# Role of Psychological Stress and Choroidal Thickness in Central Serous Chorioretinopathy

Reshmi Mathews<sup>1</sup>, Saban Horo<sup>1</sup>, Deepa Jose<sup>1</sup>, Joel Antony Kavalakatt<sup>1</sup>, Grace Rebekah J<sup>1</sup>, Sheeja Susan John<sup>1</sup> (Christian Medical College, Vellore, India

## **ABSTRACT**

Introduction: Central serous chorioretinopathy (CSCR) is a disease with a multifactorial aetiology.

**Objectives:** To evaluate the role of psychological stress and choroidal thickness in patients with CSCR.

Materials and methods: This was a hospital-based, analytical cross-sectional study, conducted at Christian Medical College, Vellore, India, from 2018 February to 2019 September, after the approval of the Institutional Review Board and Ethics Committee. Patients who satisfied the eligibility criteria of the study, were selected from the outpatient clinics of the Department of Ophthalmology. Twenty-five patients with unilateral CSCR (Group 1 - cases), and 50 age and gender-matched patients without CSCR (Group 2 - controls), randomly selected in a 1:2 ratio, were enrolled after obtaining informed consent. Psychological stress was assessed using Cohen Perceived Stress Scale (PSS-10). All patients had a Swept Source optical coherence tomography scan of the macula. Subfoveal choroidal thickness (SFCT) of both eyes of all participants was measured using a standard protocol. The data were collated and analysed, using Independent samples t-test and Chi-square/ Fisher's exact test, as appropriate, with IBM SPSS Statistics for Windows, version 21 (IBM Corp., Armonk, N.Y., USA).

**Results:** There was a significant difference between the two groups in mean stress scores (p <0.01), as well as stress categories (p <0.01). Although there was no significant difference in the prevalence of hypertension between the two groups (p = 0.33), there was a significant difference both in the mean systolic (p <0.01) and diastolic (p <0.01) blood pressure between the two groups. We found a significant difference between mean SFCT of CSCR eyes (421+/- 78.34  $\mu$ m) and control eyes (314.24 +/- 52.48  $\mu$ m, p <0.01), as well as between fellow eyes (396.20 +/- 68.79  $\mu$ m) and control eyes (314.24 +/- 52.48  $\mu$ m, p <0.01). However, there was no significant difference in the mean SFCT of CSCR eyes and fellow eyes (p =0.24).

**Conclusion:** The findings reiterate the concept that the underlying pathophysiological changes leading to CSCR involve both the eyes of the patient. Psychological stress and hypertension may be factors that play an important role in the etiopathogenesis of the pachychoroid-related changes leading to CSCR. Stress relief measures, with a holistic approach to management, should be an integral part of the therapeutic strategies for CSCR.

**Key words:** Aetiology; central serous chorioretinopathy; choroid; optical coherence tomography; psychological stress.

Financial Interest: Nil Received: 23.03.2023

Conflict of Interest: Nil Accepted: 21.09.2023

Corresponding Author Sheeia Susan John

Christian Medical College, Vellore, India E-mail: sheejajohn@rediffmail.com



Access this article online

Website: www.nepjol.info/index.php/NEPJOPH
DOI: https://doi.org/10.3126/nepjoph.v15i2.53598
Copyright © 2023 Nepal Ophthalmic Society





This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License (CC BY-NC-ND).



#### INTRODUCTION

Central serous chorioretinopathy (CSCR) is a chorioretinal disorder with multifactorial aetiology. Although the disease is usually selflimiting, some patients develop recurrent or chronic disease, resulting in varying degrees of visual loss (Manyath et al., 2018; Prakash et al., 2013). The changes seen in the choroidal vessels in patients with CSCR have led to the concept that CSCR is primarily a disease of the choroidal vasculature (Gołębiewska et al., 2017). Further, newer Optical Coherence Tomography (OCT) systems have demonstrated the thickening of the choroid in patients with CSCR (Ambiya et al., 2018; Brandl et al., 2014). CSCR is influenced exogenous several and endogenous factors that cause alterations in the choroidal vasculature. Several investigators have noted that psychological stress is associated with the development of CSCR (Agarwal et al., 2016; Bazzazi et al., 2015). Stress is associated with the activation of the hypothalamic-pituitaryadrenal axis, causing increased secretion of cortisol (Yanuzzi et al., 1987; Berger et al., 1987; Tsigos et al., 2002).

