International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2019): 7.583

# Endogenous Cortisol Profile in Patients with Central Serous Chorioretinopathy (CSCR) at a Tertiary Health Care Centre

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**Abstract:** ***Aim: To study the endogenous cortisol levels in patients with central serous chorioretinopathy (CSCR). Methods: Endogenous cortisol levels in plasma were determined in 20 patients with acute CSCR. Results: The mean values of the 8 am plasma cortisollevelis28.67µg/dl, 11 pm plasma cortisol level 21.15µg/dl, revealed significantly higher values in the patient group (p<0.001). Conclusions: Increased levels of endogenous cortisol are present in patients with CSCR.***

## 1. Introduction

Central serous chorioretinopathy (CSCR) is a localised serous detachment of the posterior pole of the retina, occurring as a result of a focal defect in the retinal pigment epithelium (RPE), which leads to leakage of fluid of choroidal origin into the subretinal space, causing a detachment of the neurosensory retina from the RPE in the macular area.

The typical clinical picture is that of a male aged 20–50 years, presenting with an acute onset of blurring of vision associated with metamorphopsia, micropsia, and a central scotoma.[1]Fundus reveals one or more leakage points at the level of the RPE. Various studies have implicated infective,[2] vascular,[3,4] toxic, immunological,[5,6]allergic,[7,8] mechanical,[9] psychological,[10.11]and endocrinological[12-16] factors in the pathogenesis of this disease. However, the exact cause remains obscure. Many conditions including emotional stress, type A personality, pregnancy, and Cushing's syndrome have been associated with an increased incidence of CSCR.[17] Furthermore, anecdotal reports exist in the literature, of the worsening or precipitation of CSCR in patients started on systemic steroid therapy,[13-15] thus supporting the association between steroids and CSCR.

## 2. Methodology

Twenty patients with CSCR who presented to the ophthalmology department at Sree balaji medical college were included in the study. The criteria for inclusion into the study were: males between 20–40 years of age, first attack of CSCR, documentation of a single leak on fluoresce in angiography, and presentation within 2 weeks of onset of symptoms. Patients with any other ocular or systemic disease, any surgery or trauma within 2 weeks of presentation, receiving any form of local or systemic steroids, obesity with a body mass index of more than 30, alcohol abuse or dependence, and major depression (DSM III R criteria) were excluded from our study, since all these conditions can independently alter the endogenous cortisol levels.

At the time of inclusion into our study, all the subjects underwent a systemic evaluation, comprising a complete medical history, general physical examination, erythrocyte sedimentation rate, liver, and kidney function tests. The ocular evaluation comprised visual acuity testing, slit lamp biomicroscopy of the anterior and posterior segment (with a +90 D lens), indirect ophthalmoscopy, and fluoresce in angiography.

Cortisol sampling was done after the patient was evaluated. For plasma cortisol estimation, 10 ml of venous blood was taken and sent for cortisol analysis. Cortisol analysis was done on the spectrophotometer based on the Porter–Silber reaction.

## 3. Results

There was no significant difference in the age and duration of symptoms in the two groups. The mean age (SD) of the patients was 28.6(5.19) years (range 20–36years).

The mean duration of symptoms before inclusion in the study was 8.56 (4.19) days in the patients

In 12patients, the right eye was involved, while 8 had CSCR in the left eye.

The best corrected Snellen visual acuities in the patient group varied from 6/6 to 6/12 in 14 cases and 6/18 to 6/36 in 6 cases.

The means (SD) of the 8 am and 11 pm plasma cortisol valuesin the patients and controls were 28.67µg/dl (8.26)µg/dl and 21.15 (11.54)µg/dl, revealing significantly higher cortisol values in CSCR patients (*t* test p<0.001).

## 4. Discussion

The exact aetiology of CSCR remains controversial. Various studies have documented raised levels of endogenous steroids in states associated with an increased incidence of CSCR, such as pregnancy[18] stress,[10,11,17,19] type A personality,[20] and Cushing's syndrome.[12]CSCR has also been shown to have been precipitated by systemic steroid therapy,[6,14,15] indirectly implicating steroids in the pathogenesis of CSCR. Recently Bouzas *et al* [12] reported the development of CSCR in three out of 60 (5%) patients with

confirmed Cushing's syndrome, during a period of active untreated disease while plasma cortisol levels were high. Since both CSCR and endogenous Cushing's syndrome are rare diseases, their coincidental association would be highly improbable, especially as Cushing's syndrome affects women more frequently while CSCR is seen predominantly in men. Our study has convincingly shown the positive association between endogenous glucocorticoids (cortisol) and CSCR. We documented the presence of high endogenous cortisol levels in CSCR patients.

