Original Article

Uveitis-associated glaucoma and treatment-requiring ocular hypertension in a tertiary referral center

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ABSTRACT

Background: Elevated uveitis-associated intraocular pressure (IOP) may result from inflammatory, structural, and corticosteroid-related mechanisms and lead to secondary glaucoma. This study aimed to describe patients with uveitis who subsequently developed glaucoma or treatment-requiring ocular hypertension (OHT) in a tertiary referral clinic.

Methods: This retrospective study was conducted at the Uveitis Clinic of Nikookari Eye Hospital, Tabriz University of Medical Sciences. Records of patients with uveitis diagnosed or managed during the preceding 10 years were reviewed. Patients were eligible if, after uveitis onset, they had IOP > 21 mmHg documented on at least two occasions separated by at least one week and requiring intervention, or documented glaucomatous findings based on available clinical assessment. Patients with IOP elevation or glaucomatous findings predating uveitis or insufficient documentation to establish the temporal relationship were excluded. Demographic, clinical, and treatment variables were extracted.

Results: Among 441 patients with uveitis, 93 (21.1%) subsequently developed glaucoma or treatment-requiring OHT. Mean (standard deviation [SD]; range) age was 42.5 (17.4; 5–80) years, and 58 patients (62.4%) were female. Mean (SD) uveitis duration was 5.2 (4.3) years, and mean (SD) interval from uveitis diagnosis to glaucoma/OHT diagnosis was 2.1 (2.6) years. Panuveitis was the most frequent anatomical category (33.3%), followed by anterior (31.2%), intermediate (29.0%), and posterior uveitis (6.5%). Chronic uveitis was present in 76.3%, infectious uveitis in 39.8%. Herpes-family infection accounted for 14.0% of the cohort, Fuchs heterochromic iridocyclitis for 16.1%. Topical and systemic corticosteroid exposure was documented in 94.6% and 50.5%, respectively. The anterior chamber angle was open in 82.8% and closed in 17.2%. Medical, laser, and surgical treatment were documented in 95.7%, 15.1%, and 10.8%, respectively.

Conclusions: In this tertiary referral cohort, approximately one in five patients with uveitis subsequently developed glaucoma or treatment-requiring OHT. The affected population was clinically heterogeneous, with chronic and panuveitic disease, infectious etiologies, Fuchs heterochromic iridocyclitis, corticosteroid exposure, and predominantly open-angle findings. These findings support continued IOP surveillance throughout uveitis, with attention to inflammatory activity, corticosteroid exposure, angle status, and glaucomatous changes. Given the retrospective design and lack of a comparator group, these characteristics cannot be considered independent risk factors. Prospective multicenter studies are needed to clarify risk factors, clinical course, and treatment outcomes.

KEYWORDS

uveitis, glaucoma, ocular hypertension, intraocular pressure, retrospective studies, tertiary referral hospital

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INTRODUCTION

Uveitis is a heterogeneous group of intraocular inflammatory disorders that may involve different anatomical compartments and follow acute, recurrent, or chronic courses. Uveitis represents an important ocular inflammatory disease with a substantial epidemiological burden [1]. Among its ocular complications, intraocular pressure (IOP) elevation and secondary glaucoma are important because they may result in optic nerve damage and visual impairment [2, 3]. Reported frequencies vary considerably between studies, reflecting differences in uveitic populations, disease characteristics, referral settings, and diagnostic definitions [2, 4, 5].

The mechanisms underlying IOP elevation in uveitis are multifactorial. Inflammatory cells, proteins, and mediators may interfere with trabecular outflow, while trabeculitis can directly impair trabecular function [3, 6]. Structural changes, including peripheral or posterior synechiae and pupillary block, may further compromise aqueous outflow [3, 4]. Corticosteroid therapy represents an additional mechanism and may increase IOP in susceptible patients [2, 3]. These mechanisms may coexist and vary by type and stage of uveitis [2, 3].

The terminology used for uveitis-associated IOP elevation also varies among studies. Ocular hypertension (OHT) is generally distinguished from glaucoma when elevated IOP occurs without documented glaucomatous optic nerve or visual-field damage, whereas uveitic glaucoma implies glaucomatous structural or functional damage [3, 7]. However, some retrospective studies have used elevated IOP or glaucomatous optic nerve damage requiring medical and/or surgical treatment as the basis for defining glaucoma [4]. In other cohorts, OHT was specifically evaluated independently of glaucomatous optic-disc or visual-field changes [2]. Consequently, reported frequencies are not always directly comparable. For example, secondary glaucoma was identified in 19.7% of patients in a large Japanese series [8], whereas secondary glaucoma or OHT was reported in 8.8% of 1997 consecutive new uveitis patients at a tertiary referral center [5].

The clinical characteristics of uveitis also appear to influence the occurrence and mechanism of IOP elevation. Chronic inflammation has been associated with a higher frequency of raised IOP than acute disease, while active inflammation showed a marginal association with raised IOP; increasing age, disease duration, and corticosteroid exposure were significantly correlated with raised IOP on univariate correlation analysis [9]. In a tertiary uveitis cohort, OHT was present in 21% of eyes, in association with older age, extensive peripheral anterior synechiae, and steroid use [10]. Anatomical and etiological patterns may also be relevant. Takahashi et al. reported particularly high frequencies of secondary glaucoma in specific uveitic entities, including herpetic anterior uveitis, and most affected eyes had active anterior inflammation at the time of pressure elevation [8]. In a retrospective study of viral keratouveitis, elevated IOP was observed in 44% of eyes and glaucomatous damage in 20% [11]. Fuchs heterochromic iridocyclitis is another recognized uveitic entity associated with secondary glaucoma [12].

Findings from specialized uveitis clinics further illustrate the importance of the anterior chamber angle and treatment exposure. A study from a tertiary uveitis clinic found that elevated IOP was associated with angle closure, inflammatory activity, and route of corticosteroid administration [7]. In contrast, another cohort showed that most cases of OHT had an open angle and that the relative contribution of inflammation and corticosteroid exposure differed between granulomatous and non-granulomatous uveitis [2]. These observations support consideration of inflammatory activity, anatomical involvement, underlying etiology, angle status, and treatment exposure when describing uveitis-associated IOP elevation.

