

Ocular Manifestations and Visual Outcomes of Patients with Giant Cell Arteritis

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Abstract

Background: Ocular involvement in giant cell arteritis (GCA) is common, yet there is limited literature addressing its specific ocular manifestations and their impact on visual outcomes. The objective was to evaluate the incidence of visual involvement, treatment, relapse rate, and visual outcome in patients with GCA.

Methods: This is a single center retrospective cohort study. The study included 35 eyes from 37 consecutive patients with biopsy-proven GCA (TAB+), 39 eyes from 39 consecutive patients with clinically diagnosed GCA and negative biopsy (TAB-), and 16 eyes from 21 consecutive patients clinically diagnosed with GCA without undergoing a biopsy (no TAB). We collected demographic and clinical presentation. 26/37 (70%) in the TAB+ group, 29/39 (74%) in the TAB- group, and 14/21 (67%) in the no TAB group underwent at least one neuro-ophthalmic exam. Eyes were grouped into no, mild, or severe ocular involvement. At each visit, patients underwent neuro-ophthalmic evaluation, ophthalmic imaging, and visual fields. The main outcome measures were **best-corrected visual acuity (BCVA), color perception (% correct)**, retinal nerve fiber layer (RNFL), ganglion cell complex (GCC), area of foveal avascular zone (FAZ), and Humphrey visual field (HVF).

Results: Within our entire cohort of 97 patients, 69 (71%) underwent neuro-ophthalmic examinations, 5 had unilateral involvement and 42 had bilateral involvement. At diagnosis, all patients were started on high dose prednisone, 9/37 (24%) intravenously and 28/37 (76%) orally. 16/37 (43%) patients were treated with adjuvant tocilizumab. Within our cohort, 5/97 (5%) patients had visual relapses, all within the first year after diagnosis. 15/50 (30%) eyes had no OI, 22/50 (44%) eyes had mild OI, and 13/50 (26%) eyes had severe OI. After 12 months of GCA diagnosis, patients with no OI compared to severe OI demonstrated: BCVA (logMAR 0.164 vs. 1.26, $p<0.001$), color perception (87% vs 6%, $p<0.001$), RNFL (93mm vs. 56mm, $p<0.001$), GCC (76mm vs. 52mm, $p<0.001$), FAZ (0.119 mm^2 vs. 0.314 mm^2 , $p<0.001$), and HVF (-2.58 vs. -19.18, $p<0.001$).

Conclusions: Visual involvement in GCA is common. The affected eyes had irreversible vision loss. GCC, RNFL, and FAZ were associated with poor visual outcome.

Introduction

Giant cell arteritis (GCA) is a large vessel vasculitis typically affecting branches of the common carotid artery and it is one of the leading causes of blindness in neuro-ophthalmology if left untreated or undiagnosed.¹ Initial clinical presentation of GCA can involve varying symptoms such as headaches, scalp tenderness, jaw claudication, fever, weight loss, proximal muscle pain, and visual loss.²⁻⁴ In addition to recognizing clinical symptoms, the use of erythrocyte sedimentation rate, C-reactive protein, and platelet counts, as well as temporal artery biopsy (TAB) can aid in diagnosing GCA.⁵ The gold standard for treatment of GCA currently involves administering high dose corticosteroids upon diagnosis to prevent further ischemic complications that can lead to permanent vision loss⁶ or even death.⁷

According to prior small cohort studies, mild manifestations of visual symptoms such as ocular pain, diplopia, and amaurosis fugax are often seen in patients with GCA.⁸⁻¹¹ Other more severe manifestations note in the literature involve various forms of ocular ischemia such as arteritic-anterior ischemic optic neuropathy (A-AION), central retinal artery occlusion (CRAO), branched retinal artery occlusion (BRAO), cilioretinal artery occlusion, and persistent choroidal ischemia.¹²⁻¹⁶ Currently, there are limited studies on the longitudinal evaluation of visual outcome and neuro-ophthalmic manifestations in patients with GCA.

The association between visual complaints and ophthalmic imaging findings for patients with GCA has not been well studied. The incidence of visual relapses after the initiation of high dose corticosteroids for patients with GCA is rare.¹⁷ The clinical question of whether the detection of visual relapse in GCA patients can be monitored through patients' visual complaints or through serial neuro-ophthalmic exams remain to be answered. We hypothesized that ocular manifestations of GCA can be better evaluated with neuro-ophthalmic exam and advanced office-based ophthalmic testing. The changes seen in the retina and optic nerve can correlate with the disease status of GCA and help predict visual outcomes. In this retrospective study, we evaluated the clinical presentation, neuro-ophthalmic examination and ophthalmic imaging data for all consecutive patients with clinically diagnosed or biopsy proven GCA.

Methods

This is a single center, retrospective, cohort study conducted in accordance with the Declaration of Helsinki at a tertiary medical center. Deidentified patient data were utilized in this study. The Institutional Review Board approved the study protocol at the University of California, Davis. All methods were carried out in accordance with relevant guidelines and regulations and the Declaration of Helsinki.

Study Population

Patients who were seen at a tertiary medical center with the ICD 10 code M31.6 between September 2013 and September 2023 were included in the study. The inclusion criteria of our search included all patients who have a diagnosis of GCA with the ICD code: M31.6. For our data analysis of neuro-ophthalmology exam parameters, exclusion criteria consisted of patients who had significant ocular comorbidities such as age-related macular degeneration, diabetic retinopathy, severe glaucoma, or corneal opacities.

