·Clinical Research·

Head trauma can cause transient elevation of intraocular pressure in patients with open angle glaucoma

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Abstract

• AIM: To describe a newly-recognized entity, illustrated by five cases of glaucoma in whom trauma to the head, but not the eye, resulted in marked, transient elevation of intraocular pressure (IOP).

• METHODS: Retrospective case series. Chart review.

• RESULTS: All five cases had a diagnosis of primary open-angle glaucoma prior to the experience of trauma to the head. All cases had an unusual elevation of IOP (around 70 percent) for days to weeks following the trauma, after which the IOP fell to pre-accident levels. No cause other than the trauma could be determined.

• CONCLUSION: The relationship between head trauma and elevation of IOP appears real.

• KEYWORDS: head trauma; open angle glaucoma; intraocular pressure
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INTRODUCTION

Intraocular pressure(IOP) is a consequence of the balance between the rate of aqueous inflow and outflow. Ocular trauma can decrease aqueous outflow causing an elevation of IOP due to blockage or "plugging" of the outflow mechanisms by iris, blood, inflammatory material, or other substances such as vitreous. What we report here is a series of individuals with primary open angle glaucoma (POAG) who had trauma to the head but not the eye, and who developed a sudden marked elevation of IOP following the trauma; this later fell to pre-accident levels. There appeared to be no cause for the elevation of IOP other than the head trauma. Possible reasons for this are discussed.

MATERIALS AND METHODS

Several charts of glaucoma patients in Wills Eye Institute have been reviewed. A transient elevation of IOP has been noticed in five patients with POAG who experienced head trauma.

CASE REPORT

Case 1 A 30-year-old woman of European extraction was first seen with far-advanced glaucomatous optic nerve damage in the right eye and early damage in the left. The IOP had been as high as 40mmHg in the right eye and in the 20's in the left. The anterior chamber angles were wide open and normal. A diagnosis of juvenile onset open angle glaucoma was made. Medicinal treatment did not control the IOP in the right eye which, due to far-advanced visual field loss, had a visual acuity of 20/200 and was exotropic. A trabeculectomy was performed, following which the IOP was in the teens. In the left eye the IOP was 35mmHg off treatment; after timolol was started the IOP was consistently in the upper teens. The optic nerve of the left eye had a cup/disc ratio of 0.6, with saucerization inferiorly; there was a probable superior nasal step in the visual field. The patient was followed for five years without change in the disc or field of the left eye, and with stable IOP in both eyes, around 14mmHg in the right eye and 16mmHg in the left eye, on no treatment for glaucoma. At a routine examination the patient's IOP was noted to be 20mmHg in the right eye and 28mmHg in the left eye. She reported that several weeks earlier she had been in a severe automobile accident which resulted in a broken rib and a concussion. She had not had any trauma to either eye and had not noted a change in her vision. A CT scan of the brain had been performed which was interpreted as within normal limits. The sudden rise in IOP was noted, but her treatment was not changed and a

repeat evaluation was scheduled for several weeks later, at which time the findings were remarkably similar, though the IOP in the right eye had risen slightly to 23mmHg. She was advised to add dorzolamide to the timolol being used in the left eye. Several weeks later the IOP fell back to the low 20's in the right eye and 16mmHg in the left eye, which was where it had been prior to the accident. Visual acuity had fallen to hand movements in the right eye. At no time during these examinations was there any evidence of trauma to either eye. Timolol 5g/L was added to the right eye. One month later the IOP was 18 mmHg in each eye. A repeat visual field examination showed what appeared to be further deterioration in the right eye, and no apparent change in the left. One month later the IOP was 14mmHg in the right eye and 16mmHg in the left. The dorzolamide was stopped in the right eye ; one week later the pressure was again 14mmHg in the both eyes. Dorzolamide was stopped in the left eye. One week later on no treatment in either eye, the IOP was 13mmHg in the right eye and 16mmHg in the left. It has remained in the mid-teens since that time.

