ORIGINAL ARTICLE

Angiographic Patterns and Tomographic findings in Central Serous Chorioretinopathy revisited

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ABSTRACT

Aim: To address angiographic patterns and optical coherence tomographic changes in central serous chorioretinopathy in a Pakistani cohort.

Study design: Observational, Descriptive, study.

Study period: 2012 to 2015

Methods: 51 pts of Central serous chorioretinopathy (CSCR) from a tertiary care hospital were included in the study. History charts, clinical examination, Optical coherence tomography (OCT) and fundus fluorescein angiograms were reviewed. The data considered for the study was age, sex, ocular manifestations with special consideration to angiographic patterns & structural changes on OCT.

Results: Male to female ratio was 9.1:1. Mean age was 40 years. Ratio of right to left eye was 1.9:1. There were five different angiographic patterns of CSCR; Inkblot, Smoke stack, diffuse, mixed and non-leaking. All patterns were seen in both acute and chronic disease. When OCT scans were analyzed, RPE (retinal pigment epithelium) thinning, Granular IS/OS junction (inner segment/outer segment), RPE detachments, dipping sign, and disrupted RPE were seen. Frank choroidal ischemia was seen in one patient. Likewise, CSCR associated with choroidal folds was observed in one patient. **Conclusion:** A reliable relationship does not exist between angiographic patterns, structural abnormalities on OCT and duration of CSCR. Some patients have non-leaking CSCR in which

abnormalities on OCT and duration of CSCR. Some patients have non-leaking CSCR in which diagnosis is confirmed on OCT.

Keywords: Central serous chorioretinopathy, Ink blot pattern, Smoke stack pattern,

INTRODUCTION

Central serous chorioretinopathy (CSCR) is an idiopathic disorder characterized by a localized serous detachment of the sensory retina at the leakage macula secondary to choriocapillaris through one or more hyperpermeable RPE sites¹. To diagnose a case of CSCR, it is important to rule out other causes of sensory retinal detachment, e.g., choroidal neovascularization, inflammation, optic disc pit and tumours . In CSCR the leakage is either focal or diffuse from areas of dysfunctional RPE. Sophisticated techniques like FFA. OCT and **ICGA** (Indocvanine Angiography) have provided an insight into the pathogenesis and leakage patterns of CSCR. This particular study describes demographic, angiographic and optical tomographic changes in this benign retinochoroidopathy. Some existing concepts about CSCR are endorsed and comments are made on the non-leaking type of CSCR.

MATERIALS AND METHODS

It was a descriptive study carried out in a tertiary care hospital in the city of Lahore. 51 Patients of Central

serous chorioretinopathy were included in the study. Provisional diagnoses of the patients were made clinically. Clinical history, including ocular as well as systemic history, was taken. Ocular examination included distance and near visual acuity, pupillay reactions to light and accommodation, Slit lamp Biomicroscopy, Ophthalmoscopy and Tonometry. All the clinical diagnostic tests were performed by an ophthalmologist. Fundus fluorescein angiogram and Optical coherence tomograms were performed for all patients. The study was in accordance with the ethical standards of the committee on human experimentation with the Helsinki Declaration of 1975, as revised in 1983. All male/female patients having CSCR were included in the study. Patients with CSCR but with poor quality FFA/ OCT (because of media opacity), with co-existing ocular diseases of anterior or posterior segment and patients with optic disc pit were included in the study. Data was compiled and statistical analysis was done.