Data Collection

Eligible patients in our study would have their following data collected including age, biological sex, race, department of initial presentation, visual complaints, ocular history at initial presentation, symptoms related to GCA, TAB, erythrocyte sedimentation rate (ESR) levels, c-reactive protein (CRP) levels,

platelet levels, and treatment (route of administration, corticosteroid dose, duration, and other immunologic therapy [tocilizumab]).

For the patients with visual involvement, we collected data from each neuro-ophthalmic exams after their initial diagnosis of GCA. The neuro-ophthalmology exam parameters were collected for best-corrected visual acuity (BCVA), intraocular pressure (IOP), pseudoisochromatic Ishihara color plates, slit-lamp examination, and binocular ophthalmoscopy at each visit. The mean deviation score (MDS) values were collected from the Humphrey visual field (HVF) testing (Humphrey Field Analyzer 750 II-I; Carl Zeiss Meditec). Additional measurements including thickness of retinal nerve fiber layer (RNFL), ganglion cell complex (GCC) were obtained with optical coherence tomography (OCT) (Cirrus HD-OCT Model 5000; Carl Zeiss Meditec). Area of foveal avascular zone (FAZ), radial peripapillary capillary vessels (RPC), microvascular density of the inner retina (ILM-IPL), and outer retina (IPL-OPL) were obtained with optical coherence tomography-angiography (OCT-A) (Optovue AngioVue OCTA; Visionix USA, Inc.). If fluorescein angiography (FA) was done upon initial diagnosis of GCA, the objective findings were documented.

The Snellen visual acuity measurements recorded for BCVA were converted into logMAR values for the purpose of statistical analysis. For patients with counting fingers (CF), the Snellen fraction was converted by: CF 1 foot = 1/200; CF 2 feet = 2/200, etc.¹⁸ For those with hand motion (HM), light perception (LP), and no light perception (NLP), the estimated logMAR equivalent was utilized American Academy of Ophthalmology IRIS ® Registry in Chicago.¹⁹

All patients were subsequently analyzed into three major groups. The first group of patients were those who had GCA pathology confirmed with TAB (TAB+). The second group of patients were those who were clinically diagnosed with GCA but had a negative TAB (TAB-). The third group of patients were those who were clinically diagnosed with GCA without receiving a TAB (no TAB).

Within each of those group, for all patients who had neuro-ophthalmic examinations, eligible eyes who met inclusion criteria were then further grouped and divided into no ocular involvement (no OI), mild ocular involvement (mild OI), and severe ocular involvement (severe OI). The group for no OI was defined as no ocular findings or symptoms for patients with a diagnosis of GCA. The group for mild OI included symptoms of transient vision loss (amaurosis fugax), neurologic symptoms (binocular diplopia), intact vision but with cotton wool spots on fundus exam, mild disc pallor, or delayed filling of the choroid plexus noted on FA. The group for severe OI included those who had ocular ischemic syndrome, ischemic optic neuropathy, CRAO, BRAO, cilioretinal artery occlusion, severe disc pallor, or persistent choroidal ischemia noted on FA. The linear regression models were computed with all eyes who had OI (mild and severe).

Statistical Analysis

Descriptive statistics were used to analyze the demographic data and baseline characteristics of our patients. The Welch's independent t-test was used as an unequal variance t-test to compare the independent samples and a p-value of <0.05 was determined as statistically significant. GraphPad Prism software version 10.3.0 (GraphPad Software Inc., San Diego, CA, USA) was used for statistical analyses and computing the linear regression models.

Results

Figure 1 demonstrates the recruitment, inclusion, and exclusion of patients in the study. Only 2 patients had an FA during their initial ophthalmic exam and the results demonstrated significant choroidal ischemia in the eye with OI. Table 1 further illustrates the demographics and clinical presentations between the three different subgroups of patients with confirmed GCA. When comparing the subgroups, the age and CRP levels were significantly different between them. A post-hoc analysis demonstrated that the TAB+ group had an increased age when compared to the TAB- group (p=0.004) and the TAB+ group had an increased CRP when compared to the no TAB group (p=0.037).

For the patients with confirmed GCA, they all initially presented in various departments: 32/97 (32.99%) at the Emergency Department, 31/97 (31.96%) at Primary Care, 22/97 (22.68%) at Rheumatology, 10/97 (10.3%) at Ophthalmology or Optometry, 1/97 (1%) at Cardiology, and 1/97 (1%) at Neurology. In addition, 23/97 (25.77%) GCA confirmed patients had a history of PMR diagnosis for a median of 90 (1-522) weeks.

TAB Positive GCA Group

Within this group, 22/37 (59%) patients were promptly started on high dose prednisone therapy based on clinical symptoms and 15/37 (41%) patients did not initiate treatment until after confirmed elevated inflammatory markers (ESR, CRP, and platelets). The initial visual presentation was blurred vision in 8/37 (22%), vision loss in 12/37 (32%), amaurosis fugax in 6/37 (16%), ocular pain in 1/37 (3%), sequential binocular vision loss in 1/37 (3%), and 1/37 (3%) presented initially with ocular symptoms bilaterally.

