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Endophthalmitis: Epidemiology, Causing Agents, Therapy and Visual Outcome with Special Focus on Glaucoma Patients

Endophthalmitis: Ursachen, Erreger, Therapie und Visusverlauf mit Fokus auf Glaukompatienten

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ABSTRACT

Background Endophthalmitis is one of the most serious emergencies in ophthalmology. In order to reduce its prevalence, it is important to have a proper understanding of potential risk. Surgical therapy with targeted, pathogen-specific medication and an intact immune system are fundamental for preserving visual acuity. As it is unclear whether an unfavourable course is more likely in the presence of underlying ocular disease, a comparison was made between glaucoma patients (G) and non-glaucoma patients (NG) in terms of causative factors, pathogens, treatment and visual acuity. Since a potential alteration of the local immune system in glaucoma has been described, it is of interest to determine whether the clinical course of endophthalmitis in glaucoma patients differ from that of non-glaucoma patients.

Patients and Methods A retrospective analysis of 75 eyes (13 G, 62 NG) who underwent treatment and surgery following a diagnosis of endophthalmitis in the Department of Ophthalmology, University of Erlangen-Nuremberg has been evaluated over a period of 5 years. Clinical characteristics, surgical treatment, microbial spectrum and visual acuity in glaucoma and non-glaucoma eyes were investigated.

Results Severe visual impairment (44 %) with inflammation of the anterior chamber (62.7 %), hypopyon (52 %) and reduced (40 %) or absent view (26.7 %) of the fundus were predominantly present at first diagnosis in all patients. Previous eye surgery was observed in a total of 53 %, primarily cataract surgery. Gram-positive cocci were seen as the most common causative agent in both groups, (G: 23.1 %; NG: 38.7 %), whereas other rare pathogens were present only in glaucomatous eyes. Pars plana vitrectomy was performed in 76 % and enucleations in 20 % of all patients, with the latter significantly more common in glaucomatous eyes ($p = 0.01$). A significant postoperative improvement in visual acuity was achieved in non-glaucoma patients ($p < 0.001$); visual acuity was worse in glaucomatous eyes.

Conclusion Although rare, early diagnosis and treatment of endophthalmitis is crucial in terms of prognosis. In the present cohort, worse visual acuity outcomes were obtained in glaucoma patients in comparison to non-glaucoma patients.

ZUSAMMENFASSUNG

Hintergrund Endophthalmitiden stellen einen der schwerwiegendsten Notfälle in der Ophthalmologie dar. Um deren Prävalenz zu minimieren, ist eine möglichst genaue Kenntnis auslösender Faktoren von Bedeutung. Eine chirurgische Therapie mit gezielter, erregerspezifischer Medikation und ein intaktes Immunsystem sind die Basis für den Visuserhalt. Ferner stellt sich die Frage, ob anhand zugrunde liegender Erkrankungen am Auge ein ungünstiger Verlauf prognostiziert werden kann, sodass ein Vergleich zwischen Glaukom- (G) und Nichtglaukompatienten (NG) im Hinblick auf ursächliche Faktoren, Erreger, Therapie und Visusverlauf gezogen wurde. Da bei Glaukompatienten eine potenzielle Alteration des lokalen Immunsystems diskutiert wird, ist von Interesse, ob sich die klinischen Verläufe einer Endophthalmitis von Nichtglaukompatienten unterscheiden.

Patienten und Methoden Es handelt sich um eine retrospektive Analyse von 75 Augen (13 G, 62 NG), die in einem 5-Jahres-Zeitraum aufgrund einer Endophthalmitis an der Augenklinik des Universitätsklinikums Erlangen-Nürnberg behandelt wurden. Auszuwertende Parameter waren u. a. das klinische Bild, operative und medikamentöse Behandlungen, das mikrobielle Spektrum und der Visusverlauf bei Glaukom- sowie Nichtglaukompatienten.