Various modalities of treatment of CSCR have been tried with variable results. The fact remains that unless the risk factors predisposing to the disease are appropriately addressed, the management may be inadequate, and the disease may recur and even become chronic, with subsequent risk of permanent visual impairment. In a disease like CSCR, with a multifactorial etiopathogenesis, holistic management which includes addressing the risk factors, is of paramount importance. However, not many studies have focused on this aspect of disease management.

The aim of this study was to evaluate the role of psychological stress and choroidal thickness in patients with Central serous chorioretinopathy, with a view to obtaining a better understanding of the two important risk factors associated with the disease in the Indian population, with implications for management strategies.

## MATERIALS AND METHODS

This was a hospital-based, analytical cross-sectional study, conducted at the Department of Ophthalmology, Christian Medical College, Vellore, in South India, over a period of 20 months, from February 2018 to September 2019, after obtaining the approval of the Institutional Review Board and Ethics Committee (Reference number 11094, dated 10.01.2018).

The study included patients with unilateral CSCR, seen in the outpatient clinics (cases -Group 1), and age and gender-matched patients without CSCR (controls - Group 2), who were randomly selected in a 1:2 allocation ratio (for every patient with CSCR in Group 1, two age and gender-matched patients without CSCR in Group 2). The selection of controls (Group 2), from the list of new registrations for OPD patients for the day, was based on a random start using a pseudo random number, generated using a calculator. The number equivalent to the last two digits from the right, of the pseudo random number generated, was chosen. Every patient in the list, starting with the random start, was considered, and the first patient who fulfilled the appropriate age and gender matching criteria, was recruited into the study. The age of each control (Group 2) was matched to within 2.5 years above or below that of the corresponding case (Group 1).

Exclusion criteria were duration of CSCRrelated symptoms more than four months, any intervention for the disease, age less than 20 years or more than 60 years, steroid medication, post-organ transplant patients, pregnant women, history or clinical features of endogenous hypercortisolism, any other retinal/ choroidal/ optic disc pathology that may interfere with the diagnosis of CSCR, or may be associated with changes in choroidal thickness, history of ocular trauma/ surgery/ intravitreal injections/ laser procedures, myopia or hyperopia of more than 2 dioptres, or a difference of more than 1 dioptre between the two eyes of the patient, axial length of less than 21.5 mm or more than 24.5 mm, or a difference of more than 0.5 mm between the two eyes of the patient, and media opacity that interfered with clear OCT image acquisition.

Information regarding the clinical and sociodemographic profile of the participants, potential risk factors for CSCR and potential confounders was elicited using a Clinical Research Form. The educational and socio-economic status of the participants of the study was assessed, based on the Modified Kuppuswamy's socio-economic scale (Kumar et al., 2012). History of dyspeptic symptoms was used as a surrogate marker for Acid peptic disease. Similarly, history of snoring was used as a surrogate marker for Obstructive sleep apnea.

All the participants of the study had a complete ophthalmological examination, including refraction, slit lamp biomicroscopy, Goldmann applanation tonometry, dilated fundus examination, OCT scan of the macula (Swept source OCT, DRI OCT Triton Plus, Topcon Inc, Tokyo, Japan), axial length measurement by Optical Biometry, and measurement of blood

pressure. Patients with CSCR (Group 1) also had fundus fluorescein angiography.

Subfoveal choroidal thickness (SFCT) of both eyes of all the study participants was measured using a standard protocol. One horizontal and one vertical 6 mm line scan of the macula of each eye of all the participants was performed. SFCT was measured at the fovea, from the outer part of the hyper reflective line corresponding to the base of the retinal pigment epithelial layer to the hypo reflective line or margin corresponding to the sclera choroidal interface. (Rahman et al., 2011) Three consecutive readings of SFCT were taken in each scan, and the mean of the six readings of SFCT in each eye was taken as the final reading of SFCT. All measurements were done by a single experienced observer.

