ORIGINAL ARTICLE

Features of central serous chorioretinopathy presenting at a tertiary care hospital in Lahore

Ahmad Zeeshan Jamil, Khurram Azam Mirza, Zaheer Uddin Aqil Qazi, Wasim Iqbal, Javed Khaliq, Fawad-ur-Rahman, Arslan Ahmed

Abstract

Objective: To evaluate the clinical, angiographic and optical coherence tomographic features of central serous chorioretinopathy in patients presenting at a tertiary care centre in Lahore.

Methods: The observational study was conducted at the Layton Rehmatulla Benevolent Trust Eye and Cancer Hospital Lahore from July 15, 2010 to December 15, 2011. Patients who had received prior treatment for the condition and allergy to fluorescein were excluded. There were 86 eyes of 64 adult patients with central serous chorioretinopathy. The following data was recorded: history, signs and symptoms, best corrected visual acuity, fundus fluorescein angiography, and central macular thickness measurement with optical coherence tomography. Data was analyzed using SPSS 17.

Results: Mean age of patients who presented during the study duration was 39.52±8.85 years. There were 53(82.8%) males and 11(17.2%) females. Of the total, 42(65.6%) cases had unilateral and 22(34.4%) cases had bilateral involvement. Chronic central serous chorioretinopathy was seen in 27(42.2%) cases while 37 (57.8%) cases were acute presentations. Retinal pigment epithelial detachment was observed in 29 (45.3%) cases. On fundus fluorescein angiography, there were 62 (72.1%) eyes that showed ink blot pattern. Median visual acuity at presentation was 0.25. Median central macular thickness at presentation was 550.5µ.

Conclusion: Central serous chorioretinopathy in the study sample was associated with pigment epithelial detachment, bilateral involvement, and presence of systemic diseases.

Keywords: Central serous chorioretinopathy, Optical coherence tomography, Retinal pigment epithelium, Visual acuity. (JPMA 63: 478; 2013)

Introduction

Central serous chorioretinopathy (CSCR) is the serous detachment of the neurosensory retina (Figure-1). CSCR is one of the 10 most common conditions that affect the macula.1It commonly occurs in young and middle-aged patients.[1]

A defect in the outer blood-retina barrier is attributed to its pathogenesis, whereby leaks in the retinal pigment epithelium (RPE) and hyper-permeability of choriocapillaries lead to separation of the eye layers.[2]

After an acute episode of the disease there is recovery of vision in most of the cases. Recurrent, longstanding and diffuse disease can lead to non-reversible vision loss.[3] Severe permanent visual loss occurs in about 5% of patients.[4] The disease is symptomatic in only one eye in most of the cases. It may be bilateral in about 18% cases.[5] Disease process in CSCR is more widespread and there is evidence of bilateral retinochoroidal dysfunction.[5]

Common associations of CSCR include Type A personality, Cushing's syndrome, systemic and intranasal steroid use, hypertension, organ transplant and systemic lupus erythematosis.[6]

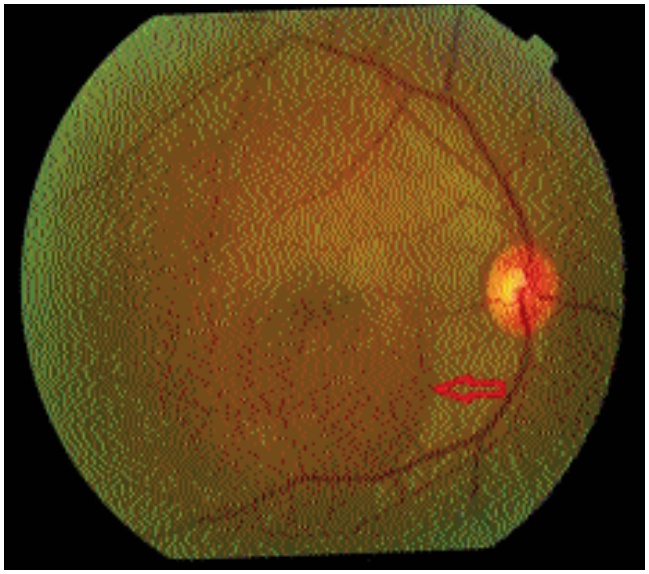

Figure-1: Colour fundus photograph of right eye showing round serous elevation of the sensory retinal at the macula (red arrow).