RESULTS

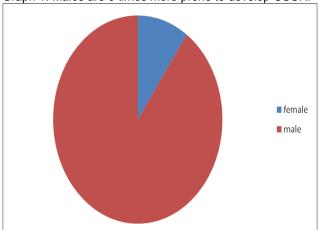
CSCR is predominantly a disease of males; male to female ratio of 9.1:1 was seen in this study (Graph 1). Age ranged from 29 to 74 years (mean 40 years). Although any eye could be involved, but in this study, right eye was more commonly affected than the left eye (Graph 2). In this particular study, there were five

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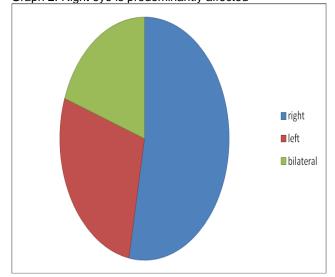
different angiographic patterns of CSCR; Inkblot (figure 1), Smoke stack (Figure 2), diffuse (figure 3), mixed and non-leaking (Graph 3). The commonest was Inkblot 27(52.94%). Mixed pattern (with smoke stack and inkblot in the same eye) was least common 2(3.9%). All patterns were seen in both acute and chronic disease and no clear cut relation could be made out between angiographic patterns and disease chronicity (Graph 4). One patient with gross Choroidal ischemia was also seen (figure 4).

When OCT scans were analyzed, RPE thinning was the commonest finding 29(56.86%). Granular IS/OS junction was the second commonest finding 24(47.10%). Other structural changes (Graph 5) were RPE detachments 18(35.30%), the dipping sign 17(33.33%) and disrupted RPE 1(1.96%). The dipping sign was caused by sub-retinal fibrin on the detached surface of neuro-sensory retina (figure 5).

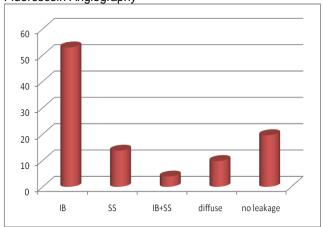
Graph 1: Males are 9 times more prone to develop CSCR.



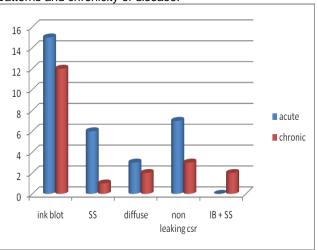
Graph 2: Right eye is predominantly affected



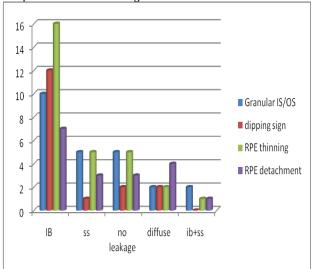
Graph 3: Percentage of different patterns of CSCR on Fluorescein Angiography



Graph 4: No relation between different angiographic patterns and chronicity of disease.



Graph 5: Structural changes in CSCR as seen on OCT



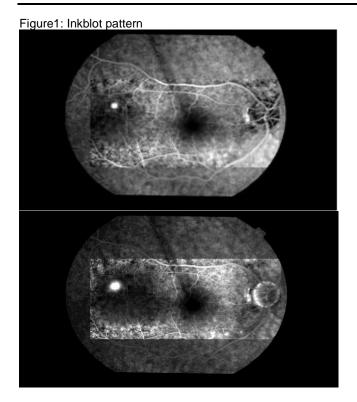


Fig. 2: Classical smoke stack

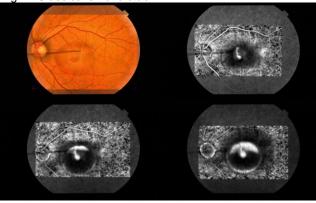


Fig.3: Diffuse leakage with small white dots indicating window defects

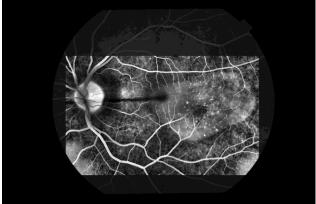


Fig. 4: Choroidal ischemia in a patient with chronic CSCR

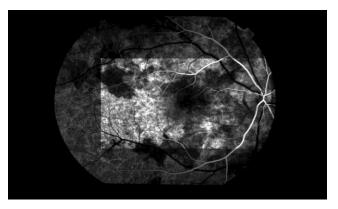
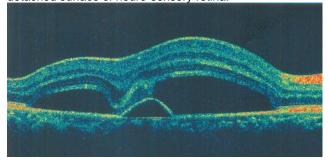


Fig. 5: Dipping sign due to sub-retinal fibrin on the detached surface of neuro-sensory retina.