Psychological stress was assessed using the tenitem version of Cohen Perceived Stress Scale (PSS-10) (Cohen et al., 1983). The original English questionnaire was translated into Tamil and Hindi. Two versions of the translation in each language were obtained, which were then back translated into English. The English questionnaire, along with the translations and back translations, were reviewed by five subject matter experts in each language, and the better of the two translations in each language was chosen. One of the investigators in the study was trained to administer the questionnaire in a standard manner. After the questionnaire was piloted in all three languages, it was verbally administered to all the study participants by this investigator. Based on the score obtained after administering the questionnaire, patients were categorised into three: low stress (0-13), moderate stress (14-26), high stress (27-40).



Age, gender, axial length, refractive error, smoking, alcoholism, hypertension, organ transplantation, pregnancy, endogenous hypercortisolism, acid peptic disease, obstructive sleep apnea, allergic airway disease, diabetes, collagen vascular disorders, cardiovascular disease, and intake of steroids and other medicines that have been implicated in the etiopathogenesis of CSCR were considered as potential confounders/ suspected effect modifiers in our study. Group 1 and Group 2 of the study were matched for age and gender. Other potential confounders like extremes of age, axial length and refractive error, pregnant women, patients on steroids, post-organ transplant patients, and patients with history or clinical features of endogenous hypercortisolism were excluded. Data regarding the risk factors such as hypertension, diabetes, smoking, alcoholism, dyspeptic symptoms, snoring, allergic airway disease, cardiovascular disease and medications implicated in the etiopathogenesis of CSCR, were meticulously documented and analysed.

Statistical analysis was done using IBM SPSS Statistics for Windows, version 21 (IBM Corp., Armonk, N.Y., USA). Descriptive statistics were reported using Mean±SD for continuous variables. Categorical variables were analysed using frequency and percentage. Continuous variables, which were normally distributed, were assessed using Independent samples t-test after checking for normality. Chisquare/ Fisher's exact test was used to analyse categorical variables.

#### RESULTS

During the study period, 38 patients with Central serous chorioretinopathy were screened for

recruitment into the study. Twenty five patients who met the eligibility criteria, were enrolled into Group 1, and 50 randomly selected, age and gender-matched controls without CSCR were recruited into Group 2 of the study, after obtaining informed consent.

All baseline demographic characteristics such as age, gender, educational and socio-economic status were comparable between the two groups. They were also not significantly different with respect to the risk factors such as hypertension, diabetes, smoking, alcoholism, dyspeptic symptoms, snoring, allergic airway disease, cardiovascular disease and medications implicated in the etiopathogenesis of CSCR (Table 1).

The right eye of the patients in Group 2 was taken as the 'control eye' for analysis of the comparison of refractive error, axial length and subfoveal choroidal thickness between the two groups. There was no statistically significant difference in the refractive error and axial length, either between CSCR eyes and control eyes, or between fellow eyes and control eyes. The mean difference in refractive error and axial length between the two eyes of the patients in Group 1 and Group 2 was also comparable (Table 2).

Decrease in vision was the most common symptom of CSCR, present in 19 patients (61%), followed by a positive central scotoma present in 11 patients (36%). Only one patient experienced metamorphopsia. The duration of the symptoms ranged from a minimum of four days to a maximum of 110 days (median: 14 days).

Table 1: Demographic profile and risk factors in the two groups.

	Group 1 n (%)/ mean (SD)	Group 2 n (%)/ mean (SD)	p	
Age (years)	38.80 (5.89)	39.08 (5.79)	0.85	
Male	21 (84)	42 (84)	1.00	
Female	4 (16)	8 (16)		
Higher educational status	9 (36)	26 (52)	0.26	
Lower educational Status	16 (64)	24 (48)		
Higher socio-economic status	12 (48)	33 (66)	0.42	
Lower socio-economic status	13 (52)	17 (34)	0.42	
Hypertension	3 (12)	2 (4)	0.22	
No hypertension	22 (88)	48 (96)	0.33	
Diabetes	2 (8)	7 (14)	0.71	
No diabetes	23 (92)	43 (86)	0.71	
Smoking	0 (0)	8 (16)	0.09	
No smoking	25 (100)	42 (84)		
Alcohol use	1 (4)	2 (4)	1.00	
No alcohol use	24 (96)	48 (96)		
Dyspepsia	0 (0)	2 (4)	0.55	
No dyspepsia	25 (100)	48 (96)		
Snoring	3 (12)	2 (4)	0.33	
No snoring	22 (88)	48 (96)		
Allergic airway disease	2 (8)	1 (2)	0.26	
No allergic airway disease	23 (92)	49 (98)	1	
Cardio vascular disease	0 (0)	1 (2)	1.00	
No cardio vascular disease	25 (100)	49 (98)	1.00	
CSCR relevant medication	2 (8)	0 (0)	0.11	
No CSCR relevant medication	23 (92)	50 (100)	0.11	



Table 2: Refractive error and axial length (AL) in the two groups.

