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Case report

Bilateral cataract formation via acute spontaneous fracture of the lens following treatment of hyperglycemic hyperosmolar syndrome

Yevgeniy V. Sychev^a, Emily M. Zepeda^a, Deborah L. Lam^{a, b, *}^a Department of Ophthalmology, University of Washington School of Medicine, Box 359608, 325 Ninth Avenue, Seattle, WA, 98104, USA^b VA Puget Sound Health Care System, Department of Ophthalmology, 1660 South Columbian Way S-112 OPH, Seattle, WA, 98108, USA

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ABSTRACT

Purpose: Acute development of cataracts that may be transient is known to occur during correction of diabetic ketoacidosis and hyperglycemic hyperosmolar syndrome. Nettleship in 1885 was the first to describe the presence of a transient cataract in three diabetic patients that grew worse and eventually cleared with treatment.¹ We present a case of irreversible cataracts formed by nuclear fracture of the crystalline lens after hyperglycemia correction, an entity that has not yet been described.

Observations: A 67 year-old Caucasian man presented with sudden bilateral vision loss one week after a week-long hospitalization in the intensive care unit for correction of hyperglycemia in the setting of hyperglycemic hyperosmolar syndrome requiring an insulin drip. This was caused by spontaneous fractures of the lens nuclei causing bilateral irreversible cataracts. The patient underwent uncomplicated bilateral cataract extraction resulting in restoration of normal vision.

Conclusions and importance: Acute transient cataracts that develop during correction of hyperglycemic hyperosmolar syndrome are thought to result from osmotic lens swelling. In this case report, internal fracture of the lens was produced by mechanical forces generated in the process of lens swelling occurring as a consequence of initial hyperglycemia and its subsequent correction. This case represents a rare ocular complication of hyperglycemia correction, and provides new evidence that mechanical forces can be part of diabetic cataractogenesis.

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1. Introduction

The consequences of elevated blood glucose levels in the diabetic patient have well known effects on the lens such as changing the refractive index and decreasing the accommodative amplitude. Diabetes is also known to have a significant effect on the clarity of the lens, specifically in the form of a cataract. While a typical diabetic cataract is a chronic process related to persistently elevated blood glucose, there are reported cases of acute and frequently reversible cataracts. These case reports describe the development of a transient cataract, typically of posterior subcapsular type, most frequently observed during aggressive treatment of diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar syndrome (HHS) (Table 1).^{1–9} The majority of these cases have been observed

in children or young adults. Here we report a previously undescribed type of cataract that occurred several days after correction of extreme hyperglycemia in an elderly adult with HHS. This new description of diabetic cataract highlights the importance of accessory glucose metabolism pathways in the lens and how osmotic forces have a role in cataractogenesis.

2. Case report

A 67 year-old Caucasian man with past medical history of coronary artery disease was hospitalized with symptoms of weakness, fatigue, loss of appetite, polydipsia and polyuria. His blood glucose (BG) on presentation was 1620 mg/dl and hemoglobin A_{1c} was 19.8%. He was diagnosed with diabetes mellitus complicated by HHS and treated without complication in an intensive care unit with an insulin drip according to established hospital protocols for normalization of blood glucose. He was discharged six days later with BG in the 100–200 mg/dl range on a combination of long and short acting insulin and oral metformin. He was previously a pre-diabetic and did not require any medication. The patient denied

* Corresponding author. Veterans Affairs Puget Sound Health Care System, Department of Ophthalmology, S-112-OPH 1660 South Columbian Way, Seattle, WA 98108, USA.

E-mail address: deblam@uw.edu (D.L. Lam).

Table 1
Case reports describing diabetic related cataractogenesis.

