Research Article

Comparison of optical coherence tomography findings in males and females with acute central serous chorioretinopathy

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Abstract

Objective: To compare the optical coherence tomography (OCT) findings in males and females with acute central serous chorioretinopathy (CSCR).

Materials and methods: In this retrospective study, the OCT scans of 8 females (Group 1) and 16 males (Group 2) cases with acute CSCR which were diagnosed based on clinical, ophthalmological and OCT findings and which was followed in our hospital were retrospectively reviewed. The existence of the OCT findings and patterns regarding CSCR and sub-foveal choroidal thicknesses (SFCTs) were compared.

Results: Serous macula detachment (SMD) was present in all of the female and male cases. The most common OCT finding was the brush-border pattern (6 cases, 75%) except SMD and retina pigment epithelium (RPE) bulging in females, and RPE bulging (14 cases, 87.5%) except SMD in males. There was no statistically significant difference in the frequency of OCT findings and patterns between both groups (p>0.05). The mean SFCTs of the eyes with CSCR in Group 1 and Group 2 were 346.08±32.1 μm (ranged from 338 to 365 μm) and 351.32±36.5 μm (ranged from 344 to 376 μm), respectively. The difference between SFCTs in the affected eyes was also not statistically significant (p>0.05).

Conclusion: This pilot study suggests that there is no difference between the frequencies of OCT findings and patterns of the female and male patients with acute CSCR.

Introduction

Central serous chorioretinopathy (CSC) is a common retino-choroidal disease characterized by serous detachment of the neurosensory retina and/or the retinal pigment epithelium (RPE) in the macula. It has a benign and self-limiting nature, with a long-term recurrence rate of 30%. CSCR usually occur in males in 20-50 years [1-6].

Although the pathogenesis of disease is well unknown, it has been considered that focal RPE defect or choroidal lobular ischemia and choroidal venous congestion may be played in the main role [5-8]. Recent studies have demonstrated that the CSC is associated with psychological stress, type A personality, glucocorticoid treatment, endogenous hypercortisolism like Cushing's syndrome, systemic hypertension, and pregnancy [1-9].

Spectral domain optical coherence tomography (SD-OCT) is a very useful and a non-invasive imaging modality for the diagnosis and follow-up of various macular diseases.

In recent studies, the descriptive OCT findings in CSCR have been demonstrated in both sexuals [10-16]. However, to our best knowledge, there is not a direct comparison of the OCT findings between both sexual in the cases with CSCR in literature. Thus, we conducted this study to compare the OCT findings in male and female cases with acute CSCR.

Material and methods

This study was designed as a retrospective comparative study. In this retrospective study, the OCT scans of 8 female (Group 1) and 16 male (Group 2) cases with acute CSCR which were diagnosed based on clinical, ophthalmological and OCT findings and followed in retina section of our hospital were retrospectively reviewed. The study was designed according to Helsinki Declaration.

Inclusion criteria

Female (Group 1) and male (Group 2) patients with acute CSCR were included to the study. Acute CSCR was defined as the accumulation of serous fluid between the photoreceptor outer segments and the RPE and serous macular detachment (SMD) of the neurosensory retina self-resolving within 6 months of symptom onset.

Exclusion criteria

The patients with epi-retinal membrane or vitreo-macular traction documented by OCT, and media opacities such as corneal opacity, lens opacity, vitreous and pre-retinal haemorrhage, uveitis, choroidal neovascularization, diabetic maculopathy/retinopathy, and patients with history of previous intraocular surgery, macular laser photocoagulation, and intravitreal injection and the patients having

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lower image quality score or low signal strength of OCT were excluded from the study.

OCT procedure and analysis

OCT examinations were performed using spectral OCT (RTVue-100 OCT, Optovue, Inc., Fremont, CA). During OCT examination the maculae were scanned by a single retina specialist (BT) on six radial sections including the horizontal, vertical, and oblique planes through the center of the fovea. Acquired OCT images were evaluated by the same retina specialist (BT). Sub-foveal choroidal thicknesses (SFCT) were measured manually by a single specialist (BT) using enhanced depth imaging (EDI) mode build-in the OCT device. Only following OCT findings to be attributed to acute CSCR but not chronic CSCR were evaluated [10-13, 16].

- Retina pigment epithelium detachment (PED) was defined as a dome-shaped elevation of the RPE typically seen overlying a homogeneously hypo-reflective space towards inner retina on RPE-choriocapillaris-Bruch membrane complex.