	Mean (SD)	p	
RE* in CSCR eyes	+0.50 (0.67)	0.33	
RE in control eyes	+0.33 (0.73)		
RE in fellow eyes	+0.39 (0.52)	0.72	
RE in control eyes	+0.33 (0.73)	0.72	
RE difference b/w 2 eyes of patients (Group 1)	0.39 (0.45)	0.12	
RE difference b/w 2 eyes of patients (Group 2)	0.26 (0.27)	0.12	
AL** in CSCR eyes	23.06 (0.55)	0.26	
AL in control eyes	23.19 (0.56)	0.36	
AL in fellow eyes	23.13 (0.58)	0.69	
AL in control eye	23.19 (0.56)	0.68	
AL difference b/w 2 eyes of patients (Group 1)	0.14 (0.11)	0.71	
AL difference b/w 2 eyes of patients (Group 2)	0.09 (0.08)	0.71	

<sup>\*</sup>Refractive error

Fundus Fluorescein Angiography (FFA) was done in 24 out of the 25 patients in Group 1. Active leakage of the dye was observed only in CSCR eyes. The majority of the patients had a single focus of leak (21 patients, 87.5%), with macula being the most common site of leak (20 patients, 83.33%). Nineteen out of the 23 patients with active leakage (83%) had an inkblot pattern of leak. None of our study patients were noted to have a choroidal neovascular membrane. Window defects due to retinal pigment epithelial (RPE) atrophic changes were noted in the CSCR eye in 20 patients (83.33%), and the macula was the most common site (18 patients). In the fellow eye, 15 patients (62.5%) had RPE atrophic changes, with the macula again being the most common site (13 patients). There was no significant difference in the prevalence of RPE atrophic changes noted on FFA, between the CSCR eye and the fellow eye (p = 0.22).

The mean subfoveal choroidal thickness (SFCT) of the eyes with CSCR (421+/- 78.34  $\mu$ m) was greater than that of the control eyes (314.24 +/- 52.48  $\mu$ m, p <0.01). We also found that the mean SFCT of the fellow eyes of CSCR patients (396.20 +/- 68.79  $\mu$ m) was significantly greater than that of the control eyes (314.24 +/- 52.48  $\mu$ m, p <0.01). There was no statistically significant difference in the mean SFCT of CSCR eyes and fellow eyes (p =0.24) (Table 3).

Stress scoring ranged from 0-40 as per Cohen's perceived stress scale (PSS-10). There was a significant difference between Group 1 patients with CSCR and Group 2 patients without CSCR in mean stress scores (p <0.01), as well as stress categories (p <0.01). Although there was no significant difference in the prevalence of hypertension between the two groups (p = 0.33), there was a significant difference both in the mean systolic (p <0.01) and diastolic (p <0.01) blood pressure between the two groups (Table 4).

<sup>\*\*</sup>Axial length

Table 3: Comparison of subfoveal choroidal thickness between the two groups.

	Mean (SD)	p
SFCT in CSCR eyes	421 (78.34)	<0.01
SFCT in control eyes	314.24 (52.48)	<b>\\0.01</b>
SFCT in fellow eyes	396.20 (68.79)	<0.01
SFCT in control eyes	314.24 (52.48)	<0.01
SFCT in CSCR eyes	421.00 (78.34)	0.24
SFCT in fellow eyes	396.20 (68.79)	0.24

SFCT was measured in µm.

Table 4: Stress scores, stress categories, and blood pressure in the two groups.

	Group 1 mean (SD)/n (%)	Group 2 mean (SD)/n (%)	р
Stress scores	23.72 (5.792)	11.12 (3.173)	< 0.01
Stress categories			
Low	0 (0)	42 (84)	<0.01
Moderate	18 (72)	8 (16)	
High	7 (28)	0 (0)	
Systolic BP	123.20 (16.45)	106.40 (11.25)	<0.01
Diastolic BP	78.08 (10.09)	70.80 (8.47)	<0.01

## **DISCUSSION**

Central serous chorioretinopathy is a disease with a multifactorial aetiology. This study did not find a statistically significant difference between the two groups in the prevalence of hypertension, diabetes, smoking, use of alcohol, acid peptic disease, obstructive sleep apnoea, allergic airway disease, cardiovascular disease and use of CSCR-relevant medications.