Within the TAB positive GCA group, 26/37 (70%) patients had at least one ophthalmologic exam after being diagnosed with GCA and 50 eyes were included in the data analysis. 2 eyes were excluded due to ocular comorbidities of severe age-related macular degeneration. Upon initial ophthalmologic exam, 15/50 (30%) eyes had no OI, 22/50 (44%) eyes had mild OI, and 13/50 (26%) eyes had severe OI, with 17/26 (65%) patients having bilateral involvement. Table 2 illustrates in detail the various neuro-ophthalmic parameters when comparing no OI with mild OI or severe OI. Figure 2 is a heat map that illustrates the distribution and trends of various neuro-ophthalmic parameters across time stratified by disease severity. Figure 3 shows a volcano plot stratifying the significant neuro-ophthalmic parameters. According to our simple linear regression models for OI (mild OI and severe OI), IOP demonstrated an upward trend for patients (F=10.09, p=0.002) (Figure 4).

For treatment of GCA, 28/37 (76%) patients were started on oral high dose prednisone therapy depending on their weight and 9/37 (24%) patients were started on 1g/day of methylprednisolone intravenously for 3-5 days prior to oral therapy. The average duration of therapy for patients who were only on high dose prednisone therapy was 284 ± 614 days and those who had initial methylprednisolone pulse therapy prior to oral high dose prednisone therapy was

582 ± 424 days. In addition, 16/37 (43%) of patients were also adjunctively treated with tocilizumab 162mg/0.9 mL for an average duration of 764 ± 405 days.

The one patient who presented with binocular visual symptoms was treated with IV methylprednisolone 1gram/day x 3-5 days pulse therapy followed with high dose oral prednisone taper prior to receiving TAB. The patient had neuro-ophthalmic examination beyond 12 months of GCA diagnosis and did not present with any objective ophthalmic findings or deterioration.

Clinically Diagnosed, TAB Negative GCA Group

For the patients who were clinically diagnosed for GCA with negative TAB, 39/44 (89%) patients were on high dose prednisone therapy prior to receiving the TAB, 4/44 (9%) had insufficient records of TAB, and 1/44 (2%) was on low dose prednisone therapy for 308 days prior to receiving the TAB. The average duration of high dose prednisone therapy prior to TAB was 12 ± 13 days. Within this group, 29/39 (74%) patients had at least one ophthalmological exam, and 1/39 (3%) patient was monocular due to a prior enucleation. Upon initial examination, 19/57 (33%) eyes had no OI, 26/57 (46%) eyes had mild OI, and 14/57 (25%) eyes had severe OI, with 17/29 (59%) of patients having bilateral involvement. Again, Table 2 further demonstrates the neuro-ophthalmic parameters when comparing no OI with mild OI or severe OI. According to our linear regression models for patients with OI (mild OI and severe OI), HVF demonstrated an improvement over time ($F=5.622$, $p=0.021$) (Figure 4).

Clinically Diagnosed, No TAB GCA Group

Of the patients who did not receive a TAB for GCA diagnosis, 15/21 (71%) were already on chronic prednisone therapy at the first eye exam, 3/21 (14%) patients were not recommended for a TAB by the physician due to clinical presentation, 1/21 (5%) patients were lost to follow up, and 1/21 (5%) patient declined the TAB. Within this group, 14/21 (67%) had at least one ophthalmological exam. Upon initial examination, 12/28 (43%) eyes had no OI, 13/28 (46%) eyes had mild OI, and 3/28 (11%) eyes had severe OI. Table 2 demonstrates the neuro-ophthalmic parameters when comparing no OI with mild OI or severe OI. According to our linear regression models for OI (mild and severe OI), IOP showed a downward trend ($F=6.646$, $p=0.012$), FAZ demonstrated an increase in size over time ($F=7.067$, $p=0.038$), and HVF progressively worsened ($F=46.47$, $p<0.001$) (Figure 4).

Visual Relapse

Within our cohort 5/97 (5%) patients experienced visual relapse and required modification of their treatment regimen. Of those who had visual relapse, 5/5 (100%) patients had reported ocular symptoms of recurrent blurriness or vision loss which prompted them to see their eye care provider. Only 3/5 (60%) patients had a neuro-ophthalmic examination before and after the visual relapse. For those patients, 2/3 (67%) patients had ocular findings found on neuro-ophthalmic exam during the episode of visual relapse and 1/3 (33%) patients no objective neuro-ophthalmic changes. One of the patients had decrease in their HVF and another patient had notable temporal blurring of the optic disc with disc swelling on fundoscopic exam. 1/5 (20%) patient was off prednisone during the episode of visual relapse and 1/5 (20%) patient was off prednisone but was on adjuvant treatment of tocilizumab. The average duration of prednisone treatment was 84 ± 16 days when patients had noted a relapse in visual symptoms. The remaining 3/5 (60%) patients' visual relapse occurred during their oral prednisone taper and required an increase in their prednisone dosage. All patients who experienced visual relapse occurred within a year of initial GCA diagnosis.

Discussion

This study presented neuro-ophthalmic outcomes in GCA patients with and without OI, emphasizing the critical role of ophthalmic examinations in detecting visual involvement in GCA. In the TAB+ group, RNFL measurements were significantly thinner upon initial diagnosis in the mild OI subgroup. The HVF and GCC measurements were significantly worse in the severe OI subgroup as early as initial diagnosis of GCA. After 6 months of diagnosis, RNFL was significantly thinner and FAZ was substantially larger in those with mild or severe OI compared to those with no OI. Additionally, the BCVA, color perception, RNFL, GCC, FAZ, and HVF showed significant deterioration 12 months after diagnosis in the severe OI group for the TAB+, TAB-, and no TAB groups. Given the low likelihood of fellow eye involvement after the rapid initiation of high-dose prednisone therapy within our cohort, this underscores that prompt treatment is necessary for visual preservation.

