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Original Research Article

Clinical study of central serous chorioretinopathy presenting in a tertiary care centre

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ABSTRACT

Background: Central Serous chorioretinopathy (CSCR) is one of the common causes of visual handicap affecting young people of highly intellectual professionals at the peak of their career which can lead to irrecoverable loss of vision.
Methods: The present prospective observational population-based study was conducted in the Department of Ophthalmology, S.C.B. Medical College, Cuttack, Odisha from October 2013 to September 2015. The total number of patients attended the Outpatient Department (OPD) during the study period were 1,83,199. Amongst which 123 patients diagnosed to have CSCR were selected for the present study.
Results: Incidence of CSCR during in this study period was 0.06%. The age group most commonly affected was 31 to 40 years. Males were affected 7 times more commonly than females. Increased incidence was noticed in bank employees (21.1 %) and IT professionals (17.8%).
Conclusions: There was increased incidence of the disease in people under stressful life condition.

Keywords: Central serous chorioretinopathy, Fundus fluorescein angiography, Ink blot pattern, Smokestack pattern

INTRODUCTION

Central Serous chorioretinopathy is one of the common causes of visual handicap affecting young people. It affects highly intellectual professionals like bank employees, IT professional, businessmen, architects at the peak of their career. It can be bilateral and recurrent. But most importantly this condition can lead to permanent damage and cause irrecoverable loss of vision. For this reason, there are constant effort from the research faculties as well as the clinicians to find out the mystery behind this disease. CSCR is the fourth most common retinopathy after age-related macular degeneration, diabetic retinopathy and retinal vein occlusions.[1] It is characterized by fairly sudden onset of visual impairment with micropsia or metamorphosia or both and also color desaturation, delayed retinal recovery time to bright light and relative scotoma. In general population the incidence of the disease is one case a year in every 22000 inhabitants.[2] Recurrence may occur after 1 year or many years later (45% or 50%). It is a self-resolving disease. Most of the cases resolve spontaneously within 1-6 months. Sometimes it may require longer than 12 months to resolve. Some cases may be associated with permanent impairment of visual acuity or scotoma and impairment of quality of vision. CSCR is characterized by idiopathic serous detachment (SD) of the sensory retina.

It is an exudative macular disease leading to neurosensory detachment. The characteristic neurosensory detachment on the posterior pole is caused by leakage of fluid seen at the level of retinal pigment

epithelium (RPE). Several theories have tried to explain the mechanism and pathogenesis of CSCR. In all of these theories increased choroidal vascular permeability is supposed to be the reason for the SD of the RPE. The fundus fluorescein angiography (FFA) findings are very important in the diagnosis of CSCR. In CSCR, there is a breakdown of the outer blood retinal barrier which allows the passage of free fluorescein molecules into the subretinal space. Various patterns of dye leakage are seen. The most common are smokestack, diffuse RPE leakage (ooze), RPE atrophic tracts.

The cause, clinical manifestations, natural course and the pathogenesis of this condition remains poorly understood and evidence on the treatment modalities which are followed at present is minimal. So, this study was carried out to know the clinical profile and the probable etiological factors of CSCR and its relation with age, sex, type of personality, occupation, natural habits and other systemic or ocular illness, to study the natural course of visual recovery and to find out the response to different available forms of treatment in randomly selected patient groups.[1,2]

METHODS

The present prospective observational population-based study was conducted in the Department of Ophthalmology, S.C.B. Medical College, Cuttack over a period of two years (October 2013 to September 2015). The selection of patients for this study were made from the patients attending the OPD.

Inclusion criteria

All newly diagnosed OPD patients of CSCR aged more than 20 years consented for follow up for 6 months were included in the study.

Exclusion criteria

Diagnosed cases of uveitis, choroidal tumors or any other macular diseases, patients suffering from confounding diseases like diabetes mellitus or patients lost to follow up for 6 months were excluded from the study.

Patients who presented to OPD with chief complaints of diminution of vision or distortion of vision underwent detailed evaluation. CSCR was diagnosed after thorough assessment of history, detailed general, systemic and ocular examination which included a 90 Diopter (90D) lens evaluation of macula. Patients with the findings like Subretinal fluid (SRF), RPE defect, small dot like deposits, RPE mottling and RPE clumping on 90 D examination of the macula underwent FFA.

The fundus photographs were taken, and fluorescein angiography was done in all the 123 patients. After dilating pupil injection fluorescein sodium (10%) 5 ml was given intravenously and multiple photographs were taken by the digital fundus camera. The patterns of CSCR on FFA were observed and recorded.

