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A Systematic Study of Cataract Surgery in People with Diabetes Mellitus

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ABSTRACT
India is regarded as the diabetes capital of the world, and diabetic patients make up a sizable share of cataract surgery patients. In light of this, we evaluated the fundamentals and recommendations for treating cataract in people with diabetes. When treating patients with diabetic cataracts, the preoperative, intraoperative, and postoperative aspects are of the utmost significance. Prior to cataract surgery, early detection and treatment of diabetic retinopathy or maculopathy have a significant impact on the final visual prognosis and are crucial in the perioperative decision-making process. We may be able to better manage these individuals overall and improve the outcomes if we have a better grasp of the numerous elements that contribute to cataract surgery in diabetic patients having a favourable outcome.

Keywords: diabetic retinopathy, treatment outcome, vitrectomy, cataract, diabetes mellitus.

1. INTRODUCTION

More than 285 million people around the world have diabetes mellitus. The International Diabetes Federation predicts that by 2030, this number would nearly double to 439 million. [1,2] As of 2015, there were 69 million Indians living with diabetes mellitus. [3] Patients with diabetes are 2-5 times more likely to acquire cataracts, and it typically does so earlier in life. [3] On the other hand, microangiopathy brought on by diabetes causes nephropathy, neuropathy, and diabetic retinopathy (DR). Retinal neurodegeneration has recently been linked to diabetes and hypothesised to precede or coexist with retinal vasculopathy, aggravating it. [4-6] Clinical signs of diabetic retinal neurodegeneration include shrinkage of the retinal nerve fibre layer, the ganglion cell layer, and the Muller cells,[7–10] loss of colour vision, and alterations in spatial frequency on electrophysiology. [11,12] Diabetes affects not just the retina but also the cornea, tear film, and crystalline lens, which affects the optical

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quality of the diabetic eye. These structural and morphological changes also affect the retina. [13]

The largest cause of blindness in the world, impacting almost 18 million people, is still cataract. [1,2] Due to the fact that cataract incidence and development are higher in patients with diabetes mellitus, cataract is regarded as a primary cause of visual impairment in diabetic patients. [14] Several clinical trials have demonstrated that cataracts affect diabetics more frequently and younger in age. [15] Up to 20% of all cataract operations are thought to be carried out on diabetic individuals. [16]

According to some research, doing cataract surgery on diabetes patients may cause DR to advance rather quickly, trigger vitreous haemorrhage, cause iris neovascularization, and ultimately impair or impair vision.

[17] Increased inflammatory cytokines are produced in the eye after uneventful cataract surgery. [18,19] According to Patel et al., significant increases in vascular endothelial growth factor (VEGF), hepatocyte growth factor, interleukin-1 (IL-1), and pigment epithelium derived factor concentrations occurred 1 day after uneventful phacoemulsification and intraocular lens (IOL) implantation. It took up to 1 month for these increases to return to preoperative levels. These cytokines may cause DR and maculopathy to deteriorate clinically or subclinically. [18] Even though there have been numerous studies on the development of DR status and the factors that affect this development after cataract surgery, more research is still needed, particularly in the longer term. Previously, biomicroscopy and fundus fluorescein angiography were used as a basis for monitoring changes in DR, particularly maculopathy.

The noninvasive monitoring of changes in retinal thickness in DR, including postcataract surgery, has improved with the recent use of optical coherence tomography (OCT), particularly spectral domain models. [20] With a focus on the connection between cataract surgery and the development of DR, this article examines the onset and evolution of cataract as well as cataract surgery and its results in diabetic patients.

2. METHODS

The Preferred Reporting Items for Systematic Reviews and Meta-analyses statement was followed when doing this literature review to ensure its accuracy.

The Cochrane library, EMBASE, and PubMed online biomedical search engines were used to conduct a thorough search of the literature for publications. Diabetes AND (cataract) AND (timing OR pathophysiology OR phacoemulsification OR consequences OR visual outcome OR visual prognosis OR retinopathy progression OR diabetic macular edoema) were the search phrases used. All pertinent publications were included in this review, which was conducted by two authors who identified the articles. Our literature search filters were limited to papers written in English and published between 1987 and June 2017. These papers could have been systematic reviews, cohort or retrospective studies, case-control series, or human studies published as randomised controlled trials or nonrandomized comparative studies.

