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An investigation on choroidal and retinal thickness alterations in Posner-Schlossman syndrome patients

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Abstract

Background We aimed to compare choroidal thickness (ChT) and retinal nerve fiber layer (RNFL) thickness in the affected and contralateral eyes of patients with Posner-Schlossman Syndrome (PSS) during acute, remission, and intermittent phases.

Methods This prospective observational study included 18 patients(36 eyes) diagnosed with PSS. These patients underwent a comprehensive ophthalmologic evaluation including slit lamp examination, visual acuity testing, intraocular pressure (IOP) measurement, and funduscopic examination, and assessment of RNFL thickness, macular thickness, and macular ChT. Patient data collected included gender, age, number of keratic precipitates (KPs), and number of episodes. Optical coherence tomography (OCT) was used to measure RNFL thickness, macular thickness, and macular ChT in both eyes during the acute, remission, and intermittent phases. The affected eye was compared with the unaffected eye at each phase.

Results In affected eyes, macular ChT was lower in the acute phase compared to the remission phase at N1500, N1000, N500, and subfoveal locations (all$p < 0.05$).The central macular recess ChT was also significantly thinner in the acute phase compared to the intermittent and remission phases. Age significantly correlated with ChT in the central recess ($p = .024$). Macular thickness was thinner during the acute phase in the affected eye ($p = .048$). The RNFL in the affected eye was thinner in the intermittent phase than in the acute phase at the inferior-temporal ($p = .011$) and global sectors ($p = .044$). During the acute phase, RNFL in the affected eye was thinner at the superior-nasal ($p = .049$), inferior-temporal ($p = .003$), and global ($p = .041$) sectors compared to the unaffected eye. In the intermittent phase, the affected eye's RNFL was thinner at the superior-nasal, inferior-temporal, inferior-nasal, and global sectors compared to the unaffected eye (all$p < 0.05$), while no difference was observed in both eyes at the nasal, superior-temporal, and temporal sectors. The number of episodes and age were significantly associated with RNFL thickness ($p < .05$).

Conclusion This study demonstrated that in eyes affected by PSS, RNFL, macular ChT, and macular thickness thinned during the acute phase. The number of episodes and age are significant factors in the development of PSS.

Keywords Posner-Schlossman syndrome, Choroidal thickness, Retinal nerve fiber layer

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Background

Elevated intraocular pressure (IOP) is the primary clinical symptom of the monocular recurrent anterior uveitis known as Posner-Schlossman Syndrome (PSS), which was initially described by Posner [1] in 1948. Anti-inflammatory and antihypertensive medications are the cornerstones of treatment.Although the pathomechanism and etiology of PSS are unknown, it was once considered to have a favorable prognosis [2]. However, more recent studies have shown that irreversible glaucomatous damage can occur in certain cases [3].

Glaucoma-related changes in retinal nerve fiber layer (RNFL) thickness often precede changes in optic papilla shape and visual field abnormalities [4], underscoring the importance of early RNFL thickness screening for patients with PSS. Research has demonstrated that eyes with early PSS exhibit more pronounced vascular damage in the pericentral sulcus region and significantly reduced macular structural and vascular parameters compared to normal eyes [5]. According to research, macular thickness can be employed as a supplementary measure in the clinical evaluation of glaucoma [6, 7].

Choroidal thickness (ChT) has been proposed as a measurable health biomarker for choroidal tissue, similar to RNFL thickness. However, its clinical utility in glaucoma remains debated [8].Studies involving individuals with acute angle-closure glaucoma have revealed that the choroidal membranes of these patients are abnormally thick [9]. Patients with PSS may have a pathologic process similar to other glaucomas when they develop optic neuropathy and visual field impairment. Notably, fewer studies have investigated ChT in PSS, both domestically and internationally.

This study, explored the clinical features of PSS across its acute, remission, and intermittent phases, focusing on how PSS progression affects macular ChT, macular thickness, and RNFL thickness and examining the associated risk factors.

Methods

Ethics statement

This observational study on patients with PSS was approved by our institution's Ethics Committee (Jszxyy202120) and adhered to the principles of the Declaration of Helsinki. All participants provided written informed consent before the study.

