Clinical and Experimental Optometry

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/tceo20

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To cite this article: Javier Orduña-Azcona, Elia Pérez-Fernández, Laura Modamio, Sofía De Manuel-Triantafilo, Carmen Fátima Rodríguez-Hernández & Pablo Gili (2022): One-year follow-up of choroidal and macular thickness in acute non-treated central serous chorioretinopathy, Clinical and Experimental Optometry, DOI: 10.1080/08164622.2021.2016022

To link to this article: https://doi.org/10.1080/08164622.2021.2016022

Published online: 14 Feb 2022.

CLINICAL AND EXPERIMENTAL OPTOMETRY
https://doi.org/10.1080/08164622.2021.2016022

RESEARCH

One-year follow-up of choroidal and macular thickness in acute non-treated central serous chorioretinopathy

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ABSTRACT

Clinical relevance: Choroidal and macular thickness assessments are essential to understand the evolution of central serous chorioretinopathy and could help in identifying patients more prone to recurrence.

Background: The aim of this work was to evaluate changes in the choroidal thickness and macular thickness in acute non-treated central serous chorioretinopathy during a one-year follow-up.

Methods: A prospective longitudinal study of 38 patients with central serous chorioretinopathy and 35 healthy controls at a tertiary care facility (Fundación Alcorcón University Hospital) was conducted. Choroidal and macular thicknesses were measured using enhanced-depth-imaging optical coherence tomography and subretinal fluid resolution and best-corrected visual acuity were evaluated, at baseline and 1, 3, 6 and 12 months. Prognostic factors determining the need for treatment were evaluated.

Results: Choroidal thickness decreased in eyes with central serous chorioretinopathy ($p < 0.001$) but not in fellow eyes ($p = 0.24$) during one-year follow-up since the acute episode. The estimated mean choroidal thickness in symptomatic eyes was 465 µm (SE: 17.18) at baseline and decreased 58.1 µm (CI 95%: 30.1-85.9) at 12 months ($p < 0.001$). Best-corrected visual acuity improved over time ($p = 0.037$), with a decrease of logMAR 0.086 (CI95%: 0-0.172).

The macular thickness changed over time ($p < 0.001$), with a decrease from baseline of 124.6 µm (CI95%: 61.4-187.9). Subretinal fluid resolved in 67% (CI 95%: 51-82) of patients at 6 months. There was no significant association between baseline choroidal thickness, macular thickness, best-corrected visual acuity, age or sex and the need for treatment.

Conclusions: The choroidal thickness decreased in acute central serous chorioretinopathy episodes during a one-year follow-up. Subretinal fluid persisted in less than 20% of patients at the end of the one-year follow-up. No prognostic factors determining the need for treatment were found.

ARTICLE HISTORY
Received 26 March 2021
Revised 11 November 2021
Accepted 1 December 2021

KEYWORDS
Central serous chorioretinopathy; choroidal thickness; follow-up study; optical coherence tomography; prognostic factor

Introduction

Central serous chorioretinopathy is a disorder characterised by serous macular detachment and retinal pigment epithelial changes. The pathogenesis of central serous chorioretinopathy is attributed to choroid hypermeability.[1] Recent optical coherence tomography (OCT) modalities, such as enhanced depth imaging OCT (EDI-OCT) and swept-source OCT, have allowed the choroid to be studied due to their greater tissue penetrance and higher definition. Previous studies reported a greater choroidal thickness in eyes with central serous chorioretinopathy[2–6] and fellow eyes[4,7,8] as compared to normal controls, supporting a role for choroidal pathophysiology in this disease. Other studies reported a reduction in the choroidal thickness in eyes with central serous chorioretinopathy.[9,10]

However, there have been no long-term follow-up studies of choroidal thickness in treatment-naive acute central serous chorioretinopathy and their fellow eyes. A long-term prospective study could aid understanding of the pathophysiology of central serous chorioretinopathy and select which patients could need treatment.

The purpose of this study was to evaluate changes in the choroidal thickness and macular thickness as measured by EDI-OCT in acute non-treated central serous chorioretinopathy during a one-year follow-up.

Methods

Study design and ethical considerations

A prospective study of 38 patients with central serous chorioretinopathy and 35 healthy controls was performed. Patients who attended the Retina Department of a tertiary care hospital (Fundación Alcorcón University Hospital, Madrid, Spain) between September 2014 and September 2017 were recruited consecutively.

This study was approved by the ethics committee of the hospital and was conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from all the patients.

Inclusion and exclusion criteria

The inclusion criteria for the central serous chorioretinopathy group were an acute central serous chorioretinopathy episode, with the symptom duration less than 30 days, with macular subretinal fluid on OCT and active leakage on fluorescein angiography. Healthy participants were consecutively selected for inclusion in the control group and one of their eyes was randomly selected for analysis.

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The exclusion criteria for both groups were as follows: a refractive error of more than ± 3.00D of spherical equivalent, the presence of other ocular disorders that could affect visual acuity, prior ocular surgery or intraocular treatment and inability of getting good retinal image quality due to media opacity or participants treated during the follow-up for unresolved subretinal fluid. Eighteen patients were excluded because of these criteria. Five of these patients received treatment with laser photocoagulation or photodynamic therapy (Figure 1).