Patients with at least one leakage point at the macula were diagnosed to have CSCR. Cases with no visual recovery after 3 months were subjected to laser photocoagulation.

RESULTS

Total 1,83,199 numbers of patients attended the OPD during the study period; amongst which 126 eyes of 123 patients those diagnosed to have CSCR were selected for the present study. The majority of cases were male. Unilateral presentation was in most of the cases (Table 1).

Table 1: Baseline characteristics of CSCR cases enrolled in the study.

Total number of cases	Male	% (Percentage)	Female	%	Unilateral	%	Bilateral	%
123	108	87.8	15	12.1	120	97.5	3	2.5

The maximum no. of patients was in age group 31-40 yrs. There were no patients in the 71-80 years age group (Table 2).

The bank employees constituted the major share followed by IT professional. (Table 3) The diminution of vision was the most common symptom and recovery from dark to light was the least common symptom (Table 4). The maximum numbers of patients were addicted to some form of tobacco. Emotional stress (type A personality) was observed in 15.9% of patients (Table 5). The maximum number of patients in this study had a VA of 6/12 at presentation. Very few patients were with VA of 6/60 or less (Table 6).

Table 2: Age wise distribution of CSCR cases in the study population.

Age group (in years)	No. of patient	Percentage
21-30	19	15.4
31-40	57	46.3
41-50	35	28.4
51-60	8	6.5
61-70	4	3.2
71-80	0	0

Table 3: Occupation wise distribution of CSCR in the study sample.

Occupation	No. of patient	Percentage
Bank employee	26	21.1%
IT professionals	22	17.8%
farmers	20	16.2%
Manual labors	2	19
Corporators	19	15.4%
Medical professionals	6	4.8%
Businessmen	8	6.5%
Teachers	8	6.5%
Housewives	4	3.2%

Table 4: Different clinical presentations of CSCR in the study group.

Complaint	Number of patients	Percentage
Diminution of vision	122	99.1
Metamorphosia	82	66.6
Micropsia	16	13.0
Macropsia	20	16.2
Positive scotoma	80	65.0
Delayed recovery from dark to light	8	6.5

Table 5: Risk factors associated with CSCR in the study population.

Risk factors	No. of patients	Percentage
No. of identifiable cause(idiopathic)	26	21.1
Emotional stress (type A personality)	19	15.4
Systemic hypertension	9	7.3
GRED	5	4.06
Pregnancy	6	4.8
Organ transplantation	0	0
SLE	0	0
Tobacco use	32	26.01
Alcohol use	10	8.1
Renal disease	4	3.2
Corticosteroids	12	9.7

The maximum number of patients who had a VA 6/6p; recovery of 6/6 was found in 46.03%, 51.5% and 59.52% at 1 month, 3 months and 6 months respectively. (Table 7). Hypermetropia was the most common refractive error at presentation in this study population. Emmetropia was least common (Table 8).

Subretinal fluid at the macula was the most common finding in this study population. The next common findings were small dot like deposits and RPE defect at the macula.

All the cases showed absent foveal reflex (Table 9). The partial thickness macular hole was the most common diagnostic dilemma encountered in this study.

Table 6: Visual acuity (VA) of CSCR cases at initial presentation.

VA	Number of patient	Percentage
6/6p	4	3.1%
6/9 to 6/9p	27	21.4%
6/12	41	32.5%
6/18	21	16.6%
6/24	17	13.4%
6/36	13	10.3%
6/60 and less	3	2.38%

Table 7: Visual acuity of CSCR cases on follow up examination.

VA	1st week follow up	1st month follow up	3rd month follow up	6 month follow up
6/6	21 (16.6%)	58 (46.03%)	65 (51.5%)	75 (59.52%)
6/9 to 6/9p	28 (22.2%)	27 (21.4%)	23 (18.2%)	22 (17.4%)
6/12	36 (28.5%)	24 (19.04%)	25 (19.5%)	20 (15.8%)
6/18	17 (13.4%)	9(7.1%)	0	0
6/24	9 (7.1%)	3 (2.3%)	0	0
6/36	9(7.1%)	2(1.5%)	1(0.7%)	1(0.7%)
6/60 and less	6(4.7%)	3(2.3%)	2(1.5%)	2(1.5%)

Table 8: Refractive error of CSCR cases at initial presentation.

Refractive error	No. of eyes	Percentage
Emmetropia	18	14.2
Hypermetropia	72	57.2
Myopia	36	28.6
Total	126	100

Table 9: Macular examination findings of CSCR cases with 90D lens at initial presentation.