Study population

The study prospectively recruited a sequential group of patients diagnosed with PSS after undergoing testing at the ophthalmology department of the Jinshan Branch of the Sixth People's Hospital in Shanghai, China, between January 2022 and January 2023. All participants who met the diagnostic criteria for PSS were recruited consecutively.

The diagnostic criteria for PSS [10], as outlined in previous research, included recurrent, unilateral symptoms such as mild discomfort, halos, and slight blurring of vision, along with transient episodes of elevated IOP, open angles without iris synechia, hoar and white suet-shaped keratic precipitate (KP), and minimal or absent cells and flare. These episodes typically last from a few hours to a few weeks. Patients with a history of ocular surgery or systemic diseases like hypertension or diabetes mellitus were excluded from the study. The unaffected eye for each patient was designated as the control group for comparative analysis.

We initially contacted 30 patients with PSS; however, after applying strict inclusion and exclusion criteria, 25 patients were recruited. Of these, 7 were lost to follow-up, leaving 18 patients (36 eyes) who were included in the final analysis.

Data collection

All participants underwent a comprehensive ophthalmologic assessment, including slit lamp examination, visual acuity testing, IOP measurement, fundus examination, and assessment of RNFL thickness, macular thickness, and macular ChT. Patient information, including gender, age, number of KPs, and PSS episodes, was also recorded. IOP was measured using a non-contact tonometer (TOPCON CT-800, Japan) three times for each eye, with the mean values used for statistical analysis. Slit lamp, indirect fundoscopy, and fundus photography were used in combination to examine the fundus.

The RNFL thickness was measured by spectral domain optical coherence tomography (OCT Spectralis, Heidelberg Engineering Co., Heidelberg, Germany). Measurements were taken in the nasal (N), superior-nasal (NS), superior-temporal (TS), temporal (T), inferior-temporal (TI), inferior-nasal (NI), and global (G) sectors within a 3.46 mm diameter centered on the optic disc, using OCT optic disc circular scanning.

Enhanced depth imaging OCT (EDI-OCT) was used to measure macular thickness and ChT. This technique improves the resolution of choroidal detail by producing inverted images when the focus is manually positioned posteriorly compared to regular retinal spectral domain OCT images [11]. A single, 30-degree linear scan centered on the fovea was performed to scan the macular region. ChT measurements were taken from the outer boundary of the hyper-reflective line corresponding to the retinal pigment epithelium to the line marking the choroidal-scleral junction perpendicularly. Macular thickness can be evaluated automatically by the OCT system software. Measurements were taken at the macular central concavity and at 500 μm, 1000 μm, and 1500 μm

from the N and T sectors of the macular central concavity. Macular thickness was measured in the same way. We manually got these measurements, averaging three measurements at each measurement location. It is important to note that the first OCT scan was a reference for subsequent imaging. At the same time, the same experienced examiner performed macular ChT, macular thickness, and RNFL thickness measurements.

Treatment and follow-up

Patients were primarily treated with antihypertensive and anti-inflammatory medications. Antihypertensive treatment included a rapid intravenous infusion of 250 mL mannitol once or carteolol drops in the affected eye twice daily. Anti-inflammatory treatment included tobramycin dexamethasone drops and pranoprofen drops in the affected eye four times daily, which were gradually discontinued after IOP control. Two additional follow-up visits after treatment were scheduled following the initial visit: 1 week later (remission phase) and 1 month later (intermittent phase). During each visit, the same thorough ocular assessments were repeated. RNFL thickness was not measured during the remission phase.

Remission phase: IOP below 21 mmHg and or significant reduction or disappearance of KP [12].

Intermittent phase: Normal IOP, significant reduction or disappearance of KP, and the cessation of anti-inflammatory and antihypertensive medications without rebound.

Statistical analysis

Statistical analyses were conducted using SPSS software, version 26. Categorical variables were expressed as percentages, while mean values were presented as mean ± standard deviation. The Shapiro–Wilk test was used to assess the normality of data distribution. These data were normally distributed. Paired-sample t-tests were used to compare differences between affected and unaffected eyes. Multiple linear regression analysis was performed to evaluate the correlation between ChT, RNFL, and various factors. A p -value of less than 0.05 was considered statistically significant.