Despite these observations, clinical profiles of patients with uveitis who subsequently develop glaucoma or treatment-requiring OHT remain heterogeneous across referral populations [4, 5, 9, 10]. The present study therefore aimed to describe the demographic and clinical characteristics of patients with uveitis who subsequently developed glaucoma or treatment-requiring OHT in a tertiary referral uveitis clinic in northwestern Iran.

METHODS

This retrospective descriptive medical-record review was conducted at the Uveitis Clinic of Nikookari Eye Hospital, Tabriz University of Medical Sciences, a tertiary ophthalmic referral center in northwestern Iran. Data were collected over a one-year period, between March 2018 and March 2019; the medical records of patients with uveitis who had been diagnosed with or managed at the clinic during the preceding 10 years were reviewed to identify patients who subsequently developed glaucoma or treatment-requiring OHT. Potential cases were initially identified through a diagnostic-code search of the medical records and were subsequently assessed by detailed review of the available clinical records. The study was approved by the Ethics Committee of Tabriz University of Medical Sciences. Because the study involved retrospective review of information already documented in patients' medical records and did not involve additional clinical intervention, the requirement for informed consent was waived. Patient confidentiality was maintained throughout the study, and the extracted clinical information was handled without disclosure of personal identifiers.

Among 441 patients with uveitis diagnosed or managed at the clinic during the preceding 10 years, 93 developed glaucoma or treatment-requiring OHT after uveitis onset and fulfilled the study's eligibility criteria. Patients were eligible if they had a documented diagnosis of uveitis and, subsequent to onset, met either of two predefined criteria. The first criterion was an IOP > 21 mmHg documented on at least two occasions separated by at least one week and requiring intervention for IOP control [8, 13]. The ≥ 1-week interval was a prespecified criterion of the present study. The second criterion was the presence of glaucomatous findings established on the basis of the available clinical assessment, including tonometry, optic

nerve head examination, optic nerve function assessment, and visual-field examination [13]. The two criteria were applied according to an "OR" rule. Hence the study population included both patients with documented glaucomatous optic neuropathy or visual-field abnormalities and patients with repeated IOP elevation requiring treatment in the absence of documented optic nerve or visual-field changes. Patients were excluded if IOP elevation > 21 mmHg had been documented before the onset of uveitis, if they had a history of glaucoma or glaucomatous findings predating uveitis [13], and when the available documentation was insufficient to establish the temporal relationship between onset of uveitis and subsequent development of glaucoma or treatment-requiring OHT. Because this was a descriptive retrospective study, missing information for individual descriptive variables did not by itself constitute an exclusion criterion.

The medical records were reviewed by an ophthalmologist, with information extracted from the clinical documentation available across multiple visits and encounters during the 10-year period. The recorded clinical history was reviewed to establish the documented onset of uveitis and the interval between uveitis onset and first documented occurrence of glaucoma or treatment-requiring OHT. Demographic variables included age and sex. The recorded characteristics of uveitis were anatomical location, clinical course, inflammatory pattern, etiology, and underlying classification. Uveitis was categorized anatomically as anterior, intermediate, posterior, or panuveitis, and the clinical course was categorized as acute or chronic according to established uveitis classification criteria [4, 7, 13–15]. Inflammatory pattern was classified as granulomatous or non-granulomatous, etiology as infectious or non-infectious, and uveitis as idiopathic or non-idiopathic according to the etiology documented in the medical record.

The available ophthalmic examination findings were reviewed for each patient. Distance visual-acuity measurements obtained during routine clinical care using a Snellen chart (auto chart projector CP 670; Nidek Co., Ltd, Gamagori, Japan) at 6 m were reviewed. When visual acuity was too poor for Snellen chart assessment, counting fingers or light perception was recorded in the clinical records. Anterior-segment examination findings obtained during routine clinical care using a slit-lamp biomicroscope (Haag-Streit BM 900 slit lamp, Haag-Streit Diagnostics, Bern, Switzerland) were reviewed, as were IOP measurements obtained during routine clinical care using a Goldmann applanation tonometer (Goldmann, AT 900 C/M; Haag-Streit, Bern, Switzerland). Particular attention was given to the temporal relationship between documented IOP elevation and onset of uveitis. Repeated IOP measurements were reviewed when available, with the predefined threshold of > 21 mmHg used for study eligibility.

Posterior-segment examination findings obtained during routine clinical care by slit-lamp biomicroscopy using a +78-diopter lens (VOLK Optical, Mentor, OH, USA) were reviewed, as was documentation of optic nerve head and optic nerve function when relevant to the diagnosis of glaucoma. Cup-to-disc ratio was considered when documented in the medical records of patients with glaucoma or OHT. Findings relevant to glaucomatous optic nerve involvement and visual-field abnormalities were considered when determining whether the clinical record supported the diagnosis of glaucomatous disease. When standard automated perimetry (SAP) had been performed as part of routine clinical care, the visual-field findings obtained using a Humphrey Field Analyzer (HFA; Carl Zeiss Meditec Inc., Dublin, CA, USA) were reviewed from the medical records. No additional visual-field examination was performed specifically for purposes of this study.

Additional reviews were conducted for gonioscopic findings obtained using a Goldmann three-mirror contact lens (Volk Optical, Mentor, OH, USA) when gonioscopy had been performed as part of routine clinical care; for documented anterior chamber angle status, which was categorized as open or closed; and for other clinically relevant anterior segment findings and lens status. No additional gonioscopic examination was performed specifically for this retrospective study.

Treatment-related reviews included treatment history, to characterize the management of both uveitis and elevated IOP; documented modality of glaucoma treatment, including medical and surgical treatment when applicable; and treatment directed at the underlying uveitis. Treatment information was considered a descriptive clinical variable rather than an outcome measure, and no attempt was made to compare treatment efficacy or establish causal relationships between individual therapies and IOP outcomes.

Data were entered and analyzed using SPSS Statistics for Windows, version 18.0 (SPSS Inc., Chicago, Ill., USA). The statistical analysis was entirely descriptive. Categorical variables were summarized as frequencies and percentages, while continuous variables were summarized using appropriate descriptive statistics. For patient-level variables, percentages were calculated using the 93 patients in the final study population as denominator, with each patient contributing once to each patient-level variable. No inferential statistical tests, hypothesis testing, regression analyses, or comparative statistical models were performed, as the objective of the study was to describe the demographic, clinical, anatomical, etiological, and treatment characteristics of patients with uveitis who subsequently developed glaucoma or treatment-requiring OHT in a tertiary referral setting.

