OPTICAL COHERENCE TOMOGRAPHY BIOMARKERS IN ACUTE CENTRAL SEROUS CHORIORETINOPATHY: RELATIONSHIP BET WEEN SUBRETINAL FLUID HEIGHT AND LOGMAR VISUAL ACUITY

Sadiqullah Khan[1], Sumayya Samad[2], Samiuddin[3], Siraj Khan[3], Zia Ullah[4]

How to cite this article

Khan S, Samad S, Samiuddin, Khan S, Ullah Z. Optical Coherence Tomography Biomarkers in Acute Central Serous Chorioretinopathy: Relationship Between Subretinal Fluid Height and Logmar Visual Acuity. J Gandhara Med Dent Sci. 2026;13(3):115-119. https://doi.org/10.37762/jgmds.13-3.824

Date of Submission: 26-11-2025
Date Revised: 01-06-2026
Date Acceptance: 29-06-2026

[2]PostGradute Resident, Pakistan Institute of Community Ophthalmology, Hayatabad Medical Complex, Peshawar
[3]Assistant Professor, Pakistan Institute of Community Ophthalmology, Hayatabad Medical Complex, Peshawar
[4]Statistical Analyst, Pakistan Institute of Community Ophthalmology, Hayatabad Medical Complex, Peshawar

Correspondence

[1]Sadiqullah Khan, Associate Professor, Pakistan Institute of Community Ophthalmology, Haytatabad Medical Complex, Peshawar
: +92-347-9000777
: drsadiqullahkhan@yahoo.com

ABSTRACT

OBJECTIVES

To evaluate the clinical significance of specific spectral-domain optical coherence tomography (SD-OCT) features, specifically subretinal fluid (SRF) height and retinal pigment epithelial (RPE) alterations, in predicting visual acuity (VA) outcomes in patients presenting with acute central serous chorioretinopathy (CSCR).

METHODOLOGY

This cross-sectional study, conducted at the Department of Ophthalmology, Hayatabad Medical Complex, Peshawar, evaluated 50 consecutive patients with acute CSCR. The sample size was statistically powered to detect a moderate-to-strong correlation ($r \geq 0.39$). Maximum subretinal fluid (SRF) height was measured quantitatively. Qualitative parameters included the frequency and location of pigment epithelial detachments (PEDs) and retinal pigment epithelium (RPE) bumps. Snellen VA was converted to continuous LogMAR units for parametric analysis. Relationships were evaluated using Pearson correlation coefficients (r).

RESULTS

The study population comprised 50 male patients with a mean age of 36.50 ± 7.42 years (SD) (range: 25-50 years). A strong, statistically significant positive correlation was observed between maximum SRF height and degraded visual acuity ($r = 0.907$, $P < 0.001$). Patients with minimal SRF height (0–200 μ, n=29, 58%) maintained baseline LogMAR vision between 0.00 and 0.30, whereas the maximum recorded SRF height (933 μ) corresponded to a severe visual reduction (LogMAR > 1.30). Concurrently, PEDs were identified in 40% (n=20) of the cohort (28% single, 12% multiple; 14% subfoveal, 26% extrafoveal), and RPE bumps were noted in 64% (n=32). No statistically significant correlation was identified between visual impairment and the presence ($r = 0.063$, $P = 0.666$), frequency ($r = 0.074$, $P = 0.608$), anatomical site ($r = 0.074$, $P = 0.607$) of PEDs, or the presence of RPE bumps ($r = 0.024$, $P = 0.807$).

CONCLUSION

In acute CSCR, visual acuity degradation is primarily driven by the physical height of subretinal fluid accumulation beneath the fovea. Secondary microstructural modifications of the RPE such as isolated pigment epithelial detachments or fine RPE contour irregularities show no immediate statistical correlation with baseline visual acuity within this cohort size.