Our study cohort demonstrated that GCA was most prevalent in females and many of them presented with a wide array of clinical characteristics that were similar to other studies.^{20,21} In addition, headaches and scalp tenderness were found to be the most common clinical symptoms manifested by our patients, which were comparable to other GCA cohorts.²²⁻²⁴ The early recognition of these clinical symptoms could aid with timelier diagnosis to prevent irreversible vision loss in patients. When comparing the three subgroups, a statistically significant relationship was noted for age and CRP levels. A post-hoc analysis showed that age was higher for the TAB+ group when compared to TAB- and CRP was higher for the TAB+ group when compared to the no TAB group. Typically, older patients have higher levels of age-related inflammation²⁵ that could lead to a positive TAB result; therefore, explaining the increased age in the TAB+ group. In addition, CRP is an acute phase reactant that is a sensitive marker of systemic inflammation; therefore, the TAB result was more likely to be positive in patients with elevated CRP compared to the other subgroups. Further studies are needed to elucidate the differences between these subgroups.

Within our cohort, most patients did not present at an ophthalmology or optometry visit when they had ocular manifestations of GCA. Given that the clinical and visual symptoms of GCA are highly diverse upon initial presentation, it re-emphasizes the importance for all clinicians from all specialties to recognize these early ocular signs to allow for prompt intervention.

According to our linear regression models, within the TAB+ group, IOP demonstrated an upward trend, while in the TAB- group, HVF also demonstrated an upward trend. The no TAB group demonstrated a significant decrease in IOP, increase in FAZ, and worsening HVF over time. Existing literature shows that IOP was lower in patients with GCA as a result of anterior ocular ischemia when compared to non-arteritic anterior ischemic optic neuropathy. Past studies have

also demonstrated that FAZ enlarges in retinal ischemic diseases and retinal vascular obstruction.^{26,27} As we know, GCA is commonly associated with ocular ischemic events (retinal and optic nerve ischemia), which may explain the increase in FAZ and decrease in HVF. The progressively increased IOP result could be due to diurnal variation or heterogeneity in obtaining the measurement through various devices such as Tono-Pen, iCare, or Goldmann applanation tonometry. Although rare, there can be some mild improvement in visual field as demonstrated in past studies.^{28,29} In addition, not all patients had regular follow up given the retrospective nature of our study and there may be anatomical differences in neuro-ophthalmic parameters between patients. Further prospective studies will need to further evaluate the trends of neuro-ophthalmic parameters.

Another noteworthy aspect of our study was that only half the eyes had OI, again, demonstrating the prudence needed for patients to receive neuro-ophthalmic examinations to monitor for OI. Failing to initiate early aggressive steroid treatment can lead to irreversible blindness for GCA patients with visual involvement.³⁰ Within our cohort, we had one patient who presented with binocular visual symptoms in the ED and the patient was promptly treated with high dose steroid prior to receiving a TAB. Even after 12 months of diagnosis, the patient did not suffer any long-term ocular manifestations when treated emergently. Other studies have also shown that failing to treat GCA after monocular vision loss can lead to irreversible vision loss in the fellow eye.³¹⁻³³ Although visual prognosis remains to be guarded after vision loss of the affected eye, it is crucial to treat GCA to prevent vision loss in the unaffected eye.

Similar to other studies, visual improvement was shown to be rare in patients who have lost vision from GCA in our cohort.³⁴⁻³⁶ While the frequency of visual relapse is rare, patients can often experience symptoms again if they are tapered off prednisone too quickly.³⁷ Within our cohort, most patients were reliable historians who could identify visual symptoms which prompted them to be seen by a clinician to change their treatment regimen accordingly. However, patients can be unreliable historians and may not be aware of subtle changes in their vision. Prior studies found that factors beyond advancing age and cognitive impairment play a role in reporting medical history in older-aged participants.³⁸ In addition, changes in visual field that may be indicative of visual relapse are not often obvious to patients until it is severe.³⁹ Within our cohort, all patients who had visual relapse occurred within one year of their GCA diagnosis. It is imperative for patients to have neuro-ophthalmic examinations subsequently after GCA diagnosis to ensure prompt identification of visual relapse and prompt treatment especially within the first year of diagnosis.

Some limitations of our study include the small sample size and retrospective nature of the study design. Not all our patients had regular interval neuro-ophthalmic exam subsequently after GCA diagnosis and there were only a limited number of patients who underwent OCT-A imaging. Additional prospective studies with larger sample sizes, standard follow-up, and standardized imaging protocols are important in addressing these limitations to improve our understanding of visual outcomes in patients with GCA.

Conclusion

In conclusion, our study provides valuable insights into the use of neuro-ophthalmic exam parameters for monitoring GCA and the importance of regular neuro-ophthalmic examination especially within the first year of diagnosis. GCC, RNFL, and FAZ are ophthalmic metrics with significant changes in involved eyes. Prompt diagnosis and treatment is imperative for patients with GCA as any delay in treatment can lead to irreversible blindness. Further studies on ophthalmic biomarkers discovery is necessary to aid in the diagnosis and management of GCA to prevent irreversible blindness.