- The RPE bulging was defined as at least two sets of peaks and troughs of RPE or a small protrusion of the RPE layer (a slight elevation of the RPE without hypo-reflective space).

- Serous macula detachment (SMD) was defined as the dome-shaped elevation of the posterior surface of the neurosensory retina over a nonreflective black cavity, with minimal shadowing of the underlying tissues and the presence of normal foveal pit but without shadowing in underlying tissues and destruction in the normal reflection of RPE.

- ''Brush border pattern'' or "elongation of photoreceptor outer segment" was defined as an irregular and serrated appearance on the outer surface of the detached neurosensory retina over sub-retinal fluid due to the accumulation of the waste products in photoreceptor outer segment.

- The intra-retinal hyper-reflective dot (IHRD) and sub-retinal hyper-reflective dot (SHRD) were defined as hyper-reflective puncta in the detached neurosensory retina and under the outer surface of the detached retina, respectively.

- ''Dipping (tenting down) pattern'' was defined as triangular hyper-reflective dipping or tenting down at the outer surface of detached neurosensory retina connecting the detached neurosensory retina and RPE. It was observed that the apex of this hyper-reflective triangular entity was at RPE line while its base was at the detached retina.

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 11.0 (Chicago, IL, USA). Results were given as the means ± standard deviations. The chi-square test was used to compare categorical variables in the study groups, respectively. A P value less than 0.05 was considered as statistically significant.

Results

Group 1 included 8 women while Group 2 included 16 men. The mean ages of the Group1 and Group 2 were 51±13.72 years (ranging between 33 and 60 years) and 43.13±10.7 (ranging between 34 and 60) years, respectively. The groups were matched for age and there was no statistically significant difference between the groups (p>0.05). The optical coherence tomographical and demographical data in the study groups are given in Table 1

Table 1. The optical coherence tomographical and demographical data in the study groups.

	Females (N = 8)	Males (N = 16)	P value
Age (years)	51.2±13.72	43.13±10.7	P>0.05
SMD (%)	8 (100%)	16 (100%)	P>0.05
PED	0 (0%)	4 (25%)	P>0.05
Dipping pattern	0 (0%)	0 (0%)	P>0.05
RPE bulging	8 (100%)	14 (87.5%)	P>0.05
Brush-border pattern	6 (75%)	6 (37.5%)	P>0.05
IHRD	2 (25%)	4 (25%)	P>0.05
SRHRD	0 (0%)	2 (12.5%)	P>0.05
Choroidal Thickness	346.08±32.1 µm	351.32±36.5 µm	P>0.05

OCT findings

OCT imaging in female cases revealed that there were single SMD in 8 cases (100%), PED with SMD in 0 case (0%), RPE bulging with SMD in 8 cases (100%), brush-border pattern with SMD in 6 cases (75%), dipping pattern with SMD in 0 case (0%), IHRDs with SMD in 2 cases (25%) and SHRDs with SMD in 0 cases (0%).

In OCT scans in males with CSCR, it was detected single SMD in 16 cases (100%), PED with SMD in 4 cases (25%), RPE bulging with SMD in 14 cases (87.5%), brush-border pattern with SMD in 6 cases (37.5%), dipping pattern with SMD in 0 case (0%), IHRDs with SMD in 4 cases (25%) and SHRDs with SMD in 2 cases (12.5%). There was no statistically significant difference in OCT findings and patterns between both groups (p > 0.05). The most common OCT finding except SMD was the brush-border pattern (75%) in females and RPE bulging (87.5%) in males.

The mean SFCTs of the affected eyes in Group 1 and Group 2 were 346.08±32.1 µm (ranged from 338 to 365 µ) and 351.32±36.5 µm (ranged from 344 to 376), respectively. The difference between SFCTs in the affected eyes was not statistically significant (p>0.05).

Additionally, the mean SFCTs in the un-effected opposite eyes in Group 1 and Group 2 were 256.22 ± 19.60 µm (ranged from 236 to 343 µm) and 246.11±26.5 µm (ranged from 229.7 to 356), respectively. It was detected that the difference in SFCTs in the un-effected eyes in both groups was not statistically significant (p>0.05).

Discussion

The most common spectral domain OCT findings detected in the patients with acute CSCR are PED, RPE bulging, SMD, dipping pattern, brush-border pattern or elongation of photoreceptor outer segments and IHRDs or SHRDs [2,13,17-21].