RESULTS

Among 441 patients with uveitis diagnosed or managed at the clinic during the preceding 10 years, 93 (21.1%) subsequently developed glaucoma or treatment-requiring OHT and met the predefined eligibility criteria. These 93 patients constituted the final study population.

Table 1. Demographic and clinical characteristics of the study population (n = 93)

Variable	Values
Age (y), Mean ± SD (Range), Median (IQR)	42.5 ± 17.4 (5–80), 46.5 (31.5–55.0)
Sex (Male / Female), n (%)	35 (37.6) / 58 (62.4)
Duration of uveitis (y), Mean ± SD	5.2 ± 4.3
Interval from uveitis diagnosis to glaucoma/OHT diagnosis (y), Mean ± SD	2.1 ± 2.6
Systemic disease (Present / Not documented), n (%)	33 (35.5) / 60 (64.5)
Corticosteroid exposure (Topical / Systemic), n (%)	88 (94.6) / 47 (50.5)
Anterior chamber angle (Open / Closed), n (%)	77 (82.8) / 16 (17.2)
Lens status (Phakic / Pseudophakic / Aphakic), n (%)	34 (36.6) / 37 (39.8) /22 (23.7)
Anatomical classification of uveitis, n (%)	
Anterior	29 (31.2)
Intermediate	27 (29.0)
Posterior	6 (6.5)
Panuveitis	31 (33.3)
Clinical course, n (%)	
Acute	22 (23.7)
Chronic	71 (76.3)
Inflammatory pattern, n (%)	
Granulomatous	29 (31.2)
Non-granulomatous	64 (68.8)
Etiological classification, n (%)	
Infectious	37 (39.8)
Non-infectious	56 (60.2)
Underlying etiology, n (%)	
Idiopathic	26 (28.0)
Non-idiopathic	67 (72.0)
Glaucoma/OHT treatment, n (%)	
Medical treatment	89 (95.7)
Laser treatment	14 (15.1)
Surgical treatment	10 (10.8)

Abbreviations: y, years; SD, standard deviation; IQR, interquartile range; n, number; %, percentage; OHT, ocular hypertension.

Mean (standard deviation [SD]; range) age of the patients was 42.5 years (17.4; 5–80), median age was 46.5 years (interquartile range, 31.5–55.0). Of the 93 patients, 58 (62.4%) were female and 35 (37.6%) male. Mean (SD) duration of uveitis was 5.2 (4.3) years, and the mean interval between diagnosis of uveitis and subsequent diagnosis of glaucoma or treatment-requiring OHT was 2.1 (2.6) years (Table 1).

Panuveitis was the most frequent anatomical category, occurring in 31 patients (33.3%), followed by anterior uveitis in 29 (31.2%), intermediate uveitis in 27 (29.0%), and posterior uveitis in 6 (6.5%). Chronic uveitis was present in 71 patients (76.3%), whereas 22 (23.7%) had acute uveitis. Twenty-nine patients (31.2%) had granulomatous and 64 (68.8%) non-granulomatous uveitis. Uveitis was classified as infectious in 37 patients (39.8%) and non-infectious in 56 (60.2%). Among those with infectious uveitis, a herpes-family virus was identified as the causative agent in 13 patients (35.1% of infectious cases; 14.0% of the total study population). Among patients with non-infectious uveitis, Fuchs heterochromic iridocyclitis was documented in 15 (26.8% of non-infectious cases; 16.1% of the total study population). Regarding underlying etiology, 26 patients (28.0%) had idiopathic uveitis and 67 (72.0%) non-idiopathic uveitis (Table 1).

Systemic disease was documented in 35.5% of patients, while 64.5% had no documentation. Regarding corticosteroid exposure, 94.6% of patients had received topical corticosteroids and 50.5% had received systemic corticosteroids (Table 1).

The anterior chamber angle was documented as open in 77 patients (82.8%) and closed in 16 (17.2%). Lens status was pseudophakic in 37 patients (39.8%), phakic in 34 (36.6%), and aphakic in 22 (23.7%). Treatment categories were not mutually exclusive, and individual patients could have received more than one treatment modality. Medical treatment was used in 89 patients (95.7%), laser treatment in 14 (15.1%), and surgical treatment in 10 (10.8%) (Table 1).

DISCUSSION

In this retrospective study of patients with uveitis managed at a tertiary referral center in northwestern Iran, 93 of 441 patients (21.1%) subsequently developed glaucoma or treatment-requiring OHT. The affected population had a mean (SD) age of 42.5 (17.4) years, was predominantly female (62.4%), and the mean (SD) interval between the diagnosis of uveitis and the subsequent diagnosis of glaucoma or treatment-requiring OHT was 2.1 (2.6) years. Panuveitis was the most frequent anatomical category (33.3%), followed by anterior uveitis (31.2%), while chronic disease predominated (76.3%).

Approximately one-third of patients had granulomatous uveitis, 39.8% had infectious uveitis, and herpes-family infection accounted for 14.0% of the total cohort. Fuchs heterochromic iridocyclitis was documented in 16.1% of patients. Corticosteroid exposure was common, with topical and systemic corticosteroids used in 94.6% and 50.5% of patients, respectively. The anterior chamber angle was open in 82.8% and closed in 17.2%. Most patients received medical treatment for elevated IOP (95.7%), whereas laser and surgical treatment were documented in 15.1% and 10.8%, respectively. Collectively, these findings describe a clinically diverse group in whom uveitis-associated pressure elevation occurred across different anatomical, inflammatory, infectious, and treatment-related contexts.

The female predominance in our cohort should be interpreted in the context of conflicting findings regarding sex and uveitis-associated OHT. Kanda et al. found a significantly higher prevalence of OHT in male than in female uveitic eyes (31.0% vs 21.6%) but noted that, contrastingly, European studies had not demonstrated a sex difference in prevalence of uveitis-associated OHT or secondary glaucoma [2]. The female predominance observed in our cohort (62.4%) therefore differs from the sex distribution they reported and may reflect differences in the underlying uveitis population, referral patterns, disease etiologies, or study design. Because our cohort consisted exclusively of patients who developed glaucoma or treatment-requiring OHT and we did not include a comparator group of uveitis patients without pressure elevation, we cannot determine whether sex was associated with these outcomes.