Findings	No. of eyes	Percentage
Subretinal fluid	56	44.4
RPE defect	29	23.01
Small dot like deposits	31	24.6
RPE mottling	5	3.9
RPE clumping	5	3.9

Table 10: Conditions simulating CSCR based on Ophthalmoscopic findings.

Disease conditions	No. of eyes	Percentage
*CNVM	10	23.8%
Choroidal melanoma	2	4.8%
Choroidal haemangioma	2	4.8%
Partial thickness macular hole	18	42.8%
Early **AMD	10	23.8%
Total	42	100%

*CNVM-Choroidal neovascular membranes, **AMD- age-related macular degeneration

Table 11: Distribution of CSCR cases according to FFA pattern (n=76).

FFA pattern	No. of eyes	Smoke stack	Ink blot
Single leak	55(72.36%)	24(31.57%)	31(40.78%)
Double leak	6(7.89%)	3(3.94%)	3(3.94%)
Multiple leak	9(11.84%)	0	0
Pigmented leak	6(7.89%)	-	-

Table 12: Recurrence of CSCR cases at 6 months of follow up examination.

Total no. of eyes	126
No. of eyes that showed recurrence	26
Percentage	19.6%

Table 13: History of drug intake associated with the development of CSCR.

Drugs	No. of patients	Percentage
Steroid	12	9.75%
Anti-Tubercular(ATT)	6	4.87%
Anti-hypertensive	9	7.31%

Authors encountered 42 eyes out of 126 where ophthalmoscopic appearance simulated other conditions which are depicted above, but later got confirmed as CSCR following FFA (Table 10). Single leak variety constituted maximum cases of FFA pattern.

Amongst single leak variety ink blot pattern outnumbered smokestack pattern. (Table 11) Recurrence of CSCR occurred in19.6% of eyes. (Table 12) Out of 123 patients 27 patients revealed the history of drug intake.

The steroid intake cases outnumbered all other drug intake cases. (Table 13). Three eyes which didn't have complete visual recovery were subjected to laser photocoagulation. Complete recovery of VA was found in 66.6% eyes. Only 1 case had PD (Table 14).

DISCUSSION

The incidence of CSCR in the general population during the study period was 0.06%. The rate of CSCR in this study was 6-fold higher in men than in women. The various studies showed the incidence was approximately 6 times higher in men than women.[3] Anna elias et al, study showed a male female ratio close to 9:1.[4] The recent literature given by Ross A, Ross AH, Mohamed Q (2011) shows a male to female ratio of 6:1.[2] Liew et al, in 2012 reviewed the epidemiology of CSCR in Australia.[5] Majority of cases in this study were having unilateral presentation. Even though Gackle et al, had observed that bilaterally in CSCR can go up to 40% of cases; at initial presentation it can be as low as 4%.[6]

This study showed a similar pattern in the initial pattern of CSCR as far as bilaterally was concerned. This is also in accordance with Tariq et al, who conducted a study in Indian population and found only 4 out of 100 cases had bilateral CSCR.[7] This study found maximum number of patients in the age group 31 to 40 years. Mitsui and Sakanashi's study shows 80% of patients were in the age range of 35 to 45 years.[8] Wang et al, and Ross A et al, found that the mean age of onset is 41 to 45 years.[1,2] Anna Elias et al, found a mean age of 42.16 years.[4] Tariq Qureshi et al, study showed that maximum patients are in the age group of 36 to 40 years.[7] Bank employees constituted the major share followed by IT professional. There are very few studies linking CSCR to professions.[9] From this study, it is evident that stress is the leading cause for CSCR. This study also found that diminution of vision was the most common symptom. The presenting symptoms in CSCR vary widely and there is considerable overlap of symptoms which was evident from this study. Yameda et al, studied 106 eyes of 53 patients and found that the commonest complaint was a central or paracentral scotoma (58%), followed by blurring of vision 34% and metamorphosia.[10] Shahid Jamal Siddique et al, (2008) who studied pattern of CSCR on FFA found that all the patients presented with blurring of vision, followed by central scotoma being the next common symptom.[11] Tariq Qureshi et al, (2013) also found that 100% of patients presented with diminution of vision and second most common symptom being a positive scotoma.[7] Emotional stress (type A personality) was observed in 15.9% of patients as found by Wang et al,[1] Corticosteroid administration and CSCR is supported by Carvalho-Recchia et al, Jonas et al where they found that all forms of exogenous corticosteroid administration, to be associated with CSCR.[12,13] Maximum patients in this study had a VA of 6/12 at presentation. Very few patients were with visual acuity of 6/60 or less (2.38%). Study by Wang et al, supported our study.[1]

Patients who had a VA 6/6p; recovery of 6/6 was found in 46.03%, 51.5% and 59.52% at 1 month, 3 months and 6 months respectively. Same was the result by Wang et al.[1] Literature on the refractive error at presentation in CSCR varies widely. Multak and Dulton found 70% of

the study population to be hypermetropic, 26% myopic and 4% emmetropic.[14] This study is in concordance with this, but the study by Tariq et al, 2013 had majority of their study population in the emmetropic group.[7]

Table 14: VA Amsler Grid finding and fundus picture of cases of recurrent CSCR just before and one month after laser photocoagulation therapy (n=3).