KEYWORDS: *Central Serous Chorioretinopathy, Subretinal Fluid Height, Pigment Epithelial Detachment, LogMAR Visual Acuity, Optical Coherence Tomography*

INTRODUCTION

Central serous chorioretinopathy (CSCR) ranks as the fourth most prevalent non-traumatic medical retinal disorder following age-related macular degeneration, diabetic retinopathy, and retinal vein occlusions.[1] The condition is characterised by a localised, circumscribed serous detachment of the neurosensory retina over the posterior pole. This phenomenon is precipitated by choroidal hyperpermeability and compromised outer blood-retinal barrier function at the level of the retinal pigment epithelium (RPE), leading to the accumulation of subretinal fluid.[2] During acute episodes of CSCR, patients typically describe symptoms including a dense central or paracentral scotoma, micropsia, metamorphopsia, localised dyschromatopsia, and a variable reduction in central distance visual acuity.[3,5] While a significant portion of acute cases undergo spontaneous resolution within three to six months, chronic or recurrent subretinal fluid retention can

trigger permanent photoreceptor degeneration, thinning of the outer nuclear layer (ONL), and structural geographic atrophy of the RPE.[4,6] The integration of spectral-domain optical coherence tomography (SD-OCT) has fundamentally shifted the management paradigm for CSCR from subjective angiographic interpretations to non-invasive, objective microstructural mapping.[7] High-resolution cross-sectional profiles allow clinicians to quantify structural biomarkers, including fluid pocket metrics and precise variations in the RPE layer, such as pigment epithelial detachments (PEDs) and focal elevations designated as RPE bumps.[8] While historical literature consistently details these qualitative morphological findings, clinical observations show substantial variation in how individual structural features impact absolute visual metrics.[6,9] Some contemporary investigations suggest that early vision loss is primarily dictated by the physical volume or vertical linear height of the subretinal fluid pocket at the foveal avascular zone. Conversely, alternative research indicates that secondary RPE changes or subfoveal PED patterns may independently compromise outer retinal alignment and worsen visual degradation. Epidemiological estimates indicate a higher prevalence of CSCR among young to middle-aged adult males globally.[7] However, regional, demographic, and cohort-specific baseline data regarding morphometric OCT variations within the Pakistani population remain limited. This cross-sectional study evaluates the quantitative relationship between SD-OCT structural features and standardised LogMAR visual acuity in a localised clinical cohort presenting with acute CSCR.

