Reversible Cataract as the Presenting Sign of Diabetes Mellitus: Report of Two Cases and Literature Review

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Abstract

Background: Type 1 diabetes mellitus (T1DM) is the most common form of diabetes in young children. Serious optic complications, e.g., diabetic retinopathy and diabetic cataract involvement, are not usually detected in T1DM patients at the onset of the disease.

Case Presentation: Two girls aged 11 years and 9 years were hospitalized in our unit in 2008 and 2009. They presented cataracts 1 and 6 months before the diagnosis of T1DM, respectively. After blood glucose level was controlled by insulin therapy, the cataract was resolved, totally in one and partly in the other girl. Meanwhile, visual acuity of both cases recovered, closely associated with fluctuation of plasma glucose level. In this study, we describe the symptoms, probable mechanism and treatment of diabetic cataract.

Conclusion: Early antihyperglycemic therapy and maintenance of stable blood glucose level may reverse acute diabetic cataract or prevent it from getting worse.

Key Words: Type 1 diabetes mellitus; Acute diabetic cataract; Visual acuity; Children

Introduction

Diabetes mellitus has a myriad of complications including cataract which is manifests more frequently and at an earlier age than in nondiabetics. Type 1 diabetes mellitus associated cataract is defined as true diabetic cataract characterized by diffuse posterior and/or anterior, subcapsular or cortical 'snow-flake opacities' [1]. The prevalence of diabetic cataract in young children and adolescents varies from 0.7% to 16%, depending on the different studies, areas and countries [1-4]. It is reported that most patients had lenticular opacity after diagnosis of diabetes mellitus for more than five years [5]. The development of acute metabolic cataract in T1DM patients at the onset of the disease is rare and infrequent in the literature, and most of them had to undergo a cataract surgery to restore vision[6-13] there were only two children whose cataract resolved completely by controlling blood glucose[14,15]. Herein, we present two similar cases, describing subsequent reinstatement of near total lenticular transparency in one girl and partly restoration in the other girl after blood glucose concentration was controlled to highlight the
clinical feature, diagnosis, treatment of this rare event, and also provide a new way to judge the severity of diabetes.

Case Presentation

Case 1
An 11-year-old girl presented with symptoms of blurry vision and progressive loss of vision for 1 month. Her vision deteriorated rapidly over the subsequent month without headache, polyuria, polydypsia or polyphagia. Body weight had no significant change. Past history of the patient was unremarkable with no trauma, radiation exposure, maternal or actual infection, or drug use.

Ophthalmological examination showed 20/50 vision in the right eye and 20/66 in the left eye, and confirmed diffuse white cortical changes as well as posterior subcapsular cataract. The pupils reacted normally to light. Fundus oculi was visible after mydriasis. Ultrasonography showed bilateral cataract without vitreous opacity or retinal detachment. Electrophysiology (ERG) showed slightly reduced amplitudes of a and b waves for the left eye. No abnormality was found by fluorescein fundus angiography and visual evoked potentials (VEP) test.

Initial laboratory tests showed a random blood glucose of 23.3 mmol/L (420mg/dL), glycated hemoglobin (HbA1c) 30% (target range <7%), C-peptide 0.38 ng/ml (normal range 0.6-3.8 ng/ml), and insulin less than 2.0 IU/l (normal range, 2.0-18.0 mIU/ml); acid-base status was normal, glucose in urine and ketonuria were negative. Head CT scan was normal.

Immediately after T1DM diagnosis was established, the patient received insulin therapy (Novolin 30R, 29 IU per day: 19 IU before breakfast and 10 IU before supper). She was followed up periodically for assessment of cataract, visual acuity, biochemical tests, plasma glucose levels, and HbA1c level every 3 months. Her uncorrected visual acuity changed as follows: improved to 20/40 in the right eye and 20/50 in the left eye during the first 5 months after therapy; to 20/25 in the right eye and 20/40 in the left eye, slit lamp showed clear corneas and quiet anterior chambers, especially in the right eye, the cataract disappeared eight months later so as if it had never happened. In examination after one year her visual acuity had regressed to 20/40 in the right eye and 20/55 in the left eye. However, most interesting was HbA1c level that was always 7-7.1% and the blood glucose concentration was almost normal. After careful inquiry and analysis, we found that her visual acuity and cataracts were closely related to the fluctuation of plasma glucose level. Her blood glucose has been under strict control with no significant fluctuations since then. The latest ocular examination showed a visual acuity of 20/22 in the right eye and 20/25 in the left eye; also it was easy to see the fundus oculi after mydriasis with a near total lenticular transparency.

Case 2
A 9-year-old girl complained of loss of vision in both eyes for 6 months without headache, polyuria, polydypsia or polyphagia. There was no relevant medical history.