Layton Rahmatullah Benevolent Trust (LRBT), Township, Lahore.
Correspondence: Ahmad Zeeshan Jamil. Email: ahmadzeeshandr@yahoo.com

In most of these conditions there is an alteration of autonomic nervous system responses. Patients exhibit hyperactivity of sympathetic system.[6] Adrenergic hormones and glucocorticoids play a role in the pathogenesis of CSCR by their effects on RPE and choroids.[7]

However, it is still not completely understood how CSCR develops,[1] and three theories have so far been presented: RPE dysfunction theory, choroid dysfunction theory, and combined choroid and RPE dysfunction theory.

According to the RPE dysfunction theory,there is a reversal in RPE polarity that results in secretion of fluid and ions in the choreoretinal direction. Fluid leaks into the subretinal space and causes neurosensory detachment.[8,9] Choroidal dysfunction theory states that it is the choroid that is the major site for the development of CSCR. Choroidal ischaemia results in the development of hyper-permeability of choroidal vessels. Leakage in the choroid may cause overlying PRE and neurosensory detachment.[3] Various studies have shown abnormalities of choroidal circulation in patients of CSCR.[8,9] Indocyanine green (ICG) angiography-based studies have also shown that the hyper-permeability of choroid may be the etiological factor.[10] The combined choroid and RPE dysfunction theory states that the retina and choroid are affected simultaneously.

Stress management, life-style modification and control of risk factors have been the cornerstone of CSCR management. Photodynamic therapy, laser photocoagulation and pharmacological agents (acetazolamide, propranolol, mifepristone and ketoconazole) have been used to treat CSCR.[5] But these treatments only decrease the disease duration and have no effect on recurrence of disease.[11] RPE may be decompensated by chronic and recurrent disease and that leads to poor visual outcome.[12]

The objective of this study was to evaluate the clinical, angiographic and optical coherence tomographic features of CSCR in patients presenting at a tertiary care centre in Lahore.

Patients and Methods

The prospective observational study was conducted at Layton Rehmatulla Benevolent Trust Eye Hospital Lahore from July 15, 2010 to December 15, 2011. This study was approved by the ethics and research board of the hospital. It was in accordance with the Helsinki Declaration. Informed consent was taken from all the patients.

Inclusion criterion was patients presenting with CSCR. Patients having the disease for less than six months were defined as acute. Chronic CSCR was defined as disease present for more than six months. Patients with prior treatment for CSCR and allergy to fluorescein were excluded from the study.

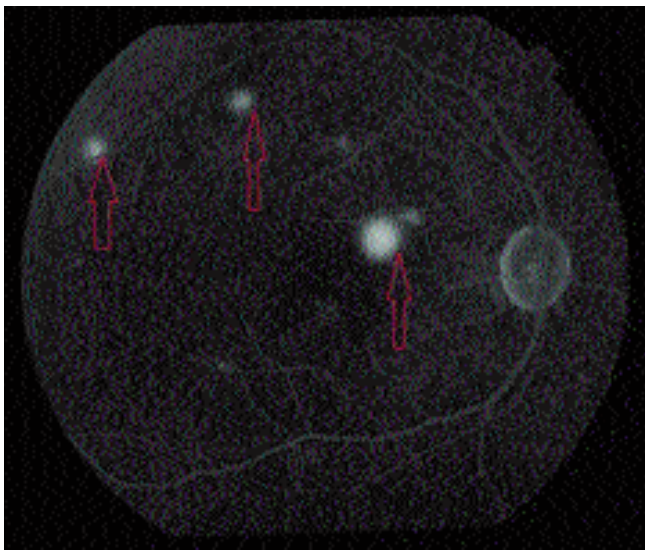

Figure-2: Fluorescein angiography image of right eye during the late phase. There are multiple areas of dye pooling in inkblot pattern (red arrows). The angiogram picture is suggestive of CSCR.