Ergebnisse Bei Erstvorstellung dominierte bei allen Patienten eine akute Visusverschlechterung (44 %) mit Vorderkammerreiz (62,7 %), Hypopyon (52 %) und reduziertem (40 %) oder fehlendem (26,7 %) Funduseinblick. Vorangehende intraokularchirurgische Eingriffe wurden bei insgesamt 53,3 % beobachtet, insbesondere Kataraktoperationen. In beiden Gruppen konnten grampositive Kokken als häufigster Erreger identifiziert werden (G: 23,1 %; NG: 38,7 %), wohingegen seltene Keime nur bei Glaukompatienten vorkamen. Bei 76 % aller Patienten wurde eine Pars-plana-Vitrektomie durchgeführt, eine Enukleation bei 20 %, Letzteres signifikant häufiger bei Glaukompatienten ($p = 0{,}01$). Postoperativ konnte eine signifikante Visusverbesserung bei Nichtglaukompatienten erzielt werden ($p < 0{,}001$); im Direktvergleich stellte sich ein schlechteres visuelles Outcome bei glaukomatös vorerkrankten Augen dar.

Schlussfolgerung Stellt die Endophthalmitis eine sehr seltene Erkrankung dar, ist eine frühzeitige Diagnosestellung und Behandlung dennoch für die Prognose entscheidend. In der vorliegenden Kohorte zeigt sich ein schlechterer Endvisus bei Glaukompatienten als bei Nichtglaukompatienten.

Introduction

Endophthalmitis is a rare, highly acute condition that poses a serious threat to visual acuity and intraocular structures. Despite intensive medical and surgical treatment, the visual outcome is often poor [1–4]. Pathogenesis may be endogenous in the context of hematogenous spread of infection to the internal structures of the eye or, much more commonly, exogenous [5]. Exogenous origin involves direct inoculation, which facilitates entry of the pathogen [1]. For the most part, this occurs following intraocular surgery, especially cataract surgery, severe ocular trauma, and by spreading per continuitatem (e.g., keratitis) [2, 6].

Endophthalmitis occurs preferentially unilaterally with involvement of the vitreous as well as adjacent structures [6]. Because the vitreous has few to no cells that act as an immune defense, its defense mechanisms are extremely limited, which predisposes it to infection [5]. Diagnosis is made on the basis of clinical criteria [4, 7].

On initial ophthalmologic presentation, visual loss, lid redness and swelling, conjunctival hyperemia or limbitis, sometimes accompanied by photophobia of the affected eye, usually predominate. In addition, slit-lamp examination usually reveals an inflammatory state with cells in the anterior chamber, fibrin accumulation, or a hypopyon [1, 5]. These initially non-specific symptoms are collectively termed endophthalmitis, although the severity can vary widely [1, 8]. Except in the case of endogenous origin, there are no associated systemic symptoms [5].

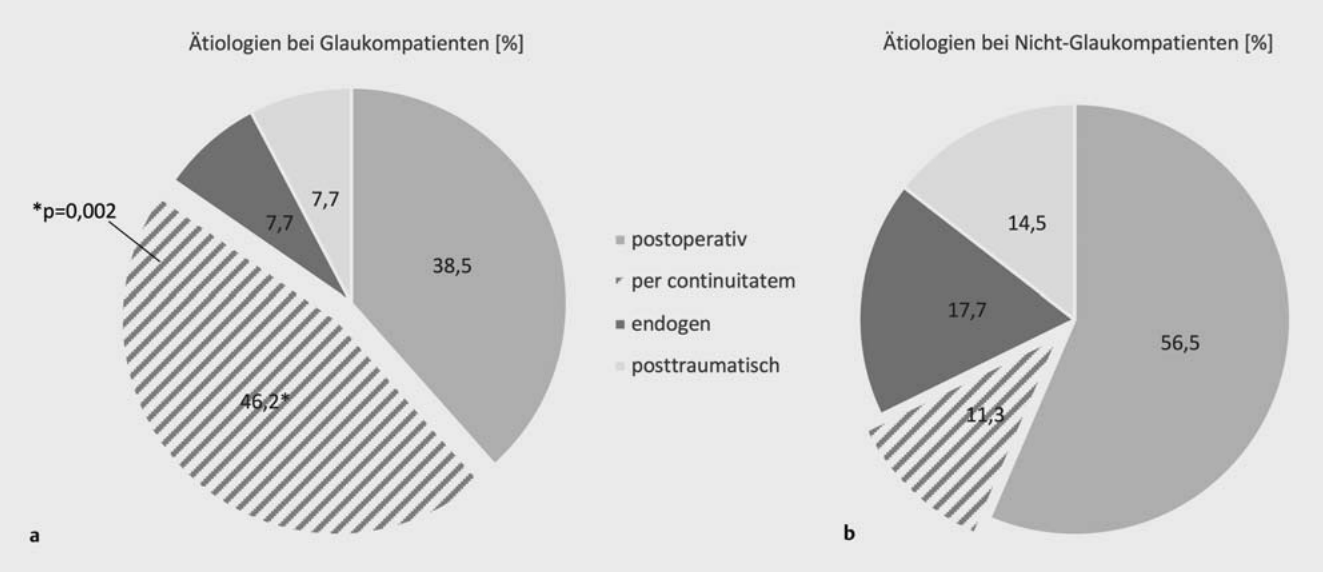
At the time of diagnosis, the causative pathogen is unknown [6]. In terms of empirical therapy, immediate application of broad-spectrum antibiotics and intravitreal antibiotic administration is performed after surgical removal of the vitreous in the course of pars plana vitrectomy (ppV) to achieve sufficient concentration hoc loco [1]. Intraoperatively, potentially infectious material is collected for the purpose of subsequent antibiotic therapy adjustment.