3. RESULTS

The visual result after cataract surgery was provided as an endpoint in 14 studies out of the 105 studies total included in this review study. Poor visual result was the outcome variable used in the meta-analysis[21]. In terms of poor visual outcomes following cataract surgery, it

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is evident that there is a large amount of heterogeneity being reported in the literature (QStatistic 386.2768, P 0.001). The proportions with 95% confidence intervals discovered in the studies included in the meta-analysis forest plot. A graphical tool for identifying bias in the meta-analysis. The funnel plot's asymmetry provides unmistakable proof of publishing bias. MedCalc Statistical Software, version 18.2.1 (MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org; 2018), was used for the meta-analysis.

4. DISCUSSION

Pathogenesis of diabetic cataract

Diabetic patients experience different kinds of lens alterations. A prevalent kind of cataract in people with type 1 diabetes is the snowflake cataract. However, senile cataracts are the most common kind to be found in diabetics. [23] Numerous studies have been conducted to investigate the type of cataract connected to diabetes. Diabetes has been proven to be substantially related with posterior subcapsular cataract. [24] In fact, several researchers have found a link between higher levels of glycated haemoglobin and a higher risk of neural and cortical cataract. [25] Further investigation had revealed that the likelihood of developing cortical cataract in diabetic patients increased with the length of their diabetes. [26]

Numerous pathways have been hypothesised for the cataract aetiology in diabetes mellitus. In the lens, the enzyme sorbitol dehydrogenase produces sorbitol more quickly than it converts it to fructose, a process that is more pronounced in diabetics than in non-diabetics. [27] An infusion of fluid is produced to counteract the osmotic gradient as a result of the hyperosmotic impact caused by the increased sorbitol buildup. Increased intracellular synthesis of cytokines/growth factors, oxidative stress, and hyperglycemia per se all contribute to osmotic retention of fluid in the lens fibres and the resulting osmotic stress. [28‑30] Osmotic stress plays a significant influence in the rapid development of cataracts in young patients with Type 1 diabetes. a result of the substantial swelling of the cortical lens fibres [31,32]. Superoxide radicals and hydrogen peroxide (H2 O2) are produced as a result of the accumulation of sorbitol and advanced glycation endproducts. Antioxidant enzymes often aid in the conversion of superoxide radicals into H2 O2 and oxygen. However, diabetes compromises lens antioxidant enzymes like superoxide dismutase and catalase, causing oxidative stress that aids in cataract development. [33‑35]

Pathophysiological changes in crystalline lens with fluctuations in blood glucose levels

Myopia shifts as a result of hyperglycemia, which is also the main factor in diabetic patients' transientrefractive alterations. Many people who receive rigorous medical treatment have a tendency to develop hyperopia as opposed to hyperglycemia. It is believed that both morphologic and functional alterations in the crystalline lens are responsible for the refractive changes that are seen during periods of unstable blood sugar. [36] In addition, adjustments for keratorefractive and cataract surgery could be inaccurate because changes in corneal topography parameters during hyperglycemia variations. Diabetes is known to cause the lens's basement membrane, also known as the lens capsule, to thicken, which can alter vision.

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Over a 5-year period, Saxena et al. discovered a two-fold increased incidence of cortical cataracts in patients with diabetes mellitus and proposed the same cause.

Timing of cataract surgery

The trend among diabetic individuals to undergo cataract surgery early has helped to enhance visual results. When cataract surgery is delayed until it is too late to properly diagnose or treat DME, the visual consequences are likely to be worse for the patient. The risk of DME is reduced, and the visual prognosis may be noticeably improved, when cataract surgery is performed before lens opacities limit appropriate macular evaluation and the detection of retinal thickening.

Preoperative considerations

Counseling before to surgery is essential. Patients should have appropriate glycemic control and no signs of an eye or periocular infection prior to surgery. Compared to non-diabetics, some conjunctival microorganisms, such as Staphylococcus aureus, Enterococci, specific Streptococci, and Klebsiella species, are more common in diabetic individuals. Therefore, in these patients, the importance of complete asepsis cannot be overstated. A thorough and comprehensive ophthalmologic examination is required, and it must include measurements of visual acuity (VA), best-corrected VA, relative afferent pupillary defect, tonometry, dilated fundoscopy and gonioscopy (in patients with high-risk PDR with a focus on new vessels), as well as slit-lamp biomicroscopy to check for iris neovascularization and corneal health. In some circumstances, ancillary diagnostic tests such fluorescein angiography, OCT, and B-scan ultrasonography may be beneficial.

