

FREQUENCY OF OCCURRENCE AND TREATMENT OF OCULAR DISEASE IN SYMPTOMATIC INDIVIDUALS WITH ACQUIRED BRAIN INJURY A CLINICAL MANAGEMENT PERSPECTIVE

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Abstract

In an earlier retrospective study of visually-symptomatic individuals with acquired brain injury (ABI), a number of ocular diseases were shown to have an elevated frequency of occurrence (FO) in the ABI sample as compared to a matched non-ABI cohort. Some conditions overlapped the subgroups of ABI, while others were exclusive to the ABI subgroups of either traumatic brain injury (TBI) or cerebrovascular accident (CVA). In the present paper, we discuss the clinical, rehabilitation, and quality of life ramifications of these specific ocular diseases, as well as their associated treatment options and management strategies.

Key Words

acquired brain injury, cerebrovascular accident (CVA), frequency of occurrence, ocular disease, relative risk (RR), quality of life, traumatic brain injury (TBI), treatment and management strategies

INTRODUCTION

Acquired brain injury (ABI) is a broad diagnostic category that primarily encompasses brain insult secondary to either traumatic brain injury (TBI) or cerebrovascular accident (CVA).¹ ABI is found in relatively high prevalence (2 in 1000; 0.2%) in the general population.²⁻⁴ There have been a number of studies investigating the frequency of occurrence of visual sequelae of ABI.^{1,5-13} Aside from anecdotal reports,^{1,8-13} there has been a paucity of rigorously conducted research regarding the frequency of occurrence of ocular disease in a relatively large ABI sample.

However, Rutner et al conducted a retrospective study at the Raymond J. Greenwald Rehabilitation Center (RJGRC) at the SUNY, State College of Optometry on a large sample of ABI patients.¹⁴ They found that these ambulatory, visually-symptomatic individuals with either mild TBI or CVA had an increased frequency of occurrence of specific ocular diseases as compared to a non-ABI cohort.¹⁴ Their findings are used as the basis for the current paper.

Ocular disease can substantially diminish the quality of life (QOL) of an individual. Even modest vision loss secondary to ocular disease appears to reduce the QOL to the same degree as a more serious medical condition, such as diabetes.¹⁵ Brown

et al speculated that anxiety suffered by patients over the potential future loss of vision leads to the diminution of QOL.¹⁵ Thus, the benefit from intervention and treatment of ocular disease has the potential to improve a patient's overall QOL irrespective of whether other more serious comorbidities exist.

In another study, Brown et al¹⁶ examined the QOL scores of patients with vision loss and comorbidities that included CVA. They compared the QOL scores of the CVA patients to those having other comorbidities. Both groups had the same degree of vision loss. The researchers found that the CVA patients had scores indicating a lower QOL than those with other comorbidities. This suggests that vision loss in patients with CVA may cause a more profound effect on QOL than vision loss accompanied by other physical conditions. Thus, it is possible that vision loss in patients with CVA may cause an even more profound diminution of QOL.

The purposes of the present paper are:

1. to summarize the ocular disease conditions that Rutner et al¹⁴ found to be noteworthy in their sample of ABI patients
2. to discuss the adverse effects these conditions have on visual functioning
3. to propose treatment options and management strategies for these conditions.

ABI Category	Age Range(years)	Mean Age(years)	Standard Deviation (years)
TBI (n=160)	8 to 91	44.9	15.8
CVA (n=60)	24 to 90	61.2	14.7

METHODS

The detailed methods that Rutner et al utilized are presented in that report.¹⁴ They are now reviewed.

A computer query for a particular time period yielded 486 records of visually symptomatic individuals, of which 300 were selected randomly. Each of three members of the RJGRC's clinical staff then randomly chose 100 of the records to be reviewed. Of these, only those of visually symptomatic individuals with either mild TBI (n=160) or CVA (n=60) were reviewed for the presence of ocular disease. In the mild TBI category, there were 73 males and 87 females. The CVA category consisted of 33 males and 27 females. Table 1 lists the age characteristics of the patients in each group.

All had received a full vision evaluation over the past year.