DISCUSSION

CSCR is considered to develop as a result of choroidal vascular compromise (as evident from ICGA studies). These vascular changes lead to RPE dysfunction, leakage and consequently ending up in serous elevation of neurosensory retina^{2,3}. There was one patient in our study where gross choroidal ischemia was seen on FFA (Figure 2). It is supported by other authors who have described hypoperfusion and choroidal ischemia to be the basic characteristics of retrobulbar blood vessels circulation in cases of CSCR⁴.

Male preponderance, predominantly affected right eye and Inkblot pattern as the commonest leakage type are now universally accepted truths and is described in almost all studies of CSCR^{5,6,7}. Male to female ratio in this study was 9.1:1 which was similar to the Egyptian population⁸. In Korean study, this ratio was 3.9:1⁹. Although mean age is around 40 years in most studies but we had a patient of 74 years who presented with CSCR for the first time. Similarly, Spaide RF has described a patient with 82.9 years at the time of first diagnosis¹⁰.

Three angiographic patterns of CSCR are generally described in literature. We have highlighted two other types; mixed pattern and non-leaking CSCR. There were two patients who had Inkblot and Smoke stack pattern in the same eye. Another finding in our study was non-leaking CSCR (19.6%). These patients were clinically diagnosed but no leakage was seen on FFA. When OCT was performed, it showed

serous elevation of neuro-sensory retina. There can be two possible explanations for non-leaking characteristic. Firstly, we found RPE detachments in the center of non-leaking CSCR, which can later on seep fluid into sub-retinal space and not seen in the 10 to 15 minutes of FFA. Secondly, the leaking site might have healed and no active leakage was seen.

In this particular study, RPE thinning, Granular IS/OS junctional layer, RPE detachments, the dipping sign and disrupted RPE could be appreciated on OCT. The dipping sign was caused by sub-retinal fibrin on the posterior surface of neuro-sensory retina, which was seen to dip on to the RPE. In concordance to this study, the dipping sign was described by other investigators as well^{11,12}. Some researchers believe that this sign corresponds to the site of leakage when seen on FFA¹³. However, further elaborative studies are required to prove it.

Some studies have described sub-retinal and intra-retinal bright dots on OCT, which might indicate Intra-retinal precipitates in eyes with CSCR resulting from the accumulation of proteins or macrophages photoreceptor phagocytized the segments¹⁴. Although one research has associated dipping sign with acute disease and hyper-reflective dots with chronic CSCR¹⁵ but we could not find any relation of OCT and FFA signs with the chronicity of the disease. Another important finding was reported by Saito M¹⁶. He described a ring-shaped, grayish white exudate in CSCR on OCT and FA findings, which he marked as a route of exudative fluid from the choroid into the subretinal space. We could not see this phenomenon in our study.

We found one case of CSCR with choroidal folds. This is in concordance to a report by G. M. Haffner¹⁷. He reported three patients with the diagnosis of CSCR with concurrent chorioretinal folds. He claimed those cases to be the first three cases in literature. According to him, Choroidal folds in chronic CSCR cases may represent more diffuse choroidal vasculature pathology. However, further research is needed in this respect as well.

Recently much work is being done on Autofluorescence. In case of CSCR a gravitation sign is also described which corresponds to the track of sub retinal fluid on autofluorescence ^{18,19}.

There are some short comings in this study. Fundus Autofluorescence and ICGA could not be performed. Our OCT equipment lacked choroidal details which would have helped finding out choroidal changes in CSCR.

CONCLUSION

Many hypothesis are found in literature relating different FFA and OCT patterns with disease chronicity. We found no reliable relationship between angiographic patterns, structural abnormalities on OCT and duration of CSCR. Some patients have non-leaking CSCR in which diagnosis is confirmed on OCT.