Abbreviations

GCA, giant cell arteritis; TAB, temporal artery biopsy; A-AION, arteritic anterior ischemic optic neuropathy; CRAO, central retinal artery occlusion; BRAO, branch retinal artery occlusion; ICD-10, International Classification of Diseases, 10th Revision; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; BCVA, best-corrected visual acuity; IOP, intraocular pressure; MDS, mean deviation score; HVF, Humphrey visual field; RNFL, retinal nerve fiber layer; GCC, ganglion cell complex; OCT, optical coherence tomography; FAZ, foveal avascular zone; RPC, radial peripapillary capillary; ILM-IPL, inner limiting membrane to inner plexiform layer; IPL-OPL, inner plexiform layer to outer plexiform layer; OCT-A, optical coherence tomography angiography; FA, fluorescein angiography; logMAR, logarithm of the minimum angle of resolution; CF, counting fingers; HM, hand motion; LP, light perception; NLP, no light perception; IRIS® Registry, Intelligent Research in Sight Registry; TAB+, temporal artery biopsy positive; TAB-, temporal artery biopsy negative; OI, ocular involvement; PMR, polymyalgia rheumatica; IV, intravenous; ED, emergency department.

Declarations

Ethics approval and consent to participate: The Institutional Review Board approved the study protocol at the University of California, Davis (UC Davis). Due to the retrospective nature of the study, the UC Davis Institutional Review Board waived the requirement for informed consent in accordance with relevant guidelines and regulations.

All methods were carried out in accordance with relevant guidelines and regulations and the Declaration of Helsinki.

Consent for publication: Not applicable

Availability of data and materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: TD, GG, HL, LH, BC, and YL report no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Authors' contributions: TD contributed to conception and design of work, acquisition, analysis, and interpretation of data, and drafted the work. GG contributed to acquisition of data and substantively revised it. HL contributed to acquisition of data and substantively revised it. LH contributed to acquisition of data and substantively revised it. BC contributed to acquisition of data and substantively revised it. YL contributed to conception and design of work, acquisition of data, interpretation of data, and substantively revised it. All authors read and approved the final manuscript.

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Tables

Table 1. Participant Characteristics		Clinically diagnosed (n=60)		TAB confirmed (n=37)
		No TAB (n=21)	Negative TAB (n=39)	
Age (mean ± SD)		71.86 ± 7.24	67.46 ± 10.11	74.84 ± 8.24
Sex, n (%)	Female	19 (90%)	27 (69%)	24 (65%)
	Male	2 (10%)	12 (31%)	13 (35%)
Race	White	19 (90%)	32 (83%)	35 (95%)
	African American/Black	1 (10%)	4 (10%)	1 (3%)
	Asian	1 (5%)	0	0
	American Indian or Alaskan Native, Native Hawaiian or other Pacific Islander	0	0	1 (3%)
	Hispanic	1 (5%)	3 (8%)	0
Symptoms	Vision loss	7 (33%)	21 (54%)	24 (65%)
	Headache and scalp tenderness	14 (67%)	29 (74%)	29 (78%)
	Jaw claudication	10 (48%)	14 (36%)	12 (32%)
	Arthralgia and myalgias	5 (24%)	7 (18%)	6 (16%)
	Fever, fatigue, and weight loss	7 (33%)	11 (28%)	8 (22%)
Labs	Erythrocyte sedimentation rate in mm/hr (mean ± SD)	62.66 ± 33.47	57.70 ± 32.97	55.69 ± 29.52
	C-reactive protein in mg/dL (mean ± SD)	18.24 ± 46.82	12.29 ± 17.78	27.29 ± 55.23
	Platelets (mean ± SD)	328,250 ± 122,361	357,375 ± 147,507	361,931 ± 112,520

TAB: Temporal artery biopsy

Table 2: Neuro-ophthalmic Parameters

		0–3 months after diagnosis				3–6 months after diagnosis			6–12 months after diagnosis			12+ months	
TAB+	No OI vs. Mild OI	BCVA	0.12	0.201	p=0.146	0.132	0.165	p=0.607	0.159	0.103	p=0.9589	0.164	0.129
		IOP	17	13	p=0.002	19	17	p=0.0341	16	15	p=0.322	16	15
		Color	87	82	p=0.597	100	92	p=0.11	100	87	p<0.001	100	87
		RNFL	96	87	p=0.02	88	88	p=0.935	95	87	p=0.022	93	87
		GCC	74	75	p=0.60	72	75	p=0.356	73	70	p=0.434	76	75
		FAZ							0.075	0.24	p<0.001		
		RPC							50	47	p=0.23		
		ILM-IPL							48	46	p=0.248		
		IPL-OPL							46	45	p=0.766		
		HVF	-2.58	-1.64	p=0.11	0.455	-4.518	p=0.029	-0.41	-4.624	p=0.016	-2.58	-1.65
	No OI vs. Severe OI	BCVA	0.12	1.696	p<0.001	0.132	1.236	p=0.427	0.106	1.752	p=0.001	0.164	1.26
		IOP	17	11	p<0.001	19	14	p<0.001	16	13	p=0.066	16	13
		Color	87	6	p<0.001	100	31	p=0.066	100	17	p<0.001	100	30
		RNFL	96	86	p=0.6	88	64	p=0.053	95	59	p<0.001	93	56
		GCC	74	60	p=0.021	72	53	p<0.001	74	47	p<0.001	76	52
		FAZ							0.075	0.276	p=0.029	0.119	0.314
		RPC							50	32	p<0.001		
		ILM-IPL							48	35	p=0.002		
		IPL-OPL							46	40	p=0.143		
		HVF	-0.05	-17.85	p=0.02	0.455	-14.49	p=0.034	-0.41	-22.84	p=0.001	-2.58	-19.2
TAB-	No OI vs. Mild OI	BCVA	0.095	0.191	p=0.001	0.093	0.193	p=0.134	0.202	0.147	p=0.177	0.726	0.206
		IOP	13	12	p=0.238	15	16	p=0.788	14	17	p=0.098	14	16
		Color	100	100	p=0.331	100	100	p=n/a	100	96	p=0.203	94	100
		RNFL	92	86	p=0.207	87	78	p=0.093	90	77	p=0.087	92	80
		GCC	80	69	p=0.03	75	71	p=0.325	81	72	p=0.244	77	67
		FAZ	0.204	0.176	p=0.706	0.185	0.176	p=0.849	0.177	0.318	p=0.095	0.357	0.438
		RPC	48	43	p=0.147				47	36	p=0.042		
		ILM-IPL	44	46	p=0.584				47	38	p=0.011		
		IPL-OPL	47	45	p=0.479				43	40	p=0.554		
		HVF	0.625	-4.153	p=0.065	0.798	-2.443	p=0.093	-1.672	-2.326	p=0.638	-0.76	-1.99
	No OI vs. Severe OI	BCVA	0.095	2.148	p<0.001	0.093	1.828	p<0.001	0.202	1.521	p<0.001	0.726	1.817
		IOP	13	16	p=0.051	15	13	p=0.156	14	17	p=0.098	14	13
		Color	100	25	p=0.058	100	0	p=n/a	100	96	p=0.203	94	2
		RNFL	92	110	p=0.213	87	73	p=0.04	90	77	p=0.087	92	64
		GCC	80	57	p=0.001	75	57	p<0.001	81	72	p=0.244	77	54
		FAZ							0.177	0.318	p=0.095		
		RPC											
		ILM-IPL											
		IPL-OPL											