Case 2 A 60-year-old woman of European extraction was referred for management of what was believed to be POAG. Her health was not good. She was asymptomatic from an ocular point of view, but had been noted to have intraocular pressures in the low 20's and discs that were believed to be suspicious for glaucoma. On no treatment, the IOP was 21mmHg in the right eye and 26mmHg in the left. There was a questionable notch in the disc of the right eye inferiorly and what appeared to be a definite notch in the left eye. There was a suspicious superior arcuate scotoma in the right eye and a superior paracentral nasal defect in the left eye. A diagnosis of open-angle glaucoma was made, and the patient was started on latanoprost once daily in the left eye. The IOP fell into the mid-teens in the left eye. As the patient was not in good health and the findings in the right eye were marginal, she was not started on medication in that eye. Over the next three years, the IOP in both eyes and the optic discs and visual fields remained unchanged, consistently around 20mmHg on no medication in the right eye, and 16mmHg in the left on latanaprost once daily. She returned urgently for evaluation after she had been in a severe automobile accident, at which time she had been "knocked unconscious." She was worried about her eyes, as she did not believe she saw as clearly as she had prior to the accident, which had occurred one week before. Her visual acuity was unchanged at 20/25 in each eye. There were no signs of ocular trauma. The anterior chamber angles were normal without angle recession. Posterior vitreous detachments were present in both eyes, but had been noted at the time of her initial examination. She was extremely apprehensive despite the use of an anti-anxiety oral medication, diazepam. The IOP in the right eye was 24mmHg and in the left eye was 29mmHg. A repeat visual field examination showed no definite change from before, though it was less reliable. Timolol 0.5% twice daily was added to each eye. Four days later the IOP was 19mmHg in the right eye and 22mmHg in the left eye. Two weeks later the findings were essentially the same. She was still "badly shaken" by her experience. The IOP was 18mmHg in the right and 23mmHg in the left eye. The same treatment was continued, and she was advised to return in one month. At that visit she was "feeling better," and notably less anxious, though she was still taking diazepam. On timolol 5g/L in each eye twice daily and latanoprost once daily at bedtime in each eye, the IOP was 14mmHg in the right eye and 16mmHg in the left eye. She was advised to stop all medications in the right eye and stop the timolol in the left eye. Two weeks later the IOP was 18mmHg in the right eye on no medication, and 19mmHg in the left eye on latanoprost. She was still taking diazepam, which was the only medication, other than her eye drops that had been changed after her accident. Over the next year, the IOP remained in the same range without trending up or down, and she continued to take diazepam. She died a year and a half after the accident.

Case 3 A 59-year-old businessman came to his ophthalmologist for an examination. He complained to his local ophthalmologist because of periodic "eye aches." When first seen his vision was 20/25 in the right eye and 20/20 in the left eye and IOP 36mmHg in the right eye and 27mmHg in the left eye. There was a suggestion of a Krukenberg spindle in the right eye. The anterior chamber angles were wide open with 3/4-plus pigmentation of the posterior trabecular meshwork in the right eye and 2-plus in the left. There were no transillumination defects of the iris, nor was there a Zentmeyer line. The right optic disc was severely cupped with no rim for approximately 60 degrees, and there were dense superior and early inferior arcuate scotomas. In the left eye the rim was narrow (cup/disc ratio . 7, DDLS 4), but there was no definite visual field loss. The diagnosis was POAG in the right eye, possibly in association with the pigment dispersion syndrome, and probable POAG, early in the left. After trying several regimens, his IOP was controlled in the 15 to 18 range in the right eye on timolol 5g/L twice daily and latanoprost once daily, and in the left in the same range on latanoprost once daily. His optic discs, visual fields and intraocular pressures remained stable for five years. At age 64, he came to us for an examination because he noted he was not seeing as well following an accident in which he had been "rear-ended" while stopped at

a stop light. He had "whiplash" and persistent neck pain and headaches following the accident. He had no known actual blow to the head at the time of the accident. Radiologic studies of his head and neck revealed no significant pathology. He was treated with physical therapy and ibuprofen. He was not given systemic corticosteroids. There were no signs of ocular trauma. Visual acuity was unchanged. There was a slight increase in the mean defect of the visual fields in both eyes. IOP was 34mmHg in the right eye and 25mmHg in the left. Dorzolamide three times daily was added to both eyes. One week later the IOP had fallen to 25 mmHg in the right eye and 20mmHg in the left. An argon laser trabeculoplasty was considered but deferred. One week later the IOP was several millimeters higher in each eye. Brimonidine twice daily was added to the right eye. One week later the IOP was essentially unchanged at 28mmHg right eye and 21mmHg left eye, and after a lengthy discussion with the patient, a trabeculectomy was scheduled and brimonidine was advised to be stopped, but the patient was reluctant to have surgery in the right eye. Two weeks later the IOP was 22mmHg in the right eye and 20mmHg in the left. The surgery was cancelled and medication continued. Two weeks and five weeks later, the IOP was essentially the same. One month later, the IOP had fallen to the pre-accident level in both eyes (16mmHg right eye and 17mmHg left eye); timolol was stopped in the left eye and dorzolamide stopped in the right eye. One month later the pressure had risen slightly in the right eye, but remained unchanged in the left eye. Two months later the IOP was 16 mmHg in the right eye and 18mmHg in the left on latanoprost once daily in each eye at bedtime and timolol 0.5% twice daily in the right eye. The pressure has remained in that range for four years.