Table 1 The characteristics of patients with PSS

	Total ($N=18$)
Age (year, mean ± SD)	54.89 ± 12.44
Sex	
Male	10
Female	8
Diseased eyes	
right	12
left	6
Number of episodes	3.33 ± 2.95
Number of KPs	3.89 ± 2.56

Results

Patient characteristics

This study was conducted in 18 patients (36 eyes) with PSS. Data from the affected eye and the contralateral (unaffected) eye were analyzed. Table 1 shows the basic information of the patients. The mean age was 54.89 ± 12.44 years (range: 33–82 years). Of the 18 patients, 10 were male (55.6%) and 8 were female (44.4%). PSS affected the right eye in 12 patients (66.7%) and the left eye in 6 patients (33.3%), with all cases presenting as monocular onset. The mean number of PSS episodes per patient was 3.33 ± 2.95. All patients presented with one or more hoar and white suet-shaped KP on the cornea during the acute phase, with a mean of 3.89 ± 2.56.

The mean IOP in the affected eye decreased from 39.17 ± 12.90 mmHg in the acute phase to 14.17 ± 2.23 mmHg in the intermittent phase. During the outpatient follow-up of these patients, we observed that at initial presentation, topical or systemic IOP-lowering and topical anti-inflammatory treatments were administered. Most patients achieved normalized IOP in the affected eye by the next day, and all of them had normalized IOP in the affected eye on the third day. The IOP in the unaffected eye was 16.61 ± 2.48 mmHg during the acute phase and 15.00 ± 2.89 mmHg during the intermittent phase. The IOP was significantly higher in the affected eye during the acute phase than in the unaffected eye ($p<.001$). However, in this study, the affected eye was found to have a lower IOP compared to the unaffected eye in the remission phase ($p<.001$), with no statistical differences observed between the two during other intervals. IOP changes for all 18 patients are specified in Table 2.

Eight patients (44%), cases 2, 3, 5, 6, 10, 11, 12, and 17 had optic nerve damage. This number increased to 10 patients (56%), with the addition of new cases (14 and 18) during the interval.

Analysis of macular ChT and macular thickness data

There was no difference in ChT at the T500, T1000, and T1500 choroidal locations ($p>.05$). In the acute phase, ChT at the N1500, N1000, N500, and subfoveal locations was thinner compared to the remission phase (all $p<.05$). Figure 1 displays changes in ChT under the macular central recess in both eyes at different phases. There was a significant difference in ChT between the acute and intermittent phases at the subfoveal locations of the affected eye ($p=.029$). When the affected and unaffected eyes were compared in the acute, remission, and intermittent phases, no statistically significant difference was observed ($p>.05$) (Table 3).

Macular thickness analysis showed that at the N1500 macula in the affected eyes, the macular thickness was thinner in the acute phase than in the remission phase ($p=.042$). During the intermittent phase, macular

Table 2 Changes in IOP in both eyes of 18 patients

Case	Affected eyes IOP (mmHg)			Unaffected eyes IOP (mmHg)		
	Acute phase	Remission phase	Intermittent phase	Acute phase	Remission phase	Intermittent phase
1	42	11	12	15	13	13
2	30	11	11	12	10	11
3	24	18	15	17	20	19
4	60	14	12	18	17	14
5	24	14	17	17	17	14
6	38	12	14	16	17	13
7	44	12	13	16	14	14
8	50	13	14	15	14	14
9	22	12	12	14	17	17
10	28	11	10	13	12	10
11	44	15	13	15	15	13
12	30	12	17	18	15	15
13	48	18	14	21	19	19
14	60	16	16	17	17	13
15	60	17	15	17	18	16
16	27	17	17	21	21	21
17	31	17	17	20	19	17
18	43	16	16	17	17	17
mean	39.17 ± 12.90	14.22 ± 2.56	14.17 ± 2.23	16.61 ± 2.48	16.22 ± 2.86	15.00 ± 2.89
P	<0.001[a]	<0.001[b]	0.164[c]			

[a]: IOP comparison in patients at the acute phase between affected and unaffected eyes

[b]: IOP comparison in patients in remission phase between affected and unaffected eyes

[c]: IOP comparison in patients in intermittent phase between affected and unaffected eyes