In physical examination cardiovascular, respiratory, digestive and neurologic systems were normal. On biomicroscopic examination, diffuse posterior subcapsular cataract with snowflake opacity was detected in the left eye and dense snowflake in the right eye. Intraocular pressure was 14 mmHg in the left eye and 16 mmHg in the right eye. B-scan ultrasonography showed slight vitreous opacity in both eyes, but no retinal detachment. VEP tests showed slightly longer peak time of both eyes’ P100, and reduced amplitude of a and b waves in ERG for the right eye. The pupils were sluggishly reacting. No abnormality was found by fluorescein fundus angiography. However, fundus oculi of the right eye was invisible after mydriasis due to cataract.

Laboratory data showed a random blood glucose level of 25.5 mmol/L, HbA1c level was 31%, blood pH 7.314, glucose and ketonuria in urine were negative.

The diagnosed T1DM was treated with insulin (15 units of regular insulin before breakfast, 10 units before lunch and 10 units before supper and 3 units before sleep). Through controlling the blood glucose, visual acuity of 20/80 improved to 20/50 after a period of 3 months, and to 20/40 after 1 year therapy in the left eye, which could be corrected to 20/20 via glass (+3.00D.S○3.00D.C×10°); while the right eye’s acuity reached
from 20/180 to 20/125 after one year and could not be corrected, so it was advised to undergo cataract surgery when the blood glucose concentration was stable. In the meantime her fasting blood glucose level was unstable and HbA1c level was 9.8%.

Discussion

Diabetic retinopathy and diabetic cataract are two most common optic complications of diabetes, which are unusual to be recognized in early stage of diabetes, especially in prepubertal children [16]. ERG is often used to evaluate retino-cortical conduction in order to discover the functional prognosis [17]. Although clinical examinations showed that these two cases could not be diagnosed as diabetic retinopathy, the ERG changed, revealing some retinal lesions.

Regressed of diabetic cataract to clear lens after good glycemic control has been rarely reported, we found only two in children with T1DM [14, 15].

Both of our cases presented vision changes as a first manifestation without any headache, polyuria, polydipsia or polyphagia, and had no history of trauma, radiation exposure, maternal or actual infection, or drug use. Such cases can easily be misdiagnosed or missed diagnosed, which may end up with complications such as diabetic retinopathy which is irreversible. In our cases, case 1 had T1DM of only 1 month duration and antihyperglycemic treatment and supportive therapy to save visual acuity initiated immediately after diagnosis. Her blood sugar level and glycated hemoglobin was almost within normal range. So after one and half years of therapy, the lens opacities resolved and visual acuity improved from 20/50 to 20/22 in the right eye and from 20/66 to 20/25 in the left eye. However, in patient 2 who had 6 months longer duration of diabetes before diagnosis, combined with poor control of blood glucose level, resulted in worse visual acuity, which regressed only partly. We advised cataract surgery of the left eye, as it could not be corrected and the lens remained cloudy. We agree with Moshe [15] that to save visual acuity, shorter duration of T1DM symptoms before treatment is essential.

It is most noteworthy that in case 1, HbA1c showed almost stable values, as the visual acuity changed during the period of treatment. Only recalling the history, we found that her blood sugar level oscillated within normal range. Studies have shown that drop in serum glucose causes a drop in aqueous humor glucose [10,19]. Fluctuations in the level of aqueous humor glucose cause marked changes in lens membrane permeability. When lens is exposed to hyperosmotic solution, volume initially decreases, and then with the entry of glucose and the accompanying water, the lens gradually swells, resulting in formate myopia. On return to exposure to hypoosmotic solution, the swollen lens encounters a hypoosmotic shock and swells further. Thus, these fluctuations are potentially damaging because there is a double osmotic shock at each change [10]. Therefore, we presume that blood sugar level oscillation is responsible for the patient’s visual acuity change.

The pathogenesis of reversible cataracts in diabetes is incompletely understood. Some researchers presumed that the transient nature of the diabetic cataract was related to changes in lens hydration and cataract may be reversible with the normalization of hydration [15,18]. In addition, it is postulated that lens can slowly metabolize its contained sorbitol and reverse the water accumulation through cellular pumping mechanisms [14]. Finally, formation of new fibers is supposed to play a prominent role in the reversibility [14,18].

There have been several case reports of acute diabetic cataracts antedate the diagnosis of diabetes and all of them had a cataract surgery to restore vision [6-13]. Although the overall outcomes of cataract surgery are excellent, patients with diabetes may have poorer vision outcomes than those without diabetes [5]. Studies showed that surgery may cause a rapid acceleration of retinopathy, induce rubeosis or lead to macular changes, such as macular edema or cystoid macular edema. Moreover, surgical trauma, stress and routine clinical application of corticosteroids will cause blood glucose to rise [5,20,21].