Case 4 An 81-year-old male real estate developer and textile mill owner had been followed for ten years with the diagnosis of advanced "average-pressure glaucoma" in the right eye and moderate in the left. His IOP's had never been found to be above 17mmHg, and were consistently asymmetric prior to starting therapy. He had a dense superior arcuate scotoma in the right eye in association with an inferior notch and an acquired pit of the optic nerve. In the left eye there was a dense superior nasal step in association with a notched optic nerve. Following argon laser trabeculoplasties and the use of timolol 5g/L% twice daily in each eye and pilocarpine 1% four times daily in each eye (he was pseudophakic), the pressures were consistently in the range of 12 to 15 in the right eye and 11 to 13 in the left eye. His discs and fields remained stable. He returned unexpectedly for an evaluation after he had been injured in a bicycle race in the mountains. One of the other contestants had run into him and he had fallen, breaking his hip, and hitting his head, taking skin off the side of his face and scalp. He had a remarkably rapid recovery following hip surgery, and he came for an examination approximately one month later. At that point the IOP was 17mmHg in the right eye and 14mmHg in the left on his therapy. He was not taking corticosteroids. His visual fields in both eyes showed what looked like definite worsening. Acetazolamide 250mg three times daily was added (he knew he would tolerate this as he took "Diamox" during his races in the mountains). One week later the IOP had fallen in both eyes to pre-accident levels and he was tolerating the Diamox without difficulty. One month later the IOP was 12mmHg in the right eye and 10mmHg in the left; the acetazolamide was stopped. One month later the pressure had risen to 15mmHg in the right eye and 14mmHg in the left eye, and the acetazolamide was resumed. Two months later the IOP was 10mmHg in the right eye and 9mmHg in the left, and acetazolamide was again stopped. Four days later the pressure was 12mmHg in the right eye and 13mmHg in the left eye, and it remained in that range until he died six months later from metastatic prostate cancer.

Case 5 When a housewife of European extraction was 62-years-old, she was told by her ophthalmologist that she had glaucoma in both eyes. Treatment with pilocarpine and epinephrine was started. When her ophthalmologist retired in 1968, she was first examined by us and found to have visual acuity of 20/60 in the right eye and 20/40 in the left and IOP of 24mmHg in the right eye and 22mmHg in the left, far-advanced glaucomatous optic nerve and visual field damage in the right eye, and moderate damage in the left. Her pupils were miotic due to the pilocarpine that was being used, and the cause for her reduced acuity was believed to be cataracts. A cataract extraction combined with a thermal sclerostomy was performed in the right eye. The addition of timolol 5g/L twice daily to the left eye lowered the pressure to 18 mmHg and a cataract extraction without a filtration procedure was performed on the left eye. Post-operatively the IOP in the right eye was 14mmHg and in the left eye, on pilocarpine and timolol, 17mmHg. The patient was followed for 20 years, using no medication in the right eye and pilocarpine and timolol in the left. During that period of time, in the right eye the visual field deteriorated slightly and acuity worsened to 20/30 due to age-related macular degeneration. In the left eye there was slightly more deterioration of the visual field and the acuity fell to 20/40 due to age-related macular degeneration. The IOP was consistently 14mmHg in the right eye, and between 14 and 17mmHg in the left eye. When 83-years-old, she fell in her bathroom and hit her head sufficiently hard that she was

disoriented, and she broke a rib. A CT scan of the brain and orbits was negative other than showing non-specific brain atrophy. She came for an ocular examination because she had struck her forehead over the right eye and was concerned. The examination of the right eye showed a visual acuity of 20/60 and IOP of 8 mmHg. She had marked ecchymosis over the right eye and the filtering bleb was leaking. In the left eye, acuity was 20/40 and IOP 27mmHg on pilocarpine and Timolol, and, when repeated, 28mmHg. There was no evidence of trauma to the left eye. Dorzolamide three times daily was added to the left eye. Over the next few months the conjunctiva over the filtering bleb of the right eye gradually healed, and the IOP in the left eye remained between 17 and 20mmHg on pilocarpine, timolol and dorzolamide. One month later the pressure in the right eye was 15mmHg on no treatment and the left eye 14mmHg. The consideration of changing to "Cosopt" was entertained, but it was decided to stop the dorzolamide, following which the pressure rose to 19mmHg. Timolol was changed to "Cosopt" and the pressure decreased to 13mmHg where it has remained since, on a combination of pilocarpine and Cosopt.

RESULTS

All five cases had a diagnosis of primary open-angle glaucoma. The IOP level before trauma was around 12-15 mmHg, having been controlled by surgery and/or medications (Table 1).