The overall frequency of 21.1% in the present study is broadly comparable with several published tertiary center cohorts, although direct comparisons should be made cautiously because definitions, denominators, and case ascertainment methods differ. More recently, Barış et al. reported glaucoma or elevated IOP in 440 of 2,176 patients with uveitis (20.2%) treated at a tertiary center [16]. Heinz et al. identified secondary glaucoma or OHT in 8.8% of 1,997 consecutive new uveitis patients at a tertiary referral center [5], whereas Takahashi et al. reported secondary glaucoma in 19.7% of patients with uveitis [8]. Mahajan et al. reported OHT in 42 of 200 patients (21%) in a non-consecutive tertiary center cohort [10]. More recently, Felfeli et al. reported OHT or uveitic glaucoma in 23.6% of 914 adults with noninfectious uveitis treated at tertiary uveitis clinics, further demonstrating that a frequency of around one-fifth to one-quarter of patients can occur in specialized referral populations [14]. Conversely, large database-based studies may produce different estimates; a 2025 US study reported that 15.5% of patients with uveitis developed glaucoma during a 10-year period [17]. These differences emphasize that the 21.1% observed in our study should be interpreted in the context of the study population and case definition rather than regarded as a universal prevalence estimate. Our eligibility criteria deliberately included both patients with documented glaucomatous structural or functional abnormalities and those with repeated IOP elevation requiring intervention, thereby encompassing a clinically relevant spectrum from treatment-requiring OHT to established glaucoma.

The time interval between uveitis diagnosis and subsequent glaucoma or treatment-requiring OHT in our cohort was 2.1 (2.6) years. This observation is clinically relevant because IOP elevation in uveitis may develop at different stages of the inflammatory disease rather than being restricted to the initial episode. Neri et al. demonstrated that the incidence of glaucoma increased with duration of uveitis, particularly among patients with chronic disease [4], while more recent evidence indicates that the risk of glaucoma may be especially pronounced during the first year following diagnosis in some noninfectious uveitis populations [18]. The 2026 multi-institutional cohort of patients with idiopathic noninfectious uveitis reported the highest relative glaucoma risk during the first year after diagnosis, although risk remained elevated thereafter [18]. These findings, together with the mean interval observed in our cohort, support continued IOP surveillance throughout the course of uveitis rather than limiting pressure monitoring to periods of overt inflammatory activity. The relatively wide standard deviation in our interval also suggests substantial heterogeneity in the timing of pressure elevation, consistent with the multifactorial nature of uveitic glaucoma.

The predominance of chronic uveitis in our cohort is consistent with previous evidence linking prolonged inflammatory disease with increased IOP elevation. Herbert et al. found raised IOP significantly more frequently in chronic than acute uveitis—46.1% versus 26.0% of eyes—while treatment-requiring IOP elevation occurred in 33.8% versus 15.1%, respectively. Active inflammation showed a marginal association with raised IOP on χ^2 testing ($P = 0.031$), whereas steroid use ($P = 0.008$), increasing age ($P = 0.022$), and years since diagnosis ($P = 0.006$) were significantly correlated with raised IOP on univariate analysis [9]. However, steroid use ($P = 0.059$) and years since diagnosis ($P = 0.213$) were not significantly associated with raised IOP in the multivariate analysis. Similarly, the Auckland cohort demonstrated that chronic inflammation was an independent risk factor for glaucoma, together with older age, higher presenting IOP, and cystoid macular edema [19]. The high proportion of chronic disease in our population may therefore reflect the cumulative effects of prolonged intraocular inflammation, repeated episodes of pressure elevation, and sustained exposure to IOP-raising treatments. Nevertheless, because our study included only patients who developed glaucoma or treatment-requiring OHT and did not include a contemporaneous comparison group of uveitis patients without pressure elevation, the present data cannot establish chronicity as an independent predictor within our population. Rather, the finding is consistent with the broader literature and supports the biological plausibility of chronic inflammatory exposure as an important contributor.

One of the most notable findings was the anatomical distribution of uveitis. Panuveitis was the most frequent subtype in our cohort, accounting for 33.3% of patients—slightly exceeding anterior uveitis (at 31.2%). This pattern differs from the anatomical distribution generally encountered in broader uveitis populations, in which anterior uveitis is frequently the

predominant anatomical category [17, 19]. The predominance of panuveitis among patients who developed glaucoma or treatment-requiring OHT may therefore reflect the characteristics of a tertiary referral population, in which patients with more extensive or complex intraocular inflammation are disproportionately represented. It may also reflect the cumulative inflammatory burden associated with involvement of multiple ocular compartments. Contemporary evidence provides some support for this interpretation. In a large multi-institutional cohort of idiopathic noninfectious uveitis, panuveitis had the highest hazard ratio for glaucoma versus matched nonuveitis controls (hazard ratio [HR], 50.49; 95% confidence interval [CI], 22.36–113.00) among the anatomical subtypes examined [18]. Similarly, the Auckland cohort found a 5-year cumulative glaucoma risk of 8.7% for panuveitis, compared with 6.2% for anterior uveitis, 5.4% for intermediate uveitis, and 1.6% for posterior uveitis [19]. Felfeli et al. also found that panuveitis was associated with poorer final visual acuity among patients with noninfectious uveitis complicated by OHT or uveitic glaucoma [14].

However, the association between anatomical subtype and glaucoma is not uniform across studies. Neri et al. did not evidence a significant difference in glaucoma incidence among anterior, intermediate, posterior, or panuveitis [4]. Furthermore, the large 2025 US database study reported the highest glaucoma frequency among patients with combined anterior and intermediate uveitis, followed by anterior uveitis, whereas panuveitis accounted for a smaller proportion of affected patients [17]. These apparently divergent findings are important because they indicate that the high proportion of panuveitis in our cohort should not be interpreted as evidence that panuveitis universally confers the highest risk of glaucoma. Differences in referral patterns, underlying etiologies, disease severity, follow-up duration, corticosteroid exposure, and definitions of glaucoma or OHT may all contribute to variation among cohorts. Our finding is therefore best regarded as a distinctive feature of the affected population rather than as evidence of an independently increased risk. Future prospective studies comparing patients with different anatomical subtypes within the same underlying uveitis population would be better suited to determine whether panuveitis independently predicts the development of pressure-related complications.