Case report	Age (years)	Sex	Clinical diagnosis and treatment	BG prior to treatment, mg/dl	Delay to development of cataract following initiation of treatment	Visual Acuity at diagnosis of cataract OD, OS	Description of cataract	Duration of follow-up	Resolution of Cataract at follow-up
Present report	67	Male	HHS treatment	1620	Subjective blurry vision developed after 13 days, cataract confirmed after 18 days	20/70, 20/50	Nuclear fracture in a “split pea” configuration	6 weeks	No
Bilginturan 1977 ⁶ case 1	15	Male	DKA treatment	550	Subjective blurry vision developed after 4 days	20/40, 20/40	Bilateral lamellar	2.5 weeks	Yes
Bilginturan 1977 case 2	10	Female	DKA treatment	645	Subjective blurry vision developed after 4 days	20/20, 20/25	Bilateral posterior subcapsular	2 weeks	No, improved
Hunt 1978 ⁷	3	Female	HHS treatment	1546	Cataract discovered on routine physical examination after 18 hours	Not obtainable	Bilateral dense opacity	3 days	Yes
Phillip 1993 ⁸	14	Female	DKA treatment	434	Subjective blurry vision developed after 8 days	20/25, 20/25	Bilateral posterior subcapsular	14 days	Yes
Brown 1973 ⁴	4	Female	HHS treatment	2000	Cataract discovered on routine physical examination after 29 hours	Not obtainable	Bilateral complete opacity	1.5 days	Yes
Lawrence 1946 ¹ case 1	39	Male	Hyperglycemia treatment	416	Symptoms developed after 16 days, cataract confirmed after 3 weeks	Not reported	Bilateral rosette cataract	4 days	Yes
Lawrence 1946 case 2	45	Female	Hyperglycemia treatment	380	Symptoms developed after 3 days, cataract confirmed after 15 days	Not reported	Bilateral rosette cataract	~35 days	Yes
Roberts 1950 ²	48	Female	Hyperglycemia treatment	Not reported	Subjective blurry vision developed after 4 days	20/100, laterality not reported	Bilateral posterior subcapsular	3 days	Yes
Sharma 2001 ⁹	62	Female	Hyperglycemia treatment	762	Symptoms developed after 10 days, cataract confirmed after 12 days	Not reported	Bilateral posterior subcapsular rosette	3 weeks	No improved
Cornwell 1995[19]	19	Female	Hyperglycemia treatment	290	Symptoms developed after 6 weeks, cataracts confirmed concurrently	LP, LP	Bilateral dense cortical cataracts	7 months	No improvement
White 1984 [20]	21	Female	Hyperglycemia treatment	455	Progressive vision loss for 4 weeks, cataracts confirmed after 1 month	20/80, 20/80	Bilateral dense cortical opacities	4 months	No improvement

Abbreviations: BG, blood glucose; DKA, diabetic ketoacidosis; HHS, hyperglycemic hyperosmolar syndrome; LP, light perception.

any subjective changes in vision during his hospitalization. Once he was on medication, he did not have any recurrence of his prior hyperglycemic symptoms. He was discharged from the hospital. Seven days after discharge, he awoke with sudden bilateral severe vision loss; he could not distinguish his facial features in a mirror. He went to the emergency room where his BG was 105 mg/dl, external ocular exam was normal, and no systemic or neurologic abnormalities were evident. He was discharged from the emergency room with a plan to follow up with ophthalmology. He presented to the ophthalmology clinic five days later where he described no prior ocular history and previous good subjective vision. Best corrected vision on exam one year ago was 20/15 in each eye, and no cataract was noted. On examination the patient's visual acuity was 20/200 uncorrected in each eye. With manifest refraction, vision improved 20/70 (+4.0 sphere) in the right eye and 20/50 (+4.0 sphere) in the left eye. Intraocular pressure was physiologic and all ocular structures displayed no pathology with the exception of unusual bilateral cataracts. There was minimal opacity in each lens; however, the nuclei were fractured along the equatorial plane in a configuration resembling a split pea (Fig. 1). The patient elected to undergo cataract surgery in the absence of improvement in vision or objective appearance of the cataract after four weeks of observation. One month after uncomplicated cataract surgery in the second eye, the patient's uncorrected visual acuity was 20/15 in the right eye and 20/20 in the left. His diabetes remained well controlled.

