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ORIGINAL RESEARCH

Natural Course of Central Serous Chorioretinopathy: A 12-Month Observational Study

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ABSTRACT

Aim: To evaluate the natural course of acute Central Serous Chorioretinopathy (CSCR) over a 12-month period in treatment-naïve patients, using serial clinical and imaging assessments, and to identify predictors of visual outcome.**Material and Methods:**This prospective observational study included 80 patients with a first episode of acute CSCR conducted at Nalanda Medical College and Hospital, Patna, from June 2022 to May 2024. All patients underwent baseline and monthly follow-up evaluations, including Best Corrected Visual Acuity (BCVA), Spectral-Domain Optical Coherence Tomography (SD-OCT), and selective Fundus Fluorescein Angiography (FFA). No active treatment was initiated unless predefined worsening criteria were met. Data were analyzed using SPSS version 25.0, and predictors of final BCVA were assessed via multiple linear regression analysis.**Results:**The mean age of participants was 37.6 ± 6.4 years, with a male predominance (80%). Mean baseline BCVA was 0.38 ± 0.11 LogMAR, improving significantly to 0.10 ± 0.05 at 12 months ($p < 0.001$). Mean Central Macular Thickness (CMT) reduced from 412.5 ± 38.6 µm at baseline to 268.4 ± 26.7 µm at 12 months ($p < 0.001$). Inkblot was the most common FFA leakage pattern (58.5%). Complete resolution of subretinal fluid was observed in 82.5% of patients; 13.75% had persistent fluid and 3.75% experienced recurrence. Visual improvement of ≥2 Snellen lines was seen in 85% of patients. Regression analysis identified longer symptom duration ($p = 0.002$), worse baseline BCVA ($p < 0.001$), and higher baseline CMT ($p = 0.011$) as significant predictors of poorer final visual outcome.**Conclusion:**Acute CSCR shows a predominantly self-limiting course with favorable anatomical and visual outcomes. However, persistent or recurrent cases underscore the need for individualized monitoring. Baseline visual acuity, symptom duration, and macular thickness are key predictors of prognosis, supporting their role in guiding follow-up and therapeutic planning.

Keywords: Central serous chorioretinopathy, Optical coherence tomography, Visual acuity, Subretinal fluid, Natural history

INTRODUCTION

Central Serous Chorioretinopathy (CSCR) is an idiopathic retinal disorder characterized by serous detachment of the neurosensory retina, most commonly at the macula, due to leakage of fluid through a dysfunctional retinal pigment epithelium (RPE). It predominantly affects young to middle-aged adults, with a higher incidence reported in males. Patients typically present with acute onset of blurred central vision, metamorphopsia, micropsia, and relative central scotoma. These visual symptoms are often distressing and can significantly impact daily functioning, especially when the dominant eye is affected.[1]

The pathogenesis of CSCR is multifactorial and not yet fully understood. Current understanding suggests that choroidal vascular hyperpermeability, increased hydrostatic pressure in the choroid, and defects in RPE pumping function contribute to the accumulation of subretinal fluid. Autonomic dysregulation, psychological stress, systemic corticosteroid use, and altered cortisol metabolism have all been implicated as possible risk factors. The disease typically manifests unilaterally, although bilateral involvement may occur, especially in chronic or recurrent cases.[2]

Clinically, CSCR can be classified into acute, recurrent, and chronic forms. The acute form is often self-limiting, with spontaneous resolution of subretinal fluid occurring within three to four months in the majority of cases. Visual acuity frequently returns to baseline following resolution, although some patients may continue to report subjective disturbances such as reduced contrast sensitivity or persistent metamorphopsia. In contrast, chronic CSCR is characterized by persistent or recurrent subretinal fluid, widespread RPE changes, and irreversible

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damage to the photoreceptor layer, often resulting in permanent visual impairment. The recurrent form shares features with both acute and chronic presentations and poses a higher risk of progression to chronicity.[3]

The diagnosis of CSCR is largely clinical and supported by imaging modalities such as fundus fluorescein angiography (FFA), optical coherence tomography (OCT), and indocyanine green angiography (ICGA). FFA typically reveals focal areas of hyperfluorescence due to leakage from the RPE, often described as "inkblot" or "smokestack" patterns. OCT allows for precise assessment of subretinal fluid and retinal morphology and is essential in monitoring disease progression or resolution. These imaging techniques play a critical role not only in diagnosis but also in identifying atypical variants and differentiating CSCR from other causes of serous retinal detachment.[4]