Four cases were involved in traffic accidents and one fell in the bathroom. All had trauma to the head, but not to the eye. Using the same anti-glaucoma medications as prior to the trauma, they had marked elevation of intraocular pressure (14-36mmHg) for several days to several weeks after the trauma. This elevated IOP then fell to pre-accident levels.

DISCUSSION

All of the cases reported had been followed for a sufficient duration of time that the level of IOP prior to their accident was well established. Four cases had accidents associated with severe blows to the head, and one with severe "whiplash." Possible explanations for the IOP elevation that followed the trauma include 1) trauma to the eye [1-3], 2) cessation of use of anti-glaucoma medications[4], 3) initiation of systemic medications that cause an elevation of IOP[5], 4) elevation of intracranial pressure [6], and 5) elevation of systemic factors affecting the neural or hormonal control of IOP [7, 8]. Regarding the possibility of number 1, there was no evidence of direct trauma to the eyes, either by history or by thorough ocular examination. Regarding point number 2 (use of glaucoma medications), all patients were well known to their physician and had a long history of proper use of medications. All patients stated they had not changed their

Table 1 Intraocular Pressure Level before and after trauma mmHg

Patient	Before		After		1 month after	
	OD	OS			OD	OS
1	14	16	20	28	14	16
2	14-16	14-16	24	29	18	19
3	15-18	15-18	36	27	16	18
4	12-15	11-13	17	14	12	13
5	14	17	8	27	15	14

medication program, and there was no evidence that medication use had been altered. Regarding the possibility of number 3 (an effect from some systemic medications), no case was treated with medications known to cause an elevation of IOP, such as systemic corticosteroids. One received diazepam, but this is not known to cause an elevation of IOP. Regarding point number 4, conceivably, head trauma could cause an increase in intracranial pressure, which could increase the pressure in the superior orbital vein or other ocular venous drainage systems, resulting in an increase in IOP. Czarnik *et al* [6] and colleagues studied the relation between intracranial pressure and IOP, and found no direct relationship between them. These authors did not study the relationship between intraorbital pressure and intraocular pressure, but there is no reason to expect that any of the present cases had an elevation of intraorbital pressure. Regarding the final point (neural or hormonal effect on intraocular pressure), there is relatively little published material. However, it is certain that relationships exist. Experimentally, it is showed that injecting hypertonic or hypotonic solutions into the area of the hypothalamus evoked a change in IOP. Monkey experiments demonstrated that localized electric stimulation in the area of hypothalamus resulted in changes in IOP. A relationship between serum cortisol and IOP was found. They showed that patients with POAG and ocular hypertension had elevated levels of serum free cortisol which were associated with a reduced cortisol binding capacity to albumin. The mechanism by which endogenous steroids increase IOP appears to be different from that responsible for elevation of IOP with topical corticosteroids. The latter may interfere with increased resistance to aqueous outflow because of the changes in the trabecular meshwork (TM) and its extracellular matrix. However, systemic steroids have been shown to increase aqueous inflow in experimental studies. Clinically patients with hypercorticoidism, as occurs in Cushing's disease, have elevated IOP which drops to normal level immediately after removal of the hyper-secreting tumor. Schwartz and others have noted significant relationships between the level of endogenous cortisol and IOP. If such changes were caused by depositions in the

trabecular meshwork, it would take weeks or months for the IOP to fall after removal of the hyper-secreting tumor. Systemic adrenal corticotrophic hormone can cause elevations of IOP from the normal range to the 40's or 50's within hours, which would not be possible due to depositions in the TM. TM regulates the drainage rate by changing the intercellular space through a combination of action [9]. Cellular contractility and cellular volume are partly controlled by cytoskeleton and junctional proteins such as F-actin. A study showed that dexamethasone induces F-actin expression and enhances fibroblast mediated contraction [10]. Contraction of the TM reduces the intercellular spaces and thus reduces aqueous humor outflow, resulting in an elevated IOP. Such changes could presumably occur quickly.

All of the individuals in the present report felt seriously threatened by what had happened to them. All were emotionally "shaken." It is reasonable to believe that all of them were experiencing "stress". Elevation of serum cortisol is a well-established aspect of the stress syndrome [11]. All patients had POAG, and as such were likely to be steroid responders. The cases reported here occurred over a period of around five years, and it was not until the fourth or fifth case was noted that a possible causal relationship between head trauma and elevation of IOP was considered.

We describe here what appears to be an entity, specifically, transient elevation of IOP following head trauma in patients with primary open-angle glaucoma. Should such cases be noted in the future, it would seem prudent to assume that there is a relationship between head trauma and elevation of intraocular pressure, and seek mechanisms to explain the relationship.

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