Various studies have shown an increase in choroidal thickness in eyes with CSCR (Brandl et al., 2014; Kuroda et al., 2013; Arora et al., 2016). Furthermore, it has been found that SFCT

in the fellow eyes was also thicker as compared to the control eyes (Arora et al., 2016; Kim et al., 2011). In the study by Arora et al., the mean SFCT of CSCR eyes, fellow eyes and normal eyes was found to be 429  $\pm$  74.18  $\mu m$ , 360  $\pm$  57.99  $\mu m$ , and 301.80  $\pm$  46.59  $\mu m$  respectively (p <0.001). Similarly, in the study done by Kim et al. on patients with CSCR and age-matched normal controls, comparing the SFCT in affected eyes, unaffected fellow eyes and normal eyes of controls, the mean SFCT of the CSCR eyes, unaffected fellow eyes, and normal control eyes was 445.58±100.25, 378.35±117.44, and 266.80±55.45  $\mu m$ , respectively.



In the current study, the mean SFCT of the CSCR eyes, fellow eyes and control eyes was 421 +/- 78.34  $\mu$ m, 396.24 +/-52.48  $\mu$ m and 314.24 +/-52.48  $\mu$ m respectively. The mean SFCT of the CSCR eyes was significantly greater than that of the control eyes (p <0.01). The mean SFCT of the fellow eyes was also significantly higher than that of the control eyes (p <0.01). However, there was no significant difference in the mean SFCT between the CSCR eyes and the fellow eyes (p = 0.24), reiterating the concept of bilaterality of the pathophysiology of CSCR.

We noted that 20 patients (83.33%) had RPE changes in the CSCR eye and 15 patients (62.5%) had RPE changes also in the fellow eye, and there was no statistically significant difference between the CSCR eyes and the fellow eyes with respect to RPE changes (p =0.22). The presence of comparable RPE changes and increased choroidal thickness in the fellow eyes of patients with unilateral CSCR reiterates the concept that the underlying pathophysiological changes leading to CSCR involve both the eyes of the patient. However, one eye tends to manifest the disease at a particular point in time. The fellow eye may have been involved in the past, or may manifest the disease in the future, if the underlying etiopathological factors are not appropriately addressed.

The association of CSCR with psychological stress and Type A personality has been extensively studied. Stress and Type A personality are associated with endogenous hypercortisolism. Stress activates the hypothalamic-pituitary-adrenal axis, causing increased secretion of cortisol (Tsigos et al., 2002; Stephens et al., 2012). Various studies, using standard stress assessment tools, have found the stress scores to be significantly

elevated in CSCR patients as compared to the normal population (Agarwal et al., 2016; Bazzazi et al., 2015). In the study by Agarwal et al., including 54 CSCR patients and 54 controls, using the National Stress Awareness Day Stress Questionnaire (International stress management association, UK), the stress scores in the CSCR group were found to be elevated (p = 0.04). Similarly, Bazzazi et al., in their study on 30 CSCR patients and 30 controls, using the Hamilton Anxiety Rating Scale (HAM-A), found the stress scores in the CSCR group to be significantly elevated as compared to the control group (p = 0.000).

In the present study, we used the Cohen Perceived Stress Scale (PSS-10) to assess the stress scores, and found significantly higher stress scores among the patients in the CSCR group (Group 1). The mean stress score was 23.72 +/-5.792 in Group 1 and 11.12 +/-3.173 in Group 2 (p <0.01). Based on the stress scores, the subjects were further classified into three categories (low, moderate, and high stress), and we found a statistically significant difference between the two groups also with respect to stress categories (p <0.01).

Several investigators have studied the role of hypertension in the etiopathogenesis of CSCR (Haimovici et al., 2004; Chatziralli et al., 2017). Untreated hypertension results in alteration in choroidal circulation, and contributes to CSCR. In the current study, although there was no significant difference in the number of hypertensive patients between the two groups (p =0.33), we found a statistically significant difference between the two groups in both the mean systolic blood pressure (p <0.01), and the mean diastolic blood pressure (p <0.01).