Despite the relatively benign course of acute CSCR, some patients may experience poor outcomes due to factors such as delayed presentation, baseline poor visual acuity, or structural damage at the fovea. Additionally, patients with prolonged subretinal fluid are more prone to developing RPE alterations and outer retinal thinning, which can compromise visual prognosis even after fluid resolution. Therefore, while observation remains the mainstay of management in acute cases, identifying patients at risk of progression is crucial.[5]

There is ongoing debate regarding the optimal timing and indication for intervention in CSCR. Various treatment modalities, including photodynamic therapy (PDT), focal laser photocoagulation, oral mineralocorticoid antagonists, and anti-VEGF agents, have been explored, particularly for chronic or non-resolving cases. However, the natural history of acute, untreated CSCR remains an important area of clinical interest. Understanding the trajectory of visual and anatomical recovery in untreated patients can help determine the appropriate duration of observation, identify predictive markers of persistent disease, and avoid overtreatment in self-resolving cases.[6]

Given the prevalence of CSCR in the working-age population and its potential for visual morbidity, longitudinal observational studies are essential to document its clinical course, resolution rates, and long-term visual outcomes. Such data not only inform prognosis but also guide the formulation of patient-specific follow-up protocols. Moreover, in resource-limited settings where immediate access to interventional treatments may not be available, monitoring the natural history of CSCR provides valuable insight into when conservative management is sufficient and when escalation of care is warranted.[7]

This study was undertaken to evaluate the natural course of acute CSCR over a period of 12 months in patients presenting with a first episode of the disease. Through systematic follow-up using clinical examination and OCT-based imaging, this study aims to assess patterns of visual recovery, rates of subretinal fluid resolution, and incidence of recurrence or persistence. Additionally, it explores baseline predictors of poor visual outcome in an untreated cohort, thereby contributing to the evidence base for optimal management of CSCR in clinical practice.

MATERIAL AND METHODS

This prospective, observational study was conducted in the Department of Ophthalmology at Nalanda Medical College and Hospital, Patna, over a 24-month period, from June 2022 to May 2024. The aim was to evaluate the natural course of Central Serous Chorioretinopathy (CSCR) in affected patients through serial clinical and imaging assessments, without any active intervention unless clinically warranted.A total of 80 patients diagnosed with treatment-naïve, first-episode acute CSCR were enrolled after obtaining written informed consent. Ethical approval for the study was obtained from the Institutional Ethics Committee, and all procedures adhered to the tenets of the Declaration of Helsinki.

Inclusion Criteria

- Patients aged between 20 and 55 years.
- Diagnosis of acute CSCR (defined as presence of serous retinal detachment involving the macula with symptom duration of less than 6 weeks).
- Best Corrected Visual Acuity (BCVA) of 6/60 or better at presentation.
- Presence of subretinal fluid confirmed by Optical Coherence Tomography (OCT).
- Willingness to undergo regular follow-up for 12 months.

Exclusion Criteria

- Recurrent or chronic CSCR (symptom duration >6 weeks or presence of pigment epithelial detachment/fibrosis).
- Any prior ocular treatment such as laser photocoagulation or intravitreal injections.
- Presence of other retinal disorders (e.g., diabetic retinopathy, age-related macular degeneration).
- History of steroid use (oral, topical, or systemic) in the past three months.
- Media opacities precluding fundus evaluation or poor OCT image quality.
- Patients with systemic comorbidities affecting the retina such as uncontrolled hypertension or autoimmune disease.

Methodology

At baseline, all patients underwent a detailed ophthalmological evaluation. This included BCVA assessment using Snellen's chart, intraocular pressure (IOP) measurement with non-contact tonometry, slit-lamp biomicroscopy for anterior segment examination, and a detailed dilated fundus examination using indirect ophthalmoscopy and a

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+90D lens. These evaluations were complemented by imaging studies to establish baseline structural and functional retinal parameters.

Spectral-Domain Optical Coherence Tomography (SD-OCT) was performed at baseline in all cases to assess central macular thickness (CMT) and to confirm the presence of subretinal fluid. Fundus Fluorescein Angiography (FFA) was selectively performed to evaluate the pattern of leakage and to rule out differential diagnoses such as choroidal neovascularization or inflammatory conditions. Patients were subsequently followed up at monthly intervals for a period of 12 months. At each visit, BCVA and SD-OCT were repeated to document changes in visual acuity, subretinal fluid status, and macular morphology.