Patients' history was taken regarding onset, duration, symptoms of disease, systemic problems, use of drugs, smoking and anxiety.

Presenting best corrected visual acuity (BCVA) was recorded by Snellen's chart and then converted into decimal notion for statistical usage.

Detailed eye examination was performed with special emphasis on the posterior segment. Optic disc was particularly examined to rule out optic disc pit.

Fundus fluorescein angiography (FFA) was done (Figure-2), and the pattern of fluorescein leakage was noted. The presence of pigment epithelial detachment (PED) and subretinal precipitates was also noted.

Optical coherence tomography (OCT) was performed (Figure-3), and the central macular thickness (CMT) measured.

Statistical analysis was done in SPSS version 17. Data was presented as simple descriptive statistics. Age was presented as mean ± standard deviation. Visual acuity and CMT were presented as median. Gender, laterality of eyes, presence of PED, sub-retinal precipitates and angiographic pattern were presented by frequency and percentage.

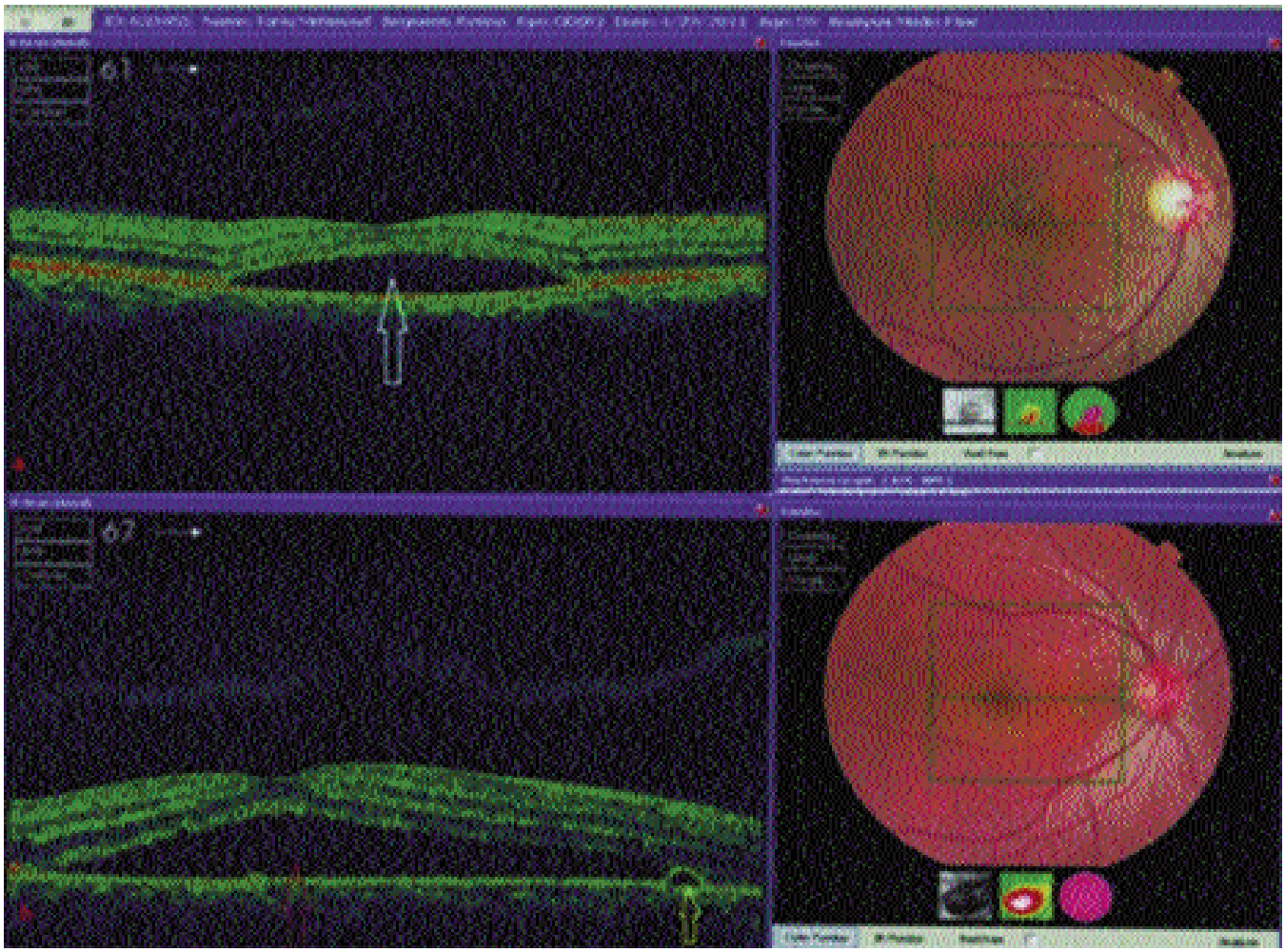

Figure-3: Optical coherence tomography. a) Optical coherence tomographic image of CSCR in right eye. There is optically empty elevation of sensory retina (white arrow). b) Optical coherence tomography of right eye. There is sensory retinal elevation along with small elevation of retinal pigment epithelium. This OCT scan shows CSCR (red arrow) with PED (yellow arrow).

Results