Since a competent immune system is required to fight pathogens and reduce the intraocular inflammatory state, it follows that the prognosis may be worse in the presence of underlying diseases (e.g., diabetes [9], glaucoma [10]).

In the highly complex pathogenesis of glaucoma, it is posited that there may be immune and autoimmune involvement in the eye in addition to raised intraocular pressure [11–15]. Through these altered molecular mechanisms and responses to antigens, it is conceivable that they may also participate in and possibly negatively influence the immune response as a consequence of endophthalmitis. The aim of the study was to analyze the development and progression of endophthalmitis, specifically in terms of causes, clinical symptoms, causative agents and visual acuity between glaucoma and non-glaucoma patients.

Patients and Methods

A retrospective analysis was performed on 75 eyes of 75 patients treated for endophthalmitis over a period from June 2013 to June 2018 at the Department of Ophthalmology, University Hospital Erlangen-Nuremberg, Germany. Epidemiologic data and pre-existing conditions included patient age, sex, underlying diseases, and ocular pathologies. Furthermore, surgical data were evaluated with respect to time and type of surgery, any revision surgery, and systemic and intraocular medications. To assess the success of the treatment, the best corrected visual acuity of the patients was recorded for up to 1 year postoperatively. Before per-



▶ **Fig. 1** Etiologies of endophthalmitis in patients with (**a**, n = 13) and without glaucoma (**b**, n = 62): postoperative (gray), per continuitatem (shaded), endogenous (dark gray), post-traumatic (light gray); data given as percentages.

forming statistical tests, a test for normal distribution was first performed using a Kolmogorov-Smirnov test. When considering normally distributed quantitative characteristics, a paired T-test for dependent samples was used. If the analysis was based on a qualitative characteristic, a chi-square test was performed. For cell counts below 5, on the other hand, Fisher's exact test was used. A p-value of < 0.05 was considered statistically significant.

Results

In the Department of Ophthalmology of the University Hospital Erlangen-Nuremberg, 75 eyes of 75 patients (40 male, 35 female) were treated for endophthalmitis during the 5-year study period. The mean age at admission was 69.3 ± 15.8 years (17–94 years). 60% (n = 45) of patients had underlying cardiovascular disease and 32% (n = 24) had Type II diabetes mellitus.

Underlying etiologies for the development of endophthalmitis were classified (by descending frequency) as postoperative (G 38.5%/NG 56.5%), per continuitatem (G 46.2%/NG 11.3%), endogenous (G 7.7%/NG 17.7%), and post-traumatic (G 7.7%/NG 14.5%) (▶ **Fig. 1**).

In 53.3% (n = 40/75) of all patients, there was an underlying intraocular postoperative etiology: These cases occurred predominantly after cataract surgery (29.3%, n = 22/75, mean 20.7 ± 18.9 days postoperatively [first presentation within 6 weeks postoperatively: n = 19/22, 86.4%; after 6 weeks postoperatively: n = 3/22, 13.6%]), after intravitreal intraoperative medication (IVOM; 17.3%, n = 13/75, mean at 8.2 ± 7.6 days postoperatively), and after pars plana vitrectomies (6.7%, n = 5/75).