In a meta-analytic comprehensive review, Daruich et al presented the frequencies of these OCT findings in acute CSCR as follows: the elevation or bulging of RPE 19-68%, PED 32-71%, SHRDs 20.3-52%, sagging or dipping pattern 13-43%, elongated photoreceptor outer segments or brush-border pattern 73%, SHRDs 65%-82%, IHRDs 21-100% [2]. However, PED prevalence may vary from 8% to 71% [11,18,19,22]. In our study, OCT did not show any PED in female cases. This is very interesting finding to be considered in the comparison of OCT findings in both sexual.

Fundus angiography (FA) and OCT studies showed that both PED and the small bump/bulging areas are consistent with the leakage points localization of the defect, thickening, or protrusion in the RPE in the FA in active CSCR [2,5,10-16,23]. These bulging lesions should not be confused with PED. Hirami et al reported that the RPE bulging was

presented in 89% of the patients and that these were located in areas of choroidal vascular hyperpermeability [24]. Montero et al reported that the hyperreflective small RPE bulging is present in 90% of the cases and they were related to leaking spots in FA [11]. However, it has been reported in an *en face* OCT study that it was detected in 35 % of the eyes with acute CSCR [19]. Additionally, RPE layer mapping study using OCT demonstrated that RPE bulging was in 94% of asymptomatic eyes in patients with CSR [25]. RPE bulging was observed in all of the females and a percent of 87.5 in males in our study. This is compatible with previous reports.

Hyperreflective spots or dots (HRD) are new findings previously unseen in OCT demonstrated firstly by Coscas et al [26]. The HRDs are scattered, punctiform, small in size, mainly located in the outer retinal layers. It has been reported in the patients with CSCR. It has been demonstrated that HRDs in the patients with CSCR may be located as subretinal, intraretinal or at inner regions of the detached retina [13,17,26,27]. Although etiology of HRD is not clear, there are various theories of the pathogenesis of HRD. They might be caused focal accumulations of pigment or lipofuscin granules, small intraretinal proteins or lipid/lipoprotein exudates/deposits/extravasation due to the breakdown of the blood-retinal barrier, derived the degenerated photoreceptors or the macrophages phagocyted their outer segments. It has been suggested that HRD might be a marker of the inflammatory response or the breakdown of the blood-retina barrier [13,17,26,27]. OCT showed that HRDs accumulate at the posterior surface of the detached neurosensory retina in a vast majority of the patients with CSCR and that HRDs might have non-phagocyte-bearing rhodopsin and/or due to previous inflammatory mediators and/or dysfunctional RPE [28]. In our study, we observed IHRDs in 25% of the cases while as we did not detect any SHRD in female cases. However, 25% of male cases had IHRD and 12.5% of males had SHRDs. These prevalences seem lower compared to previous reports. This may be related to the evaluation of only acute CSCR cases in our study.

Dipping (tenting down) pattern may be observed in some acute CSCR patients. It is characterized by dipping or tenting at the outer surface of the detached neurosensory retina due to hyper-reflective material accumulation such as sub-retinal fibrin or fibrinous exudate connecting the detached neurosensory retina and RPE at its opposite [13,17,29]. In our study, OCT did not reveal dipping pattern in any case. This may be due to the absence of chronic recurrent cases in our study.

Brush-border pattern or elongation of photoreceptor outer segments is due to the accumulation of the waste products in photoreceptor outer segments and also the elongation of photoreceptor outer segments. In the normal turn, waste products of photoreceptors are removed by RPEs via phagocytosis. However, in CSCR, sub-retinal fluid and serous macular detachment cause loss of the contact between photoreceptor outer segments and RPE. Thus, waste products in photoreceptor outer segments accumulate on the outer surface of the detached neurosensory retina. This provides an irregular appearance of the detached neurosensory retina [10-13 16,18-20,30]. In the other hand, intraretinal precipitates may result from proteins or accumulation of macrophages that may have phagocytized photoreceptor outer segments [16]. The most common finding except SMD in OCT was the brush-border pattern (75%) in female cases in our study.

Although there is no descriptive and comparative study in literature belonging to only female case group with CSCR, some case reports present OCT findings in females with CSCR. In the acute stage of the disease, it has been observed sub-retinal hyper-reflective material except for SMD [24].

