Journal of Pharmaceutical Research International

33(61A): 480-485, 2021; Article no.JPRI.80336
ISSN: 2456-9119
(Past name: British Journal of Pharmaceutical Research, Past ISSN: 2231-2919, NLM ID: 101631759)

Changes in Eyes in a Diabetic Patient

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Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JPRI/2021/v33i61A35881

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/80336

Review Article

Received 22 October 2021
Accepted 27 December 2021
Published 28 December 2021

ABSTRACT

Introduction: Diabetes mellitus (DM) (i.e., diabetes), a set of metabolic illnesses defined by chronically increased blood sucrose levels, is becoming more common worldwide. As a result of a pancreatic beta-cell failure, inadequate insulin is formed. Type 1 insulin is generated to efficiently apparent blood sucrose; type 2 insulin is formed to effectively clear blood glucose; type 3 insulin is produced (T2DM), characterized by insulin resistance. When the hormone fails to affect the target cells, gestational diabetes mellitus (GDM) arises while pregnant. Insulin resistance develops in pregnant women. Diabetes is the leading cause of visual defects in adults in the world. Diabetes causes problems in maximum parts of the eye, like in retina it causes retinopathy, in the lens it causes cataract, in lids- lashes the xanthelasma is more common, in conjunctiva the bacterial infections are more common as in diabetes they receive more nutrition and easy to increase, in cornea it causes keratopathy, and it is more severe it is also linked to tear function abnormality. Diabetes is a well-known significant factor for visual impairment as there are 12000 to 24000 new cases of visual loss every year. By 2030, it is expected that more than 342 million individuals worldwide will have diabetes, with the degree of diabetic consequences in various organs determining the total health burden.

Keywords: Diabetes; retinopathy; keratopathy; insulin.

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1. INTRODUCTION

Diabetes can damage your eyes over time, resulting in blurry vision or possibly blindness. Taking care of your diabetes, on the other hand, can help you avoid diabetic eye illness or keep it from worsening. Diabetic patients are more likely to develop glaucoma, cataracts, and other eye issues [1].

Hyperglycemia is hazardous to practically all of the body's cells. The cornea and retina are the most affected by hyperglycemia ophthalmic consequences [1]. In diabetic tear film, glucose levels are four times greater than in control tears. Patients with high blood glucose levels with corneal problems, also known as diabetic keratopathy, are approx 70% of all people with diabetes. Diabetic retinopathy has a higher prevalence and cause of blindness in adults over 50, with the retina accounting for the high amount of visual loss [2]. Furthermore, vascular alterations in the diabetic choroid are comparable to those seen in the diabetic retina. High blood glucose levels and the formation of high-level glycation final products significantly affect different areas of the cornea, which form tissue dysfunction that can be assessed physiologically.

(1) Inadequate corneal epithelial wound healing, (2) anomalies of subbasal nerves and (3) decrease of corneal endothelial pump function (1) Hyperglycemia stimulates IGFBP3 release, which competes with IGF1 to regulate it, whereas hyperglycemia suppresses TGFb3, EGFR, and CNTF [2].

The resultant decrease in epithelial cell division and increase in natural cell death affects epithelial wound healing. The high blood glucose level in diabetes causes nerve damage, which is the major flaw (2). Hyperglycemia for an extended period causes the buildup of highly modified glycation end products, which promote inflammation and oxidative damage.

NGF and sphingolipids are essential for brain health and myelin formation, but hyperglycemia inhibits their production.

Endothelial cell loss and pump dysfunction are also side effects of long-term hyperglycemia [2].

Aside from these factors, a decrease in the epithelial barrier, crosslinking of stroma, collagen, and matrix, and a decrease of the endothelial pump can cause an increase in the size of the corneal stroma (the maximum bulk of the cornea) [3]. NGF, nerve growth factor; TGFb3, transforming growth factor beta3; CNTF, ciliary neurotrophic factor; EGFR, epithelial growth factor receptor; IGF-1, insulin-like growth factor 1; NF-kB, nuclear factor kappa-light-chain-enhancer of activated B cells transcription factor; NF-kB, nuclear factor kappa-light-chain enhancer of activated B cells transcription factor; NF-k Activation/promotion is represented by solid blue arrows, while inhibition of negative regulation is represented by red stop arrows [3]. DR's etiology is complicated and not fully understood. On the other hand, the processes include vascular, neural, and immune systems [3]. The retina contains two sources of blood supply and is subjected to high metabolic demand during the visual cycle [3]. The retinal-colored epithelial cells and the outside 1/3 of the retina are supplied by retinal arteriole arteries, whereas the choroid provides the retinal-colored epithelial cells and the superficial 1/3 of the retina. A decline in retinal perfusion is one of the first alterations in DR. The patient may not notice the microvascular alterations, although they are noticeable on a fundus examination [4].