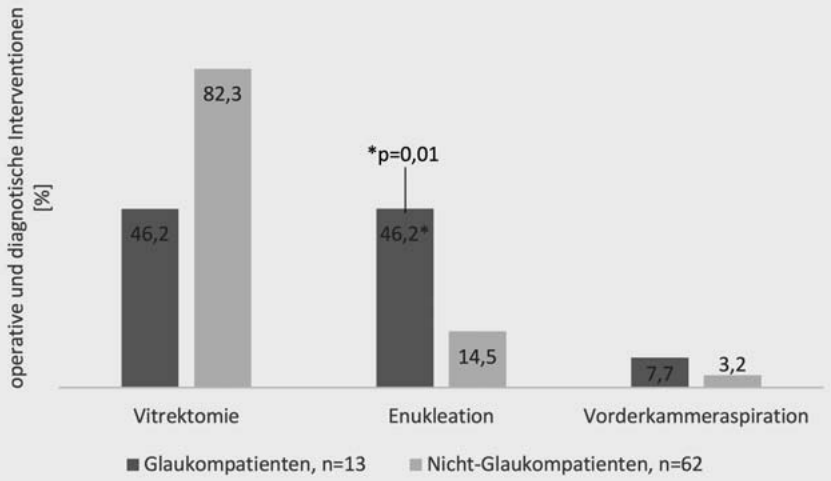
In 17.3% (n = 13/75), endophthalmitis per continuitatem developed after corneal migration due to a pre-existing corneal ulcer (n = 9), chronic corneal endothelial-epithelial decompensation (n = 3), and after long-term contact lens use (n = 1).

Furthermore, an endogenous focus was found in 12 of 75 patients, including systemic candida infections, viremia, sepsis (n = 2 each), and endocarditis (n = 1). Immunosuppression (due to concomitant disease of chronic hepatitis, n = 3; drug-induced, n = 2) was present in 5 additional patients. Of these, one patient reported intravenous drug abuse.

The rarest observed cause was severe ocular trauma (13.3%, n = 10).

The most prominent symptom at initial presentation within the entire cohort was a loss of visual acuity (44%, n = 33). 22.7% (n = 17) of patients complained of pain in the affected eye. Clearly visible conjunctival injection was seen in 19 eyes. The main finding on initial slit-lamp examination was cells in the anterior chamber (62.7%, n = 47), hypopyon (52%, n = 39), or a reduced or absent view of the fundus (66.7%, n = 50).

As the surgical method of choice, 57 of 75 eyes underwent a pars plana vitrectomy. In 15 patients, preservation of the globe was no longer possible due to the advanced intraocular inflammatory state, so enucleation or evisceration had to be performed. The diseased eyes that had to be treated with one of these interventions were significantly more likely to have a corneal ulcer (p = 0.01). Diagnostic anterior chamber aspiration was performed in 3 patients with sterile endophthalmitis (n = 2) and after previous ppV (n = 1), but no extensive intraocular surgery was performed (▶ **Fig. 2**). Primary intervention (62.7%, n = 47/75; ppV: n = 43, enucleation/evacuation: n = 1; anterior chamber aspiration: n = 3) was performed within 24 hours of admission. Vancomycin (1 mg/0.1 ml) was administered intravitreally during vitrectomies, as well as gentamicin (n = 32) or amphotericin B (5–7.5 µg/0.1 ml, n = 7) in a proportion of patients. Topical therapy was continued in the form of eye drops or ointment (mainly containing ofloxacin, n = 46). Until the pathogen was identified, empiric broad-spectrum antibiotics were used, predominantly cefuroxime (3 × 1.5 g intravenously, n = 66). In the case of a partic-



▶ **Fig. 2** Surgical and diagnostic interventions performed in patients with (dark gray; n = 13) and without glaucoma (light gray; n = 62): there was a significantly increased number of enucleations in the glaucoma group (p = 0.01); data given as percentages.

ularly pronounced intraocular inflammatory state, antibiotic treatment was extended to include vancomycin (n = 21). Furthermore, after obtaining antibiotic sensitivities, antibiotic therapy was changed in 9 patients. If fungal endophthalmitis was suspected, weight-adjusted voriconazole or fluconazole (n = 8) or caspofungin (n = 1) were used intravenously. Furthermore, 2 patients received aciclovir in addition to antibiotics because of a history of recurrent herpes keratitis.

Postoperative complications occurred in a total of 14 patients. In these cases, there was marked fibrin formation (n = 6), impaired wound healing (n = 4), bullous retinal detachment (n = 3) with subsequent repeat vitrectomy in each case, and hypotonia bulbi (n = 1/14). One of the patients developed a fibrin membrane after anterior chamber aspiration. Similarly, a conjunctival scleral defect was evident in the form of a wound healing disorder after evisceration. The remaining complications were observed after ppV.