Patients who have PDR before to cataract surgery are more likely to worsen and experience vitreous haemorrhage and other retinal problems quickly. PRP is therefore advised prior to surgery. When preoperative PRP is not possible due to lens opacity, indirect PRP may be provided either right away after cataract surgery or within a week of the treatment.

In contrast, combining cataract surgery with vitrectomy and endolaser photocoagulation may be performed, particularly in situations of extensive vitreous haemorrhage or in eyes with posterior pole tractional retinal detachment (TRD). According to several research, simple phacoemulsification contemporary surgery does not cause the development of DR, and the development of DR following cataract surgery is primarily attributable to inadequate glycemic control. In light of the contradicting findings on this particular subject, extensive investigations may provide a clearer picture in the future.

Prior to surgery, ME should receive proper treatment because preexisting maculopathy may worsen, leading to a poor visual prognosis. Laser photocoagulation or intravitreal injections of steroids or anti-VEGF medications are two possible treatments for maculopathy. Increased retinal vascular permeability factors including IL-6 and VEGF are well-known to have a significant role in diabetic ME (DME). Cataract surgery can raise the risk of ME progression or redevelopment by 20% to 50% in patients who already have DME or have previously received therapy for it.

Therefore, in these situations, intravitreal steroids and anti-VEGF medications are a viable choice. Although these patients require continued care, it has been demonstrated that intraoperative intravitreal ranibizumab with cataract surgery is more successful than preoperative or postoperative injection for DME. Contrarily, steroids have demonstrated efficacy in treating persistent or resistant DME. Comparing dexamethasone to fluocinolone

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acetonide and triamcinolone acetate, there may be less of a chance for cataract development and an increase in intraocular pressure (IOP) with dexamethasone. In addition, less frequent injections are needed for fluocinolone and intravitreal dexamethasone implants. Over a period of three years, studies on intravitreal dexamethasone implants have demonstrated a significant improvement in clinically significant ME (CSME). In fact, it has also been demonstrated to slow the development of DR. Furthermore, a recent study demonstrated that individuals who underwent cataract surgery after receiving a preoperative fluocinolone acetonide 0.2 g implant had positive visual outcomes. Recent research has raised the possibility that nonsteroidal anti-inflammatory drug use before to surgery could lessen the likelihood of postoperative ME in patients with DR. Examples of these treatments include diclofenac and nepafenac.

Before having cataract surgery, patients with neovascularization of the iris (NVI) require immediate treatment to cause regression. Traditionally, PRP was used to do this. Medical treatment alone typically is ineffective when neovascular glaucoma (NVG) develops. IOP may be lowered and inflammation may be reduced with the help of topical beta-adrenergic antagonists, alpha-2-adrenergic agonists, carbonic anhydrase inhibitors, cycloplegics, and corticosteroids. When anti-VEGF drugs were injected intravitreally, eyes with NGV revealed a dramatic short-term reversal of neovascularization and a reduction in IOP. Phacoemulsification should be considered as soon as NVI starts to retreat, either with or without vitrectomy, to allow for the therapy of the posterior segment pathology. In contrast, phacoemulsification and endoscopic diode laser cyclophotocoagulation offer an alternative treatment for eyes with closed anterior chamber angles and mature (fibrosed) new arteries. When NVI has receded, trabeculectomy and phacoemulsification may also be planned in eyes with concurrent NVG and cataract. However, in eyes with NVG, the visual outcomes after phacoemulsification are typically subpar.

Cataract surgery (intraoperative considerations)

In comparison to extracapsular cataract surgery, phacoemulsification with IOL insertion produces better visual results and less irritation. In diabetic eyes, anterior capsular phimosis is more prevalent. To avoid anterior IOL displacement and posterior capsular opacification, the capsulorhexis size should be bigger than usual but smaller than IOL optic diameter (PCO). Additionally, postoperative detection and treatment of peripheral retinal pathology are made easier with a big diameter optic (6.0 mm or larger). Longer and more difficult cataract surgeries carry a higher risk of retinopathy progression and ensuing vision impairment. Due to disruption to the pupillary parasympathetic supply, inadequate pupillary dilation is quite likely in diabetes patients. For intraoperative use in these patients, iris hooks, malyugin rings, or other iris expanders should be taken into consideration. Additionally, problems such intraoperative hyphema brought on by rubeosis iridis must be considered. According to some research, intravitreal anti-VEGF medications used prior to surgery may reduce the risk of iris neovascularization-related haemorrhage. According to Cetinkaya et alresearch .'s, diabetes patients were more likely than non-diabetics to get photic retinopathy during cataract surgery. The risk of intraoperative complications including posterior capsular rupture, zonular dehiscence, and vitreous loss is not increased by the mere presence of diabetes. However, keratoepitheliopathy, including corneal epithelial defects/abrasions, which may heal slowly, is more common in diabetic eyes.