METHODOLOGY

It was a hospital-based cross-sectional study carried out at the Department of Ophthalmology, Hayatabad Medical Complex, Peshawar, following formal institutional ethical approval. The study duration lasted from November 28, 2019, to March 24, 2022. A total of 50 consecutive patients presenting an initial episode of acute clinical CSCR were enrolled. All included participants had acute CSCR and had unremarkable slit lamp examination results of anterior segments and posterior segments except for CSCR. The sample size was determined based on the statistical power required to detect a significant correlation between macular microstructural features and LogMAR visual acuity. Assuming a two-tailed α of 0.05, a statistical power 1-β of 80%, and an anticipated moderate-to-strong correlation coefficient ($r = 0.39$), based on prior architectural SD-OCT literature, a minimum sample size of 50 patients was calculated. Diagnosis was established through comprehensive clinical ophthalmoscopy combined with confirmatory cross-sectional SD-OCT mapping showing classic subsensory retinal detachment at the macula. Subjects were excluded from participation for the presence of any significant co-existing ocular or systemic disease known possibly to affect visual acuity (e.g. diabetic retinopathy, hypertensive neuroretinopathy, choroidal neovascularisation, age-related macular degeneration, high refractive errors (3.00 DS or 1.50 DC), dense media opacities, amblyopia, or significant nystagmus) preventing stable structural fixation. All imaging sequences were captured utilising a standardised SD-OCT platform (Heidelberg Engineering) following uniform pharmacological pupil dilation with tropicamide (1%). The scanning matrix comprised a series of high-definition horizontal, vertical, and radial line protocols centered directly over the fovea, capturing 10 co-averaged high-definition frames per scan line to optimise the signal-to-noise ratio. The structural parameters evaluated were defined as follows: Subretinal Fluid (SRF) Height: Defined as the maximum vertical distance between the posterior margin of the neurosensory retina and the anterior reflection of the RPE layer at the center of the fovea, measured in micrometers.[10] Retinal Pigment Epithelial Detachment (PED): Defined as a well-demarcated, dome-shaped elevation separating the RPE monolayer from the underlying Bruch‘s membrane. PEDs localised within 200µ of the foveal avascular zone centre were classified as *subfoveal*, while those between 200µ and 2500µ were classified as *extrafoveal.*[10] Retinal Pigment Epithelium (RPE) Bumps: Defined as localised, small micro-protuberances or irregular architectural contours along the surface of the RPE layer without clear detachment.[10] To address methodological limitations regarding the mathematical evaluation of Snellen fractions, all baseline visual acuities were converted to the Logarithm of the Minimum Angle of Resolution (LogMAR) scale. The cohort data were grouped based on clinical vision impairment categories modified for LogMAR equivalence : Normal / Minimal Impairment: LogMAR 0.00 to 0.30 (Snellen 6/6–6/12), Mild Loss: LogMAR > 0.30 to 0.48 (Snellen < 6/12–6/18), Moderate Loss: LogMAR > 0.48 to 1.00 (Snellen < 6/18–6/60), Severe Loss: LogMAR > 1.00 to 1.30 (Snellen < 6/60–3/60) and Profound Loss (Blindness Category): LogMAR > 1.30 (Snellen < 3/60). Statistical processing was executed via SPSS version 20.[11] Quantitative parameters are stated as means accompanied by standard deviations (SD). Because LogMAR conversions provide a continuous linear scale, parametric assumptions were utilised to assess linear relationships via Pearson correlation coefficients (r). Two-tailed testing was conducted throughout, with the alpha threshold for statistical significance defined at

$P < 0.05$, and highly significant associations denoted at $P < 0.01$.

RESULTS

The study evaluated 50 eyes of 50 consecutive patients presenting with acute unilateral CSCR. The cohort was entirely male, exhibiting a mean age of 36.50 ± 7.42 years. The youngest participant was 25 years old, and the oldest was 50 years old. Age distribution frequencies are compiled in Table 1.

Table 1: Age Distribution of the Study Cohort (N = 50)

Age Brackets (Years)	Frequency (n)	Percentage (%)
25 – 30	11	22.0%
31 – 35	16	32.0%
36 – 40	12	24.0%
41 – 45	2	4.0%
46 – 50	9	18.0%

The mean baseline SRF height across the entire sample was 297.92 ± 175.22 µ (Median: 244.50 µ; Range: 76–933 µ). Stratifying visual function against fluid volume showed that 58% (n=29) of patients presented with minor fluid elevations (200 µ) and preserved functional vision (LogMAR 0.00–0.30). In contrast, advanced accumulation (500–600 µ) was associated with severe vision drop, and the single patient tracking above 900 µ exhibited profound visual loss (LogMAR 1.30). The complete distribution is detailed in Table 2.

Table 2: Functional LogMAR Stratification by Subretinal Fluid Height

Clinical Impairment Category	LogMAR Range Equivalent	Associated SRF Height Range (µm)	Patient Count (n)	Relative Percentage (%)
Normal / Minimal Impairment	0.00 to 0.30	0 – 200	29	58.0%
Mild Loss	> 0.30 to0.48	> 200 – 300	7	14.0%
Moderate Loss	> 0.48 to1.00	> 300 – 500	11	22.0%
Severe Loss	> 1.00 to1.30	> 500 – 600	2	4.0%
Profound Loss / Blindness	> 1.30	> 600 –1000	1	2.0%

Parametric analysis demonstrated a strong and statistically significant positive linear relationship between increasing maximum SRF height and higher LogMAR values (indicating poorer visual acuity), with $r = 0.907$ and $P < 0.001$ (Table 3).