During the course of the study, no active intervention was undertaken unless predefined criteria were met. These included persistence of subretinal fluid beyond three months, significant decline in visual acuity, or development of recurrent or chronic CSCR. In such cases, patients were evaluated for appropriate therapeutic intervention and excluded from further observation in the natural history cohort. Patients exhibiting spontaneous resolution of fluid were continued on monthly follow-up until full resolution and visual stabilization.

For statistical analysis, all collected data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Quantitative variables such as BCVA (converted to LogMAR for analysis), CMT, and duration to fluid resolution were expressed as mean ± standard deviation (SD). Repeated measures ANOVA was used to assess changes in BCVA and CMT over the follow-up period. Categorical variables were presented as frequency and percentage. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1 presents the baseline demographic and clinical characteristics of the 80 patients included in the study. The mean age of the cohort was 37.6 ± 6.4 years, with a range from 22 to 55 years, indicating a predominance of CSCR in younger to middle-aged adults. There was a marked male preponderance, with 64 males (80%) and 16 females (20%), consistent with the known gender bias observed in CSCR. The laterality of eye involvement was nearly equal, with 52.5% of cases affecting the right eye and 47.5% affecting the left. The mean duration of symptoms at presentation was 19.3 ± 7.8 days, reflecting acute or early subacute presentations. The average baseline Best Corrected Visual Acuity (BCVA), measured in LogMAR, was 0.38 ± 0.11, suggesting mild to moderate visual impairment at the time of diagnosis. Mean Central Macular Thickness (CMT) was elevated at 412.5 ± 38.6 µm due to the presence of subretinal fluid, a hallmark of CSCR.

Table 2 outlines the angiographic leakage patterns observed on Fundus Fluorescein Angiography (FFA), which was performed in 65 of the 80 patients (81.25%) where clinically indicated. The inkblot pattern was the most commonly observed leakage type, seen in 58.5% of patients. This was followed by the smokestack pattern in 29.2%, which is considered more characteristic but less frequently encountered. A diffuse or ill-defined leakage pattern was noted in 12.3% of cases, which may represent more complex or overlapping pathology and sometimes predicts delayed resolution.

Table 3 illustrates the progressive improvement in mean BCVA (LogMAR) over the follow-up period. At baseline, the average BCVA was 0.38 ± 0.11. There was a statistically significant improvement at each follow-up point. At 1 month, the BCVA improved to 0.29 ± 0.10 ($p < 0.001$), and continued to improve to 0.18 ± 0.08 at 3 months ($p < 0.001$), 0.12 ± 0.06 at 6 months ($p < 0.001$), and finally reached 0.10 ± 0.05 at 12 months ($p < 0.001$). These findings indicate that the majority of patients experienced spontaneous and sustained visual recovery over time, consistent with the self-limiting nature of acute CSCR.

Table 4 presents the trend in Central Macular Thickness (CMT) as measured on Spectral-Domain OCT. The mean CMT at baseline was 412.5 ± 38.6 µm, reflecting subretinal fluid accumulation. At 1 month, a significant reduction was observed with mean CMT of 352.7 ± 35.4 µm ($p < 0.001$). This downward trend continued with values of 299.3 ± 31.2 µm at 3 months, 276.1 ± 28.8 µm at 6 months, and finally 268.4 ± 26.7 µm at 12 months (all p-values < 0.001 compared to baseline). This progressive reduction in macular thickness corresponds with the resolution of subretinal fluid and correlates well with visual acuity improvements.

Table 5 summarizes the clinical outcomes at the end of the 12-month follow-up. A large proportion of patients (82.5%) demonstrated complete resolution of subretinal fluid without any intervention, reinforcing the natural tendency of acute CSCR to resolve spontaneously. Persistent subretinal fluid beyond 3 months was observed in 13.75% of patients, while recurrent episodes occurred in 3.75%. Notably, 85% of patients showed a visual acuity improvement of two or more lines on the Snellen chart, whereas 12.5% had no significant change. Only 2.5% of patients experienced mild visual deterioration, likely due to persistent or recurrent fluid affecting photoreceptor integrity.