In more than half of patients (50.7 %, n = 38/75), a pathogen could be identified from the retained material. 71.1 % (n = 27/38) of the pathogens detected were gram-positive bacteria, with Staphylococcus epidermidis (n = 16) being the most common, and Staphylococcus aureus (n = 7) the second most common. Furthermore, endophthalmitis was shown to be caused by streptococci (n = 4), bacillus species (n = 1), and in isolated cases, gram-negative rods (n = 6) and fungi (n = 4) (▶ **Fig. 3**).

Postoperatively, there was a significant overall increase in visual acuity (hereafter rounded to decimal visual acuity) from 0.08 ± 0.19 at initial presentation to 0.2 ± 0.27 ($p < 0.001$) at the most recent examination, i.e., 1 year after diagnosis. If the etiology is taken into account when considering visual performance, the best outcome was seen in cases where the cause had arisen postoperatively, with an average visual acuity of 0.32 ± 0.28 (min: 0, max.: 1, p = 0.003). In contrast, post-traumatic and endogenous endophthalmitis resulted in poorer visual recovery, but this did not differ significantly from the other etiologies (post-traumatic: 0,2 ± 0.33; endogenous: 0.16 ± 0.21; $p > 0.05$). We found the worst ultimate visual outcomes in eyes in which the etiology of endophthalmitis was spreading per continuitatem (0.04 ± 0.11; $p < 0.001$), including loss of the eye in 9 of 13 cases.

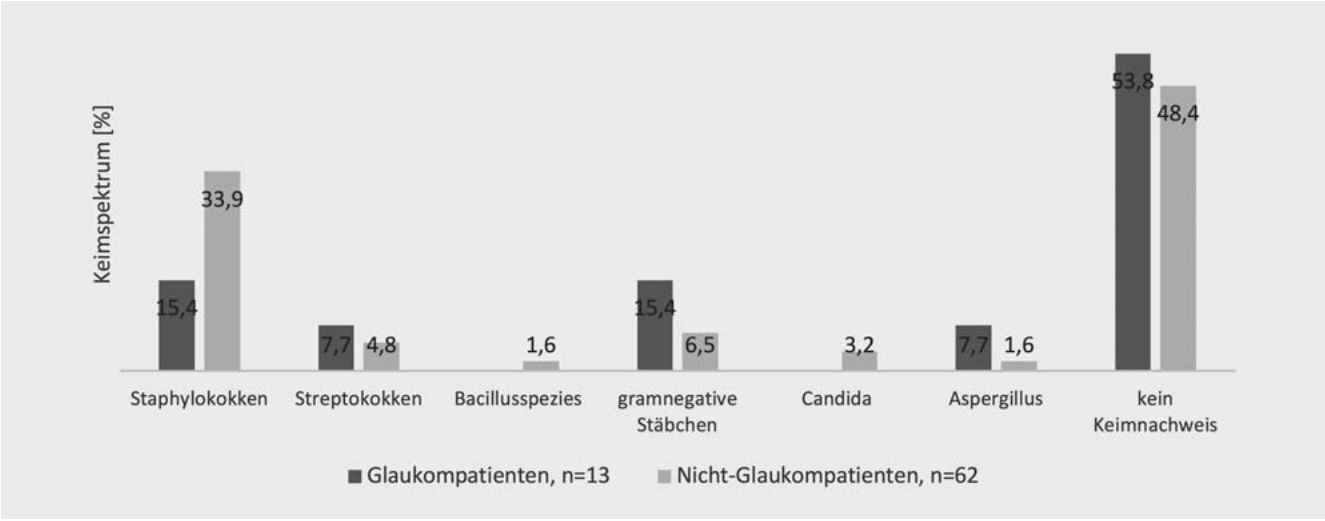
The course of a total of 13 glaucoma patients (primary open-angle glaucoma: n = 3, secondary open-angle glaucoma: n = 8, secondary angle-closure glaucoma: n = 2) was analyzed retrospectively.

Regarding the causes, this group of patients showed significantly more frequent spread per continuitatem (n = 6/13, p = 0.002), whereas endogenous etiologies were significantly less frequent (n = 1/13, p = 0.02) (▶ **Fig. 1 a**). However, post-traumatic as well as postoperative endophthalmitis, independent of the preceding intervention, did not occur significantly more or less frequently ($p > 0.05$). Recent glaucoma surgery was not noted in any patient's medical records. Similarly, no significant difference in the clinical symptoms described above and findings at first presentation could be identified between the two groups ($p > 0.05$), with the exception of a corneal ulcer. This was significantly more frequent (n = 5/13, p = 0.006) in glaucoma patients.