Table 3: Parametric Correlation of Structural SD-OCT Variables with LogMAR Visual Acuity

Structural SD-OCT Parameter Under Evaluation	Pearson Correlation Coefficient (r)	Statistical Significance Level (P)
Subretinal Fluid (SRF) Height (µ)	0.907	< 0.001*
Presence of RPE Detachment (PED)	0.063	0.666
Total Quantity of Detachments (PED Number)	0.074	0.608
Anatomical Sub-site (Subfoveal vs. Extrafoveal)	0.074	0.607
Presence of Distinct RPE Bumps	0.024	0.807

Qualitative RPE analysis revealed that 40% (n=20) of the study participants had distinct PEDs. Within this subset, isolated singular detachments were noted in 14 patients (28%), while multiple tracking zones occurred in 6 patients (12%). Anatomically, 7 patients (14%) had subfoveal PED placement, whereas 13 patients (26%) demonstrated extrafoveal localisations. Statistical analyses showed no significant correlation between LogMAR visual acuity and the presence of PEDs ($P = 0.666$), the number of lesions ($P = 0.608$), or their location relative to the fovea ($P = 0.607$). Furthermore, distinct microstructural RPE bumps were tracked in 32 patients (64%) but demonstrated no significant statistical relationship with baseline visual presentation ($r = 0.024$, $P = 0.807$).

DISCUSSION

This investigation evaluated the relationships between central microstructural changes captured on high-resolution SD-OCT and baseline distance visual acuity, standardised using continuous LogMAR transformations. Our clinical data show a strong, highly significant linear correlation between increased maximum subretinal fluid height at the fovea and poorer LogMAR visual scores ($r = 0.907$, $P < 0.001$). This indicates that fluid accumulation mechanically disrupts outer retinal positioning, which serves as a primary driver of functional vision loss during the acute phase of central serous chorioretinopathy. The mean age of 36.50 years observed within this cohort aligns with established regional trends showing a higher frequency of acute presentations among young to middle-aged adult males.[12] The lack of female participants in this sample reflects consecutive operational presentation patterns within this localised facility during the study window. It should be interpreted as a cohort characteristic rather than a broader demographic indicator, as acute chorioretinal fluid shifts occur across both sexes globally.[13,14] The

absence of female participants may be attributed to the lower incidence of CSCR in females than males; additionally, the COVID-19 lockdown in Pakistan and worldwide limited hospital visits, as people were encouraged to stay indoors and seek only essential medical services. Our quantitative documentation of a strong correlation between fluid pocket height and acute visual degradation aligns with several previous imaging studies.[15,17,20] Manna and Kuli reported corresponding trends where elevated fluid pockets (261.12 to 67.81µ) were associated with notable drops in early functional vision.[16] Similarly, foundational research by Nair et al. demonstrated that during acute presentations, absolute fluid volume shifts correlate with immediate visual performance, whereas localised cellular changes may remain clinically silent.[17] In contrast, secondary structural developments along the RPE layer, including the presence, frequency, and anatomical location of subfoveal or extrafoveal PEDs, did not show a statistically significant relationship with baseline visual acuity in this study. These results align with observations by Daruich et al., who noted that structural alterations like isolated PEDs often indicate underlying choroidal congestion rather than serving as direct causes of immediate, independent vision loss.[18,21] While some researchers, including Mudvari et al. and Nguyen et al., have reported that specific tracking configurations or persistent subfoveal PED components can affect visual acuity, their cohorts often include mixed, chronic, or recurrent cases. In those advanced stages, chronic mechanical stretching may induce secondary changes in the overlying outer nuclear layer or disrupt the ellipsoid zone.[19] The lack of statistical significance regarding PED variations and structural RPE bumps within our data suggests these secondary parameters do not directly impact visual acuity during early acute presentations. However, because our sample size was limited to 50 patients, these specific evaluations may be underpowered to detect subtle relationships. Therefore, the absence of statistical significance should not be definitively interpreted as an absolute absence of biological effect.