Patient assessments

All the participants underwent a full ophthalmic examination, including best-corrected visual acuity, refractive correction, slit-lamp biomicroscopy, fundus photography and EDI-OCT (Zeiss Cirrus spectral-domain OCT Model 4000-1616; version 6.5.0.772, Carl Zeiss Ophthalmic Systems, Dublin, CA, USA). Central serous chorioretinopathy was diagnosed based on the findings of fluorescein angiography and indocyanine angiography.

Fundus images were obtained using a mydriatic fundus camera (FF450 IRplus; Carl Zeiss Ophthalmic Systems, Dublin, CA, USA) with a digital archiving system (Visupac 452, version 4.4.4; Carl Zeiss Ophthalmic Systems, Dublin, CA, USA). Colour photographs were taken with a 3 CCD AVT ZK-5 high-resolution colour camera (2.588 x 1.958 resolution). Red-free, fluorescein angiography and indocyanine angiography images were obtained using a Kodak Megaplus 1.6 camera (Kodak, Rochester, NY, USA). The participants were asked to score their stress level from 0 (absent) to 10 (maximum) and report any previous corticosteroid use. Best-corrected visual acuity was measured using an ETDRS chart and the logMAR scale was used for analysis.

The choroidal thickness, macular thickness, subretinal fluid resolution and best-corrected visual acuity in central serous chorioretinopathy eyes and fellow eyes at baseline and 1, 3, 6 and 12 months were evaluated. In control eyes, these variables were studied only at baseline according to the findings of another study, which showed that the choroidal thickness did not change in healthy participants during a one-year follow-up.[11] Recurrence was considered as a new episode of central serous chorioretinopathy that occurred during the follow-up after complete resolution of subretinal fluid in a previous episode or when a new leakage was observed by fluorescein angiography in non-resolved central serous chorioretinopathy after 6 months.

Persistent cases with non-resolved subretinal fluid after 6 months were offered to be treated with laser photocoagulation if the leak visualised by fluorescein angiography was located more than 375 microns from the fovea or photodynamic therapy if the leakage was located less than or equal to 375 microns from the fovea. The data of patients who accepted treatment were not analysed from this point forwards. Patients who decided not to be treated were followed up until the end of the study and their data were analysed.

All participants underwent OCT, with a horizontal 9-mm single high-definition scan crossing the fovea using an eye-tracker system, as described previously by other authors.[2,7] A 512 × 128 macular cube scan was also performed in all participants and the macular central thickness evaluated from the automated Cirrus macular thickness map. In follow-up visits, the auto-repeat and auto-alignment features were used to reduce possible inaccuracies in intra-individual comparisons. The subfoveal choroidal thickness was measured manually in all participants using a software calliper built into a custom-

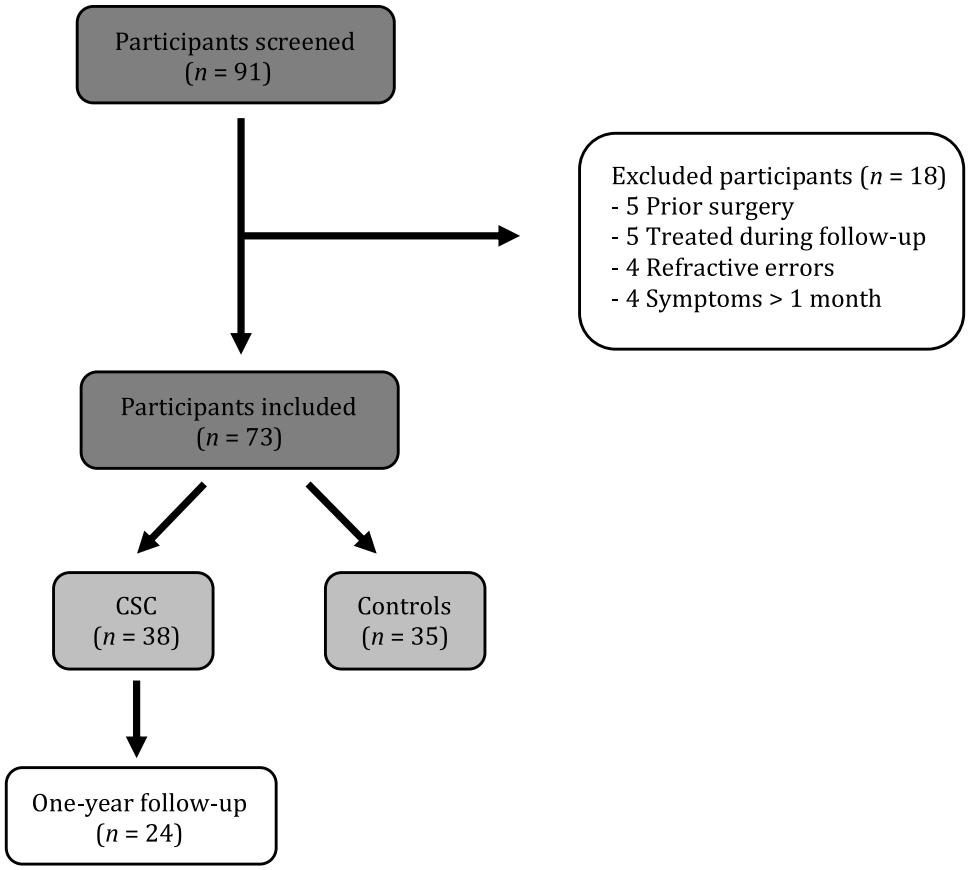

Figure 1. Flow diagram of the patient selection process. CSR, Central serous chorioretinopathy.

made OCT image viewer, from the outer part of the retinal pigment epithelium to the choroidoscleral interface, making a 90º angle with the retinal pigment epithelium (Figure 2).