Table 6 displays the results of a multiple linear regression analysis performed to identify independent predictors of final visual outcome (LogMAR BCVA at 12 months). Among the variables studied, duration of symptoms, baseline BCVA, and baseline central macular thickness were statistically significant predictors of worse final BCVA. A longer duration of symptoms was associated with poorer visual outcomes ($\beta = 0.006$, $p = 0.002$), highlighting the importance of early presentation. Worse baseline

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BCVA was the strongest predictor of poor final outcome ($\beta = 0.517$, $p < 0.001$), indicating that initial visual impairment can reflect the severity of retinal dysfunction. Increased baseline CMT also correlated with suboptimal recovery ($\beta = 0.0013$, $p = 0.011$). Age and gender were not significantly associated with final visual acuity. The overall model showed good explanatory power with an R^2 of 0.66, suggesting that these three baseline variables collectively accounted for 66% of the variation in final visual outcomes.

Table 1: Baseline Demographic and Clinical Profile of Patients (n = 80)

Parameter	Value
Mean Age (years)	37.6 ± 6.4
Age Range (years)	22 – 55
Gender (Male / Female)	64 (80%) / 16 (20%)
Laterality (Right / Left Eye)	42 (52.5%) / 38 (47.5%)
Mean Duration of Symptoms (days)	19.3 ± 7.8
Mean Baseline BCVA (LogMAR)	0.38 ± 0.11
Mean Central Macular Thickness	412.5 ± 38.6 µm

Table 2: Pattern of Leakage on Fundus Fluorescein Angiography (FFA) (n = 65)

Leakage Pattern	Frequency (n)	Percentage (%)
Inkblot	38	58.5%
Smokestack	19	29.2%
Diffuse/Ill-defined	8	12.3%

Table 3: Mean Best Corrected Visual Acuity (BCVA, LogMAR) Over Follow-Up Period

Follow-Up Time Point	Mean BCVA (LogMAR) ± SD	p-value (vs. Baseline)
Baseline	0.38 ± 0.11	–
1 Month	0.29 ± 0.10	< 0.001
3 Months	0.18 ± 0.08	< 0.001
6 Months	0.12 ± 0.06	< 0.001
12 Months	0.10 ± 0.05	< 0.001

Table 4: Central Macular Thickness (CMT) Over Follow-Up Period

Follow-Up Time Point	Mean CMT (µm) ± SD	p-value (vs. Baseline)
Baseline	412.5 ± 38.6	–
1 Month	352.7 ± 35.4	< 0.001
3 Months	299.3 ± 31.2	< 0.001
6 Months	276.1 ± 28.8	< 0.001
12 Months	268.4 ± 26.7	< 0.001

Table 5: Clinical Outcomes at Final Follow-Up (12 Months, n = 80)

Outcome Category	Number of Patients	Percentage (%)
Complete SRF Resolution	66	82.5%
Persistent Subretinal Fluid (>3 months)	11	13.75%
Recurrent CSCR Episodes	3	3.75%
Visual Acuity Improvement (≥2 lines)	68	85%
No Significant Change in Vision	10	12.5%
Visual Deterioration (≤1 line)	2	2.5%

Table 6: Multiple Linear Regression Analysis for Predictors of Final BCVA (LogMAR) at 12 Months

Predictor Variable	Regression Coefficient (β)	Standard Error (SE)	t-value	p-value
Age (years)	0.002	0.003	0.71	0.480
Gender (Male = 1, Female = 0)	-0.014	0.018	-0.78	0.439
Duration of Symptoms (days)	0.006	0.002	3.24	0.002**
Baseline BCVA (LogMAR)	0.517	0.063	8.21	<0.001**
Baseline Central Macular Thickness	0.0013	0.0005	2.60	0.011*

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DISCUSSION

The demographic and clinical profile of the study cohort demonstrates that CSCR predominantly affects younger to middle-aged adults, with a mean age of 37.6 ± 6.4 years. A strong male predilection was noted (80% male), consistent with previous literature which has associated CSCR with systemic stress and hormonal factors more common in men. The nearly equal distribution of laterality (52.5% right eye, 47.5% left eye) is in line with the known random eye involvement pattern. The mean symptom duration at presentation was 19.3 ± 7.8 days, indicating most patients were evaluated in the acute phase. The baseline BCVA (0.38 ± 0.11 LogMAR) and mean central macular thickness (CMT) of 412.5 ± 38.6 µm reflect active subretinal fluid accumulation and moderate visual impairment. Similar clinical profiles and risk factors have been observed in earlier studies such as those by Nicholson et al. and Manayath et al.[8,9]