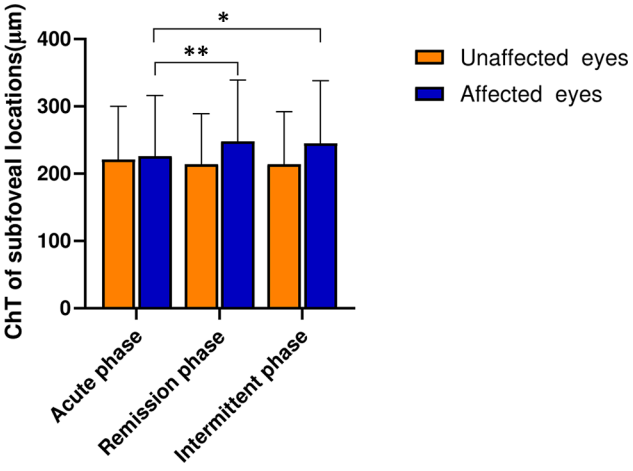

Fig. 1 Analysis of ChT in the macular subfoveal locations. Data were presented as the mean ± standard deviation.$^{*}P<.05$,$^{**}P<.01$

thickness at macula N1000 was thinner in the affected eye compared to the acute and remission phases ($p<.05$). During the acute phase, macular thickness at T1500 in the affected eye was thinner than in the unaffected eye ($p=.048$) (Table 4).

Analysis of RNFL thickness data

Table 5; Fig. 2 list the analysis of RNFL thickness in the affected and unaffected eyes during the acute and intermittent phases. The affected eye's RNFL was thinner in the acute phase compared to the unaffected eye's TI and G sectors (all $p<.05$). However, significant differences in the N, TS, T, or NI sectors were not observed. There was no difference in the other sectors of the affected eye's RNFL between the intermittent and acute phases. However, there was a thinner RNFL in the G ($p=.044$) and TI ($p=.011$) sectors. Significant differences in the N, TS, and T sectors were not observed. However, the affected eyes were thinner compared to the unaffected eyes during the interval in the NS ($p=.048$), TI($p=.001$), NI ($p=.019$), and G($p=.001$) sectors.

Multiple linear regression analysis of various factors with macular ChT

A multiple linear regression analysis of macular ChT at the center recess was conducted in relation to the number of episodes, age, and maximum IOP in the acute phase. Age was significantly and negatively correlated with ChT in the center recess (R2=0.480,$p=.024$) (Table 6).

Multiple linear regression analysis of various factors with RNFL thickness

Multiple linear regression analysis was also performed to assess the correlation between RNFL thickness in affected eyes and the number of episodes and KPs, age, and maximum IOP during the acute phase. (Tables 7

Table 3 ChT analysis of the affected and unaffected eyes

	Affected eyes (μm)						Unaffected eyes (μm)					
	Acute phase	Remission phase	Intermittent phase	p^d	P^e	p^f	Acute phase	Remission phase	Intermittent phase	p^h	p^i	p^j
N1500	187.83 ± 70.91	203.22 ± 79.0	200.06 ± 94.11	0.019*	0.197	0.649	197.17 ± 82.65	189.94 ± 78.84	186.11 ± 83.43	0.494	0.341	0.381
N1000	211.44 ± 77.95	232.33 ± 94.12	227.78 ± 100.93	0.037*	0.111	0.586	209.72 ± 82.23	204.00 ± 80.06	199.78 ± 80.03	0.895	0.128	0.150
N500	224.89 ± 83.58	244.22 ± 98.73	237.72 ± 90.67	0.044*	0.138	0.456	218.06 ± 81.02	214.44 ± 78.63	207.61 ± 77.56	0.659	0.117	0.091
subfoveal locations	225.94 ± 90.24	247.72 ± 91.12	244.83 ± 93.45	0.009*	0.029*	0.286	221.44 ± 78.34	214.39 ± 75.02	214.39 ± 77.43	0.802	0.051	0.097
T500	231.44 ± 78.96	242.89 ± 88.35	240.06 ± 83.26	0.167	0.322	0.678	222.67 ± 76.22	212.72 ± 73.46	216.11 ± 79.00	0.612	0.110	0.210
T1000	226.06 ± 74.84	234.22 ± 81.84	233.44 ± 75.93	0.365	0.482	0.906	224.33 ± 86.18	209.17 ± 77.32	211.28 ± 74.80	0.937	0.187	0.256
T1500	215.56 ± 66.32	224.94 ± 85.25	224.06 ± 71.57	0.425	0.301	0.922	227.50 ± 85.21	207.72 ± 74.55	213.22 ± 73.34	0.555	0.414	0.576