2. EPIDEMIOLOGY

Type one diabetes, which has an autoimmune etiology, affects about 10% of people and is most common in children and younger age groups. On the other hand, type two diabetes accounts for ninninety percentage of cases and is linked to increased weight and insulin resistance. Up to twenty% of patients with type 2 diabetes may have type 1.5 or latent autoimmune diabetes; these patients are not fat and show no signs of insulin resistance [4]. Diabetes affects 422 million people worldwide, up from 108 million in 1980. Low- and middle-income countries have seen a faster increase in prevalence than high-income countries [4].

3. DISCUSSION

High blood glucose is not likely to cause visual damage in the short term. It causes damage when patients change their diabetes treatment plan or prescription; they may have blurry vision for a few days or weeks. Increased blood glucose levels might affect fluid levels or cause inflammatory processes in the tissues that help you focus, resulting in blurred vision [1]. This form of hazy vision is only temporary and will disappear as your blood glucose levels return to normal.

Blood sugar levels that remain increased for a briefed period can harm the tiny blood vessels in the back of your eyes. Prediabetes is a condition in which blood sucrose echelons are increased than usual but not increased plenty to be called diabetes [1]. Arteries and veins that have been damaged may leak intracellular fluid and produce edema. It's also possible that new, weak arteries and veins will form. These veins can go into the eye's center, the optic disc, or macula, causing scarring or dangerously high pressure inside the eye [1].

Blood vessel issues are the root of most significant diabetic eye disorders.

3.1 Diabetic Cornea

Recurrent erosions, delayed wound healing, ulcers, and edema are symptoms of cellular dysfunction and faulty repair processes in the diabetic cornea. Changes in the epithelial basement membrane also happen, probably related to epithelial dysfunction [2]. In diabetic corneas, neuropathy causes a decrease in corneal sensitivity and innervation, which can be linked to corneal epithelial abnormalities. Corneal epithelial deficiencies are thought to be caused by neuropathy [2].

Nephropathy, final-stage renal failure, peripheral neuropathy, and vision loss are examples of diabetes-related microvascular consequences. The frequency of these consequences enormously depends on the length of the condition and the patient's age. Dry eye, superficial punctate keratitis, recurrent corneal erosion syndrome, and persistent epithelial abnormalities are more common in diabetic eyes [2]. Because the corneal epithelium is the eye's initial layer, it is continually vulnerable to wear and tear and must be replaced.

Although the mechanisms governing IGFBP3 secretion from corneal epithelial tissue are unknown, it was discovered in trials with immortalized human corneal epithelial cells that elevated planes of sugar level in the culture medium can bring IGFBP3 creation, suggesting that hyperglycemia in patients could be the cause of IGFBP3 upregulation [3]. Because the normal corneal epithelium plays such a crucial role in preventing liquid from inflowing the stroma, a decrease in the blockade role will result in edema and distension of the typically dehydrated stroma. Tight junctional complexes among cornea epithelial cells, visible as electron compact structures, primarily serve the epithelium that forms the barrier [3]. Loss of epithelial function might be explained by the damage or disruption of these tight junction structures, as well as the injury of basal corneal epithelial cells on imaging [3].

3.2 Starting Fluctuations in Diabetic Retina

Diabetic retinopathy is a microvascular condition in which serum seeps from the microvasculature, vascular leakage increases, and capillaries are destroyed starting in the disease. Endothelial cells, pericytes, and neurons are toxic to heightened blood sucrose level and mitochondrial and outside the cellular region, reactive oxygen species, resulting in their demise early in Diabetic Retinopathy(4). According to mounting data, low-grade inflammation appears to be at the root of diabetic retinopathy vascular issues. Inflammation is the body's general response to organ injury, during which white blood cells are drawn to the inflammatory area [4]. Diabetic retinopathy is finest described as a persistent decreased-level inflammation with heightened systemic inflammation. The macula is the region of your retina that you use for reading, driving, and seeing faces [5]. Diabetic macular edema is the distension of the macula triggered by diabetes. This illness can progressively demolish the tubular vision in this zone of the eye, causing visualization loss. Macular edema is widespread in diabetic retinopathy people with different symptoms [5].

Diabetic retinopathy is happened by a disturbance to the retina's blood vessels induced by diabetes. The retina is the back layer of tissue in the inner eye. Light and photos from the eye are converted into nerve impulses delivered to the brain. Diabetic retinopathy is the leading cause of visual loss or blindness in 20 to 74 [6]. This illness can disturb persons with type one or type two diabetes.

Diabetic retinopathy typically has no symptoms in the early stages. Some people report changes in their vision, such as difficulty reading or seeing objects far away. These shifts can occur at any time [6].