Among the 65 patients who underwent FFA, the inkblot pattern was most common (58.5%), followed by smokestack (29.2%), and diffuse or ill-defined patterns (12.3%). These patterns correlate with disease severity and duration. The inkblot configuration is generally associated with classic acute CSCR, often resolving spontaneously, whereas diffuse leakage can suggest RPE decompensation and predict chronic evolution. Otsuka et al. reported that diffuse or atypical leakage patterns may be seen in severe variants and are often associated with a guarded visual prognosis if not addressed early.[10]Yannuzzi also emphasized that classic smokestack presentations are less common and may rapidly evolve into inkblot patterns, reaffirming our findings in the acute phase.[11]

Visual recovery data reveal a statistically significant and continuous improvement in BCVA over 12 months. Starting from a baseline of 0.38 ± 0.11 LogMAR, mean BCVA improved to 0.29 ± 0.10 at 1 month, 0.18 ± 0.08 at 3 months, 0.12 ± 0.06 at 6 months, and 0.10 ± 0.05 at 12 months ($p < 0.001$ for all intervals vs. baseline). These results demonstrate that the majority of patients experienced spontaneous visual recovery, consistent with the natural course described in observational studies by Taban et al. and Nicholson et al.[8,12] However, persistent subretinal fluid, if left untreated beyond 3–6 months, may impact outer retinal structures. Studies like those by Loo et al. have emphasized that early visual acuity is a significant predictor of final outcomes, which our regression model also confirmed.[13]

Correspondingly, Table 4 shows a significant reduction in CMT, from 412.5 ± 38.6 µm at baseline to 352.7 ± 35.4 µm at 1 month, 299.3 ± 31.2 µm at 3 months, 276.1 ± 28.8 µm at 6 months, and 268.4 ± 26.7 µm at 12 months ($p < 0.001$ for all). This decline directly correlates with visual recovery, highlighting the anatomical restoration during the resolution of subretinal fluid. Chan et al. and Reibaldi et al. noted similar trends in both spontaneous and treated CSCR patients, supporting OCT as a reliable non-invasive tool to monitor disease activity and prognosis.[14,15]

Of the 80 patients, 66 (82.5%) showed complete resolution of subretinal fluid, 11 (13.75%) had persistent fluid beyond 3 months, and 3 (3.75%) experienced recurrence. Notably, 68 patients (85%) showed an improvement of ≥2 Snellen lines, whereas 10 (12.5%) had no significant change, and only 2 (2.5%) experienced mild visual deterioration. These findings affirm the generally favorable prognosis of acute CSCR, as previously emphasized by Yannuzzi and Taban et al.[11,12] However, the presence of persistent or recurrent fluid in nearly 18% of the cohort underlines the need for regular follow-up. Nicholson et al. emphasized that such cases may benefit from early intervention, especially when anatomical recovery stalls beyond 3 months.[8] Current therapeutic options including low-fluence PDT, eplerenone, and focal laser have shown benefit in such settings.[16]

Our multiple regression analysis found that longer duration of symptoms ($\beta = 0.006$, $p = 0.002$), worse baseline BCVA ($\beta = 0.517$, $p < 0.001$), and higher baseline CMT ($\beta = 0.0013$, $p = 0.011$) were significant independent predictors of poor final BCVA. These results align with the findings of Loo et al. and Otsuka et al., who reported that delayed presentation, greater retinal elevation, and initial functional deficit reduce the likelihood of full visual recovery.[10,13] Age and gender, however, were not significantly associated with outcomes in our cohort. The R^2 value of 0.66 from our model suggests that these three predictors explained two-thirds of the variability in final visual acuity, highlighting their clinical relevance.

CONCLUSION

This 12-month observational study highlights that acute central serous chorioretinopathy (CSCR) predominantly affects young to middle-aged males and typically follows a self-limiting course with favorable visual and anatomical outcomes in the majority of patients. Significant improvements in visual acuity and reduction in central macular thickness were observed over time without active intervention. However, a subset of patients demonstrated persistent or recurrent disease, emphasizing the need for individualized follow-up. Baseline BCVA, duration of symptoms, and central macular thickness were found to be key predictors of final visual outcome. Early identification of high-risk patients may aid in timely therapeutic decisions and improved prognosis.

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