The major ocular sites for the disease entities were: anterior segment, pupil, crystalline lens, and posterior segment. Targeted conditions included under each category were determined by consensus.

The reviewers then recorded the number of times that each of the targeted conditions was found in each sample group (TBI, CVA, ABI) at the patient's initial visit. These sums were then divided by the number of subjects in each group; the resulting ratios determined the frequency of occurrence (FO), which was then converted to a percentage.

The next step was to compare these FOs to the FOs of these conditions in a general, non-brain-injured general population (GP). The researchers conducted a Medline computer based search (years 1970-2005), as well as other sources.^{7,15-19} These FOs were also used as percentages. The relative risk (RR) is obtained by dividing the particular ocular disease's FO by that of the GP's FO for that condition.²⁰ Thus, if a disease was present in 20% (FO) of the study's sample, and the occurrence of that disease was found to be 10% in the GP, the RR is 2.

An RR >1 indicates an increased FO of a particular ocular disease in the brain injured sample relative to the GP cohort.

An RR <1 indicates that the FO of the particular ocular disease is less in the brain injured sample than in the GP cohort, and represents a protected condition.

An RR of exactly one (1) indicates that the FO of the ocular disease is the same in the ABI sample and the GP cohort.

Table 2.
Summary of Rutner et al's¹⁴ findings for the TBI sample (n=160) and the present authors' suggested treatment options.

FO TBI= percentage of TBI subjects having the particular disease
FO GP= percentage of the particular disease found in a general, non-brain-injured population

* indicates that the prevalence of the particular disease in a general, non-brain-injured population was not available
RR=relative risk.

Under Treatment: AB= antibiotics, AG = anti-glaucoma medication, AT=artificial tears, LC=lid crutch, LH= lid hygiene, M= monitor, S=steroids, sx = surgery, w/u = work up

Categories	FO TBI	FO GP	FO TBI/GP (Relative Risk)	Treatment
ANTERIOR SEGMENT				
Blepharitis	18.8%	11.2%	1.7	LH
Dry eye	15.6%	15%	1.04	AT/LH
Pinguecula	6.9%	*		AT/M/S/sx
Superficial epithelial keratitis	3.8%	0.18%	21.1	AT
Lagophthalmos	1.9%	0.6%	3.2	AT
Ptosis	1.3%	5.1%	0.25	LC/sx
Lid lesion	1.3%	0.93%	1.4	M/sx
Pterygium	1.3%	1.5%	0.87	M/sx
Corneal abrasion	1.3%	0.05%	26	AB
Chalazion/hordeolum	0.6%	0.5%	1.2	LH
PUPIL				
Fixed pupil	2.5%	*		M
Afferent pupillary defect	1.9%	*		M
CRYSTALLINE LENS				
Posterior subcapsular cataract	1.9%	*		M/sx
Traumatic cataract	0.6%	0.04%	15	M/sx
POSTERIOR SEGMENT				
Vitreous degeneration	7.8%	0.12%	65	M
Peripheral retinal degeneration	4.9%	0.5%	9.8	M
Optic atrophy	4.2%	0.14%	30	M
Traumatic optic atrophy	2.1%	0.12%	17.5	M
Vitreous prolapse	0.7%	0.01%	70	M
Vitreous hemorrhage	0.0%	0.04%		M/sx
Optic neuritis	0.0%	*		w/u
Drug-induced glaucoma	0.0%	*		AG/sx
Traumatic glaucoma	0.0%	0%		AG/sx
Retinal detachment	0.0%	0.08%		Sx
Retinal hole	0.0%	0.02%		M/Sx

RESULTS

The data are summarized for the TBI, CVA, and ABI samples in Tables 2-4 respectively. In each table the FO of diseases found in that sample and their corresponding RRs are shown. The extreme right column of each table summarizes recommended management and treatment strategies for each condition that are subsequently more fully discussed in the text. Table 5 presents conditions with clinically significant RRs that were unique to the

TBI sample, to the CVA sample, and that were common to both samples.