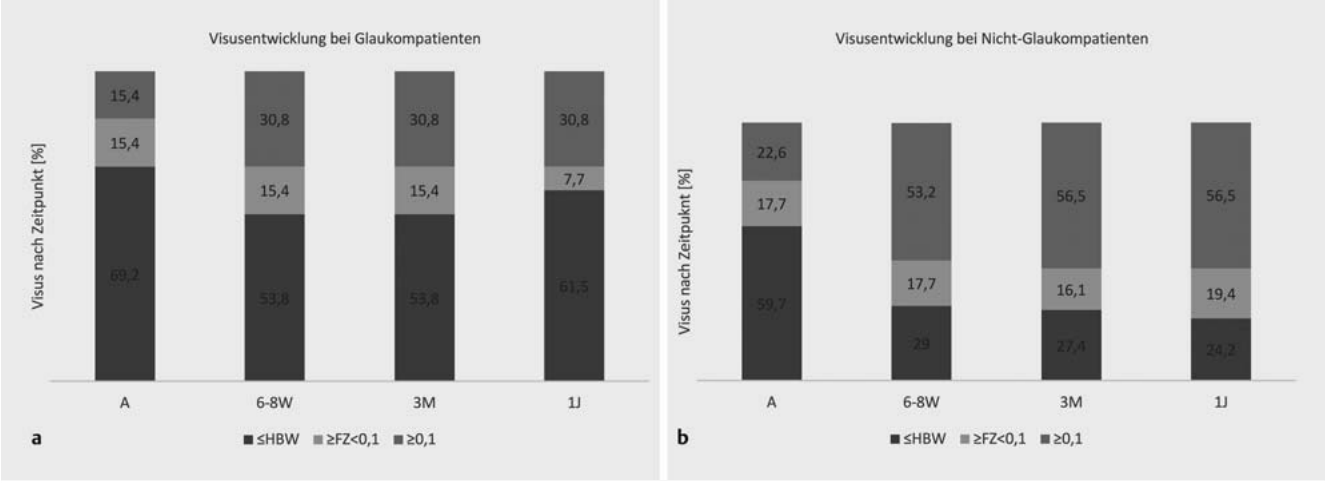
Regarding the surgical methods chosen, enucleation had to be performed significantly more often (6/13; p = 0.01) in glaucoma patients compared to non-glaucoma patients (▶ **Fig. 2**).

The distribution of causative pathogens yielded a comparable profile in both groups ($p > 0.05$), but the gram-negative rods Pantoea agglomerans and Moraxella nonliquefaciens (n = 1 each) were detected exclusively in glaucoma patients (▶ **Fig. 3**).

More than 60 % of the glaucoma patients had a visual acuity at their last examination that detected, at most, hand movements (HBW), whereas this was the case in only 24.2 % of eyes that were not glaucomatous. However, visual acuity was already significantly worse in the glaucoma group at baseline (▶ **Fig. 4**).



▶ **Fig. 3** Distribution of causative pathogens for endophthalmitis in patients with (dark gray; n = 13) and without glaucoma (light gray; n = 62); data in percentages.



▶ **Fig. 4** Changes in visual acuity after endophthalmitis in patients with (**a**, n = 13) and without glaucoma (**b**, n = 62) (A = initial visual acuity, 6–8 W = after 6–8 weeks, 3 M = after 3 months, 1 Y = final visual acuity after up to 1 year, HM = hand movement, FC = finger counting); data in percentages.

Discussion

Endophthalmitis is a rare but severe and rapidly progressive form of intraocular inflammation. It often results in irreparable damage to intraocular structures or even loss of the entire globe, even in young patients without previous ophthalmologic disease [1].