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Numerous factors, including neurogenic (anomalies of the subbasal nerve) and defective corneal stem cell and epithelial cell division, contribute to the impaired corneal wound healing. Additionally, studies have indicated that diabetics have a higher propensity to lose corneal endothelial cells than non-diabetics. For this reason, routine specular microscopy is advised for all diabetics, and extra caution should be used to protect endothelial cells while operating on diabetic patients.

Intraocular lens choice

For easier viewing and treatment of the peripheral retina in DR, large diameter IOLs are preferred. Patients with diabetes appear to experience more severe PCO than non-diabetics. Diabetes patients, especially those with PDR, have much greater levels of phosphorus in their serum and aqueous humour than healthy people, which might cause hydrophilic acrylic IOLs to become opaque. In the early postoperative phase, anterior chamber flare is slightly more common with hydrophobic acrylic IOLs. Hydrophobic acrylic lenses should be the IOL of choice in diabetic patients who anticipate vitreoretinal surgery since they have the lowest propensity for silicone oil adhesion and PCO develops less commonly than these lenses.

Visual prognosis following cataract surgery

Due to improved preoperative retinopathy management, advancements in operative techniques, improved glycemic and hypertensive control, and improved surgical technique of phacoemulsification, recent studies on cataract surgery in diabetics tend to report a lower incidence of complications and better visual outcomes. Patients with diabetes who have little to no retinopathy have a good visual prognosis comparable to that of people without diabetes. Poor preoperative VA (representing diabetic maculopathy, ischemia, and traction) and the presence of DME have both been identified as risk factors for poor postoperative VA after cataract surgery.

Cataract surgery and intravitreal injection

When doing cataract surgery on eyes with DME but no epiretinal membrane or tractional component, intravitreal steroids may be taken into account, especially if the patient has never received treatment before. Since 2005, neovascular and exudative ocular disorders have been treated with intravitreal injections of bevacizumab (Avastin, Roche). Since then, a number of modest studies have examined how intravitreal bevacizumab affects diabetic neovascular problems.

Combined cataract surgery and vitrectomy

Cataracts frequently accompany with diabetic individuals undergoing vitrectomy. Furthermore, after vitrectomy, lens opacities frequently get worse. In cases of coexisting cataract and non-clearing vitreous haemorrhage, macular TRD, mixed mechanism retinal detachment, and persistent DME that is not responding to intravitreal anti-VEGF drugs and/or steroids, phacoemulsification may be paired with pars plana vitrectomy. Numerous studies have showed that the vitreoretinal interface contributes to the formation of chronic DME after laser photocoagulation and have shown that combination surgery, when appropriate, significantly improves anatomic and visual outcomes.

Effect of cataract surgery on retinopathy

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After phacoemulsification surgery, some studies have shown DR advancement, while others have shown no discernible change. Patients with elevated haemoglobin A1c have a higher chance of developing DR after cataract surgery, according to Squirrell et al.. Krepleret al. discovered in a retrospective analysis that DR was linked to male sex, a protracted illness, and inadequate glycemic management.

Effect of cataract surgery on macular edema

Following cataract surgery, altered angiogenic factor concentrations may exacerbate maculopathy. After a trouble-free cataract procedure, OCT imaging revealed thicker retinas in diabetes eyes without retinopathy compared to non-diabetic eyes.

Differentiating diabetic macular edema from pseudophakic macular edema in diabetic patients

DME and pseudophakic cystoid ME (PCME) should be distinguished from one another, particularly because PCME is more common in patients with diabetes. In these two entities, the pathophysiology, therapy, prognosis, and natural history are all quite different: (1) The presence of underlying DR, exudates, and ME indicates DME (2) A PCME may be present if there is no or little DR and no exudates in the posterior pole. When in doubt, perform a fundus fluorescein angiography, which shows optic disc staining (also known as a "hot disc") and typical petalloid dye pooling in PCME, whereas the disc is often normal and leaking micro-aneurysms and/or capillary plexus are recognised as the causes of ME in DME.

