Pregnancy-associated retinal diseases and their management*. Surv Ophthalmol. 2013 Mar-Apr; 58(2): 127–142.
2. Gotovac M, Kastelan S, Lukenda A: *Eye and pregnancy*. Coll Antropol. 2013 Apr; 37 Suppl 1: 189–193.
3. Normalina M, Zainal M, Alias D: *Central serous choroidopathy in pregnancy*. Med J Malaysia. 1998 Dec; 53(4): 439–441.
4. Daruich A, Matet A, Dirani A, Bousquet E, Zhao M, Farman N, et al.: *Central serous chorioretinopathy: Recent findings and new physiopathology hypothesis.* Progress in Retinal and Eye Research 2015, http://dx.doi.org/10.1016/j.preteyeres.2015.05.003.
5. Bartczak A, Kraśnicki P, Urban R, Laudaiński T, Mariak Z: *Bilateral serous retinal detachment in preeclampsia – a case report*. Klin Oczna. 2014; 116(1): 21–23.
6. Dewilde E, Huygens M, Cools G, Van Calster J: *Hypertensive choroidopathy in pre-eclampsia: two consecutive cases*. Ophthalmic Surg Lasers Imaging Retina. 2014 Jul-Aug; 45(4): 343––346.
7. Handor H, Daoudi R: *Images in clinical medicine. Hypertensive retinopathy associated with preeclampsia*. N Engl J Med. 2014 Feb 20; 370(8): 752.
8. Hafidi Z, Handor H, Oudanane S, Laghmari M, Derrar R, Sabrane I, et al.: *Bilateral exudative retinal detachment as presenting sign of preeclampsia*. J Fr Ophtalmol. 2014 May; 37(5): e65–66.
9. Haimovici R, Koh S, Gagnon DR, Lehrfeld T, Wellik S: *Central Serous Chorioretinopathy Case-Control Study Group. Risk factors for central serous chorioretinopathy: a case-control study*. Ophthalmology. 2004 Feb; 111(2): 244–249.
10. Chuang EL, Sharp DM, Fitzke FW, Kemp CM, Holden AL, Bird AC: *Retinal dysfunction in central serous retinopathy*. Eye (Lond). 1987; 1 (Pt 1): 120–125.
11. Mazzuca DE, Benson WE: *Central serous retinopathy: variants*. Surv Ophthalmol. 1986 Nov-Dec; 31(3): 170–174.
12. Gilbert CM, Owens SL, Smith PD, Fine SL: *Long-term follow-up of central serous chorioretinopathy*. Br J Ophthalmol. 1984 Nov; 68(11): 815–820.

ISSN 0023-2157 Index 362646

13. Levine R, Brucker AJ, Robinson F: *Long-term follow-up of idiopathic central serous chorioretinopathy by fluorescein angiography*. Ophthalmology. 1989 Jun; 96(6): 854–859.
14. Konstantinidis L, Mantel I, Zografos L, Ambresin A: *Intravitreal ranibizumab in the treatment of choroidal neovascularization associated with idiopathic central serous chorioretinopathy*. Eur J Ophthalmol. 2010 Sep-Oct; 20(5): 955–958.
15. Lim JW, Kim MU: *The efficacy of intravitreal bevacizumab for idiopathic central serous chorioretinopathy*. Graefes Arch Clin Exp Ophthalmol. 2011Jul; 249(7): 969–974.
16. Al-Mujaini A, Wali U, Ganesh A, Montana C: *Natural course of central serous chorioretinopathy without subretinal exudates in normal pregnancy*. Can J Ophthalmol. 2008 Oct; 43(5): 588–590.
17. Mayo GL, Tolentino MJ: *Images in clinical medicine. Central serous chorioretinopathy in pregnancy*. N Engl J Med. 2005 Aug 18; 353(7): e6.
18. Rezai KA, Eliott D: *Optical coherence tomographic findings in pregnancy-associated central serous chorioretinopathy*. Graefes Arch Clin Exp Ophthalmol. 2004 Dec; 242(12): 1014–1016.
19. Sunness JS, Haller JA, Fine SL: *Central serous chorioretinopathy and pregnancy*. Arch Ophthalmol 1993; 111: 360–364.
20. Yadav NK, Jayadev C, Mohan A, Vijayan P, Battu R, Dabir S, et al.: Medscape. *Subthreshold micropulse yellow laser (577nm) in chronic central serous chorioretinopathy: safety profile and treatment outcome.* Eye (Lond). 2015 Feb; 29(2): 258–265.
21. Gupta B, Elagouz M, McHugh D, Chong V, Sivaprasad S: *Micropulse diode laser photocoagulation for central serous chorio-retinopathy*. Clin Experiment Ophthalmol. 2009 Nov; 37(8): 801–805.
22. Lanzetta P, Furlan F, Morgante L, Veritti D, Bandello F: *Nonvisible subthreshold micropulse diode laser (810 nm) treatment of central serous chorioretinopathy*. *A pilot study.* Eur J Ophthalmol. 2008 Nov-Dec; 18(6): 934–940.

The study was originally received 01.04.2015 r. (KO-00004-2015)/
Praca wpłynęła do Redakcji 01.04.2015 r. (KO-00004-2015)
Accepted for publication 29.12.2015 /
Zakwalifikowano do druku 29.12.2015 r.

Adres do korespondencji (Reprint requests to):
dr n. med. Maciej Gawęcki
Oddział okulistyczny Szpitala Specjalistycznego
im. J.K. Łukowicza w Chojnicach
ul. Leśna 10
89-600 Chojnice
e-mail: gawecki@use.pl