The etiologies observed in the present cohort were consistent with literature data [7]: In most patients, endophthalmitis occurred exogenously, especially after previous intraocular surgery (53.3%), but rarely endogenously (16%) or post-traumatically (13.3%). Furthermore, a comparatively high incidence due to infection per continuitatem, especially on the background of a corneal ulcer, could be unmasked (17.3%), in glaucoma patients to a significant degree ($p = 0.002$). A corneal ulcer can facilitate the penetration of pathogens into the deeper structures of the eye (e.g., the anterior chamber) and their further proliferation, especially in the vitreous body. In the Endophthalmitis Vitrectomy Study (Observation of Acute Postoperative Endophthalmitis), corneal infiltrates or ring ulcers are described as predictors of poor visual outcome [8]. Furthermore, this condition is associated with an increased enucleation rate, which could be corroborated in the context of the observations presented here: One third of our enucleated patients had a pre-existing corneal ulcer, and in one study the proportion was as high as 50% ($p < 0.001$) [16].

Less than 25% of all patients initially reported pain in the affected eye, which is described much more frequently in the literature [8]. It should be borne in mind, however, that the clinical picture and the findings at the initial examination may vary con-

siderably in terms of severity and do not necessarily correlate with a correspondingly fulminant course. Hypopyon, on the other hand, is regularly found at diagnosis, in up to 75% of all patients in the literature and thus more often than in the present cohort (52%) [8].

Postoperative endophthalmitis was preceded primarily by cataract surgery (29.3%), the most common type of eye surgery performed worldwide [4], and IVOM. The incidence of endophthalmitis after the latter procedure is very low (0.06% in the largest meta-analysis to date with more than 350,000 anti-VEGF injections [17]), but is steadily increasing due to its exponential use, especially in the treatment of age-related macular degeneration [4,7]. With regard to the occurrence of post-injection endophthalmitis, the safety of administration in a clinic setting is comparable to that in the operating room [18]. If this complication occurs anyway, there is usually an ophthalmologic follow-up within a few days, as in our patient population [2]. Given the repetitive nature of this treatment, the risk of developing endophthalmitis persists. Any subjective alteration in the patient's sensation during such an injection should always be taken seriously, as this may be the only indication of the onset of infection [4].

The rate of endophthalmitis after cataract surgery is also very low (0.03–0.2%) in the literature. This can be further divided into acute-onset (within the first 6 weeks) and delayed-onset (after 6 weeks) postoperative endophthalmitis [4]. In the present study, 19 of 22 patients presented within 6 weeks. The rarest postoperative development of endophthalmitis occurred after pars plana vitrectomy (present 6.7%, n = 5). However, endophthalmitis can occur after any intraocular procedure [3].

Unilateral involvement was present in all patients studied, regardless of etiology. However, particularly in cases of endogenous etiology, bilateral endophthalmitis with a prevalence of 15% has been described [3,6], as infection reaches intraocular structures through an impaired blood-chamber aqueous barrier [19], which favors the spread of pathogens.

The microbiological analysis revealed gram-positive organisms as the main pathogens, and in individual cases gram-negative rods and fungi were detected. Viruses could not be detected, nor were they clinically suspected as the trigger of an exogenous etiology. However, the classification of etiologies can also be based on the causative agent (primarily bacterial, viral, fungal) or on successful pathogen detection (positive culture vs. clinical diagnosis) [18]. The predominance of gram-positive pathogens has been described many times by other authors [5, 18, 20], including Staphylococcus epidermidis in postoperative endophthalmitis. The prevalence and natural resistance of the pathogen can be understood in terms of its colonization of the skin and its ability to form a biofilm on the lens [20]. Although the microorganisms detected in glaucoma patients and non-glaucoma patients yielded a similar profile, rare pathogens were detected mainly in glaucomatous pre-diseased eyes (Pantoea agglomerans, Moraxella nonliquefaciens). Pantoea agglomerans is a gram-negative rod that has rarely been associated with endophthalmitis, however it is becoming more common and is associated with a poor visual outcome [21].

There is a strong correlation between the visual outcome and the causative agent. Depending on the underlying etiology, this results in a heterogeneous spectrum [4,6,8]. Posttraumatic endophthalmitis, for example, is associated with particularly poor final visual acuity. This is based on the high virulence of the causative pathogen, usually Bacillus cereus, which causes fulminant inflammation and rapid visual deterioration or loss within 24 to 48 hours [6].

The present study aimed to determine whether there were differences in the clinical courses of endophthalmitis between glaucoma and non-glaucoma patients. To the best of our knowledge, there are no case reports or retrospective studies describing and analyzing endophthalmitis in glaucomatous eyes in direct comparison to non-glaucoma patients. Nor can a comparison with non-glaucoma patients be made based on case reports of postoperative endophthalmitis after glaucoma surgery. In addition, there is plenty of evidence of an association between uveitis and secondary glaucoma, but not the other way around.