The study comprised 86 eyes of 64 patients with CSCR. There were 53(82.8%) males and 11(17.2%) females. The mean age was 39.52±8.85 years. There were 42(65.6%) cases with unilateral involvement and 22(34.4%) cases with bilateral involvement. Out of the unilateral cases, there were 20(31.3%) right eyes and 22(34.4%) left eyes. Of the total, there were 37(57.8%) cases with acute CSCR and 27(42.2%) cases with chronic CSCR. PED was present in 29(45.3%) cases,16(59.26%) cases had chronic CSCR and 13(35.13%) had acute CSCR. In terms of presentation, 31(48.43%) patients presented with complaint of decrease in vision; 13(20.31%) with complaint of scotoma, 9(14.06%) with metamorphopsia; 8(12.5%) with micropsia; and 3(4.68%) patients reported change in colours as their presenting complaint. Seven (10.93%) patients were having anxiety; 6(9.37%) were using naswar, gutka or smoking; 6(9.37%) were using sex-arousal medicines; 6(9.37%) were using systemic steroids, 4(6.25%) were having history of infertility; 2(3.12%) had acid peptic disease; 2(3.12%) patients were cases of viral hepatitis; 2(3.12%) were hypertensive; 2(3.12%) female patients were having menstrual irregularities; 2(3.12%) were undergoing psychiatric treatment; 2(3.12%) patients were involved with aggressive weight-reduction measures; 1(1.56%) patient was having full-arm burn; 1(1.56%) was using oxymetazoline nasal spray, and in 21(32.81%) patients we could not identify any systemic problem. Subretinal precipitates were present in 25(39.1%) patients, while 39(60.9%) showed no such finding. On FFA, 62(72.1%) eyes showed ink blot pattern; 15(17.44%) patients showed diffuse leakage; and 9(10.46%) had smoke-stack pattern. Median visual acuity at presentation was 0.25. Median CMT at presentation was 550.5µ.