[d]: Comparison of ChT in affected eyes during acute and remission phase

[e]: Comparison of ChT in the affected eyes during the acute and intermittent phase

[f]: Comparison of ChT in affected eyes during remission and intermittent phase

[h]: Comparison of ChT between affected and unaffected eyes in the acute phase

[i]: Comparison of ChT between affected and unaffected eyes during the remission phase

[j]: Comparison of ChT between affected and unaffected eyes during the intermittent phase

*: $p < .05$

and 8) The RNFL thickness at the TI sector in the acute and intermittent phases of the affected eye was negatively correlated with the number of episodes and age (R2 = 0.573, $p < .001$ and R2 = 0.543, $p < .001$). An increase in age and the number of episodes were associated with a thinner RNFL in the TI sector. Additionally, a negative correlation was observed between RNFL thickness at the G sector in the intervals of the affected eye and the number of episodes (R2 = 0.499, $p < .001$) (Table 9). The higher the number of episodes, the thinner the RNFL at the G sector.

Discussion

PSS is a rare ophthalmic disease with a higher prevalence in Asia than in Europe. Paivonsalo Hietanen et al. [13] examined 1122 patients with uveitis in Finland between 1980 and 1982 and 1988, finding that PSS accounted for 18% of the cases. Between 2013 and 2017, Bro et al. [14] analyzed 2,483 patients with uveitis in southern Sweden; PSS comprised 0.7% of the study population. Siak et al. 's [15] study of 1249 patients with uveitis in Singapore from 1997 to 2010 revealed that PSS was responsible for 4.9% of uveitis cases. PSS is prevalent in males and usually develops between the ages of 20 and 60 [10], aligning with our research. Most patients with PSS are reported to present with monocular onset [16].

Previous studies have shown a unique feature of IOP in PSS known as the crossover phenomenon [17], which means that during an episode of PSS, the IOP of the affected eye is significantly higher than that of the contralateral eye, while during intervals, the decrease in IOP is less pronounced in the contralateral eye. The present study demonstrated that IOP was significantly higher in the acute phase compared to the unaffected eye, and lower in the remission phase, likely due to the use of antihypertensive medications during the remission phase. No difference in IOP was observed between the affected and contralateral eyes during the intervals. Since IOP changes dynamically, we measured IOP according to the patient's different disease stages: acute, remission, and intermittent. Although all measurements were made during the day, different patients had their IOP measured at different times, so it is unclear if these variables had an impact on the phenomenon of IOP crossover. Even though all study participants had normal IOP during the intervals, some experienced irreversible optic nerve damage. Investigating whether this had impacted the overall IOP crossover phenomenon is necessary. Nevertheless, once the IOP crossover phenomenon disappears, PSS may progress to glaucoma.

It is unclear whether inflammation solely affects the anterior portion of the eye or if posterior components like the choroid and retina are also affected by PSS, as the pathophysiology of the condition is not entirely