Blood vessels in the retina begin to bleed into the vitreous in the latter stages of the illness (the gel-like fluid that fills your eye). If this happens, you may notice black, floating dots or streaks that resemble cobwebs. The spots may clear up on

their own, but it's critical to get treatment as soon as possible(6). Without therapy, the bleeding may recur, worsen, or result in scarring.

Diabetic retinopathy can progress to proliferative diabetic retinopathy, a more advanced manifestation. Damaged blood vessels seal off in this kind, foremost the retina to develop new, aberrant blood vessels. These new blood vessels are fragile, and they may leak into the transparent, jellylike fluid that fills your eye's center (vitreous) [7].

The retina might ultimately separate from the posterior of your eye due to scar tissue formed by the establishment of new blood vessels. If the new blood vessels barricade the usual flow of liquid out of the eye, compression in the eyeball might rise. As a result, glaucoma develops.

4. MACULAR OEDEMA

Macular edema is characterized as thickening of the retina or the presence of hard exudates at the macula's two disc diameter. Diabetic macular edema (DME) is the most common cause of diabetic individuals' moderate tosevere vision loss. DME develops independently of the DR stage and should be assessed accordingly [4]. Although central macular thickness does not directly correlate with visual acuity in diabetic eyes, there is a strong correlation between photoreceptor inner/outer segment junction unity and visual understanding [4].

The new blood vessels may outflow into the transparent, jelly-like liquid that fills your eye's center. Only a few black patches may seem if the amount of bleeding is negligible (floaters). Blood can plug the vitreous cavity and entirely obstruct your vision in different severe situations [5].

In most cases, a vitreous hemorrhage does not outcome in permanent visual loss. Within a few weeks or months, the blood in the eye usually clears. Your eyesight will most likely recover to its original sharpness unless your retina is injured. Diabetic retinopathy causes abnormal blood vessels to form, causing scar tissue to form and the retina to retract away from the back of the eye [5]. Floating dots in your vision, blinding flashes, or significant vision loss are possible outcomes. New blood vessels can grow in the iris (front part of the eye), obstructing the normal flow of fluid out of the eye and causing pressure to build up [5]. This pressure (optic nerve) might spoil the nerve that communicates pictures from your eye to your brain might be spoiled by this pressure (optic nerve).

5. PREVENTION

Almost all existing therapies for DR are more successful when given sooner than later, which adds to the need for a comprehensive screening progra. It's all the more terrible since inm one research, more than half of the people who became blind from DR had never been examined [6]. Fundamental lifestyle changes have been known to decrease the risk of type 2 diabetes or delay its onset.

The public should do these things to save and prevent type two diabetes and its complications: Have an average body weight; be physically active by engaging in at least thirty minutes of more than normal-intensity activity on most days [6].

For weight loss, increase physical activity; consume a balanced diet low in sucrose and saturated fats; and eliminate smoking, which raises the danger of diabetes and cardiovascular disease. Other cost-diminishing measures include:
Retinopathy (which causes blindness) screening and treatment; blood cholesterol control (to maintain cholesterol levels); and early detection and prevention of diabetes-related kidney damage [6]. Type one diabetes necessitates the practice of insulin, but type two diabetes can be treated with oral drugs but may necessitate the practice of insulin, as well as blood pressure regulate and foot care (patient self-care by keeping foot hygiene; wearing suitable footwear; looking for professional care for ulcer managing; and consistent examination of feet by health professionals) [6].

Request a glycosylated hemoglobin test with your doctor. The glycosylated hemoglobin test, frequently known as the hemoglobin A1C test, measures your usual blood sugar level over the earlier 2 to 3 months [7]. The A1C purpose for most persons with diabetes is to keep it around 7%. Uphold a healthy blood pressure and cholesterol level. Eating well-proportioned meals, exercising frequently, and diminishing weight can all help [7]. Medication is occasionally compulsory as well. If you smoke or use other tobacco products, talk to your doctor about quitting. Smoking increases your probability of developing diabetes complications, including diabetic retinopathy [8-13].

5.1 Symptoms of Diabetic eye disease

1) Blurry or wavy vision
2) Frequently changing vision
3) Poor color vision
4) Spots or dark strings
5) Flashes of light

5.2 Diabetic Cataract

In diabetes, two types of cataract occur the senile cataract and True diabetic cataract. In senile cataract, it appears at in early age and progress rapidly. In true diabetic cataract also known as snowflake cataract or snowstorm cataract, it is a rare condition in which there is osmotic over hydration of lens and it usually occur in teenagers [3]. Osmotic over hydration of lens occur due to accumulate or attachment of sorbitol, when glucose is metabolized by NAPDH+ dependent aldose reductase. At first there is appearance of fluid vacuoles at very number beneath the anterior and posterior capsules, which is soon after that presence of bilateral snowflake- like white opaqueness in the cortex [4]. Although cataract surgery is reasonably safe and has a high success rate in healthy people, it is not the same for diabetics. Following cataract surgery, posterior capsular opacification (PCO) is a typical finding. When the lens is removed during cataract surgery, the capsule in which the lens is housed remains, and it might hinder vision in some situations due to opacification(5). As with DR, there are worries that cataract surgery may hasten the growth of macular edoema in diabetics.