		HVF	0.625	-20.61	p=0.054	0.798	-22.63	p=0.018	-1.672	-19.27	p<0.001		
No TAB	No OI vs. Mild OI	BCVA	0.231	0.327	p=0.363	0.086	0.23	p=0.133	0.1582	0.188	p=0.644	0.142	0.162
		IOP	15	17	p<0.008	18	22	p=0.022	16	19	p=0.045	17	17
		Color	96	95	p=0.807	96	100	p=0.351				93	97
		RNFL	84	84	p=0.934	82	89	p=0.037	79	90	p=0.06	77	78
		GCC	71	74	p=0.436	67	80	p=0.03	60	78	p=0.092	63	73
		FAZ											
		RPC											
		ILM-IPL											
		IPL-OPL											
		HVF	-1.6	-1.892	p=0.828	-0.15	-0.007	p=0.835	-0.382	-1.35	p=0.378	-0.37	-2.83
	No OI vs. Severe OI	BCVA	0.231	2.126	p=0.027	0.086	1.427	p<0.001	0.1582	1.301	p<0.001	0.142	1.589
		IOP	15	15	p>0.999	18	20	p=0.169	16	19	p=0.299	16	17
		Color				96	0	p<0.001				93	0
		RNFL	84.44	117.3	p=0.396	82	53	p<0.001	79	53	p<0.001	77	52
		GCC										63	43
		FAZ											
		RPC											
		ILM-IPL											
		IPL-OPL											
		HVF	-1.6	-2.77	p=0.293								