Table 4 Macular thickness analysis of the affected and unaffected eyes

	Affected eyes (μm)						Unaffected eyes (μm)			p^4	p^5	p^6
	Acute phase	Remission phase	Intermittent phase	p^1	p^2	p^3	Acute phase	Remission phase	Intermittent phase			
N1500	351.35 ± 13.92	353.65 ± 13.35	352.47 ± 13.19	0.042*	0.290	0.219	353.94 ± 15.20	354.82 ± 14.02	353.00 ± 13.39	0.258	0.573	0.810
N1000	344.12 ± 15.91	344.24 ± 15.24	341.29 ± 16.64	0.899	0.018*	0.020*	345.76 ± 19.09	346.76 ± 16.73	344.82 ± 16.16	0.576	0.176	0.168
N500	286.18 ± 17.47	287.24 ± 21.11	283.59 ± 20.45	0.718	0.371	0.056	283.00 ± 24.20	285.76 ± 23.24	284.00 ± 26.48	0.394	0.559	0.909
subfoveal locations	221.41 ± 16.17	221.41 ± 14.42	219.94 ± 16.57	1.00	0.561	0.421	223.18 ± 14.82	222.41 ± 16.70	222.47 ± 17.43	0.679	0.794	0.505
T500	288.89 ± 18.17	289.00 ± 17.15	287.76 ± 15.49	0.951	0.567	0.461	289.29 ± 14.10	287.52 ± 15.50	283.24 ± 15.41	0.895	0.675	0.224
T1000	328.65 ± 16.60	329.76 ± 14.43	328.76 ± 14.05	0.460	0.929	0.336	333.59 ± 16.58	332.47 ± 14.72	331.00 ± 14.17	0.064	0.298	0.492
T1500	321.00 ± 14.49	323.06 ± 13.74	321.00 ± 13.07	0.141	1.00	0.106	326.59 ± 14.91	328.06 ± 15.95	326.35 ± 16.41	0.048*	0.139	0.093

[1]: Comparison of macular thickness in affected eyes during acute and remission phase

[2]: Comparison of macular thickness in the affected eyes during the acute and intermittent phase

[3]: Comparison of macular thickness in affected eyes during remission and intermittent phase

[4]: Comparison of macular thickness between affected and unaffected eyes in the acute phase

[5]: Comparison of macular thickness between affected and unaffected eyes during the remission phase

[6]: Comparison of macular thickness between affected and unaffected eyes during the intermittent phase

*: $p < .05$

understood. Measuring ChT and optic nerve fiber layer thickness may help detect changes in the posterior segment of the eye's histology in patients with PSS. Research indicates that measuring retinal thickness could improve our understanding of ocular physiopathology [18, 19]. According to recent research, some cases of PSS result in irreparable glaucomatous damage, including impairment to the optic nerve and visual field [3]. Structural damage often precedes visual field loss detected by the conventional visual field technique. As a result, we noticed alterations in RNFL thickness from the acute to the intermittent phase. Some of the affected eyes had new damage to the optic nerve fiber layer after a single episode. Compared to the N and T sectors, the superior and inferior sectors of the RNFL are thicker [20]. Similar to our findings, RNFL degradation develops earlier in the superior and inferior quadrants than in the N and T sectors [21]. A previous study indicated that primary open-angle glaucoma progresses twice as fast as glaucoma due to uveitis [22].

During a follow-up period of 32.8 ± 28.3 months, a retrospective case study revealed a substantial reduction in the overall RNFL thickness in eyes affected by PSS [23]. Tsai [24] observed a 30-year-old man with PSS over 12 months and discovered that even while his IOP returned to normal, the RNFL thickness in the afflicted eye was declining. In patients with PSS, RNFL thinning is linked to cytomegalovirus positivity [23].

This association may be explained by the possibility that IOP is greater and ocular inflammation is more severe in patients with PSS who test positive for the virus [25–27]. We analyzed RNFL thickness using multiple linear regressions, and our analysis showed that RNFL thickness decreases with age and morbidity frequency, consistent with Gao et al.'s findings; the length of the illness and the frequency of recurrences are significant factors in PSS progression [28]. Longitudinal observations are required in PSS to enable the analysis of changes in RNFL following multiple relapses.

It has been shown that glaucoma causes thinning of macular thickness [29], although there is limited research on macular thickness in PSS. Our investigation revealed that the affected eye's macular thickness changed during various PSS stages, but there was no discernible variation between the two eyes. There was a significant difference in the macular thickness at T1500 between the affected and contralateral eyes during the acute phase. However, no significant difference was observed during the remission or intermittent phases. This may be due to increased IOP during the acute phase. We only measured the macular thickness at localized points in the macular region and did not analyze the whole region of the macula in terms of the whole retinal thickness, limiting our