The etiological composition of our cohort provides another important perspective. Infectious uveitis accounted for 39.8% of patients, and herpes-family infection was documented in 14.0% of the entire study population. This proportion is clinically relevant because viral anterior uveitis is particularly associated with recurrent or marked IOP elevation [3, 20, 21]. Takahashi et al. reported that secondary glaucoma occurred in 30.4% of eyes with herpetic anterior uveitis in their Japanese cohort, with most eyes developing secondary glaucoma while active anterior inflammation was present at the time of IOP elevation [8]. In the Auckland cohort, viral uveitis had the highest 5-year cumulative glaucoma risk among the etiological categories examined, reaching 15.1% [19]. More detailed studies have also demonstrated important differences between herpes simplex virus, varicella-zoster virus, and cytomegalovirus. A multicenter study of 259 herpetic anterior uveitis patients found that elevated IOP and glaucoma surgery were significantly more frequent in CMV-associated disease than in HSV- or VZV-associated anterior uveitis [20]. A 270-patient study of virus-associated anterior uveitis found glaucomatous damage in 19% of patients and glaucoma surgery in 10%, while evidencing clinically meaningful differences among viral entities [21]. These findings are relevant to our cohort because grouping herpes-family infections together may conceal clinically important differences in the propensity for recurrent inflammation, pressure elevation, and surgical treatment. CMV-associated disease may be associated with more sustained or recurrent IOP elevation than HSV- or VZV-associated disease [20, 21].

The relatively high proportion of Fuchs heterochromic iridocyclitis in our cohort is another finding that warrants specific consideration. Fuchs heterochromic iridocyclitis was documented in 15 patients, representing 16.1% of the total study population. This proportion is notable because Fuchs heterochromic iridocyclitis may be relatively prominent in some cohorts of uveitic glaucoma compared with its frequency among general uveitis populations [13, 19]. In a cohort of 88 patients with Fuchs heterochromic iridocyclitis, Toniolo et al. found OHT or glaucoma in 26% at presentation and estimated that 39% of patients would develop OHT or glaucoma by four years; increasing age and cataracts at presentation were associated with greater risk [22]. In a Turkish tertiary-center cohort of uveitic glaucoma, Fuchs accounted for approximately 14% of cases—a proportion close to the 16.1% observed in our study [13]. These observations suggest that the relatively high representation of Fuchs heterochromic iridocyclitis in our cohort may have contributed to the overall burden of glaucoma and treatment-requiring OHT. However, because the present study was descriptive and did not include a comparison group, it cannot establish that Fuchs heterochromic iridocyclitis independently increased the risk of pressure elevation in our population.

The high frequency of corticosteroid exposure is also important when interpreting the mechanisms underlying IOP elevation. Nearly all patients in our cohort had received topical corticosteroids (94.6%), and approximately half had received systemic corticosteroids (50.5%). Corticosteroid exposure is well recognized as an important contributor to elevated IOP in patients with uveitis, although its contribution is difficult to separate from that of the underlying inflammatory disease. Herbert et al. [9] identified steroid use as a significant correlate of raised IOP, while Mahajan et al. [10] identified steroid use as a significant risk factor for uveitis-related OHT. In a large multicenter cohort of adults with noninfectious uveitis, current topical corticosteroid use, current prednisone use at doses > 7.5 mg/day, and recent periocular corticosteroid exposure were independently associated with incident OHT [23]. Subsequently, Liu et al. reported higher hazard ratios for glaucoma among patients with noninfectious uveitis who used systemic corticosteroids (HR, 10.19; 95% CI, 6.34–16.37) or ophthalmic corticosteroids (HR, 5.67; 95% CI, 3.88–8.30)—estimates that represent comparisons of uveitis patients with matched

nonuveitis controls within the respective corticosteroid-use strata [18]. The authors noted that the higher estimate associated with systemic corticosteroid use likely reflected greater underlying disease severity [18]. Mechanistically, inflammatory cells, inflammatory proteins, changes in trabecular outflow, and corticosteroid-induced alterations of trabecular function may operate simultaneously [6]. Consequently, the very high corticosteroid exposure observed in our study should not be interpreted as demonstrating steroid-induced glaucoma in most patients. Rather, it illustrates the therapeutic dilemma inherent in uveitis management: corticosteroids are necessary to suppress potentially vision-threatening inflammation, yet exposure may simultaneously increase the risk of pressure elevation in susceptible eyes.

The predominantly open anterior chamber angle observed in our cohort is consistent with previous evidence that open-angle mechanisms account for a substantial proportion of uveitis-associated pressure elevation. In our study, 82.8% of patients had an open angle and 17.2% had a closed angle. Kanda et al. reported that 92% of eyes with uveitis-associated OHT had an open angle [2], while Takahashi et al. found that only 7.5% of eyes with secondary glaucoma had peripheral anterior synechiae extending over more than 180° of the trabecular meshwork [8]. However, angle closure is also an important mechanism in selected patients. In a specialized uveitis clinic, Shaaban et al. found that 36.4% of eyes with elevated IOP had closed angles and reported a significant association between angle closure and raised IOP ($P < 0.001$) [7]. Similarly, Mahajan et al. reported peripheral anterior synechiae extending over more than 180° in 26.2% of eyes with uveitic OHT [10]. Thus, although open-angle mechanisms predominated in our cohort, these findings from other uveitis populations indicate that angle closure and synechial disease may contribute substantially to IOP elevation in selected patients. Inflammation-related trabecular dysfunction, inflammatory cells and proteins within the aqueous, trabeculitis, and corticosteroid-related impairment of trabecular outflow may contribute to open-angle pressure elevation, whereas inflammatory structural changes, peripheral or posterior synechiae, pupillary block, and alterations in the iris-lens diaphragm may contribute to angle closure. The retrospective nature of our study and the limited documentation of gonioscopic findings, however, prevent more detailed attribution of the mechanism of angle closure in individual patients.

The distribution of lens status in our cohort also deserves cautious interpretation. Pseudophakia was present in 39.8%, phakia in 36.6%, and aphakia in 23.7% of patients. These findings probably reflect the complex interaction between chronic uveitis, cataract formation, previous intraocular surgery, and severity and duration of disease rather than a simple relationship between lens status and glaucoma. Cataracts at presentation have been associated with subsequent development of glaucoma in Fuchs heterochromic iridocyclitis [22], while cataract extraction has also been proposed as one of several factors associated with IOP elevation in Fuchs uveitis syndrome [24]. However, not having a comparator group of uveitis patients without glaucoma or treatment-requiring OHT means the present data cannot determine whether pseudophakia or aphakia was associated with increased or decreased risk. Lens status is therefore best interpreted as part of the clinical phenotype of this tertiary referral population rather than as an independent risk factor.