For better visualisation of this interface, brightness and contrast parameters were adjusted to obtain the best possible definition. In cases where the interface was blurred, the centre of the line was always traced and measured. When a pigment epithelial detachment was present, Bruch's membrane was used as the internal choroid limit instead of the retinal pigment epithelium. Two masked observers (PG and JO) measured the baseline scans of the central serous chorioretinopathy group in order to assess the inter-rater agreement. The scans were presented to them, so they did not know any patient information and the other observer measurement. For the control group, follow-up visits and other statistical analyses, the measurements made by the most experienced observer (PG) were used. All the measurements were made between 9 am and 12 pm to reduce possible choroidal thickness circadian changes observed previously.[12,13]

Statistical analysis

Statistical analysis was performed by using SPSS 17.0 for Windows (SPSS, Inc., Chicago, IL, USA). Data are presented as the mean ± standard deviation. First, a univariate analysis was performed to evaluate differences between central serous chorioretinopathy and controls: a χ^2 or Fisher's exact test was applied for qualitative variables and a Student's t-test or non-parametric Mann-Whitney *U* test was applied for quantitative variables. A paired *t*-test was used to analyse differences between symptomatic and fellow eyes in the central serous chorioretinopathy group. Multivariate ANOVA models were adjusted to analyse gender effect in the mean choroidal thickness differences between groups.

Variance analysis with mixed models was applied to study changes over time in the choroidal thickness, macular thickness and best-corrected visual acuity. Time was studied as a repeated measures factor and group as a fixed factor. The pairwise comparison was adjusted by the Bonferroni method. A statistically significant interaction effect between the time and group factors was interpreted as the trend over time was different for each group.

The percentage of relative change at the 6-month follow-up was calculated and Pearson's R correlation coefficients and R^2 determination coefficients were estimated for the choroidal thickness, macular thickness and best-corrected visual acuity between each variable. Pearson's R coefficient between 0.4 and 0.7 was considered moderate and a coefficient higher than 0.7 was considered good.

To study subretinal fluid resolution, survival analysis techniques were used. The survival function was estimated by the Kaplan-Meier method.

The baseline choroidal thickness, macular thickness, best-corrected visual acuity, sex, age, corticosteroid use and presence of ellipsoid changes on OCT were evaluated as possible prognostic factors of the need for treatment using subretinal fluid persistence at 6 months as the cut-off point. Modified Poisson regression was used to estimate the relative risk (RR) and 95% confidence intervals (CI).

To evaluate inter-rater agreement, the intra-class correlation coefficient was calculated using a two-way mixed analysis of variance and Bland-Altman graphics were plotted. All statistical tests were two-sided and probability values of < 0.05 were considered statistically significant.

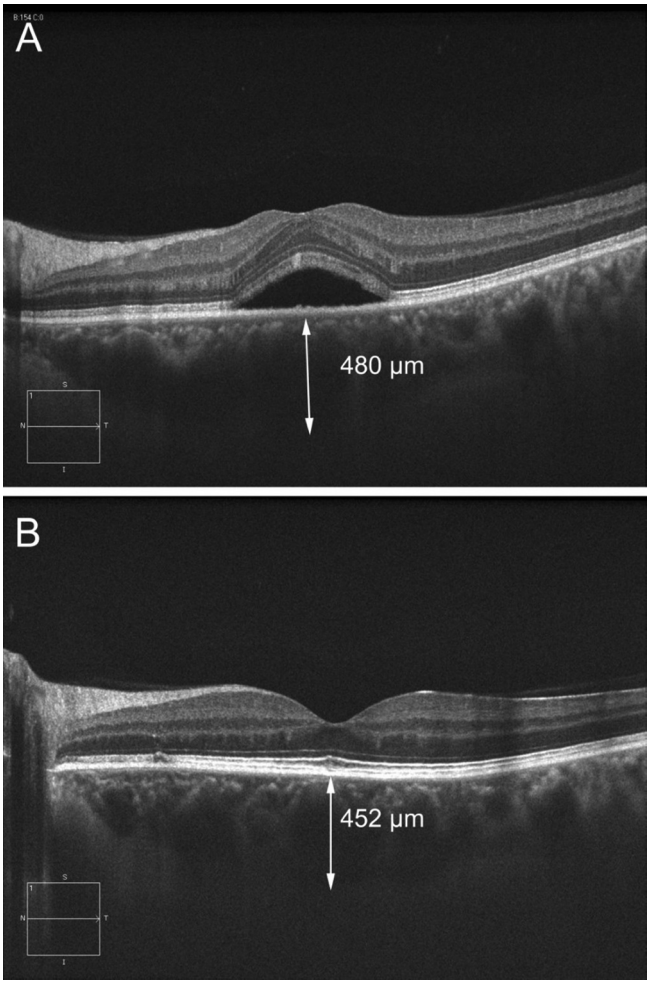

Figure 2. Enhanced depth-imaging optical coherence tomography (EDI-OCT) horizontal 9 mm scan. The double-arrow line indicates the choroidal thickness measured manually in the same patient A: at baseline and B: at 6 months