As described above, glaucoma patients had to undergo enucleation significantly more often, so that the final visual prognosis was significantly worse than in non-glaucoma patients. In addition, a pre-existing corneal ulcer as well as the identification of rare, aggressive pathogens stood out in the direct comparison with glaucoma patients. In contrast, significant visual improvement was achieved in non-glaucoma patients.

One possible explanation for these differences is the special immune status of glaucomatous eyes. Indeed, glaucoma has a highly complex, multifactorial etiology. In addition to the main risk factor of raised intraocular pressure (IOP), a disturbed blood-aqueous barrier is present, but there is also some evidence of an immune [22] and autoimmune [11, 23] component. In the serum of glaucoma patients, there was increased activation of the complement system (e.g., increased C3) [24], which in turn plays a significant role in pathogen defense [25]. In addition, both T and B cells have been described as agents in the pathogenesis of glaucoma: Transiently raised IOP showed significant infiltration of CD4+ T cells into the retina (mouse model) [22]. This can be mediated through human but also bacterial heat-shock proteins (HSP), causing apoptosis of retinal ganglion cells [26].

B-cell-mediated antibody formation to a wide range of proteins, such as enzymes or even matrix structures, is found in glaucoma patients [27, 28]. Specific antibodies against HSP [29] or receptors [11] have also been described. The latter (i.e., agonist autoantibodies against the β2-receptor, β2-AAb) are of particular interest in this regard, as they represent a link to microcirculation and to IOP [30]. The triad of inflammation in the sense of "rubor, dolor, tumor" describes the clinical picture of involvement of the microcirculation (rubor). Hyperemia is found in inflamed tissue, due to vasodilation in response to the local incident.

The influence on the microcirculation on the surrounding tissue, e.g., in the context of a systemic disease (diabetes, β2-AAb), may further exacerbate the inflammatory process, e.g., in endophthalmitis. As a result, the body's own directed immune response to the eye can be impaired.

As a result of the inflammatory processes, many glaucoma patients also present with ocular surface diseases (OSD). OSD is caused by the disease itself, but also by the need for long-term glaucoma therapy. For the patient, it can manifest as foreign body sensation, redness, itching, photophobia, pain and blurred vision

in the affected eye, among other symptoms. In addition, conjunctival hyperemia and damage to the conjunctiva as well as cornea, e.g., the ulcers detected here, are frequently observed [25]. It is quite conceivable that glaucoma patients, who are familiar with these non-specific ocular symptoms to a certain degree, do not initially notice any difference at the onset of endophthalmitis and, as a result, may present late to ophthalmology for adequate treatment.

The data presented may offer explanations for the prognostic course of intraocular infections or inflammation in glaucoma patients. Alteration of the immune system in the eye may be involved in the pathogenesis and local response to infection, even if it is not a generalized autoimmune disease. However, in the present cohort, glaucoma patients represent a minority, so no general hypotheses should be derived from the observations.

Conclusion

Endophthalmitis is a rare but serious intraocular infection in ophthalmology that requires immediate attention. Patients with pre-existing glaucoma had a more severe course overall. Their poor prognosis was favored by pre-existing corneal ulcers and the presence of rarer, more aggressive pathogens. Last but not least, enucleation of the affected eye, as a last resort, was performed significantly more often in glaucoma patients than in non-glaucoma patients.

CONCLUSION BOX

Already known:

- Despite intensive treatment, endophthalmitis is usually associated with poor visual outcomes.
- Since this has not been described previously, a comparison was made between the clinical courses of glaucoma and non-glaucoma patients.

New findings:

- Visual outcomes after endophthalmitis in glaucoma patients was worse than in non-glaucoma patients.
- This could be due to the presence of rare, aggressive pathogens as well as pre-existing corneal ulcers, which is why glaucoma patients underwent enucleation significantly more often.
- Pathologic changes in the immune system may also play a role in glaucoma patients.

Note

This thesis was written to fulfill the requirements for the scientific degree “Dr. med.” at the Friedrich-Alexander University Erlangen-Nuremberg (FAU). Ethics review and approval was not required for the study in human participants according to local legislation and institutional requirements. Written informed consent from patients was not required for participation in this study, according to national laws and institutional requirements.

Conflict of Interest

The authors declare that they have no conflict of interest.

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