Undoubtedly, psychological stress continues to be a major risk factor for CSCR. Stress is also known to predispose to hypertension, but the exact mechanism is unclear. It has been proposed to be through the constant activation of the sympathetic system by the stress hormones (Spruill, 2010). Studies have also shown that young patients undergoing stress are more likely to develop essential hypertension as they approach midlife (Matthews et al., 2004). Various investigators have found hypertension to be a risk factor for CSCR (Haimovici et al., 2004; Chatziralli et al., 2017). Therefore, stress and hypertension may be factors that play an important role in the etiopathogenesis of the pachychoroid-related changes leading to CSCR.

Various modalities of management of CSCR are currently available. However, concerns about adverse effects of treatment and chances of recurrence continue. Unless the risk factors predisposing to the disease are appropriately addressed, the management would be inadequate.

In the current scenario, systemic morbidities like cardiovascular disease and cerebrovascular accidents are on the rise, and rising levels of psychological stress have been implicated as one of the major risk factors. Unemployment, financial burden, family disputes, long working hours, the quest for excellence, lack of a social support system and the changing socio-cultural scenario are some of the causes for increase in psychological stress levels. Stress management, has therefore, become the need of the hour.

Therapeutic strategies should target measures to relieve stress and equip the affected individuals to manage stress effectively. There is a lacuna in scientific literature regarding the role of effective stress management measures in patients with CSCR. Yoga, meditation, prayer, lifestyle modification, development of a new hobby, walking or other forms of physical exercise or sports, and psychological counselling are various modalities that could be considered. Involvement of the services of a psychiatrist or psychologist in the management of patients with CSCR would be of the utmost importance. Adequate long term follow up and continued provision of holistic care are also important in the management of these patients.

We did not do ICG angiography for our study patients. We have, therefore, not studied choroidal vascular hyperpermeability in the eyes of these patients. Although the possibility of intra-observer variability in the manual measurement of subfoveal choroidal thickness was taken into account during the design of the study, and measures to address this issue were taken, it is possible that, in spite of this, some intra-observer variability would have remained. This was a cross-sectional study, with a single point of contact with each study patient, when the assessment of stress scores and choroidal thickness was done. This would not have been able to capture the variation in stress levels and choroidal thickness, and their effect on the evolution of the pathophysiological changes of the disease.

### **CONCLUSION**

Our study has reiterated the identity of CSCR as part of the pachychoroid disease spectrum. CSCR is a bilateral disease, related to a thick choroid with altered histopathological characteristics, closely associated with autonomic dysregulation of the choroidal vasculature. It is a disease with multifactorial aetiopathogenesis. Psychological



stress and hypertension may be factors that play an important role in the etiopathogenesis of the pachychoroid-related changes leading to CSCR. Although generally self-limiting, CSCR has a potential for recurrence and chronicity, with subsequent risk of permanent visual impairment. Unless the risk factors predisposing to the disease are appropriately addressed, the management may be inadequate.

In the present-day scenario, in which rising levels of psychological stress are being

implicated in the etiopathogenesis of a variety of diseases, stress management is the need of the hour. Therapeutic strategies for CSCR should target measures to relieve stress and equip the affected individuals to effectively manage stress. Adequate long term follow up and continued provision of holistic care are also important in the management of these patients.



#### REFERENCES

Agarwal A, Garg M, Dixit N, et al. (2016). Evaluation and correlation of stress scores with blood pressure, endogenous cortisol levels, and homocysteine levels in patients with central serous chorioretinopathy and comparison with agematched controls. Indian Journal of Ophthalmology; 64(11): 803-805. DOI: 10.4103/0301-4738.195591

Ambiya V, Kumar A, Baranwal VK, et al. (2018). Change in subfoveal choroidal thickness in diabetes and in various grades of diabetic retinopathy. International Journal of Retina and Vitreous; 4: 34. DOI: 10.1186/s40942-018-0136-9

Arora S, Pyare R, Sridharan P, et al. (2016). Choroidal thickness evaluation of healthy eyes, central serous chorioretinopathy, and fellow eyes using spectral domain optical coherence tomography in Indian population. Indian Journal of Ophthalmology; 64(10): 747-751. DOI: 10.4103/0301-4738.194999