Results

Baseline data

Thirty-eight patients with central serous chorioretinopathy and 35 healthy controls were included in this study. Two patients (5.26%) presented with bilateral central serous chorioretinopathy. The demographic characteristics and mean OCT parameters of the symptomatic, fellow and control eyes at baseline are shown in Table 1.

Significant differences between groups were found in best-corrected visual acuity ($p = 0.039$), stress level ($p < 0.001$), sex ratios ($p = 0.042$) and corticosteroid use ($p = 0.012$). There were no significant differences between groups in terms of age ($p = 0.623$) or refractive errors ($p = 0.71$). No statistically significant interaction effect of sex on the mean choroidal thickness was found ($F = 0.350$, $p = 0.557$).

The mean choroidal thickness was greater in the central serous chorioretinopathy group than in the control group both in symptomatic ($p < 0.001$) and fellow eyes ($p = 0.039$). The choroidal thickness in central serous chorioretinopathy eyes was greater than that in fellow eyes ($p < 0.001$). Fundus colour photography revealed that 15% of eyes presented

Table 1. Central serous chorioretinopathy (CSC) and control group result comparison.

	CSC	Controls	*p*-value
No. of eyes	40	35	
No. of patients	38	35	
Age (years)			
Mean ± SD	45.32 ± 12	43.17 ± 12.49	.623[a]
Median (p25–p75)	42 (37–54.25)	43 (34–49)	
RE (D)			
Mean ± SD	−0.11 ± 1.37	−0.11 ± 1.33	.71[a]
Median (p25–p75)	0.13 (−0.94–0.84)	0 (−0.75–0.33)	
BCVA (logMAR)			
Mean ± SD	0.180 ± 0.602	0.032 ± 0.886	**.039**[a]
Median (p25–p75)	0.201 (0.102–0.42)	0 (0–0.102)	
Sex (males:females)	31:7	21:14	**.042**[b]
Stress level			
Mean ± SD	6.01 ± 2.34	3.51 ± 1.65	**< .001**[a]
Median (p25–75)	7 (4.25–8)	3 (2–5)	
Corticosteroids use	31 (81.6%)	0	**< .012**[b]
CT symptomatic eye (µm)			
Mean ± SD	465.45 ± 115.42	317.54 ± 72.04	**< .001**[a]
Median (p25–p75)	481 (392.25–557.5)	326 (260–373)	
CT fellow eye (µm)			
Mean ± SD	363.44 ± 113.5	317.54 ± 72.04	**.039**[a]
Median (p25–p75)	376.5 (282.25–454.25)	326 (260–373)	
Macular thickness (µm)			
Mean ± SD	402.13 ± 108.07		
Median (p25–p75)	382.5 (330–485.5)		

RE: refractive error; BCVA: best-corrected visual acuity; CT: choroidal thickness.
[a]Mann-Whitney U test.
[b]X^2 test.

with retinal pigment epithelium atrophy and 42.5% eyes presented with granular deposits. In the OCT images, ellipsoid changes were observed in 77.5% of the eyes.

Measures evolution during the one-year follow-up

During the one-year follow-up, the mixed models showed a time effect in choroidal thickness in symptomatic eyes ($p < 0.001$), but not in fellow eyes ($p = 0.24$) (Figure 3A). At baseline, the estimated mean choroidal thickness in symptomatic eyes was 465 µm (SE: 17.18) and it decreased 58.1 µm (CI 95%: 30.1-85.9) at 12 months. This decrease was reached at 3 months and remained stable thereafter.

Best-corrected visual acuity improved ($p = 0.037$) from a baseline estimated mean of logMAR 0.216 (SE: 0.027) to an estimated mean of logMAR 0.130 (SE: 0.029) at 3 months (Figure 3B).

The baseline estimated mean of macular thickness was 402.13 (SE: 14.62) and decreased at 12 months124.5 µm (CI 95%: 61.4-187.9) ($p < 0.001$) (Figure 3C). This decrease was reached at 3 months and remained stable thereafter.

At the 6-month follow-up, the choroidal thickness in eyes with central serous chorioretinopathy was greater than that in controls ($p = 0.001$). The choroidal thickness in fellow eyes was also greater than that in controls, but these differences were not statistically significant ($p = 0.343$).

The percentage of recurrence was 17.5%, with 57% of recurrences occurring at 6 months and the remainder at 5, 10 and 11 months. The choroidal thickness in recurrent cases at baseline was greater (511.83 ± 104.75 µm) than that in non-recurrent cases (434.62 ± 119.28 µm), although the difference was not statistically significant ($p = 0.169$).