In previous reports, it has been considered that CSCR in women was associated with subretinal precipitates and absence of PED or longer duration for development of PED [31-38]. The absence of PED in OCT examinations in female cases as seen in our study is compatible with this association.

In conclusion, this pilot study suggests that there is no difference between the frequencies of OCT findings and patterns of the female and male patients with acute CSCR. However, female patients with CSCR are usually presented with RPE bulging but not PED compared to the males. Further studies are needed to understand the exact cause of presentation finding in female cases.

Acknowledgments

References

1. Yannuzzi LA (1987) Type-A behavior and central serous chorioretinopathy. *Retina* 7: 111-131. [Crossref]

2. Daruich A, Matet A, Dirani A, Bousquet E, Zhao M, et al. (2015) Central serous chorioretinopathy: Recent findings and new physiopathology hypothesis. *Prog Retin Eye Res* 48: 82-118. [Crossref]

3. Wang M, Munch IC, Hasler PW, Prünte C, Larsen M (2008) Central serous chorioretinopathy. *Acta Ophthalmol* 86: 126-145. [Crossref]

4. Liegl R, Ulbig MW (2014) Central serous chorioretinopathy. *Ophthalmologica* 232: 65-76. [Crossref]

5. Nicholson B, Noble J, Forooghian F, Meyerle C (2013) Central serous chorioretinopathy: update on pathophysiology and treatment. *Surv Ophthalmol* 58: 103-126. [Crossref]

6. Abouammoh MA (2015) Advances in the treatment of central serous chorioretinopathy. *Saudi J Ophthalmol* 29: 278-286. [Crossref]

7. Prünte C, Flammer J (1996) Choroidal capillary and venous congestion in central serous chorioretinopathy. *Am J Ophthalmol* 121: 26-34. [Crossref]

8. Iida T, Kishi S, Hagimura N, Shimizu K (1999) Persistent and bilateral choroidal vascular abnormalities in central serous chorioretinopathy. *Retina* 19: 508-512. [Crossref]

9. Gass JDM. (1991) Central serous chorioretinopathy and white subretinal exudation during pregnancy. Arch Ophthalmol 109: 677–681. [Crossref]

10. Ojima Y, Hangai M, Sasahara M, Gotoh N, Inoue R, et al. (2007) Three-dimensional imaging of the foveal photoreceptor layer in central serous chorioretinopathy using high-speed optical coherence tomography. *Ophthalmology* 114: 2197-2207. [Crossref]

11. Montero JA, Ruiz-Moreno JM (2005) Optical coherence tomography characterization of idiopathic central serous chorioretinopathy. *Br J Ophthalmol* 89: 562-564. [Crossref]

12. Iida T, Hagimura N, Sato T, Kishi S (2000) Evaluation of central serous chorioretinopathy with optical coherence tomography. *Am J Ophthalmol* 129: 16-20. [Crossref]

13. Turgut B, Ergen I (2013) Santral seröz koryoretinopatinin optik koherens tomografik paternleri (Optical Coherence Tomographic Patterns of Central Serous Chorioretinopathy). *Firat Medical Journal* 18: 39-43.

14. Rezai KA, Eliott D (2004) Optical coherence tomographic findings in pregnancy-associated central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol* 242: 1014-1016. [Crossref]

15. Theodossiadis PG, Kollia AK, Gogas P, Panagiotidis D, Moschos M, et al. (2002) Retinal disorders in preeclampsia studied with optical coherence tomography. *Am J Ophthalmol* 133: 707-709. [Crossref]

16. Kon Y, Iida T, Maruko I, Saito M (2008) The optical coherence tomography-ophthalmoscope for examination of central serous chorioretinopathy with precipitates. *Retina* 28: 864-869. [Crossref]

17. Turgut B, Demir T (2016) The new landmarks, findings and signs in optical coherence tomography, *New Front Ophthalmol* 2: 131–136.