It is hypothesised that increased endogenous glucocorticoids may act as the inciting or precipitating factor for the development of CSCR by their effect on the RPE and the choroidal vasculature by the following mechanisms:

1) Cortisol, by suppressing synthesis of extracellular matrix components and inhibiting fibroblastic activity, may cause direct damage to the RPE cells or their tight junctions and may even inhibit any reparative activity in the RPE after damage by another aetiological agent.
2) Cortisol excess may cause increased capillary fragility and hyperpermeability, leading to choroidal circulation decompensation and leakage of fluid into the subretinal space. Furthermore, endogenous hypercortisolism, by inhibiting proliferation of T and B lymphocytes and the migration of leucocytes and macrophages[35] may induce a state of immunosuppression, which may increase the proneness to a subclinical acquired infection or reactivation of a latent infection, which may then damage the RPE barrier.
3) Cortisol by its direct effect on ion transport may be responsible for the reversal of polarity of the RPE cells, causing them to secrete ions into the subretinal space, leading on to osmotic fluid attraction and theserous macular detachment.

It is our belief that raised levels of endogenous steroids could set off a chain of events, which could result in damage to the RPE barrier and the choroidal vasculature, consequently precipitating CSCR. We, therefore feel that CSCR may be a multifactorial disorder, with increased levels of endogenous steroids playing a pivotal role in the pathogenesis of the disease.

## References

[1] Spitznas M. Central serous retinopathy. In: Ryan SJ,Schachat AP, Murphy RP, Paty A, eds. *Retina.* St Louis: CVMosby, 1989;**2**:217–27.
[2] Von Graefe. Uber centrale recidivierende Retinitis AlbrechtVon Graefes. *Arch Ophthalmol*1866; **12:211**.
[3] Gifford SR, Marquadt G. Central angiospastic retinopathy.*Arch Ophthalmol* 1939; **21:211**–28.
[4] Guyer DR, Yanuzzi LA, Slakter JS, Sorenson JA, Ho A,Orlock D. Digital indocyanine green video angiography ofcentral serous chorioretinopathy. *Arch Ophthalmol* 1994; **112:1057**–62.
[5] Diddie K, Aronson A, Ernest I. Chorioretinopathy in a caseof systemic lupus erythematosus. *Trans Am Ophthalmol Soc*1977; **75:122**–31.
[6] Eckstein MB, Spalton DJ, Holder G. Visual loss fromcentral serous retinopathy in systemic lupus erythematous.*Br J Ophthalmol* 1993; **77:607**–9.
[7] Klien BA. Macular and extramacular serous chorioretinopathy.*Am J Ophthalmol* 1961; **51:231**.
[8] Lowestein A. Central angiospastic retinopathy. *Br JOphthalmol* 1941; **25:269**.
[9] Dellaporta A. Central serous retinopathies. *Trans AmOphthalmol Soc* 1976; **74:144**–53.
[10] Harrington DO. Psychosomatic interrelationships in ophthalmology.*Am J Ophthalmol*1948; **31:1241**–51.
[11] Harrington DO. Psychosomatic manifestations in ophthalmology.*Trans Am Acad Ophthalmol* 1947; **52:78**.
[12] Bouzas EA, Scott MH, Mastorakos G, Chrousos GP, KaiserKupfer MI. Central serouschorioretinopathy in endogenoushypercortisolism. *Arch Ophthalmol* 1993; **111:1229**–32.
[13] Gass JD. Central serous chorioretinopathy and whitesubretinal exudation during pregnancy. *Arch Ophthalmol*1991; **109:677**–81.
[14] Wakakura M, Ishikawa S. An evaluation of corticosteroidtreatment for central serous chorioretinopathy. *RinshoGanka* 1980; **34:123**–9.*Endogenous cortisol profile in patients with central serous chorioretinopathy* 963
[15] Wakakura M, Ishikawa S. Central serous retinopathycomplicating systemic corticosteroid treatment. *Br JOphthalmol* 1984; **68:329**–31.
[16] Yoshika H, Katsume Y, Akune H. Experimental centralserous chorioretinopathy. *Jap J Ophthalmol* 1981; **25:112**–8.
[17] Gelber GS, Schatz H. Loss of vision due to central serouschorioretinopathy following psychological stress. *Am J Psychiatry*1987; **144:46**–50.
[18] Chumbley LC, Frank RN. Central serous retinopathy andpregnancy. *Am J Ophthalmol* 1974; **77:158**–60.
[19] CordesFC. A type of foveomacular retinitis observed in theUS Navy. *Am J Ophthalmol* 1944:**27**:803.
[20] Yanuzzi LA. Type A behaviour and central serousretinopathy. *Retina* 1987; **7:111**–30. Nonprotein bound cortisol in pregnancy. *J Clin EndocrinolMetab* 1969; **29:757**–9.