Table 5 RNFL thickness analysis of the affected and unaffected eyes

RNFL thickness	Affected eyes (μm)			Unaffected eyes (μm)	P^n	P^o
	Acute phase	Intermittent phase	P^m			
N	66.67 ± 25.04	63.28 ± 15.68	0.338	65.56 ± 12.80	0.843	0.567
NS	98.67 ± 25.78	98.28 ± 25.21	0.912	117.22 ± 21.54	0.049*	0.048*
TS	125.94 ± 30.08	124.28 ± 28.71	0.620	138.56 ± 26.24	0.146	0.061
T	80.50 ± 17.82	76.83 ± 13.76	0.093	82.61 ± 19.05	0.584	0.083
TI	132.61 ± 25.06	126.33 ± 28.04	0.011*	156.17 ± 21.90	0.003*	0.001*
NI	106.00 ± 27.63	101.33 ± 20.51	0.165	113.72 ± 21.34	0.271	0.019*
G	94.78 ± 17.83	91.28 ± 13.53	0.044*	102.72 ± 9.69	0.041*	0.001*

[m]:Comparison of RNFL thickness between acute and intermittent phases in affected eyes

[n]:Comparison of RNFL thickness between the affected eyes and the unaffected eyes in the acute phase

[o]:Comparison of RNFL thickness between the affected eyes and the unaffected eyes during the intermittent phase

*:$p < .05$

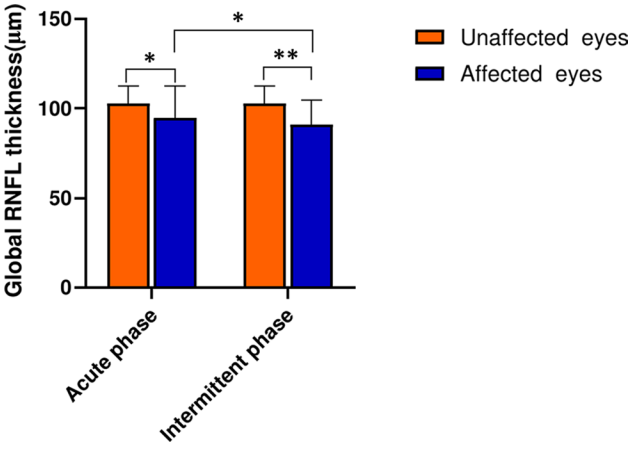

Fig. 2 RNFL thickness analysis at Global. Data were presented as the mean ± standard deviation. $^*P < .05$, $^{**}P < .01$

Table 6 Multiple linear regression analysis of the macular ChT center recess in acute phase

Variable	Estimate	Standard error	B0	*p*
Number of episodes	12.259	6.105	0.401	0.064
Age	-3.893	1.543	−0.537	0.024
Maximum IOP	2.427	1.467	0.347	0.120

Table 7 Multivariate regression analysis of TI sector RNFL thickness in the acute phase

Variable	Estimate	Standard error	B0	*p*
Number of episodes	-5.377	2.046	−0.633	0.021
Number of KPs	0.184	2.565	0.019	0.944
Age	-1.076	0.449	−0.534	0.032
Maximum IOP	0.676	0.422	0.348	0.133

Table 8 Multivariate regression analysis of TI sector RNFL thickness during the intermittent phase

Variable	Estimate	Standard error	B0	*p*
Number of episodes	-5.823	2.368	−0.613	0.029
Number of KPs	0.583	2.969	0.053	0.847
Age	-1.203	0.519	−0.534	0.037
Maximum IOP	0.751	0.488	0.345	0.148

Table 9 Multivariate regression analysis of G RNFL thickness during the intermittent phase

Variable	Estimate	Standard error	B0	*p*
Number of episodes	-3.182	1.197	−0.694	0.020
Number of KPs	0.903	1.501	0.171	0.558
Age	−0.291	0.263	−0.268	0.288
Maximum IOP	0.319	0.247	0.304	0.219

findings. Future research should explore changes in total retinal thickness.

Different types of glaucoma cause the choroids to be thinner or vary in thickness [30]. When comparing the uncontrolled IOP group to the controlled IOP group, a study by Singh et al. revealed a statistically significant increase in subfoveal ChT in primary angle-closure glaucoma [9]. Nonetheless, it has also been demonstrated that ChT is similar in glaucomatous and healthy eyes [31]. According to Guo et al., PSS-affected eyes during the acute and remission phases had macular ChT values that were noticeably thinner than normal [32]. Our research revealed that the affected eye's ChT was thinner during the acute phase compared to the remission phase. However, no difference was observed between the affected and contralateral eyes during the acute, remission, and intermittent phases. An ongoing debate exists on the connection between glaucoma and ChT [8]. It is evident, therefore, that while high IOP in patients during the acute phase causes choroidal thinning [33], acute inflammation causes choroidal thickening. We speculate that varying levels of these components' interplay may result in varying levels of ChT, warranting further research.