There was a strong negative correlation between the percentage of relative change at 6 months in best-corrected visual acuity and the macular thickness ($R = -0.655$, 95% CI: −0.829 to −0.366, $p < 0.001$). A moderate or low correlation was found between the macular thickness and choroidal thickness ($R = 0.375$, 95% CI: −0.006 to 0.661, $p = 0.054$) and choroidal thickness and best-corrected visual acuity ($R = -0.275$, 95% CI: −0.593 to 0.117, $p = 0.165$) (Figure 4).

Resolution of subretinal fluid

The median time of subretinal fluid resolution estimated with a Kaplan-Meier curve (Figure 5) was 3 months (CI 95%: 1.04-4.96 months). Subretinal fluid resolved in 67% (CI 95%: 51-82%) of eyes at 6 months and in 80% (CI 95%: 65-92%) at 9 months. In the sample of this study, subretinal fluid persisted for more than 6 months in nine eyes.

Prognostic factors

No significant association between the baseline choroidal thickness, macular thickness, best-corrected visual acuity, sex, age, corticosteroid use, ellipsoid zone changes on OCT and the need for treatment was found (Table 2).

Inter-rater agreement

An intra-class correlation coefficient for choroidal thickness measurement of 0.87 (CI 95%: 0.74-0.93) was found. Observation from the Bland-Altman plot (Figure 6) revealed a systematic difference between the two observers of 22 µm (CI 95%: 4.6-39.7) and limits of agreement of −85 and 130 µm.

Discussion

Evolution of the choroidal thickness

The results of this study revealed that the choroidal thickness decreased in acute central serous chorioretinopathy over a one-year follow-up period. They agree with those of previous investigations.[9,10,14–19] However, the majority of these studies focused on changes in the choroidal thickness following treatment in acute and chronic forms