6. CONCLUSION

Finally, hyper-glycemia has a wide range of impacts on the eye. It grounds diabetic keratopathy in the cornea, and it destroys vascular and neuronal cells in the retina and choroid, either straight or meanderingly. The most obivious treatment is maintaining normo-glycemia or patient compliance. Basement membranes have collected hazardous progressive glycosylation finish products and cell death has happened after the eye has been exposed to hyper-glycemia for a long time. Despite improved understanding of these visual disorders and the development of effective treatments, diabetes mellitus and associated ocular consequences remain a chief cause of blindness. All diabetic ocular problems can be avoided with early detection and treatment. As a result, regular eye check-ups are crucial to prevent diabetes- related vision loss. The chief goal in preventing occular consequences of diabetes is to maintain good blood glucose management and to manage other systemic risk factors such as hypertension and hyperlipidemia.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Achtsidis V, Eleftheriadou I, Kozanidou E, Voumvourakis KI, Stamboulis E, Theodosiadis PG, Tentolouris N. Dry eye syndrome in subjects with diabetes and association with neuropathy. Diabetes care. 2014;37(10):e210-1..
2. Nagai N, Murao T, Okamoto N, Ito Y. Kinetic analysis of the rate of corneal wound healing in Otsuka long-evans Tokushima Fatty rats, a model of type 2 diabetes mellitus. J Oleo Sci; 2010.
3. Xu KP, Li Y, Ljubimov AV, Yu FS. High glucose suppresses epidermal growth factor receptor/phosphatidylinositol 3-kinase/Akt signaling pathway and attenuates corneal epithelial wound healing. Diabetes;2009.
4. Kim J, Kim CS, Sohn E, Jeong IH, Kim H, Kim JS . Involvement of advanced glycation end products, oxidative stress and nuclear factor-kappaB in the development of diabetic keratopathy. Graef Arch Clin Exp Ophthalmol=Albrecht von Graef Arch Klin Exp Ophthalmol;2011.
5. Funari VA, Winkler M, Brown J, Dimitrijevich SD, Ljubimov AV, Saghizadeh M . Differentially expressed wound healing-related microRNAs in the human diabetic cornea. PLoS One;2013.
6. Wu YC, Buckner BR, Zhu M, Cavanagh HD, Robertson DM . Elevated IGFBP3 levels in diabetic tears: A negative regulator of IGF-1 signaling in the corneal epithelium. Ocul Surf;2012.
7. Clark JB, Grey RH, Lim KK, Burns-Cox CJ. Loss of vision before ophthalmic referral in

blind and partially sighted diabetics in Bristol. Br J Ophthalmol;1994.

8. Jameel, Patel Zeeshan, Sham Lohiya, Amol Dongre, Sachin Damke, and Bhavana B. Lakhkar. "Concurrent Diabetic Ketoacidosis and Pancreatitis in Paediatric Acute Lymphoblastic Leukemia Receiving L-Asparaginase." Bmc Pediatrics. 2020;20(1). Available:https://doi.org/10.1186/s12887-020-02136-3.
9. Kaple, Meghali Narayan, Chandrashekhar C. Mahakalkar, Anita Kale, and Swati Shambharkar. "Correlation of Metal Ions in Diabetic Patients." Journal of Clinical and Diagnostic Research. 2020;14(5):BC14–16. Available:https://doi.org/10.7860/JCDR/2020/43798.13730.
10. Thakare, Pratiksha, Ruchira Ankar. To assess the knowledge regarding prevention of sign and symptoms of diabetic ketoacidosis among diabetes patients in selected hospitals of Wardha District. International Journal of Modern Agriculture. 2020;9(3):125–30.
11. Thakare PS, Ankar R. To Assess the Knowledge Regarding Signs and Symptoms of Diabetic Ketoacidosis and Its Prevention among Diabetes Patients in Wardha District, Maharashtra, India. Journal of Evolution of Medical and Dental Sciences-Jemds. 2021;10(19):1413–6.
12. Thool AR, Dhande NK, Daigavane SV. Study of correlation between renal function test and severity of diabetic retinopathy in patients with type 2 diabetes mellitus. Journal of Evolution of Medical and Dental Sciences-Jemds. 2021;10(20):1511–4.
13. David P, Yeola M, Ankar R. Efficacy of Nursing Skin Care Protocol on Prevention of Skin Related Problems among Newly Diagnosed Diabetic Patients. Journal of Pharmaceutical Research International. 2021;33(31A):1–8.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/80336