REFERENCES

- Bowing B. Acquired macular disorders In: Kanski's Clinical Ophthalmology: a systematic approach. 8 Edi. Elsevier Butterworth Heinemann: 2015: P 624
- Okushiba U, Takeda M. Study of choroidal vascular lesions in central serous chorioretinopathy using indocyanine green angiography. Nihon Ganka Gakkai Zasshi. 1997; 101(1):74.
- lijima H, lida T, Murayama K, Imai M, Gohdo T. Plasminogen activator inhibitor 1 in central serous chorioretinopathy. Am J Ophthalmol. 1999: 127(4):477-8.
- Chen GF, Ma JX, Zhang TD, Wang CL, Li SF, Yang AQ. The analysis of fundus fluorescein angiography, indocyanine green angiography and hemodynamics of retrobulbar blood vessels in central serous chorioretinopathy. Zhonghua Yan Ke Za Zhi. 2009; 45(3):243-7
- Oh JH, Oh J, Togloom A, Kim SW, Huh K Biometric Characteristics of Eyes With Central Serous Chorioretinopathy Investigative Ophthalmology & Visual Science. 2014;55: 1502-1508.
- Siddiqui SJ, Ali SI, Pechuho MA, Abbasi SA. Pattern of Central Serous Chorioretinopathy (CSCR) on Fundus Fluorescein Angiography. Pak J Ophthalmol 2008; (24) 4
- Jamil AZ, Mirza KA, Qazi ZA, Iqbal W, Khaliq J, Rahman F, Features of central serous chorioretinopathy presenting at a tertiary care hospital in Lahore. JPMA 2013; 63: 478
- Shahin MM. Angiographic characteristics of central serous chorioretinopathy in an Egyptian population. Int J Ophthalmol. 2013; 6(3): 342–345.
- Eom Y, Oh J, Kim SW, Huh K. Systemic Factors Associated with Central Serous Chorioretinopathy in Koreans. Korean J Ophthalmol. 2012 Aug; 26(4): 260–264
- Spaide RF, Campeas L, Haas A, et al. Central serous chorioretinopathy in younger and older adults. Ophthalmology. 1996; 103(12):2070-9
- Kim HC, Cho WB. Morphologic changes in acute central serous chorioretinopathy using spectral domain optical coherence tomography. Korean J Ophthalmol. 2012t; 26(5): 347 -54.
- Hussain N, Baskar A, Ram LM, Das T. Optical coherence tomographic pattern of fluorescein angiographic leakage site in acute central serous chorioretinopathy. Clin Experiment Ophthalmol. 2006; 34(2):137-40.
- Hussain N, Baskar A, Ram LM, Das T. Optical coherence tomographic pattern of fluorescein angiographic leakage site in acute central serous chorioretinopathy. Clin Experimental Ophthalmol 2006; 34: 137
- Kon Y, Iida T, Maruko I, Saito M. The optical coherence tomography-ophthalmoscope for examination of central serous chorioretinopathy with precipitates. Retina. 2008; 28(6):864-9.
- Song S, Shin YU, Lee BR, et al. Time Periodic Characteristics in the Morphology of Idiopathic Central Serous Chorioretinopathy Evaluated by Volume Scan Using Spectral-Domain Optical Coherence Tomography. Am J Ophthalmol.2012;154:366-375
- Saito M, Iida T, Kishi S. Ring-shaped subretinal fibrinous exudate in central serous chorioretinopathy. Jpn J Ophthalmol. 2005;49(6):516-9.
- Haffner GM, Chaudhry NA, Duker JS, Liggett PE, Tom D, Luquez JM. Central Serous Chorioretinopathy Associated with Chorioretinal Folds. Investigative Ophthalmology & Visual Science, 2006: 47: 5182.
- Framme C, Walter A, Gabler B, Roider J, Sachs HG, Gabel VP. Fundus autofluorescence in acute and chronic-recurrent central serous chorioretinopathy. Acta Ophthalmol Scand. 2005; 83(2):161-7.
- Bowing B. Acquired macular disorders In: Kanski's Clinical Ophthalmology: a systematic approach. 8th Edi. Elsevier Butterworth Heinemann; 2015. P 62

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