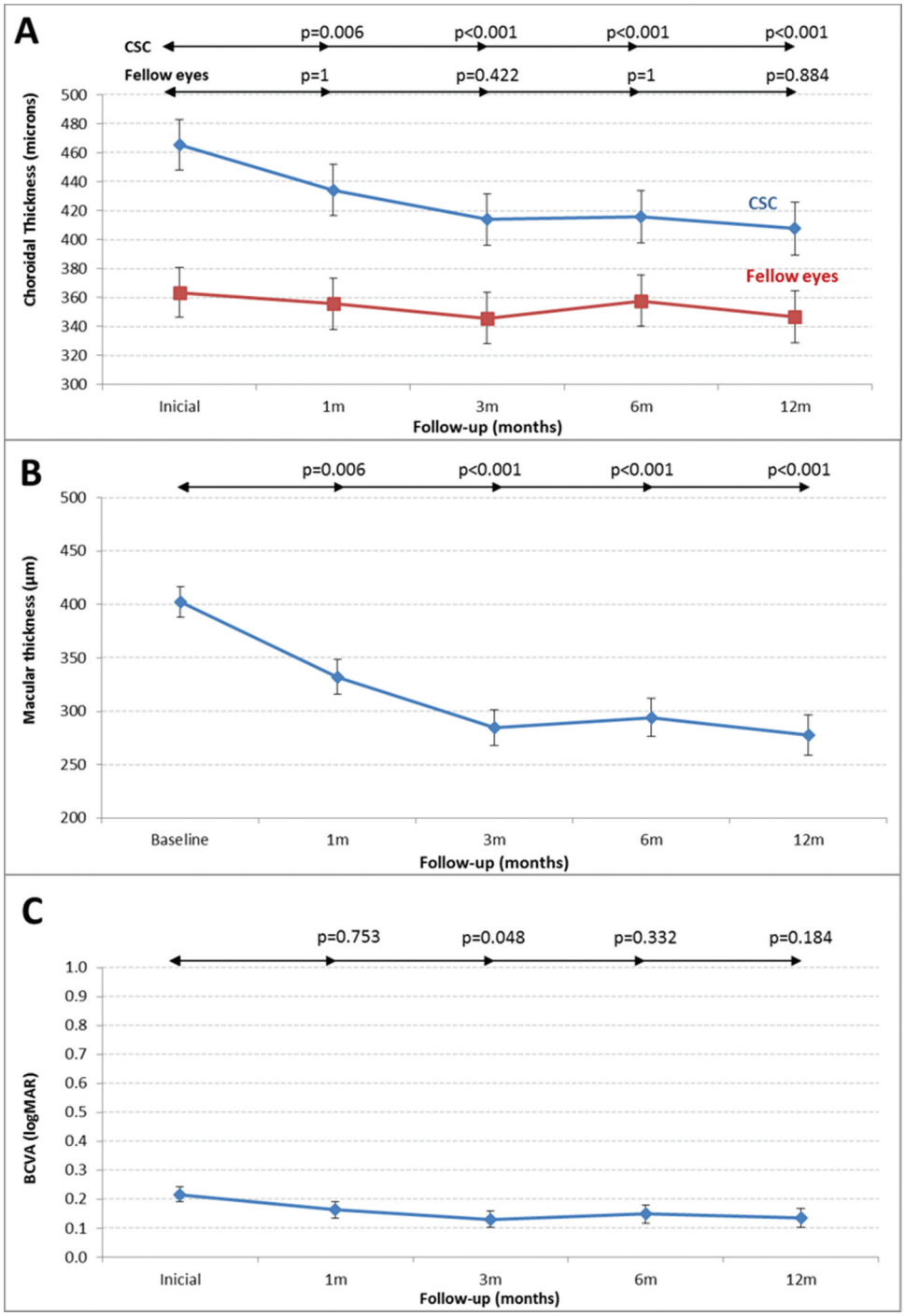

Figure 3. Time course change in A: choroidal thickness, B: macular thickness and best-corrected visual acuity (best-corrected visual acuity) C:. Mixed models estimated means and standard errors and time effect *p*-values in central serous chorioretinopathy eyes (blue line) and fellow eyes (red line).

of central serous chorioretinopathy rather than changes in the choroidal thickness in non-treated patients for a long follow-up.

Some researchers retrospectively evaluated the choroidal thickness in non-treated central serous chorioretinopathy. Kang and Kim[9] found that the choroidal thickness decreased both in non-treated central serous chorioretinopathy and central serous chorioretinopathy treated with photodynamic therapy. In their study, which included both acute and chronic patients, the choroidal thickness returned to normal only in the photodynamic treatment group.

Chung et al.[19] found a decrease in the choroidal thickness in acute treated and non-treated cases from the active to resolved state. However, other authors did not find a decrease in the choroidal thickness. Pertl et al.[20] found that an increase in subretinal fluid was associated with an increase in the choroidal thickness in non-treated central serous chorioretinopathy eyes. However, their short follow-up period of 4-6 weeks may explain this finding.

Another retrospective study found no change in the choroidal thickness during a 6-month follow-up in non-treated and photocoagulation-treated patients.[21] The baseline choroidal thickness in this study (260 ± 65.87 µm) was lower than that in the present study (465.45 ± 115.42 µm) and previous studies (505 ± 124,[2] 445 ± 100,[4] 475 ± 138 µm[6]). Also, the authors excluded participants with poor visibility of choroidal boundaries, which could bias towards cases with smaller choroidal thicknesses as OCT image penetration could be worse in cases with greater choroidal thicknesses.

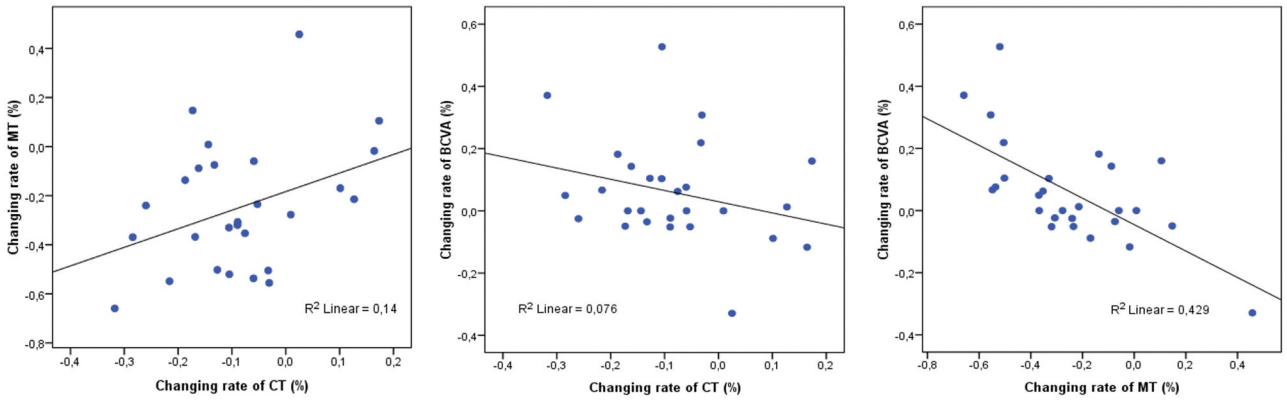

Figure 4. Correlations between the changing rate of BCVA (best-corrected visual acuity), MT (macular thickness) and CT (choroidal thickness) at 6 months. R^2, coefficient of determination

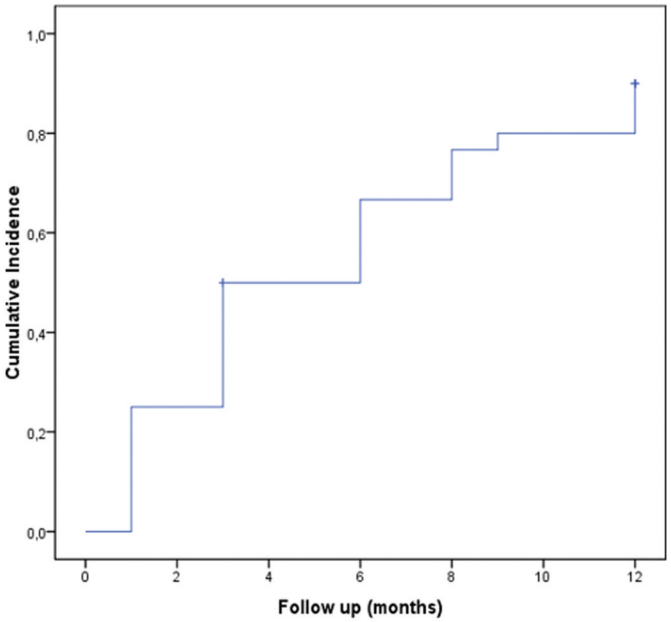

Figure 5. Kaplan-Meier curve for the cumulative incidence of subretinal fluid resolution during the one-year period.