Postoperative consideration

Due to decreased corneal innervation, diabetic individuals are more likely to develop corneal epithelial abnormalities and chronic erosions; these occur more frequently as patient age and diabetes duration rise. Following cataract surgery, diabetes patients' eyes had more severe corneal endothelial cell loss and took longer to recover from corneal edoema than normal. Diabetic patients are more likely to experience other anterior-segment problems include severe iritis, posterior synechiae, pupillary block, and pigmented precipitates on the IOL.

Modern cataract surgery, which is less stressful than older procedures, has helped to lower the occurrence of NVI, the most dreaded anterior segment consequence in diabetes patients. Additionally, it has been claimed that PRP and intravitreal anti-VEGF injections can temporarily suppress NVI. [57] Patients with diabetes may be more likely to develop postoperative endophthalmitis, which is linked to a poor prognosis for vision.

5. CONCLUSION

People who have diabetes mellitus are becoming more and more numerous. Diabetes patients have not always experienced the same positive results from cataract surgery as people without diabetes. According to the severity of the DR, diabetic patients with visually significant cataracts pose particular obstacles during surgery and subsequent rehabilitation. However, these patients recover successfully and regain great vision just as other cataract patients without diabetes with appropriate pretreatment of the DR and minimally invasive surgical procedures. Systemic and ocular diseases require special attention.

Surgery on diabetes patients might be safer and more successful with the use of contemporary pharmacologic and surgical treatments. This demonstrates how crucial patient education is prior to surgery.

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6. REFERENCES

1. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030. Diabetes Care 2004;27:1047-53.
2. World Health Organization.Available from: http://www.who.int/ blindness/causes. [Last accessed on 2006 Sep 07].
3. International Diabetes Federation (IDF). DiabetesAtlas. 7th Edition, International Diabetes Federation, Brussels, Belgium. 2015. http:// www.diabetesatlas.org. [Last accessed on 2015 Nov 06].
4. Fle t che r EL, Phipps JA, Ward MM, Puthussery T, Wilkinson-Berka JL. Neuronal and glial cell abnormality as predictors of progression of diabetic retinopathy. Curr Pharm Des 2007;13:2699-712.
5. Simó R, Hernández C; European Consortium for the Early Treatment of Diabetic Retinopathy (EUROCONDOR). Neurodegeneration is an early event in diabetic retinopathy: Therapeutic implications. Br J Ophthalmol 2012;96:1285-90.
6. Adams AJ, Bearse MA Jr. Retinal neuropathy precedes vasculopathy in diabetes: A function-based opportunity for early treatment intervention? Clin Exp Optom 2012;95:256-65.
7. Mizutani M, Gerhardinger C, Lorenzi M. Müller cell changes in human diabetic retinopathy. Diabetes 1998;47:445-9.
8. van Dijk HW, Kok PH, Garvin M, Sonka M, Devries JH, Michels RP, et al. Selective loss of inner retinal layer thickness in type 1 diabetic patients with minimal diabetic retinopathy. Invest Ophthalmol Vis Sci 2009;50:3404-9.
9. van Dijk HW, Verbraak FD, Kok PH, Garvin MK, Sonka M, Lee K, et al. Decreased retinal ganglion cell layer thickness in patients with type 1 diabetes. Invest Ophthalmol Vis Sci 2010;51:3660-5.
10. van Dijk HW, Verbraak FD, Stehouwer M, Kok PH, Garvin MK, Sonka M, et al. Association of visual function and ganglion cell layer thickness in patients with diabetes mellitus type 1 and no or minimal diabetic retinopathy. Vision Res 2011;51:224-8.
11. Di Leo MA, Falsini B, Caputo S, Ghirlanda G, Porciatti V, Greco AV, et al. Spatial frequency-selective losses with pattern electroretinogram in type 1 (insulin-dependent) diabetic patients without retinopathy. Diabetologia 1990;33:726-30.
12. Di Leo MA, Caputo S, Falsini B, Porciatti V, GrecoAV, Ghirlanda G, et al. Presence and further development of retinal dysfunction after 3-yearfollow up in IDDM patients without angiographically documented vasculopathy. Diabetologia 1994;37:911-6.
13. Calvo-Maroto AM, Perez-Cambrodí RJ, Albarán-Diego C, Pons A, Cerviño A. Optical quality of the diabetic eye: A review. Eye (Lond) 2014;28:1271-80.
14. Harding JJ, Egerton M, van Heyningen R, Harding RS. Diabetes, glaucoma, sex, and cataract: Analysis of combined data from two case control studies. Br J Ophthalmol 1993;77:2-6.
15. Benson WE. Cataract surgery and diabetic retinopathy. Curr Opin Ophthalmol 1992;3:396-400.
16. HamiltonAM,UlbigMW,Polkinghorne P, Sharma S.Management of Diabetic Retinopathy. Can Med Assoc J 1997;157:192.