We suggest diabetes-induced acute cataract not to be operated when visual acuity is greater than 20/66 in the early stage, since monitoring visual acuity can reflect patient’s blood glucose situation, especially the fluctuation. After all, it is feasible.
and simple to monitor the visual acuity through visual acuity chart for the families.

**Conclusion**

Good control of plasma glucose level as well as no gross fluctuation can regress acute cataract totally or partly. Heightened awareness of monitoring visual acuity frequently can not only contribute to reflect the state of cataract, but also can help to know whether the plasma glucose fluctuates.

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**References**

1. Falck A, Laatikainen L. Diabetic cataract in children. *Acta Ophthalmol Scand* 1998;76(2):238-40.
2. Montgomery EL, Batch JA. Cataracts in insulin-dependent diabetes mellitus: sixteen years' experience in children and adolescents. *J Ped Child Health* 1998;34(2):179-82.
3. Klein BE, Klein R, Moss SE. Prevalence of cataracts in apopulation-based study of persons with diabetes mellitus. *Ophthalmology* 1985;92(9): 1191-6.
4. Wilson ME Jr, Levin AV, Trivedi RH, et al. Cataract associated with type-1 diabetes mellitus in the pediatric population. *JAPOS* 2007;11(2):162-5.
5. Pollreisz A, Schmidt-Erfurth U. Diabetic cataract-pathogenesis, epidemiology and treatment. *J Ophthalmol* 2010; 2010:608751.
6. Patel CM, Plummer-Smith L, Ugrashul F. Bilateral metabolic cataracts in 10-yr-old boy with newly diagnosed type 1 diabetes mellitus. *Pediatr Diabetes* 2009;10(3):227-9.
7. Wilson ME Jr, Levin AV, Trivedi RH. Cataract associated with type-1 diabetes mellitus in the pediatric population. *JAPOS* 2007;11(2):162-5.
8. Costagliola C, Dell'Omo R, Prisco F, et al. Bilateral isolated acute cataracts in three newly diagnosed insulin dependent diabetes mellitus young patients. *Diabetes Res Clin Pract* 2007;76(2):313-5.
9. Taskapili M, Gulkilik G, Oztucu M, Kucukahsin H. Acute bilateral dense cortical cataracts as a first manifestation of juvenile diabetes mellitus in a 12-year-old girl. *J Pediatr Ophthalmol Strabismus* 2008;45(3):177-8.
10. Santiago AP, Rosenbaum AL, Masket S. Insulin-dependent diabetes mellitus appearing as bilateral mature diabetic cataracts in a child. *Arch Ophthalmol* 1997;115(3):422-3.
11. Scarppita AM, Perrone P, Sinagra D. The diabetic cataract: an unusual presentation in a young subject: case report. *J Diabetes Complications* 1997;11(4):259–60.
12. Niederland T, Futo G, Gal V. Acute metabolic cataract as a first manifestation of IDDM in an adolescent girl. *Orv Hetil* 1999;140(35):1953–6.
13. Lebinger TG, Goldman KN, Saenger P. Bilateral cataracts as the initial sign of insulin-dependent diabetes mellitus in a child. *Am J Dis Child* 1983;137(6):602–3.
14. Trindade F. Transient cataract and hypermetropization in diabetes mellitus: case report. *Arg Bras Oftalmol* 2007;70(6):1037-9.
15. Phillip M, Ludwick DJ, Armour KM, et al. Transient subcapsular cataract formation in a child with diabetes. *Clin Pediatr (Phila)* 1993;32(11):684-5.
16. Silverstein J, Klingensmith G, Copeland K, et al. Care of children and adolescents with type 1 diabetes: a statement of the American Diabetes Association. *Diabetes Care* 2005;28(1):186-212.
17. Tormene AP, Riva C. Electroretinogram and visual-evoked potentials in children with optic nerve coloboma. *Doc Ophthalmol* 1998-99;96(4):347-54.
18. Sharma P, Vasavada AR. Acute transient bilateral diabetic posterior subcapsular cataracts (1). *J Cataract Refract Surg* 2001; 27(5):789-94.
19. Obrosova IG, Chung SS, Kador PF. Diabetic cataracts: mechanisms and management. *Diabetes Metab Res Rev* 2010; 26(3):172-80.
20. Shah AS, Chen SH. Cataract surgery and diabetes. *Curr Opin Ophthalmol* 2010;21(1):4-9.
21. Goldman DA, Trattler WB. Cataract surgery and diabetic retinopathy. *Ophthalmology* 2010;117(4): 850-1.