Specific independent parameters, including age, axial length, refractive error, diurnal fluctuation, and perfusion pressure, have been found to impact subfoveal ChT [34]. Moreover, medications may impact ChT [35]. Similar to other research, our multivariate regression analysis of ChT in the central macular recess revealed a negative connection between age and macular ChT. However,

even if our study's ChT and RNFL vs. age and onset frequency results correspond with other research, it is still worth examining whether the degree of variations is the same. Guo et al. found associations between macular whole-image vascular density, whole-image perfusion density, and factors such as age, central cornea thickness, and signal intensity index in patients with PSS [36]. Patients with PSS also showed lower macular surface vessel density and perfusion density. The choroid covers the bulk of the perfusion of ocular tissue, and its thickness may be a significant indication of functional health. The investigation of the pathophysiologic changes in PSS-affected eyes requires more longitudinal observations to examine the ChT changes following many recurrences.

In the present study, we prospectively evaluated the changes in ChT, macular thickness, and RNFL in PSS. First, our study is a detailed study of the whole process of a single episode in patients, in which we observed not only the IOP changes, but also the changes in macular ChT, macular thickness, and RNFL. The results also show that a single episode impacts the local optic nerve fiber layer thickness, which suggests that patients with PSS should be taken seriously by clinicians at each episode. Second, our results indicate that the optic nerve fiber layer on the TI sector of patients with PSS is more likely to be affected, which may be helpful for future related studies. Finally, although our findings on choroidal and optic nerve fiber layer versus age and onset frequency are consistent with previous studies, the extent of these variations requires further investigation. Due to the low prevalence of PSS and the small sample size, our study inevitably has some limitations. We did not compare patients with PSS with normal participants and only observed structural changes in affected and unaffected eyes, leaving unanswered whether PSS affects choroidal perfusion in both eyes simultaneously. Clinicians should pay attention to the routine optic nerve fiber layer thickness examination for patients with PSS at the first visit and observe whether the phenomenon of bilateral IOP crossing exists, which is especially significant for patients with PSS who experience recurrences frequently. It is also recommended to observe the change in optic nerve fiber layer thickness for an extended period, as in glaucoma patients, to detect the turning point of optic nerve damage in time. At the same time, the patient's acute IOP should be reduced to normal as soon as possible to reduce the probability of optic nerve damage. Additionally, it is crucial to find the cause of the disease as soon as possible to reduce its frequency.

Longitudinal long-term follow-up and monitoring of ChT, macular thickness, and RNFL alterations in patients is recommended to learn more about the features of PSS.

Conclusion

In this study, the RNFL thinned in the affected eyes, and ChT was thinner in the acute phase compared to the remission phase in the affected eyes. In the acute phase, the macular thickness was thinner in the affected eye compared to the unaffected eye. However, ChT differences between the affected and unaffected eyes were not observed. The likelihood of optic nerve injury in a patient with PSS increases with age and frequency of episodes. The number of occurrences and age are significant factors in the development of PSS.

Abbreviations

ChT	Choroidal thickness
RNFL	Retinal nerve fiber layer
PSS	Posner–Schlossman syndrome
IOP	Intraocular pressure
KPs	Keratic precipitates
OCT	Optical coherence tomography
EDI-OCT	Enhanced depth imaging optical coherence tomography
N	Nasal
T	Temporal
TI	Inferior-temporal
G	Global
NS	Superior-nasal
NI	Inferior-nasal
TS	Superior-temporal

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Author contributions

HL and MJ participated in the planning, execution, analysis, and writing of the study as well as the data interpretation.The data was collected by HL, CS, LXQ, FQ, and LHL, and all authors have read and approved the final text.

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Data availability

The text's tables contain all pertinent data for the study's duration.

Declarations

Ethics approval and consent to participate

The Jinshan Branch of the Shanghai Sixth People's Hospital Review Committee gave its approval for this study, which was carried out in compliance with the Declaration of Helsinki. Written informed permission was acquired from the individuals involved.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

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