The treatment profile provides further insight into the clinical spectrum represented by our cohort. Medical treatment was documented in 95.7% of patients, whereas laser and surgical treatment were documented in 15.1% and 10.8%, respectively. The relatively low proportion receiving surgery compared with some tertiary referral series should be interpreted carefully. First, the treatment categories in our study were not mutually exclusive; therefore, the presence of medical treatment in 95.7% of patients does not indicate that all of these patients were successfully controlled medically, and the 10.8% surgical proportion does not represent a conventional rate of medical/treatment failure. Second, our population included patients with treatment-requiring OHT in addition to established glaucomatous damage. This distinction is important because patients with established uveitic glaucoma are more likely to require surgery than patients with OHT. Felfeli et al. demonstrated that uveitic glaucoma, as opposed to OHT, was strongly associated with the need for glaucoma surgery [14].

The surgical frequency in our cohort is nevertheless within the range reported by some clinical studies. Mahajan et al. reported trabeculectomy in 11.9% of eyes with uveitis-related OHT after six months of follow-up; however, this eye-level estimate is not directly comparable with the 10.8% patient-level surgical treatment frequency observed in our cohort [10]. In contrast, Heinz et al. reported a substantially higher need for surgery, with glaucoma surgery required in 35% of adults and 59% of children with secondary uveitic glaucoma or OHT [5]. Such differences may reflect differences in disease severity, age distribution, referral characteristics, duration of follow-up, and proportion of patients with established glaucomatous damage. Surgical management of uveitic glaucoma is also intrinsically challenging because postoperative inflammation, fibrosis, scarring, and hypotony may influence outcomes. Comparative studies have shown that trabeculectomy and glaucoma drainage devices can both achieve substantial IOP reduction, but their complication and failure profiles differ [25]. In virus-associated anterior uveitis, approximately 10% of patients in one large series required glaucoma surgery, while some required additional procedures after failure of the initial intervention [21]. Similarly, comparative studies of Fuchs-associated glaucoma have reported conflicting findings on the relative effectiveness of trabeculectomy and Ahmed glaucoma valve implantation. Esfandiari et al. reported a higher cumulative success rate with Ahmed valve implantation than with trabeculectomy (85.7% vs 41.7%), with mean IOP decreasing from 24.0 to 17.1 mmHg after Ahmed valve implantation but only from 23.4 to 21.6 mmHg after trabeculectomy [12]. In contrast, Nilforushan et al. reported substantially higher cumulative success with trabeculectomy than with Ahmed valve implantation at 36 months (76% vs 9% according to criterion

A) [24]. These discrepant findings may reflect differences in study populations, surgical approaches, outcome definitions, and follow-up duration, and support individualized surgical decision-making based on type of uveitis, degree of inflammation, angle status, severity of glaucomatous damage, previous surgery, and response to medical therapy.

The demographic characteristics of our cohort also merit contextual interpretation. The mean age of 42.5 years is similar to the median age reported in the Auckland uveitis/scleritis cohort, although the populations are not directly comparable [19]. The demographic composition of our cohort should be interpreted in the context of its tertiary referral setting and the underlying heterogeneity of uveitis. Similarly, the presence of systemic disease in 35.5% and idiopathic disease in 28.0% of patients reflects the complexity of tertiary uveitis populations but does not establish that systemic disease or idiopathic classification independently influence glaucoma risk. The proportion of idiopathic disease in our cohort (28.0%) was similar to that reported in the Auckland uveitis/scleritis cohort (30.9%) [19] but lower than the proportions reported by Neri et al. in chronic (51.9%) and acute (61.1%) uveitis [4] and by Felfeli et al. in a tertiary noninfectious uveitis cohort with OHT or uveitic glaucoma (61.6%) [14]. These differences may reflect variation in diagnostic evaluation, referral characteristics, study populations, and classification methods. Importantly, the high proportions of infectious disease, Fuchs heterochromic iridocyclitis, chronic disease, and panuveitis indicate that the present cohort represents a selected clinical spectrum rather than the epidemiology of uveitis in the general population.

From a clinical perspective, the findings emphasize that IOP surveillance should remain an integral component of long-term uveitis care. The coexistence of chronic inflammation, corticosteroid exposure, infectious etiologies, Fuchs heterochromic iridocyclitis, and predominantly open-angle pressure elevation means that clinically significant IOP elevation may occur even in the absence of obvious angle closure [2, 12]. Other evidence supports continued IOP surveillance throughout the course of uveitis [18, 23]. In the Systemic Immunosuppressive Therapy for Eye Diseases (SITE) cohort, inflammatory activity, previous OHT in the fellow eye, peripheral anterior synechiae, and corticosteroid exposure were associated with an increased risk of OHT [23]. In practical terms, the present findings support repeated IOP assessment during both active and quiescent phases, careful review of corticosteroid exposure, attention to angle status, and early recognition of glaucomatous structural or functional changes. Particular attention may be warranted in patients with chronic or panuveitic disease and in those with viral or Fuchs-associated uveitis. Importantly, the need to monitor IOP should not lead to inappropriate reduction of anti-inflammatory therapy when corticosteroids remain clinically necessary; rather, pressure management and inflammatory control should be considered complementary components of uveitis care.