Patients	VA before laser	VA after laser	Amsler grid before laser	Amsler grid after laser	Fundus before laser	Fundus after laser
1st patient	6/18	6/9	CS	Normal	SD	Normal
2nd patient	6/36	6/12	CS	Metamorphosia	*SD,SRF,PD	**PD
3rd patient	6/24	6/9	Metamorphosia	Metamorphosia	SD,***SRF	Normal

*SD -Serous Detachment, CS -Central Scotoma, ***SRF -Subretinal fluid, **PD - pigment epithelial defect.

The most common specific finding was the serous detachment of macula with underlying subretinal fluid which was seen in 44.4% of cases. RPE defect was found in 23.01% of cases. Studies done by Laatikainen et al, in 1991, Castro-correia et al, in 1992, Van Velthoven et al, in 2005, Mitarai et al, in 2006 shows a wide variation of this finding from 5% to 63%.[15-17] The condition that produced major diagnostic dilemma clinically was a partial thickness macular hole, followed by CNVM and early AMD in 10 cases each. Gass JD IN 1967 had observed that choroidal haemangioma, polypoidalchoroidal vasculopathy, choroidal melanoma can simulate CSCR and cause diagnostic dilemma as they can also lead to subretinal fluid.[18] Macular hole was also considered by him as an important differential diagnosis. This study shows that early AMD can also cause diagnostic confusion especially in bilateral cases. It is also evident that the speed of recovery is dependent on the visual acuity at presentation. Gilbert et al, long term follows up study of 68 eyes showed that 57% of cases returned to 6/6 vision even though the follow up period was up to 3 years. They found that the visual recovery was highly dependent on initial VA.[19] In this study, 75.8% of eyes regained 6/6 vision by 6 months. Both the patients with presenting VA of 6/60 didn't regain 6/6 vision while only 28.6% of patients with presenting VA 6/36 regained 6/6 vision. 19.6% of eyes had recurrence. According to Loo et al, and Yap et al, recurrence rate in the absence of intervention can range from 15 to 50% depending upon the study type and length of follow up.[20,21]

In general approximately 1/3rd to 1/2 of the patients have a second recurrence often within a year of first episode while 10% have 3 or more recurrence when followed up for 15 years (Gass JD, Ficker et al, Anna Elias et al,) showed a recurrence rate of 13.37% in 6 month follow-up period.[4,18,22] In this study the recurrence rate was found to be 19.6%. Small (250 micron) extra foveal leaks on FFA may be treated with argon laser photocoagulation to the leakage site, which, in a small randomized controlled trial of 42 eyes, was found to have shortened the duration of CSCR by approximately 2 months compared with Sham laser Robertson et al, and Hussain D et al,.[23,24] Long term (6-12 years) follow up of participants showed no advantage of laser over observation in terms of final VA, color hue discrimination, rate of recurrence (slightly under 50% in treated group and over 50% in untreated group) or progression to chronic CSCR.[22]

CONCLUSION

Central serous chorioretinopathy, even though labelled as a disease of unknown etiology, gave us so many clues to the probable etiopathogenesis during this study. It affects mainly young males with recovery of normal vision in most of the cases; but there exits the potential to cause irreversible diminution of vision and can at times run a long course with recurrences as well. The disease even though may be confused with other conditions affecting the macula, can be well differentiated with a meticulous clinical examination and FFA. The increased incidence of the disease in people under stressful life condition, type A personality and smokers could point towards a possible clue to the cause of the disease. This study has thrown some light into this enigmatic condition and we conclude that more and more research need to be done to find the exact etiopathogenesis of this potentially sight threatening condition.

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REFERENCES

1. Wang M, Munch IC, Hasler PW, Prünte C. Larsen M Central serous chorioretinopathy. Acta Ophthalmol. 2008;86(2):126-45.
2. Ross A, Ross AH, Mohamed Q. Review and update of central serous chorioretinopathy. Curr Opin Ophthalmol. 2011;22(3):166-73.
3. Kitzmann AS, Pulido JS, Diehl NN, Hodge DO, Burke JP. The incidence of central serous chorioretinopathy in Olmsted County, Minnesota, 1980-2002. Ophthalmol. 2008;115(1):169-73.