Figures

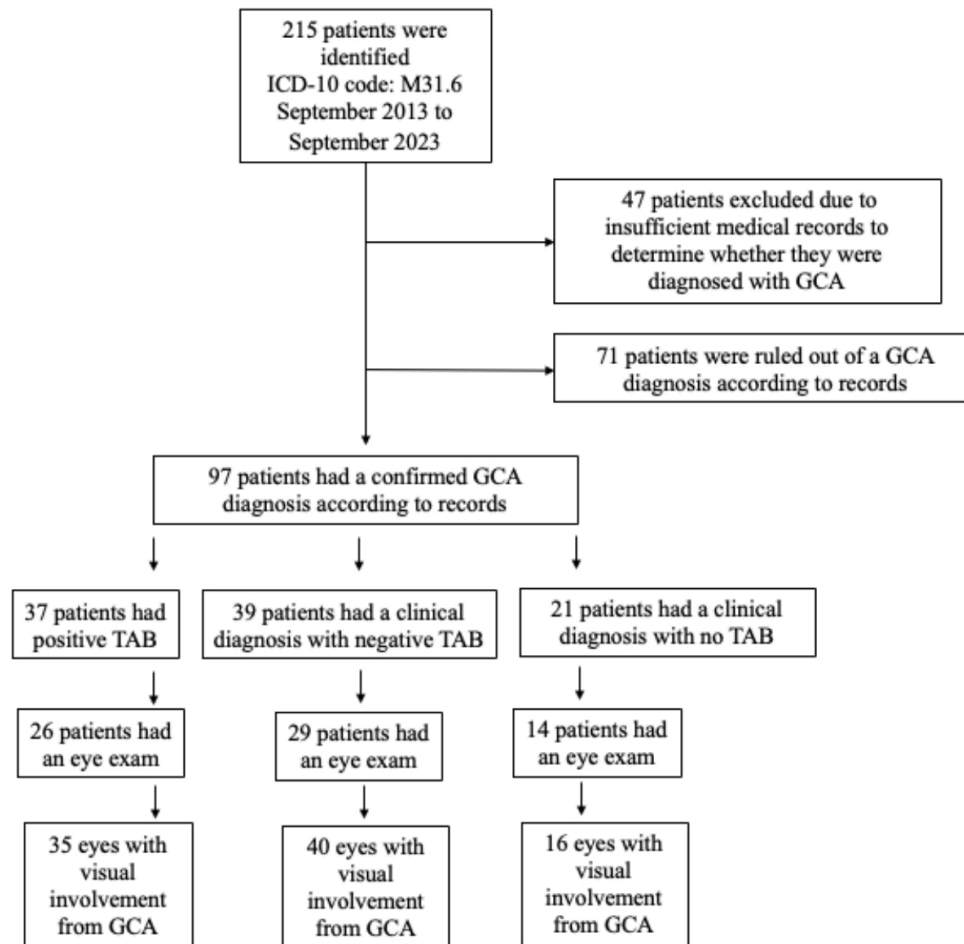


Figure 1

Flow diagram on recruitment, exclusions, and inclusions

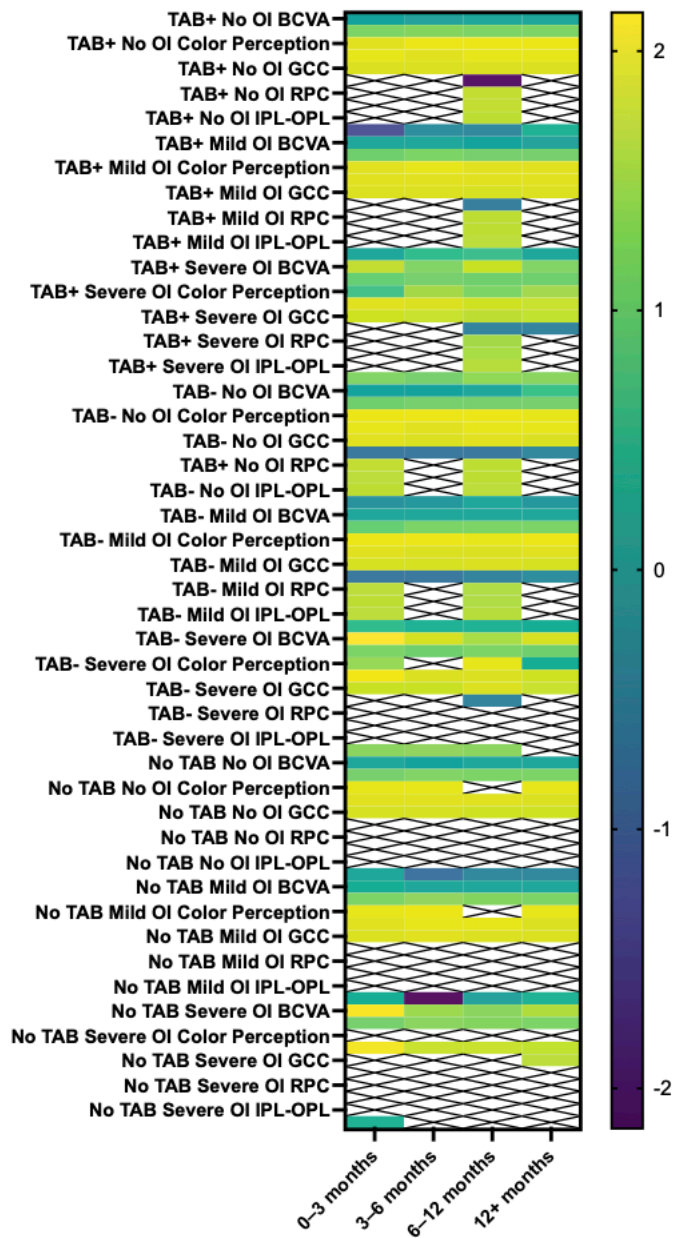


Figure 2

Heat map demonstrating the distribution and trends of various neuro-ophthalmic parameters across time stratified by disease severity.