18. Mitarai K, Gomi F, Tano Y (2006) Three-dimensional optical coherence tomographic findings in central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol* 244: 1415–1420. [Crossref]

19. Fujimoto H, Gomi F, Wakabayashi T, Sawa M, Tsujikawa M, et al (2008) Morphologic changes in acute central serous chorioretinopathy evaluated by fourier-domain optical coherence tomography. *Ophthalmology* 115: 1494–1500. [Crossref]

20. Kim HC, Cho WB, Chung H (2012) Morphologic changes in acute central serous chorioretinopathy using spectral domain optical coherence tomography. *Korean J Ophthalmol* 26: 347–354. [Crossref]

21. Shin YU, Lee BR (2012) Retro-mode Imaging for retinal pigment epithelium alterations in central serous chorioretinopathy. *Am J Ophthalmol* 154: 155–163. [Crossref]

22. Van Velthoven MEJ, Verbraak FD, Garcia PM, Schlingemann RO, Rosen RB, de Smet MD (2005) Evaluation of central serous retinopathy with en face optical coherence tomography. *Br J Ophthalmol* 89: 1483-1488. [Crossref]

23. Lehmann M, Wolff B, Vasseur V, Martinet V, Manasseh N, Sahel JA, Mauget-Faysse M (2013) Retinal and choroidal changes observed with 'En face' enhanced-depth imaging OCT in central serous chorioretinopathy. *Br J Ophthalmol* 97: 1181–1186. [Crossref]

24. Hirami Y, Tsujikawa A, Sasahara M, Gotoh N, Tamura H, et al. (2007) Alterations of retinal pigment epithelium in central serous chorioretinopathy. *Clin Exp Ophthalmol* 35: 225-230. [Crossref]

25. Gupta P, Gupta V, Dogra MR, Singh R, Gupta A (2010) Morphological changes in the retinal pigment epithelium on spectral-domain OCT in the unaffected eyes with idiopathic central serous chorioretinopathy. *Int Ophthalmol* 30: 175-181. [Crossref]

26. Coscas G, De Benedetto U, Coscas F, Li Calzi CI, Vismara S, et al (2013) Hyperreflective dots: a new spectral-domain optical coherence tomography entity for follow-up and prognosis in exudative age-related macular degeneration. *Ophthalmologica* 229: 32-37. [Crossref]

27. Turgut B, Yildirim H (2015) The causes of hyperreflective dots in optical coherence tomography excluding diabetic macular edema and retinal venous occlusion§. *Open Ophthalmol J 9*: 36-40. [Crossref]

28. Ahlers C, Geitzenauer W, Stock G, Golbaz I, Schmidt-Erfurth U, et al. (2009) Alterations of intraretinal layers in acute central serous chorioretinopathy. *Acta Ophthalmol* 87: 511-516. [Crossref]

29. Hussain N, Baskar A, Ram LM, Das T (2006) Optical coherence tomographic pattern of fluorescein angiographic leakage site in acute central serous chorioretinopathy. *Clin Exp Ophthalmol* 34: 137-140. [Crossref]

30. Matsumoto H, Kishi S, Otani T, Sato T (2008) Elongation of photoreceptor outer segment in central serous chorioretinopathy. *Am J Ophthalmol* 145: 162-168. [Crossref]

31. Gobuty M, Adhi M, Duker JS (2013) Central serous chorioretinopathy in a 91-year-old woman. *Br J Ophthalmol* 97: 1607-1608, 1615-6. [Crossref]

32. Said-Ahmed K, Moustafa G, Fawzy M (2012) Incidence and Natural Course of Symptomatic Central Serous Chorioretinopathy in Pregnant Women in a Maternity Hospital in Kuwait. *Middle East Afr J Ophthalmol* 19:273-276. [Crossref]

33. Quillen DA, Gass DM, Brod RD, Gardner TW, Blankenship GW, et al. (1996) Central serous chorioretinopathy in women. *Ophthalmology* 103: 72-79. [Crossref]

34. Al-Mujaini A, Wali U (2014) Alternating central serous chorioretinopathy in two consecutive pregnancies. *Oman J Ophthalmol* 7: 87-89. [Crossref]

35. Musetti D, Nicolo M, Traverso C (2013) Central serous chorioretinopathy in women. *Invest Ophthalmol Vis Sci.* 54: 3842.

36. Lin D, Chen W, Zhang G, Huang H, Zhou Z, et al (2014) Comparison of the optical coherence tomographic characters between acute Vogt-Koyanagi-Harada disease and acute central serous chorioretinopathy. *BMC Ophthalmol* 14:87. [Crossref]

37. Perkins SL, Kim JE, Pollack JS, Merrill PT (2002) Clinical characteristics of central serous chorioretinopathy in women. *Ophthalmology* 109: 262-266. [Crossref]

38. Uhumwangho OM, Momoh RO (2015) Central serous chorioretinopathy in Benin City, Nigeria. *Ann Afr Med* 14: 163-168. [Crossref]