3. Discussion

The presented case describes a novel type of cataract that resulted from spontaneous gross structural disruption of the lens nucleus without an associated diminution of lens translucency. It is not possible to definitively establish the nature of the intrinsic

forces within the lens that lead to splitting of the nuclei. However, given the time course of rapid cataract formation following correction of the patient's hyperglycemia, it is likely that they were a product of osmotic fluid shifts in the lens. An osmotic mechanism of cataract formation has been well studied in animal models where hyperglycemia leads to accumulation of sorbitol inside the lens resulting in lens swelling, disruption of individual lens fibers, and cataractogenesis.^{10,11} This process is mediated by the enzyme aldose reductase whose activity in the human lens is reduced relative to the animal models.¹² Therefore, it has been suggested that hyperglycemia must be severe in order to cause similar changes in the human lens.^{12,13} On the other hand, reversal of the hyperglycemic state in the course of treatment can further accentuate lens swelling by decreasing the osmolality of the aqueous thereby driving more water into the sorbitol rich lens. With eventual resolution of serum hyperglycemia, the lens can slowly metabolize the accumulated sorbitol and achieve deturgescence by cellular pumping mechanisms, leading to resolution of the cataract.^{9,16}

A thorough literature search from 1946 to present including the terms “transient diabetic cataract”, “hyperglycemia”, “Diabetic Ketoacidosis” and “hyperglycemic hyperosmolar syndrome” revealed 11 case reports describing acute diabetic related cataractogenesis (Table). The majority of previously reported cases of acute cataract formation during hyperglycemia treatment were reversible and were predominantly observed in younger patients. The typical cataract observed in these cases is of a posterior subcapsular or cortical type. It has been hypothesized that formation of these cataracts reflects damage of individual lens fibers induced by osmotic stress and subsequent hydropic changes.¹⁴ The case presented here is different in a number of respects. Significantly, the patient was elderly; therefore, his lens nucleus should have been considerably more sclerotic, even though it did not yet demonstrate