MANAGEMENT AND TREATMENT OPTIONS

At the time of acute brain injury, eye care professionals are primarily concerned with the immediate major ocular and visual consequences. However, during the subsequent subacute and rehabilitative phases of care, the possibility of residual and/or new and less dramatic, but still important, ocular diseases may not be

Table 3.**Summary of Rutner et al's¹⁴ findings for the CVA sample (n=60) and the present authors' suggested treatment options.**

FO CVA= percentage of CVA subjects having the particular disease
 FO GP= percentage of the particular disease found in a general, non-brain-injured population
 * indicates that the prevalence of the particular disease in a general, non-brain-injured population was not available
 RR= relative risk
 Under Treatment: AB= antibiotics, AG = anti-glaucoma medication, AT= artificial tears, LC= lid crutch, LH= lid hygiene, M= monitor, S= steroids, sx= surgery, w/u= work up

Categories	FO CVA	FO GP	FO CVA/GP (Relative Risk)	Treatment
ANTERIOR SEGMENT				
Blepharitis	10%	11.2%	0.89	LH
Dry eye	10.0%	15%	0.67	AT LH
Ptosis	6.7%	5.1%	1.31	LC/M
Superficial epithelial keratitis	5.0%	0.18%	27.8	AT
Lid lesion	3.3%	0.93%	3.5	M/sx
Pinguecula	3.3%	*		AT/M/S/sx
Lagophthalmos	1.7%	0.6%	2.8	AT
Subconjunctival hemorrhage	1.7%	0.1%	17	M w/u
PUPIL				
Afferent pupillary defect	3.3%	*		M W/U
CRYSTALLINE LENS				
Posterior subcapsular cataract	3.3%	*		M/sx
POSTERIOR SEGMENT				
Vitreous degeneration	10%	0.12%	83.3	M
Optic atrophy	10%	0.14%	71	M
Peripheral retinal degeneration	6.0%	0.5%	12	M

adequately addressed. Ocular diseases can significantly impact visual acuity and subsequently effect QOL and mobility options. These eventualities consequently result in increased risk of subsequent trauma and/or falls.²¹⁻²³ This may be of greater consequence in those with mild TBI and CVA, as it may also effect their overall rehabilitative process.²⁴

Anterior Segment Conditions Pingueculae

Anterior segment disease encompasses the external lids, cornea, and conjunctiva. In some conditions, it was not possible to calculate the RR because normative data were not available, despite their relatively high FO in the various sample subgroups. For example, pingueculae were present in 5.9% of the ABI group, and in 6.9% and 3.3% of the mild TBI and CVA subgroups, respectively. Pingueculae are the result of collagen degeneration within the substantia propria. They are described as a raised yellowish lesion that enlarges but does not encroach upon the cornea.²⁵ While they

are sometimes cosmetically bothersome, they pose no adverse effect on vision. Occasionally, pingueculae can produce an irritable and/or itchy eye that can be treated with artificial tears, antihistamine/mast cell stabilizers, or a mild steroid depending on the severity of the symptoms.^{25,26} Referral for surgical removal is warranted when the patient is bothered by its cosmetic appearance.²⁶

Blepharitis and dry eye

Blepharitis and dry eye had TBI RRs of 1.7 and 1.04, respectively; however, the CVA's RR were 0.89 and 0.67, respectively. Nevertheless, both conditions presented with a significant FO (10%) in the CVA sample. Thus, one in every 10 patients had signs and/or symptoms of blepharitis and/or dry eye.

It is well documented that the FO of dry eye increases with age and is more common in women than men.^{27,28} The prevalence of dry eye syndrome increases with age, from 5.7% among women < 50 years of age to 9.8% among women aged ≥ 75 years of age.²⁷ In another study,²⁸ a dry

eye questionnaire was administered to a population-based sample of 2520 volunteers, ages 65 to 84 years; 14.8% reported one or more of the six dry eye symptoms either often or all the time. In the Rutner et al study,¹⁴ patients with mild TBI (mean age 44.9 yrs) had an RR of 15.6% for dry eye that is more characteristic of an older population.