Discussion

The prospective study looked at the presenting clinical, angiographic and OCT features of CSCR. The mean age of presentation of 39.52 years in this study group was similar to mean age of 41 years reported in an earlier study.[13] Another study whose sample comprised only of women reported older mean age at presentation, which was 47 years.[6] In our study male to female ratio was 4.8:1 that was less than described by How and coauthors[13] who found male to female ratio of 5.7:1 while it was greater than studied by Spaide and coauthors[14] who described male to female ratio of 2.6:1.0.

In this study, the disease was bilateral in 34% cases that was less than 44% reported in other Asian population.[13]

PED was present in 45.3% cases, which was higher than 8% reported earlier.[6] One important finding in this study was that patients with chronic CSCR showed more PED than patients with acute disease. This may be due to the reason that more advanced RPE damage leads to more prolonged disease.[13]

There were only 34% cases in the current study in which we could not identify any systemic problem associated with CSCR. This was far less than reported by other authors[6] who reported no associated medical condition in 76% cases. Anxiety was the most common systemic problem that was present in patients with CSCR as it was mentioned by Caccavale.[15] Use of systemic steroids was the second most common known factor associated with CSCR. This finding was in accordance with literature as well.[2] There were patients of CSCR with history of use of naswar, gutka, smoking, use of sex-arousal medicines and history of treatment for infertility. Their percentage in each category was 9.4%. We could hypothesize that all these factors might affect the choroidal circulation and/or RPE function. But these factors were not compared with their incidence in the general population. We recommend further research to determine the possible role of these systemic associations with CSCR. Other systemic problems known as risk factors for CSCR that we could identify were hypertension, peptic ulcer disease and treatment for psychotic problem.[2] In this study other systemic problems that were present in patients with CSCR were also identified. They included burn, viral hepatitis, menstrual irregularities, use of decongestant nasal spray and weight-reduction measures. Further research is needed to determine their role in cases of CSCR. We could propose that CSCR might be the ocular manifestation of widespread systemic disturbances. CSCR could be more commonly the result of systemic factors.[15]

Viral hepatitis needs special mention as its prevalence in Pakistan is around 7%.[16] But in this study only 3.1% patients with viral hepatitis also had CSCR. Whether there is less risk of CSCR in patients of viral hepatitis is an issue that needs to be investigated in further research.

Decrease in visual acuity was the most common presenting symptom followed by central scotoma and metamorphopsia. This is in accordance with the previous published results.[17]

On FFA, ink-blot pattern was the most common angiographic finding followed by diffuse leakage, while smoke stack was the least common angiographic pattern. This is in contrast to the work that described ink-blot as the most common and smoke-stack as the second most common pattern.[13] This difference might be due to the fact that the earlier work studied acute CSCR, and the current work included both acute and chronic cases. Chronicity of disease could be responsible for the diffuse damage to RPE that led to diffuse pattern of leakage.[18]

Median visual acuity at presentation in this study was 0.25 decimal (20/80) that was less than the presenting visual acuity of 20/30 in a study.[6] The difference might be due to the reason that the other study was done in females and in predominantly white patients.

Median CMT at presentation was 550.5µ that was more than the presenting central retinal thickness of 456µ in a study.[5]

Sub-retinal precipitates were present in 30.1% that was comparable to the earlier work.[6]

Although our hospital is a tertiary care unit where patients come from all over the country, but it might not be the true representation of the whole population of Pakistan. This was the limitation of our study. The small sample size was another limitation. To the best of our knowledge, this was the first work to be done of its kind in Pakistan so we could not compare it with local literature. The systemic problems in patients of CSCR were not compared with their prevalence in the general population. This was another limitation of our work. We had not done ICG angiography due to temporary non-availability of this facility. Further studies should be conducted to address these issues. Nonetheless, our study identified the systemic problems present in patients of CSCR, presenting

clinical, angiographic and optical coherence tomographic features of this disease in Pakistani population.

Conclusion

CSCR associated with PED is a common feature of the disease in our study sample. Bilateral involvement is a common presentation. Evidence of systemic problem is encountered in a large number of CSCR cases.