Table 2. Prognostic factors determining the need for treatment.

	RR	CI 95%	*p*-value
Sex (males:females)	1.38	0.91–2.10	.133
Age (years)	1	0.98–1.02	.922
Corticosteroid use	0.86	0.4-1.84	.693
Initial CT (µm)	1	1	.846
Initial MT (µm)	1	1	.362
Initial BCVA (logMAR)	0.98	0.96–1	.112
Ellipsoid zone changes on OCT	0.79	0.37-1.69	.543

RR: relative risk; CI: confidence interval; CT: choroidal thickness; MT: macular thickness; BCVA: best-corrected visual acuity.
Relative risk was estimated by a modified Poisson regression.

Maruko et al.[22] found that the choroidal thickness did not decrease in patients treated with laser photocoagulation after 4 weeks follow-up. The baseline choroidal thickness of this retrospective study was also lower than that in the present study (345 ± 127 µm), probably because patients were older and no time since symptoms beginning was established. Also, patients were no treatment-naïve and were followed for a short time. This could explain the discrepancies with the present study.

Pathophysiology hypotheses

Choroidal vascular hyperpermeability and increased hydrostatic pressure have been widely proposed as the pathophysiology of the disease.[8,23] It is found in both eyes of unilateral central serous chorioretinopathy choriocapillaris hyperpermeability and venous dilation by indocyanine angiography, which would cause an increased choroidal thickness found by OCT.

More recently, the mineralocorticoid hypothesis has been proposed, suggesting that the activation of the mineralocorticoid receptor would cause choroidal vascular dilation by the activation of calcium-dependent potassium channel KCa2.3 present in choroidal endothelial cells.[24] Because of this, mineralocorticoid antagonists like eplerenone and spironolactone and steroid antagonists like mifepristone have been studied.[25,26] However, the efficacy results of these drugs have been inconclusive.

Another plausible theory is the implication of the autonomous system in choroidal blood flow. Chung et al.[19] proposed that the choroidal non-vascular smooth muscle cells present in Haller's layer could stretch out under this sympathetic input and lead to interstitial oedema and an increase in the choroidal thickness. After the autonomic stimulation ceased, the non-vascular smooth muscle cells would contract, leading to a reduction in the choroidal thickness and reabsorption of subretinal fluid. In the present study, patients with central serous chorioretinopathy referred higher stress levels than controls ($p < .001$), which could stimulate the sympathetic system. Association of central serous chorioretinopathy with stress has been previously described.[27]

The authors of the present study believe that all these theories are complementary. Susceptible individuals with thicker choroids would be affected by factors such as corticosteroids, stress or sympathetic dysregulation, causing an increase in the choroidal thickness and hyperpermeability and consequent leakage of fluid. When the stimulus ceased, the choroid would return to its basal state that could predispose again to new episodes. In the present study, at the moment of the active central serous chorioretinopathy, the choroidal thickness of eyes with central serous chorioretinopathy (465.45 ± 115.42 µm, $p < 0.001$) and fellow eyes (363.44 ± 113.5 µm, $p < 0.039$) was greater than controls (317.54 ± 72.04 µm). During the follow-up, the choroidal thickness in central serous chorioretinopathy eyes decreased ($p < 0.001$) but still was greater than controls ($p = 0.001$) at 6 months.