Blepharitis results from an inflammation of the eyelids associated with staphylococcal infection.^{29,30} It is frequently asymptomatic, but it can lead to hordeolum or chalazion development.^{29,30} Blepharitis is associated with inflammation of the conjunctiva, and it presents with symptoms of soreness of the lid margin coupled with symptoms of irritation, burning, and dry eye symptoms.^{29,30}

Dry eye can be caused by decreased tear production of the lacrimal gland, poor tear quality and/or lid margins, or evaporation of the tear film.³¹ Untreated blepharitis and dry eye may result in keratinization of the lid margin and corneal compromise resulting in secondary punctate keratopathy.²⁹⁻³¹ This would negatively impact the refractive system of the eye by disrupting its optical quality and impeding protection of the fragile structures of the anterior segment. This can affect vision function by producing a decrease in visual acuity and contrast sensitivity due to loss of corneal transparency.³¹ Treatment options for blepharitis include: hot compresses and lid massage, general hygiene measures, lid scrubs, and in recalcitrant cases the use of oral antibiotics. Dry eye treatment options include artificial tears (gels or ointments for longer lasting relief), punctual plug occlusion, and/or cyclosporine topical medication (i.e. Restasis).^{29,31,32} Failure to treat these conditions may also adversely impact upon their activities of daily living and the overall rehabilitative process.^{33,34}

Chalazion and Hordeolum

These conditions were found in the mild TBI group, but were absent in the CVA sample.

Chalazion is a relatively benign granuloma of the Zeiss and Meibomian glands, and when acute, it is referred to as a hordeolum or a sty.³⁵ When inflamed, both conditions, chalazion/hordeolum, can be painful and decrease overall QOL. Treatment options include hot compresses if less than 10mm in size; if greater than 10mm, chalazion can be surgically excised or treated with direct steroid injections.^{35,36}

Ptosis

This condition was present with higher FO ratio in the CVA sample versus the mild TBI sample. Ptosis presented with a FO of 6.7% in CVA and 1.3% in mild TBI, as well as RRs of 1.31 and 0.25 for CVA and mild TBI, respectively. However, ptosis can restrict the superior aspect of one's peripheral vision, thereby negatively impacting safety and ambulation in multiply-visually stimulating environments (e.g., reading street signs while driving).³⁷ Ptosis can be treated with a lid crutch attached to the spectacles to elevate the eyelid mechanically to enhance the visual field-of-view and improve cosmesis.³⁷ In addition, once the condition is stable, lid surgery may be a viable option.³⁷

Subconjunctival Hemorrhage

The condition was unique to CVA (FO=1.7%, RR=17). While subconjunctival hemorrhage may be secondary to various conditions, it is more likely to occur in those taking anticoagulant medications (e.g., Warfarin, Coumadin, and Aspirin). These medications may be prescribed to those with vascular compromise, such as those with CVA.³⁸ Although subconjunctival hemorrhages are typically benign and rarely impact adversely upon visual acuity or functional vision, when present in the CVA population, consultation with the primary care physician is advised to rule out clotting problems and permit adjustment of medications.

Crystalline Lens

Crystalline lens changes are a common development with increased age.³⁹ Two particular cataract types were found: posterior subcapsular cataract (PSC) and traumatic cataracts.

PSCs were present in the ABI, mild TBI, and CVA samples with FOs of 2.6%, 1.9%, and 3.3%, respectively. Associated RRs were not calculable because reference data for the non-ABI cohort were not available. PSC has a number of possible etiologies: age-related changes, ocular disease (i.e., inflammation), ocular trauma, and pharmacologically induced conditions (i.e., systemic steroids).⁴⁰ Traumatic cataracts were present only in those with mild TBI with a FO of 15%. Both forms of lens opacity can produce devastating effects on vision, especially if located centrally along the pupillary axis.⁴⁰ Referral for cataract surgery is warranted when vision affects activities of daily living or is inhibiting the rehabilitative process.

Table 4.
Summary of Rutner et al's¹⁴ findings for the ABI sample (n=220) and the present authors' suggested treatment options.