Acknowledgement

We are grateful to Mian Zulfiqar who contributed to the process of FFA and OCT.

References

1. Li XJ, Zhang JS. Intravitreal bevacizumab injection for chronic central serous chorioretinopathy. Chin Med J 2010; 123: 2145-7.
2. Haimovici R, Koh S, Gagnon DR, Lehrfeld T, Wellik S. Risk factors for central serous chorioretinopathy: a case-control study Ophthalmology 2004; 111: 244-9.
3. Jampol L M, Weinreb R, Yannuzzi L. Involvement of corticosteroids and catecholamines in the pathogenesis of central serous chorioretinopathy: A rationale of new treatment strategies. Ophthalmology 2002; 109: 1765-6.
4. Gass JDM. Stereoscopic atlas of macular diseases: Diagnosis and treatment, 4th ed. St. Louis: Mosby, 1997; vol.1, 52-70.
5. Mehany SA, Shawkat AM, Sayed MF, Mourad KM. Role of Avastin in management of central serous chorioretinopathy. Saudi J. Ophthalmol 2010; 24: 69-75.
6. Perkis SL, Kim JE, Pollack JS, Merrill PT. Clinical characteristics of central serous chorioretinopathy in women. Ophthalmology 2002; 109: 262-6.
7. Tewari H K, Gadia R, Kumar D, Venkatesh P, Garg SP. Sympathetic-parasympathetic activity and reactivity in central serous chorioretinopathy: a case-control study. Invest. Ophthalmol Vis Sci 2006; 47: 3474-8.
8. Guyer DR, Yannuzzi LA, Slakter JS, Sorenson JA, Ho A, Orlock D. Digital indocyanine green videoangiography of central serous chorioretinopathy. Arch Ophthalmol 1994; 112: 1057-62.
9. Prunte C, Flammer J. Choroidal capillary and venous congestion in central serous chorioretinopathy. Am J Ophthalmol 1996; 121: 26-34.
10. Taban M, Boyer DS, Thomas EL, Taban M. Chronic central serous chorioretinopathy: photodynamic therapy. Am. J. Ophthalmol 2004; 137: 1073-80.
11. Schaal KB, Hoeh AE, Scheuerle A, Schuett F, Dithmar S. Intravitreal bevacizumab for treatment of chronic central serous chorioretinopathy. Eur J Ophthalmol 2009; 19: 613-7.
12. Jalkh AE, Jabbour N, Avila MP, Trempe CL, Schepens CL. Retinal pigment epithelium decompensation I. Clinical features and natural course. Ophthalmology 1984; 91: 1544-8.
13. How AC, Koh AH. Angiographic characteristics of acute central serous chorioretinopathy in an Asian population. Ann Acad Med Singapore 2006; 35: 77-9.
14. Spaide RF, Campeas L, Haas A, Yannuzzi LA, Fisher YL, Guyer DR, et al. Central serous chorioretinopathy in younger and older adults. Ophthalmology 1996; 103: 2070-80.
15. Caccavale A, Romanazzi F, Imparato M, Negri A, Morano A, Ferentini F. Central serous chorioretinopathy: a pathogenic model. Clin Ophthalmol 2011; 5: 239-43.
16. Ahmed W, Qureshi H, Arif A, Alam SE. Changing trend of viral hepatitis — " A 21 year report from Pakistan Medical Research Council Research Centre, Jinnah Postgraduate Medical Centre, Karachi". JPMA 2010; 60: 86-9.
17. Dnniston AKO, Murray PI, editors. Oxford handbook of ophthalmology. New York: Oxford University Press; 2006.
18. Bujarborua D, Chatterjee S, Choudhury A, Bori G, Sarma AK. Fluorescein angiographic features of asymptomatic eyes in central serous chorioretinopathy. Retina 2005; 25: 422-9.