4. Elias A, Gopalakrishnan M, Nair D, Bhat S, Gudapati R, Anantharaman G. A 10-Year Study of Central Serous Chorioretinopathy: Recurrence Rate and Factors affecting Recurrence. Wor J Reti Vitr. 2011;1(2):69-74.
5. Liew G, Quin G, Gillies M, Fraser-Bell S. Central serous chorioretinopathy: a review of epidemiology and pathophysiology. Clin Exp Ophthalmol. 2013;41(2):201-14.
6. Gackle HC, Lang GE, FreisslerKA, Lang GK. Central serous chorioretinopathy. Clinical, fluorescein angiography and demographic aspects. Ophthalmol. 1998;95:529-33.
7. Qureshi T, Abdulah N, Fazili. A Clinical profile, fundus fluorescein angiographic and optical coherence tomographic finding in Central serous chorioretinopathy. J Evol Medi Dent Sci. 2013;2(34):6497-501.
8. Mitsui Y, Sakanashi R. Central angioplastic retinopathy. Am J Ophthalmol. 1956;41(1):105-14.
9. Lajmi H, Hmaied W, Ben Jalel W, Akremi A, El FekihL. Central serous chorioretinopathy: Professional repercussions among agents of the internal security forces. J Ophtalmol. 2018;41(8):739-43.
10. Yamada K, Hayasaka S, Setogawa T. Fluorescein-angiographic patterns in patients with central serous chorioretinopathy at the initial visit. Ophthalmologica. 1992;205:69-76.
11. Siddiqui SJ, Shah SIA, Pechuho MA, Abbasi SA, Shaikh FF. Pattern of Central Serous Chorioretinopathy (CSCR) on Fundus Fluorescein Angiography. Pak J Ophthalmol. 2008;24(4).
12. Carvalho-Recchia CA, YannuzziLA, Negrao S. Corticosteroids and central serous chorioretinopathy. Ophthalmol. 2002;109:1834-7.
13. JB Jonas, BA Kamppeter. Intravitreall triamcinolone acetonide and central serous chorioretinopathy. Br J Ophthalmol. 2005;89(3):386-87.
14. Mutlak JA, Dutton GN, Zeini M, Allan D, Wail A. Central visual function in patients with resolved Central Serous retinopathy. A long-term follow-up study. Acta Opthalmol. 1989;67:532-6.
15. Laatikainen L, Hoffren M. Long- term follow-up study of nonsenile detachment of the retinal pigment epithelium. Eur J Ophthalmol. 1991;1:79-84.
16. Castro-correia J, Coutinho MF, Rosas V, Maia J. Long-term follow-up of Central serous retinopathy in 150 patients. Doc Ophthalmol. 1992;81:379-86.
17. Van velthoven ME, Verbraak FD, Garcia PM, Schlingemann RO, Rosen RB, de SmetMD. Evaluation of Central serous retinopathy with enface optical coherence tomography. Br J Ophthalmol. 2005;89(11):1483-8.
18. Gass JD. Pathogenesis of disciform detachment of the neuroepithelium. Am J Ophthalmol. 1967;63:1-139.
19. Gilbert CM, Owens SL, Smith PD, Fine SL. Long-term followup of Central serous chorioretinopathy. Br J Ophthalmol. 1984;68(11):815-20.
20. Loo RH, Scott IU, FLYNN Jr HW, Gass JD, Murray TG, Lewis ML, et al. Factors associated with reduced visual acuity during long-term follow-up of patients with idiopathic central serous chorioretinopathy. Retina. 2002 Feb 1;22(1):19-24.
21. Yap EY, Robertson DM. The long-term outcome of Central serous chorioretinopathy. Arch Ophthalmol. 1996;114(6):689-92.
22. Ficker L, Vafidis G, While A, Leaver P. Long-term follow-up of a prospective trial of argon laser photocoagulation in the treatment of Central serous retinopathy. Br J Ophthalmol. 1988;72(11):829-34.
23. Robertson DM, IIstrup D. Direct, indirect and sham laser photocoagulation in the management of central serous chorioretinopathy. Am J Ophthalmol. 1983;95(4):457-66.
24. D Hussain, JD Gass. Idiopathic central serous chorioretinopathy. Indian J Ophthalmol. 1998; 46(3):131-7.

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