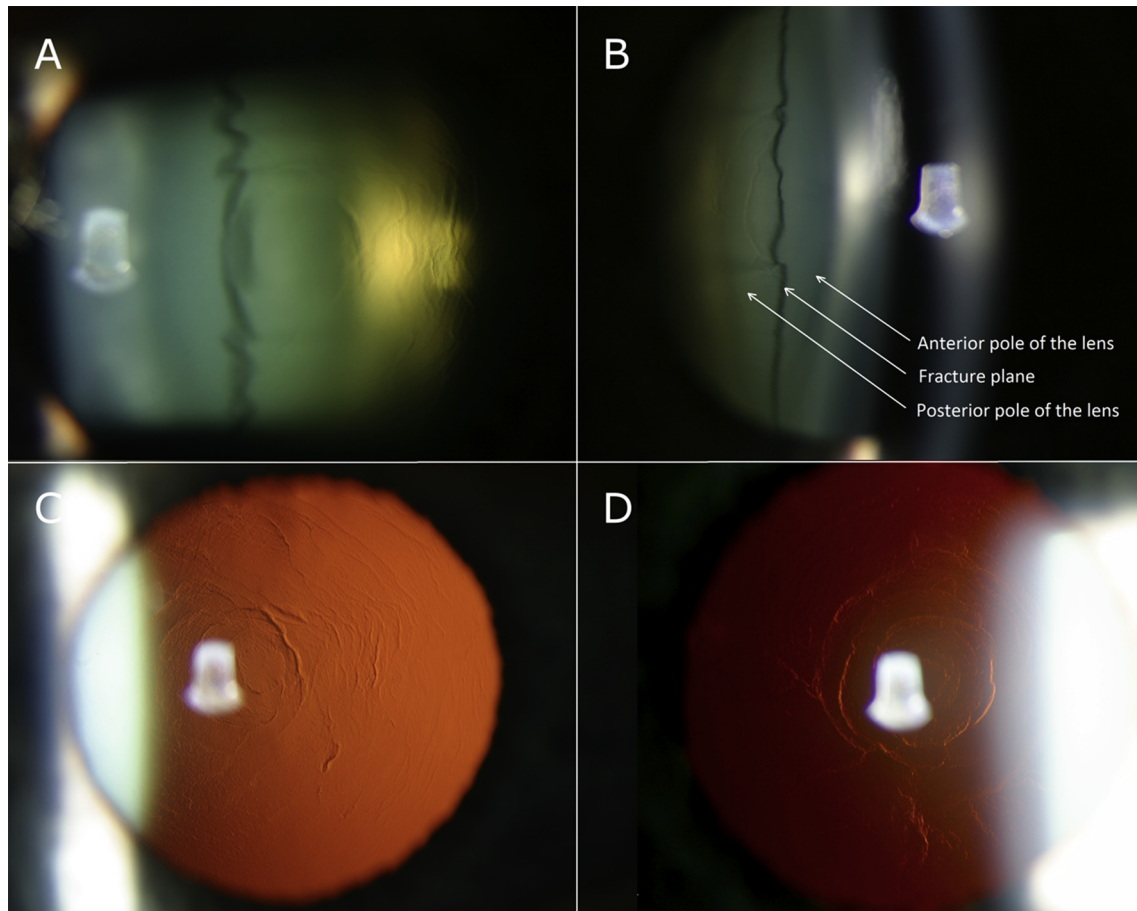


Fig. 1. Color photographs demonstrating bilateral cataracts created by lens fracturing. Narrow slit beam illumination demonstrates cross section of the fracture planes within crystalline lenses of the right eye (panel A) and the left eye (panel B). Retroillumination technique is used to highlight the extent of the cataracts in the right eye (panel C) and the left eye (panel D).

characteristic signs of advanced sclerosis clinically. His cataracts were not a reversible lens opacification that could be attributable to acute swelling of individual lens fibers but rather were caused by gross fracture. We hypothesize that forces leading to lens fracture in our patient developed in the course of intumescence of the lens as a unit, which resulted in a mechanical strain on a relatively sclerotic and, therefore, non-elastic and brittle nucleus. This mechanism is supported by experimental evidence in animal models. When exposed to osmotic stress, the rabbit lens displays swelling of not only individual lens fibers but as a unit also by accumulating water in the interfibrillary spaces.¹⁵ A similar observation had been made in the amphibian lens exposed to a hyperglycemic medium.^{16,17} Additionally, acute lens swelling resulting in acute angle closure glaucoma had been reported in a human patient undergoing treatment for HHS.¹⁸

While no direct biometric data that would explain the observed hyperopic shift were collected, we speculate that this phenomenon is the result of the lens fracture itself. The fracture plane essentially split each of the biconvex crystalline lenses into two approximately plano-convex lenses with the convex surfaces facing away from one another. This resulted in an increase in the antero-posterior dimension of the lens apparatus. In the absence of changes in the convexity of the anterior or posterior surfaces of the apparatus, this increased separation between the two poles of the lens would result in the posterior focal point of the system being displaced farther back relative to the anterior principle plane of the previous solid lens (or, alternatively, the principle plane of the anterior lens

of the newly created system). Unless there exists a net forward displacement of the entire lens apparatus, the described changes are expected to result in a hyperopic shift, which is consistent with our clinical observation.

4. Conclusions

Herein we have described a novel type of bilateral irreversible cataract that formed via a lens fracture in the coronal plane, a configuration reminiscent of a “split pea”. The cataract had developed shortly after correction of HHS in an elderly diabetic patient. This highlights the possibility of cataract formation as a rare ocular complication of treatment of hyperglycemic states. Additionally, this clinical presentation is consistent with and supports the notion of osmotic stress force generated in the lens as a whole as opposed to stress of only individual lens fibers as a mechanism that may play a role in diabetic cataract formation.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Conflict of interest

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Disclaimer requirement

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