LIMITATIONS

Converting visual data to LogMAR allowed for parametric analysis; our model did not incorporate other critical SD-OCT structural biomarkers. Parameters such as ellipsoid zone (EZ) line disruption, external limiting membrane (ELM) integrity, outer nuclear layer (ONL) thickness, and subfoveal choroidal thickness are established predictors of visual outcomes that were not quantified here. Additionally, the sample size was limited to 50 male patients recruited via consecutive sampling over an extended period, which may introduce selection bias and limit the generalizability of these findings to female populations or chronic CSCR phenotypes. Confounding clinical risk factors including endogenous cortisol levels, systemic corticosteroid exposure, systemic hypertension, and psychological stress profiles were not tracked or controlled within this analysis.

CONCLUSIONS

This study shows a significant statistical correlation between increased subretinal fluid heights on SD-OCT and poorer LogMAR visual acuity in patients with acute central serous chorioretinopathy. Concurrently, secondary microstructural variations of the RPE layer such as the presence, number, and subfoveal or extrafoveal location of pigment epithelial detachments and RPE bumps showed no independent statistical correlation with baseline visual acuity within this cohort. These structural changes likely reflect systemic choroidal congestion rather than acting as primary drivers of acute visual loss.

CONFLICT OF INTEREST: None

FUNDING SOURCES: None

REFERENCES

1. Nkrumah G, Paez-Escamilla M, Singh SR, Rasheed MA, Maltsev D, Guduru A, et al. Biomarkers for central serous chorioretinopathy. Ther Adv Ophthalmol. 2020;12:2515841420950846. doi:10.1177/2515841420950846. PMID: 32952883.
2. Mohabati D, van Dijk EHC, van Rijssen TJ, de Jong EK, Breukink MB, Martinez-Ciriano JP, et al. Clinical spectrum of severe chronic central serous chorioretinopathy and outcome of photodynamic therapy. Clin Ophthalmol. 2018;12:2167-2176. doi:10.2147/OPTH.S176885. PMID: 30410394.
3. Wang M, Munch IC, Hasler PW, Prünte C, Larsen M. Central serous chorioretinopathy. Acta Ophthalmol. 2008;86(2):126-145. doi:10.1111/j.1600-0420.2007.00889.x. PMID: 17854419.
4. Rim TH, Kim HS, Kwak J, Lee JS, Kim DW, Kim SS. Association of corticosteroid use with incidence of central serous chorioretinopathy in South Korea. JAMA Ophthalmol. 2018;136(10):1164-1169. doi:10.1001/jamaophthalmol.2018.3612. PMID: 30193203.
5. Nair U, Ganekal S, Soman M, Nair KGR. Correlation of spectral-domain optical coherence tomography findings in acute central serous chorioretinopathy with visual acuity. Clin Ophthalmol. 2012;6:1949-1954. doi:10.2147/OPTH.S36435. PMID: 23271964.
6. Mudvari SS, Goff MJ, Fu AD, McDonald HR, Johnson RN, Ai E, et al. The natural history of pigment epithelial detachment associated with central serous chorioretinopathy. Retina. 2007;27(9):1168-1173. doi:10.1097/IAE.0b013e3180dbb64a. PMID: 18046232.
7. Song IS, Shin YU, Lee BR. Time-periodic characteristics in the morphology of idiopathic central serous chorioretinopathy evaluated by spectral-domain optical coherence tomography. Am J Ophthalmol. 2012;154(2):366-375.e1. doi:10.1016/j.ajo.2012.02.019. PMID: 22652038.