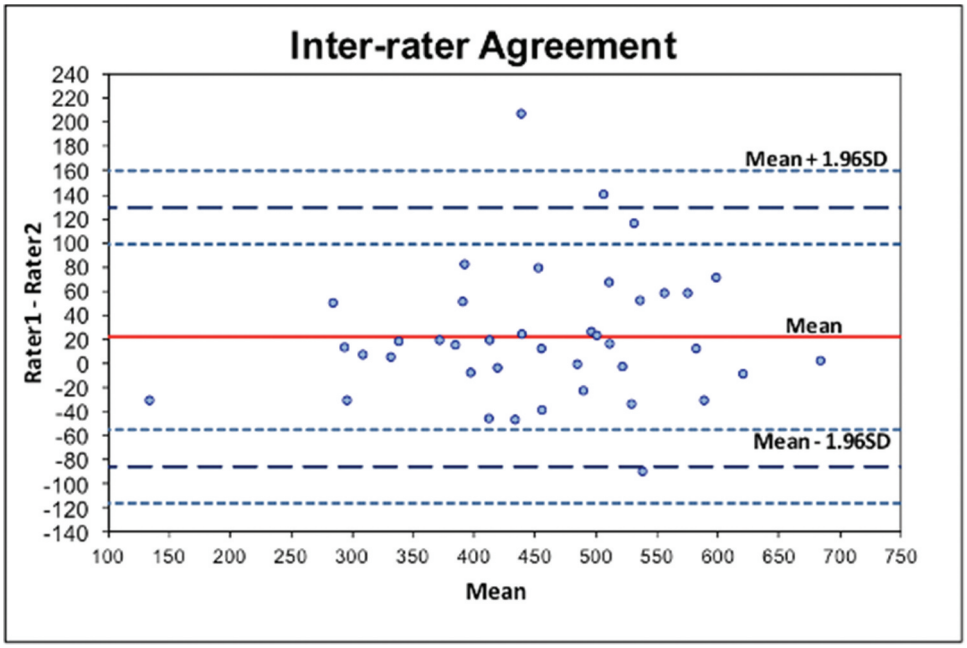

Figure 6. Bland-Altman plot for differences in the choroidal thickness measurements between observer 1 and 2.

Recurrences

This predisposed basal state is supported by the findings of Kim et al.[28] and the present results that those inactive cases with a greater choroidal thickness could be predisposed to recur. The authors observed that the reduction in the choroidal thickness over time was lower in recurrent than in non-recurrent cases. Furthermore, in cases of recurrence, the choroidal thickness increased towards the baseline value. In their study, all eyes were treated with intravitreal bevacizumab or half-fluence photodynamic therapy.[28] In the present study, the choroidal thickness in recurrent cases at baseline was greater than that in non-recurrent cases (511.83 ± 104.75 vs. 434.62 ± 119.28 µm). It is possible that these differences did not reach statistical significance (p = 0.169) given the small sample size of the recurrence sub-group. Further studies are needed to confirm these hypotheses and to shed light on which cases are more prone to recur and require earlier or more aggressive treatment to prevent it.

Correlation between variables

A strong negative correlation between the changing rate at 6 months in best-corrected visual acuity and macular thickness was found. This finding was to be expected, as it is also correlated with other macular diseases. However, at the 6 months follow-up a moderate to low correlation between the macular thickness and choroidal thickness and choroidal thickness and best-corrected visual acuity was found. Thus, changes in the choroidal thickness over time cannot serve as an indicator of best-corrected visual acuity or subretinal fluid resolution.

Prognostic factors

Ambiya et al.[29] found that central serous chorioretinopathy with the choroidal thickness lower than 356 µm was more likely to require treatment. In the present study, the baseline choroidal thickness, macular thickness, best-corrected visual acuity, sex, age and corticosteroid use were not prognostic factors for subretinal fluid persistence at 6 months. Subretinal fluid persistence at 6 months was used as the cut-off point for treatment need. At this point, treatment was offered to patients, in common with previous research.[30] The study by Ambiya et al.[29] was retrospective and patients were treated at baseline, which could have biased the results. In addition, as mentioned by the authors, no treatment protocol was established in their study. A prospective study with treatment-naive patients could better establish the role of choroidal thickness as a predictor for treatment.

Effect of age, sex and axial length on choroidal thickness

Previous studies have found association between the choroidal thickness and age, sex and axial length.[31–34] In the present study, no statistically significant differences between groups of age (p = 0.623) or refractive error (p = 0.71) were found, but differences of sex ratios were found (p = 0.042). As these could bias the comparison of the choroidal thickness between groups, a possible interaction effect of sex in the mean choroidal thickness was analysed. No statistically significant interaction effect was found (F = .350, p = 0.557).

Study limitations

The current study has several limitations. First, the symptom duration was less than 30 days. Thus, the results apply only to acute central serous chorioretinopathy. It would have been desirable to have a bigger sample with a subgroup of chronic central serous chorioretinopathy as well as calculate the sample size before initiating the recruitment. In addition, strict inclusion criteria for refractive error limit extrapolation of the results to the general population. Furthermore, the choroidal thickness was measured manually rather than using an automated software programme. However, as the inter-observer

correlation was good, errors attributed to the manual measurement were minimised. In addition, other studies reported good interobserver correlations for manual choroidal thickness measurements.[35–38] Finally, the choroidal thickness was measured only at the fovea. It would have added more information to measure it at different parts of the macula and at the leaking points on fluorescein angiography.

Contributions of this study

This is thought to be the first prospective study to evaluate changes in the choroidal thickness in treatment-naive eyes with central serous chorioretinopathy and fellow eyes as compared with that in healthy controls during a one-year follow-up and to investigate factors predicting the need for treatment. The findings enhance understanding of the role of the choroid in the pathophysiology of central serous chorioretinopathy and may help to detect patients with an elevated risk of recurrence.

Further studies are needed to confirm these data and evaluate prospectively the change in the choroidal thickness over time in response to different treatment modalities. In addition, studies on the choroidal thickness in chronic central serous chorioretinopathy could help to differentiate them from acute central serous chorioretinopathy or other pathologies.

Conclusion

This study showed that the choroidal thickness decreased in acute non-treated central serous chorioretinopathy eyes during one-year follow-up and that subretinal fluid persisted in less than 20% of patients at the end of this follow-up period.

Acknowledgements

We would like to thank Scribendi (Scribendi.com) for English language editing. The authors declare no potential conflicts of interest or funding sources.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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