17. Jaffe GJ, Burton TC, Kuhn E, Prescott A, Hartz A. Progression of nonproliferative diabetic retinopathy and visual outcome after extracapsular cataract extraction and intraocular lens implantation. Am J Ophthalmol 1992;114:448-56.
18. Patel JI, Hykin PG, Cree IA. Diabetic cataract removal: Postoperative progression of maculopathy – Growth factor and clinical analysis. Br J Ophthalmol 2006;90:697-701.
19. Dong N, Xu B, Wang B, Chu L, Tang X. Aqueous cytokines as predictors of macular edema in patients with diabetes following uncomplicated phacoemulsification cataract surgery. Biomed Res Int 2015;2015:126984.
20. Denniston AK, Chakravarthy U, Zhu H, Lee AY, Crabb DP, Tufail A, et al. The UK diabetic retinopathy electronic medical record (UK DR EMR) users group, report 2: Real-world data for the impact of cataract surgery on diabetic macular oedema. Br J Ophthalmol 2017;101:1673-8. 21.
21. Borenstein M, Hedges LV, Higgins JP, Rothstein HR. Introduction to Meta-Analysis. Chichester, UK: Wiley; 2009. 22.
22. Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. BMJ 1997;315:629-34. 23.
23. Pollreisz A, Schmidt-Erfurth U. Diabetic cataract-pathogenesis, epidemiology and treatment. J Ophthalmol 2010;2010:608751.
24. Rowe NG, Mitchell PG, Cumming RG, Wans JJ. Diabetes, fasting blood glucose and age-related cataract: The Blue Mountains Eye Study. Ophthalmic Epidemiol 2000;7:103-14.
25. Klein BE, Klein R, Lee KE. Diabetes, cardiovascular disease, selected cardiovascular disease risk factors, and the 5-year incidence of age-related cataract and progression of lens opacities: The Beaver Dam Eye Study. Am J Ophthalmol 1998;126:782-90.
26. Klein BE, Klein R, Wang Q, Moss SE. Older-onset diabetes and lens opacities. The beaver dam eye study. Ophthalmic Epidemiol 1995;2:49-55.
27. Kador PF, Wyman M, Oates PJ. Aldose reductase, ocular diabetic complications and the development of topical Kinostat(®). Prog Retin Eye Res 2016;54:1-29.
28. Obrosova IG, Chung SS, Kador PF. Diabetic cataracts: Mechanisms and management. Diabetes Metab Res Rev 2010;26:172-80.
29. Zhang P, Xing K, Randazzo J, Blessing K, Lou MF, Kador PF,et al. Osmotic stress, not aldose reductase activity, directly induces growth factors and MAPK signaling changes during sugar cataract formation. Exp Eye Res 2012;101:36-43.
30. Hashim Z, Zarina S. Osmotic stress induced oxidative damage: Possible mechanism of cataract formation in diabetes. J Diabetes Complications 2012;26:275-9.
31. Wilson ME Jr., Levin AV, Trivedi RH, Kruger SJ, Elliott LA, Ainsworth JR, et al. Cataract associated with type-1 diabetes mellitus in the pediatric population. J AAPOS 2007;11:162-5.
32. Datiles MB 3rd, Kador PF. Type I diabetic cataract. Arch Ophthalmol 1999;117:284-5.
33. Behndig A, Karlsson K, Reaume AG, Sentman ML, Marklund SL. In vitro photochemical cataract in mice lacking copper-zinc superoxide dismutase. Free Radic Biol Med 2001;31:738-44.
34. Olofsson EM, Marklund SL, Karlsson K, Brännström T, BehndigA. In vitro glucose-induced cataract in copper-zinc superoxide dismutase null mice. Exp Eye Res 2005;81:639-46.

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35. OlofssonEM,MarklundSL,BehndigA.Enhanceddiabetes-induced cataract in copper-zinc superoxide dismutase-null mice. Invest Ophthalmol Vis Sci 2009;50:2913-8.
36. Saito Y, Ohmi G, Kinoshita S, Nakamura Y, Ogawa K, Harino S, et al. Transient hyperopia with lens swelling at initial therapy in diabetes. Br J Ophthalmol 1993;77:145-8.