FO TBI= percentage of ABI subjects having the particular disease

FO GP= percentage of the particular disease in a general, non-brain-injured population

* indicates that the prevalence of the particular disease in a general, non-brain-injured population was not available

RR= relative risk

Under Treatment: AB= antibiotics, AG =anti glaucoma medication, AT =artificial tears, LC= lid crutch, LH= lid hygiene, M= monitor, S= steroids, sx= surgery, w/u= work up

Categories	FO ABI	FO GP	FO ABI/GP (Relative Risk)	Treatment
ANTERIOR SEGEMENT				
Blepharitis	16.3%	11.2%	1.46	LH
Dry eye	14.1%	15%	0.94	AT
Pinguecula	5.9%	*		M/S/sx
Superficial epithelial keratitis	4.1%	0.18%	22.11	AT
Ptosis	2.7%	5.1%	0.53	LC
Lagophthalmos	1.9%	0.6%	3.16	AT
Lid lesion	1.8%	0.93%	1.94	M/sx
Pterygium	1.0%	1.5%	0.67	M/sx/S
Corneal abrasion	1.0%	0.05%	20	AB
Chalazion/hordeolum	0.5%	0.5%	1	LH
Subconjunctival hemorrhage	0.5%	0.1%	5	M w/u
Scleritis	0%	*		S
Conjunctivitis	0%	0.75%		AB
Melanoma/carcinoma	0%	*		Refer to oncology
Angle recession	0%	0.01%		AG/ sx
PUPIL				
Afferent pupillary defect	2.6%	*		M w/u
Fixed pupil	2.0%	*		M w/u
Abnormal fluctuating pupil	0.0%	*		M w/u
Horner's pupil	0.0%	2.5%		M w/u
Adie's pupil	0.0%	0.2%		M w/u
Syphillitic pupil	0.0%	*		M w/u
CRYSTALLINE LENS				
Posterior subcapsular cataract	2.6%	*		M/sx
Traumatic cataract	0.5%	0.04%	12.5	M/sx
Subluxated lens	0.0%	0.03%		M/sx
POSTERIOR SEGEMENT				
Vitreous degeneration	8.3%	0.12%	69.17	M
Optic atrophy	5.8%	0.14%	41.43	M
Peripheral retinal degeneration	5.2%	0.5%	10.4	M
Traumatic optic atrophy	1.6%	0.12%	13.33	M
Vitreous prolapse	0.5%	0.01%	50	M
Retinal detachment	0.5%	0.08%	6.25	Sx
Vitreous hemorrhage	0%	0.04%		M/sx
Optic neuritis	0%	*		M w/u
Drug-induced glaucoma	0%	*		AG/ sx
Traumatic glaucoma	0%	0%		AG/ sx
Retinal hole	0%	0.02%		M/sx

Table 5.
Ocular diseases with clinically significant RRs that were unique to the mild TBI sample, the CVA sample, and those that occurred in both samples.¹⁴

Ocular diseases unique to the mild TBI sample	Ocular diseases unique to the CVA sample	Ocular diseases common to the mild TBI and CVA samples
corneal abrasion blepharitis chalazion/hordeolum dry eye traumatic cataract vitreal prolapse traumatic optic atrophy	subconjunctival hemorrhage ptosis	superficial epithelial keratitis lagophthalmos lid lesion vitreal degeneration optic atrophy peripheral retinal degeneration

Table 6.
Comparison of the Suchoff et al⁷ study to the Rutner et al study.¹⁴
FO = frequency of occurrence, reported as a percentage; ABI = acquired brain injury; TBI = traumatic brain injury; CVA = stroke.

Suchoff et al		Rutner et al		
Ocular Disease Classification	FO (%) in the combined ABI sample	Ocular Disease Classification	FO (%) mild TBI	FO (%) CVA
Anterior segment disease	22.6	Blepharitis	18.8	10
		Dry eye	15.6	10
Lid defects	4.8	Ptosis	1.3	6.7
		Lagophthalmos	1.9	1.7
Peripheral retinal degeneration/vitreal-retinal degenerations	9.7	Vitreal-retinal degeneration	6.9	8.3