This study has several strengths. It was conducted in a dedicated tertiary uveitis clinic and included a 10-year clinical population, allowing identification of patients with subsequent development of glaucoma or treatment-requiring OHT rather than limiting analysis to a single episode of elevated IOP. The study also used a predefined temporal relationship between uveitis onset and subsequent IOP elevation or glaucomatous findings, which helped distinguish uveitis-associated pressure elevation from abnormalities documented before uveitis. The inclusion of both treatment-requiring OHT and established glaucoma allowed characterization of a clinically relevant spectrum of pressure-related complications. Furthermore, the study incorporated anatomical, inflammatory, infectious, etiological, systemic, corticosteroid-exposure, angle, lens, and treatment variables, providing a broad clinical profile. Nevertheless, several limitations should be acknowledged. The retrospective design inevitably depended on the completeness and consistency of historical medical documentation. The study was conducted at a single tertiary referral center, and referral bias may have enriched the cohort for chronic, complex, panuveitic, infectious, or treatment-intensive disease, limiting generalizability to community-based uveitis populations. Because there was no control group of uveitis patients who did not develop glaucoma or treatment-requiring OHT, the observed characteristics cannot be interpreted as independent risk factors. The descriptive design also precluded multivariable assessment of the relative contribution of age, chronicity, anatomical subtype, etiology, corticosteroid exposure, angle status, and lens status. The broad treatment definition and non-mutually exclusive treatment categories prevented assessment of treatment success, treatment failure, or comparative effectiveness. Finally, the retrospective documentation of gonioscopy and visual-field findings limits the ability to characterize the precise mechanism and severity of glaucomatous disease in every patient. An additional limitation is that the retrospective records did not permit reliable separation of patients with established glaucomatous optic neuropathy from those with treatment-requiring OHT; therefore, the frequency, clinical characteristics, and outcomes of these two entities could not be evaluated separately. Future studies should distinguish OHT from established uveitic glaucoma using standardized structural and functional diagnostic criteria and report their clinical courses and outcomes separately. Future studies should therefore use prospective, multicenter designs with standardized definitions of OHT and glaucoma, standardized inflammatory and anatomical classifications, serial IOP and visual-field assessments, optic nerve imaging where appropriate, detailed documentation of corticosteroid exposure and inflammatory activity, and longer follow-up. Comparative analyses should specifically evaluate whether panuveitis, Fuchs heterochromic iridocyclitis, viral etiologies, chronic inflammation, and corticosteroid exposure are independently associated with the development of glaucoma after adjustment for potential confounders. Studies of viral uveitis should distinguish CMV, HSV, and VZV, while prospective studies of Fuchs-associated glaucoma could clarify the roles of cataracts, age, inflammation, and surgical timing. These approaches may determine whether this cohort's clinical profile reflects local referral patterns or broader trends in uveitis-associated glaucoma.

CONCLUSIONS

In this tertiary referral cohort, glaucoma or treatment-requiring OHT developed in 21.1% of patients with uveitis. Affected patients were predominantly female and had chronic disease, with panuveitis and anterior uveitis as the most common anatomical patterns. Fuchs heterochromic iridocyclitis and viral etiologies were notable among non-infectious and infectious cases, respectively. Most patients had received topical corticosteroids and treatment was largely medical, although laser and surgical interventions were also used. The predominance of open-angle disease suggests that pressure elevation in this cohort was not primarily attributable to extensive synechial angle closure. However, the descriptive retrospective design does not permit determination of independent risk factors or causal relationships. These findings support careful IOP monitoring and assessment for glaucomatous damage throughout the course of uveitis. Future prospective, multicenter studies with appropriate comparator groups are needed to identify independent predictors and clarify the clinical course and treatment outcomes of uveitis-associated glaucoma and treatment-requiring OHT.

ETHICAL DECLARATIONS

Ethical approval: The study was approved by the Ethics Committee of Tabriz University of Medical Sciences. Because the study involved retrospective review of information already documented in patients' medical records and did not involve additional clinical intervention, the requirement for informed consent was waived. Patient confidentiality was maintained throughout the study, and the extracted clinical information was handled without disclosure of personal identifiers.

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REFERENCES

1. Gritz DC, Wong IG. Incidence and prevalence of uveitis in Northern California; the Northern California Epidemiology of Uveitis Study. Ophthalmology. 2004 Mar;111(3):491-500; discussion 500. doi: 10.1016/j.ophtha.2003.06.014. PMID: 15019324.
2. Kanda T, Shibata M, Taguchi M, Ishikawa S, Harimoto K, Takeuchi M. Prevalence and aetiology of ocular hypertension in acute and chronic uveitis. Br J Ophthalmol. 2014 Jul;98(7):932-6. doi: 10.1136/bjophthalmol-2013-304416. Epub 2014 Mar 28. PMID: 24682184.
3. Moorthy RS, Mermoud A, Baerveldt G, Minckler DS, Lee PP, Rao NA. Glaucoma associated with uveitis. Surv Ophthalmol. 1997 Mar-Apr;41(5):361-94. doi: 10.1016/s0039-6257(97)00006-4. PMID: 9163835.
4. Neri P, Azuara-Blanco A, Forrester JV. Incidence of glaucoma in patients with uveitis. J Glaucoma. 2004 Dec;13(6):461-5. doi: 10.1097/01.ijg.0000146391.77618.d0. PMID: 15534470.
5. Heinz C, Koch JM, Zurek-Imhoff B, Heiligenhaus A. Prevalence of uveitic secondary glaucoma and success of nonsurgical treatment in adults and children in a tertiary referral center. Ocul Immunol Inflamm. 2009 Jul-Aug;17(4):243-8. doi: 10.1080/09273940902913035. PMID: 19657977.
6. Baneke AJ, Lim KS, Stanford M. The Pathogenesis of Raised Intraocular Pressure in Uveitis. Curr Eye Res. 2016;41(2):137-49. doi: 10.3109/02713683.2015.1017650. Epub 2015 May 14. PMID: 25974243.
7. Shaaban AM, Youssef MM, Eltanamly RM, AbdelAziz MS, Kotb MS. The pattern of intraocular pressure elevation in a specialized uveitis clinic. Delta Journal of Ophthalmology. 2018 Jan 1;19(1):46-52. doi:10.4103/DJO.DJO_36_17.
8. Takahashi T, Ohtani S, Miyata K, Miyata N, Shirato S, Mochizuki M. A clinical evaluation of uveitis-associated secondary glaucoma. Jpn J Ophthalmol. 2002 Sep-Oct;46(5):556-62. doi: 10.1016/S0021-5155(02)00549-x. PMID: 12457916.
9. Herbert HM, Viswanathan A, Jackson H, Lightman SL. Risk factors for elevated intraocular pressure in uveitis. J Glaucoma. 2004 Apr;13(2):96-9. doi: 10.1097/00061198-200404000-00003. PMID: 15097253.
10. Mahajan D, Sharma R, Garg SP, Venkatesh P, Sihota R, Dada T. Clinical profile of uveitis-related ocular hypertension. Int Ophthalmol. 2014 Dec;34(6):1221-6. doi: 10.1007/s10792-014-0008-8. Epub 2014 Oct 10. PMID: 25301079.
11. Sudhakar P, Menon M, Ck M, Balasubramaniam A. Glaucoma in Viral Keratouveitis: A Retrospective Review at a Tertiary Eye Hospital. J Curr Glaucoma Pract. 2022 Jan-Apr;16(1):65-70. doi: 10.5005/jp-journals-10078-1359. PMID: 36060041; PMCID: PMC9385385.
12. Esfandiari H, Loewen NA, Hassanpour K, Fatourechi A, Yazdani S, Wang C, Yaseri M, Pakravan M. Fuchs heterochromic iridocyclitis-associated glaucoma: a retrospective comparison of primary Ahmed glaucoma valve implantation and trabeculectomy with mitomycin C. F1000Res. 2018 Jun 22;7:876. doi: 10.12688/f1000research.15244.2. PMID: 30410728; PMCID: PMC6198260.
13. Altan C, Basarir B. Aetiology and clinical characteristics of uveitic glaucoma in Turkish patients. Int Ophthalmol. 2021 Jun;41(6):2225-2234. doi: 10.1007/s10792-021-01783-4. Epub 2021 Mar 17. PMID: 33730317.