8. Liu B, Deng T, Zhang J. Risk factors for central serous chorioretinopathy: a systematic review and meta-analysis. Retina. 2016;36(1):9-19. doi:10.1097/IAE.0000000000000737. PMID: 26352556.
9. Daruich A, Matet A, Dirani A, Bousquet E, Zhao M, Farman N, et al. Central serous chorioretinopathy: recent findings and new physiopathology hypothesis. Prog Retin Eye Res. 2015;48:82-118. doi:10.1016/j.preteyeres.2015.05.003. PMID: 26028504.
10. van Rijssen TJ, van Dijk EHC, Yzer S, Ohno-Matsui K, Keunen JEE, Schlingemann RO, et al. Central serous chorioretinopathy: towards an evidence-based treatment guideline. Prog Retin Eye Res. 2019;73:100770. doi:10.1016/j.preteyeres.2019.07.003. PMID: 31325502.
11. van Rijssen TJ, van Dijk EHC, Scholz P, Breukink MB, Blanco-Garavito R, Souied EH, et al. Chronic central serous chorioretinopathy: long-term follow-up and vision-related quality of life. Retina. 2019;39(10):1851-1860. doi:10.1097/IAE.0000000000002254.
12. Sly J, Madhavi C, Kumar MA. Visual outcome of central serous retinopathy. Int J Res Med Sci. 2015;3(8):1885-1888. doi:10.18203/2320-6012.ijrms20150306.
13. Jamil AZ, Rahman FU, Iqbal K, Ansari HM, Iqbal W, Mirza KA. Intravitreal bevacizumab in central serous chorioretinopathy. J Coll Physicians Surg Pak. 2012;22(6):363-366. PMID: 22647691.
14. Khalil M, Malik TG, Chaudhary QL, Shafiq MM. Angiographic patterns and tomographic findings in central serous chorioretinopathy revisited. Pak J Med Health Sci. 2015;9(4):1409-1413.
15. Manna A, Kuli JJ. Correlation of spectral-domain optical coherence tomography findings with visual acuity in central serous chorioretinopathy. J Evid Based Med Healthc. 2016;3(84):4560-4564. doi:10.18410/jebmh/2016/963.
16. Malik A, Gupta A, Mithal C, et al. Central serous chorioretinopathy: correlation of structural changes on optical coherence tomography with visual outcomes. Delta J Ophthalmol. 2017;18(1):37-43. doi:10.4103/1110-9137.201619.
17. Pan CK, Vail D, Bhattacharya J, Cao M, Mruthyunjaya P. The effect of obstructive sleep apnea on absolute risk of central serous chorioretinopathy. Am J Ophthalmol. 2020;218:148-155. doi:10.1016/j.ajo.2020.05.034. PMID: 32437786.
18. Kitzmann AS, Pulido JS, Diehl NN, Hodge DO, Burke JP. The incidence of central serous chorioretinopathy in Olmsted County, Minnesota, 1980-2002. Ophthalmology. 2008;115(1):169-173. doi:10.1016/j.ophtha.2007.02.037. PMID: 17445801.
19. Nicholson B, Noble J, Forooghian F, Meyerle C. Central serous chorioretinopathy: update on pathophysiology and treatment. Surv Ophthalmol. 2013;58(2):103-126. doi:10.1016/j.survophthal.2012.07.004. PMID: 23410815.
20. Sartini F, Menchini M, Posarelli C, Casini G, Figus M. Optical coherence tomography biomarkers in central serous chorioretinopathy: a review. Diagnostics (Basel). 2023;13(4):720. doi:10.3390/diagnostics13040720. PMID: 36832234.
21. Liew G, Quin G, Gillies M, Fraser-Bell S. Central serous chorioretinopathy: a review of epidemiology and pathophysiology. Clin Exp Ophthalmol. 2013;41(2):201-214. doi:10.1111/j.1442-9071.2012.02848.x. PMID: 22882553.

AUTHORS CONTRIBUTION

Sadiqullah Khan *- Concept & Design; Data Acquisition; Drafting Manuscript; Final Approval*

Sumayya Samad *- Concept & Design; Data Acquisition; Drafting Manuscript; Final Approval*

Samiuddin *- Concept & Design; Data Acquisition; Drafting Manuscript; Critical Revision; Final Approval*

Siraj Khan *- Concept & Design; Data Acquisition; Drafting Manuscript; Supervision; Final Approval*

The authors accept responsibility for all aspects of the work and will ensure that any concerns regarding the accuracy or integrity of any part are properly investigated and resolved.