Pupil

Pupil assessment is an important part of the basic evaluation of the neurological function of the optic nerve. An afferent pupillary defect (APD) was present in 1.9% and 3.3% of the mild TBI and CVA subgroups, respectively. APD is indicative of a decrease in the pupil-constrictive drive to the brain stem from one eye compared to the other. Its presence suggests an efferent third nerve problem. It is seen most often in: either unilateral or asymmetric bilateral optic nerve diseases (i.e., optic neuritis, ischemic optic neuropathy, or compressive optic neuropathy), glaucoma, optic chiasm or optic tract damage, retinal detachment, traumatic mydriasis, or vitreal hemorrhage. Although APD can be present in more benign situations such as unilateral dense cataract or functional amblyopia, its presence is an indication for further testing including: fundus dilation, visual field testing, color-vision testing, and possibly radiological imaging. There are no treatment options for the APD per se, but rather treatment of the underlying condition.⁴¹

Fixed pupil was present in 2.5% in the mild TBI sample, but it was absent in the CVA sample. Fixed pupils usually occur in the presence of an inflammatory process, such as anterior and/or posterior uveitis, with the formation of posterior synechiae. This condition is often present in TBI when the source of impact is either frontal or orbital, and the ocular inflammation remains untreated.⁴² Moreover, the parasympathetic function of pupillary constriction is controlled by the third cranial nerve, which is anatomically adjacent to the brainstem. Hence, any trauma to this pathway may result in a fixed and unresponsive pupil to a light stimulus. Further query for a fixed pupil should include fundus dilation, visual fields, and possible radiological testing to rule out other pathological causes. Treatment options include opaque contact lenses that minimize glare and photosensitivity, as well as surgical options.⁴² Normative data were not available for the above conditions. Hence, the RR was not calculable. However, the FO of APD was 3.3% in the CVA sample, and combined

FO of APD and fixed pupil of 4.4% in the TBI sample, is high enough to be considered clinically significant, and require further evaluation and monitoring.

Posterior Segment

Several posterior segment conditions presented with relatively high FO in the TBI group: vitreal degeneration (7.8%), peripheral retinal degeneration (4.9%), optic atrophy (4.2%), and traumatic optic atrophy (2.1%). Each of these conditions showed RRs ranging from 9.8 to 30. See Table 2. It is also noteworthy that while vitreal prolapse had a FO of only 0.7%, its RR is 70.

These conditions can impair visual acuity (e.g., causing legal blindness), contrast sensitivity, and relative depth perception. Furthermore, they are a direct result of trauma to the eye, face, or head. These posterior segment conditions should be referred to a retinal specialist for evaluation and possible surgical treatment.

Comparison with a Non-selected Study

Due to differences in classification and the sample type (i.e., selected versus non-selected), the Rutner et al¹⁴ findings cannot be directly compared to those of an earlier study on a non-selected ABI sample. Suchoff et al⁷ reported on 62 ABI patients who were examined, not because they were symptomatic, but rather as part of an annual medical physical. In this respect, the sample can be considered as non-selected. Further, in addition to ocular disease, that study reported on areas that were not included in the Rutner et al¹⁴ report; it included the frequency of oculomotor, binocular, and visual field defects. Nevertheless, some comparisons and observations can be made in terms of ocular disease. Table 6 shows similarities in FOs between the two studies.

SUMMARY

Over the past decade, there have been reports in the optometric and rehabilitation literature on the presence and consequences of visual sequela of ABI. These have included oculomotor, accommodative, binocular, and visual field deficiencies.^{6-13,43,44} Nevertheless, Rutner et al¹⁴ provided significant evidence that various ocular diseases occur with a clinically important frequency in ABI. The present paper has presented a discussion of these diseases along with clinical management and treatment strategies.

There is no doubt that it is beneficial to know which ocular diseases are more frequent in TBI and CVA. It is equally important to be aware of the impact on rehabilitation and QOL if certain ocular diseases remain unaddressed, as well as the possible treatment approaches for these ocular diseases. This is particularly true for those optometrists who are increasing called upon to treat the visual consequences of ABI. A heightened awareness of the more frequently occurring ocular diseases in TBI and CVA among all members of the rehabilitative team may serve to establish proper and prompt referrals to members of the vision rehabilitative team for evaluation and management. The result can be that the identification, diagnosis, and management of these ocular diseases will impact positively on an individual's rehabilitation, as well as overall quality of life.

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