14. Felfeli T, Rhee J, Eshtiaghi A, Balas M, Tai F, Kaplan AJ, Christakis PG, Mandelcorn ED, Rubin LA, Bakshi NK, Derzko-Dzulynsky LA. Characteristics of ocular hypertension and uveitic glaucoma among patients with noninfectious uveitis. Can J Ophthalmol. 2024 Dec;59(6):430-438. doi: 10.1016/j.jcjo.2024.02.003. Epub 2024 Feb 29. PMID: 38431271.
15. Jabs DA, Nussenblatt RB, Rosenbaum JT; Standardization of Uveitis Nomenclature (SUN) Working Group. Standardization of uveitis nomenclature for reporting clinical data. Results of the First International Workshop. Am J Ophthalmol. 2005 Sep;140(3):509-16. doi: 10.1016/j.ajo.2005.03.057. PMID: 16196117; PMCID: PMC8935739.
16. Esen Barış M, Ateş H, Güven Yılmaz S. Prevalence and Prognosis of Glaucoma/Elevated Intraocular Pressure in Patients with Uveitis. Turk J Ophthalmol. 2025 Aug 21;55(4):215-220. doi: 10.4274/tjo.galenos.2025.53498. PMID: 40838536; PMCID: PMC12372549.
17. Marshall RF, Lee D, Thorne JE, Kaleem M, Berkenstock MK. Incidence and Prevalence of Glaucoma, Corticosteroid Response, and Ocular Hypertension in Uveitis and its Anatomical Subtypes. Am J Ophthalmol. 2025 Oct;278:13-21. doi: 10.1016/j.ajo.2025.05.046. Epub 2025 Jun 4. PMID: 40480344.
18. Liu DY, Kuo HT, Hsu AY, Lin CJ, Shao YC, Chen HS, Chiang CC, Hsia NY, Lai CT, Wang YH, Wei JC, Tsai YY. Risk of Glaucoma in Patients With Idiopathic Noninfectious Uveitis: A Multi-Institutional Real-World Retrospective Cohort Study. Am J Ophthalmol. 2026 May;285:63-72. doi: 10.1016/j.ajo.2026.01.020. Epub 2026 Jan 25. PMID: 41592690.
19. Niederer RL, Wong ABC, Ma T, Chew S, Sims J. Predictors of glaucoma in patients with uveitis and scleritis. Eye (Lond). 2023 Apr;37(6):1254-1257. doi: 10.1038/s41433-022-02101-7. Epub 2022 May 24. PMID: 35610358; PMCID: PMC10101954.
20. Terada Y, Kaburaki T, Takase H, Goto H, Nakano S, Inoue Y, Maruyama K, Miyata K, Namba K, Sonoda KH, Kaneko Y, Numaga J, Fukushima M, Horiguchi N, Ide M, Ehara F, Miyazaki D, Hasegawa E, Mochizuki M. Distinguishing Features of Anterior Uveitis Caused by Herpes Simplex Virus, Varicella-Zoster Virus, and Cytomegalovirus. Am J Ophthalmol. 2021 Jul;227:191-200. doi: 10.1016/j.ajo.2021.03.020. Epub 2021 Mar 25. PMID: 33773985.
21. Pohlmann D, Pahlitzsch M, Schlickeiser S, Metzner S, Lenglinger M, Bertelmann E, Maier AB, Winterhalter S, Pleyer U. Virus-associated anterior uveitis and secondary glaucoma: Diagnostics, clinical characteristics, and surgical options. PLoS One. 2020 Feb 24;15(2):e0229260. doi: 10.1371/journal.pone.0229260. PMID: 32092116; PMCID: PMC7039515.
22. Toniolo JT, Hall AJ, Smith JG, Levy J, Lim LL. Risk Factors for Glaucoma in a Cohort of Patients with Fuchs Heterochromic Iridocyclitis. Ocul Immunol Inflamm. 2017 Dec;25(6):753-759. doi: 10.1080/09273948.2016.1199712. Epub 2016 Aug 5. PMID: 27494676.
23. Daniel E, Pistilli M, Kothari S, Khachatryan N, Kaçmaz RO, Gangaputra SS, Sen HN, Suhler EB, Thorne JE, Foster CS, Jabs DA, Nussenblatt RB, Rosenbaum JT, Levy-Clarke GA, Bhatt NP, Kempen JH; Systemic Immunosuppressive Therapy for Eye Diseases Research Group. Risk of Ocular Hypertension in Adults with Noninfectious Uveitis. Ophthalmology. 2017 Aug;124(8):1196-1208. doi: 10.1016/j.ophtha.2017.03.041. Epub 2017 Apr 19. PMID: 28433444; PMCID: PMC5522760.
24. Nilforushan N, Yadgari M, Alemzadeh SA. Surgical management of glaucoma in Fuchs uveitis syndrome: Trabeculectomy or Ahmed glaucoma valve. J Curr Ophthalmol. 2018 Dec 28;31(1):24-30. doi: 10.1016/j.joco.2018.10.005. PMID: 30899842; PMCID: PMC6407154.
25. Chow A, Burkemper B, Varma R, Rodger DC, Rao N, Richter GM. Comparison of surgical outcomes of trabeculectomy, Ahmed shunt, and Baerveldt shunt in uveitic glaucoma. J Ophthalmic Inflamm Infect. 2018 Jun 18;8(1):9. doi: 10.1186/s12348-018-0150-y. PMID: 29915970; PMCID: PMC6006003.