Bazzazi N, Ahmadpanah M, Akbarzadeh S, et al. (2015). In patients suffering from idiopathic central serous chorioretinopathy, anxiety scores are higher than in healthy controls, but do not vary according to sex or repeated central serous chorioretinopathy. Neuropsychiatric Disease and Treatment; 11: 1131-1136. DOI: 10.2147/NDT. S83216

Berger M, Bossert S, Krieg, JC, et al., (1987). Interindividual differences in the susceptibility of the cortisol system: an important factor for the degree of hypercortisolism in stress situations? Biological Psychiatry; 22(11): 1327-1339. DOI: 10.1016/0006-3223(87)90067-9

Brandl C, Helbig H, Gamulescu MA, (2014). Choroidal thickness measurements during central serous chorioretinopathy treatment. International Ophthalmology; 34: 7-13. DOI:10.1007/s10792-013-9774-y

Chatziralli I, Kabanarou S.A, Parikakis E, et al., (2017). Risk factors for central serous chorioretinopathy: multivariate approach in a case-control study. Current Eye Research; 42: 1069-1073. DOI: 10.1016/j.ophtha.2003.09.024

Cohen S, Kamarck T, Mermelstein R, et al. (1983). A global measure of perceived stress. Journal of Health and Social Behaviour; 24: 385-396. DOI: 10.2307/2136404

Gołębiewska J, Brydak-Godowska J, Moneta-Wielgoś J, et al., (2017). Correlation between choroidal neovascularization shown by OCT angiography and choroidal thickness in patients with chronic central serous chorioretinopathy. Journal of Ophthalmology; 2017: 3048013. DOI: 10.1155/2017/3048013

Haimovici R, Koh S, Gagnon DR, et al. (2004). Risk factors for central serous chorioretinopathy: A case–control study. Ophthalmology; 111(2): 244-249. DOI: 10.1016/j.ophtha. 2003.09.024

Kim YT, Kang SW, Bai KH (2011). Choroidal thickness in both eyes of patients with unilaterally active central serous chorioretinopathy. Eye (London); 25(12): 1635-1640. DOI: 10.1038/eye.2011.258

Kumar N, Gupta N, Kishore J, (2012). Kuppuswamy's socioeconomic scale: Updating income ranges for the year 2012. Indian Journal of Public Health; 56: 103-104. DOI: 10.4103/0019-557x.96988

Kuroda S, Ikuno Y, Yasuno Y, et al., (2013). Choroidal thickness in central serous chorioretinopathy. Retina; 33: 302-308. DOI: 10.1097/ iae.0b013e318263d11f

Manayath GJ, Ranjan R, Karandikar SS, et al., (2018). Central serous chorioretinopathy: current update on management. Oman Journal of Ophthalmology; 11: 200-206. DOI: 10.4103/ojo.OJO 29 2018

Matthews KA, Katholi CR, McCreath H, et al. (2004). Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. Circulation; 110: 74-78. DOI: 10.1161/01.CIR.0000133415.37578.E4

Prakash G, Chauhan N, Jain S, et al., (2013). Central Serous Chorioretinopathy: A Review of the Literature. The Asia-Pacific Journal of Ophthalmology; 2: 104-110. DOI: 10.1097/APO. 0b013e31829069ee

Rahman W, Chen FK, Yeoh J, et al., (2011). Repeatability of manual subfoveal choroidal thickness measurements in healthy subjects using the technique of enhanced depth imaging optical coherence tomography. Investigative Ophthalmology & Visual Science; 52: 2267-2271. DOI: 10.1167/iovs.10-6024

Spruill TM (2010). Chronic psychosocial stress and hypertension. Current Hypertension Reports; 12: 10-16. DOI: 10.1007/s11906-009-0084-8

Stephens MAC, Wand G (2012). Stress and the HPA axis: Role of glucocorticoids in alcohol dependence. Alcohol Research; 34(4): 468-83. PMid: 23584113

Tsigos C, Chrousos GP (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. Journal of Psychosomatic Research; 53: 865-871. DOI: 10.1016/s0022-3999(02)00429-4

Yannuzzi LA (1987). Type-A behavior and central serous chorioretinopathy. Retina; 7: 111-131. DOI: 10.1097/00006982-198700720-00009