Volcano Plot

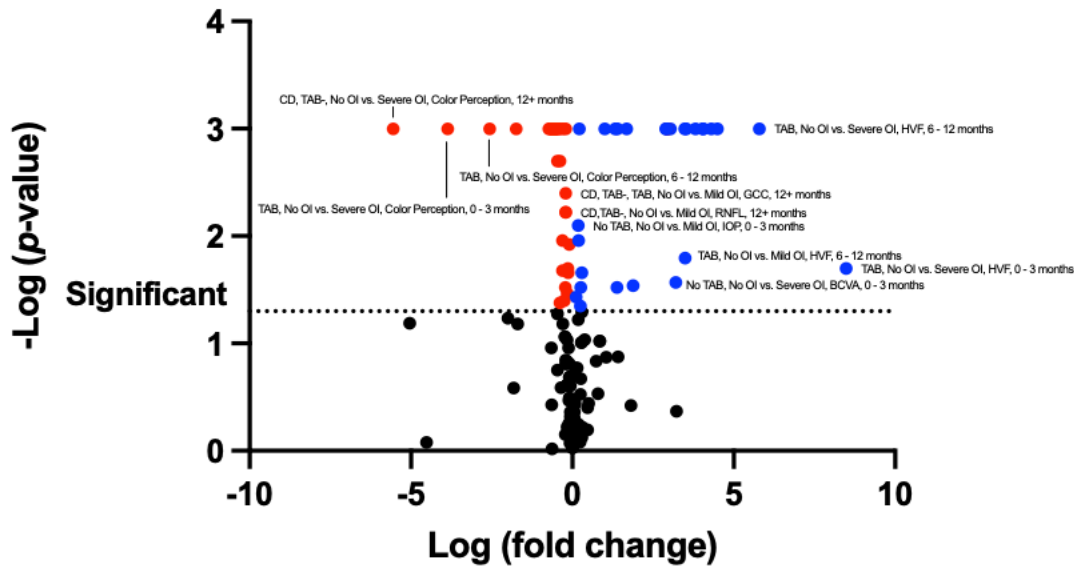


Figure 3

Volcano Plot demonstrating longitudinal changes between no OI eyes with mild or severe OI eyes in all subgroups (TAB+, TAB-, No TAB). This plot illustrates changes in neuro-ophthalmic parameter values between eyes with no OI and eyes with OI (mild or severe) at four different time intervals (0–3 months, 3–6 months, 6–12 months, and 12+ months after GCA diagnosis). Red dots represent decreases in values and blue dots represent increases in values. The dotted line delineates the significance in difference between the no OI and mild or severe OI eyes.

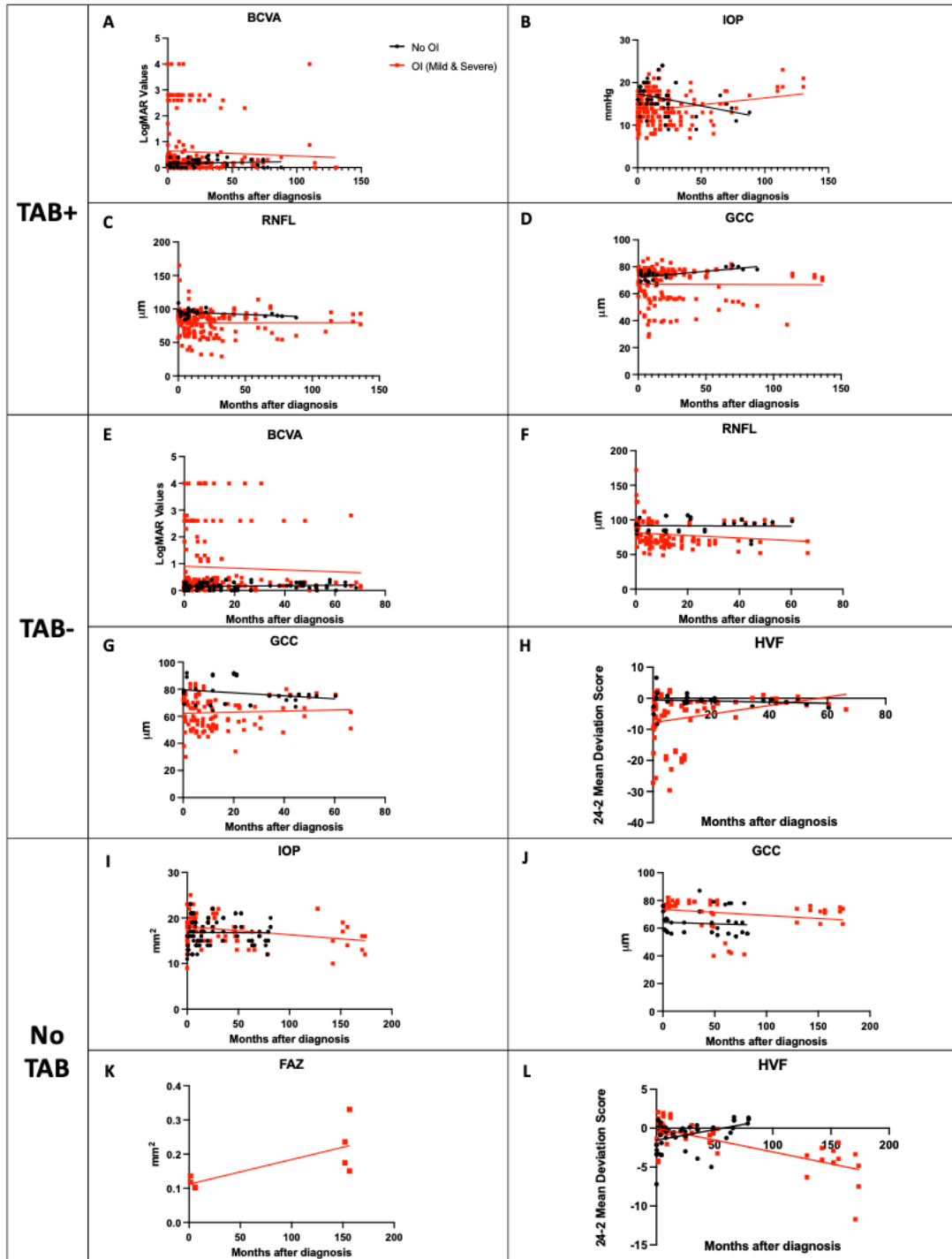


Figure 4

Simple linear regression models of various neuro-ophthalmic parameters in eyes with no OI and OI (mild and severe). (A) TAB+ BCVA trend, (B) TAB+ IOP trend, (C) TAB+ RNFL trend, (D) TAB+ GCC RNFL trend, (E) TAB- BCVA trend, (F) TAB- RNFL trend, (G) TAB- GCC trend, (H) TAB- HVF trend, (I) No TAB IOP trend, (J) No TAB GCC trend, (K) No TAB FAZ trend, (L) No TAB HVF trend.