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Serum Electrolytes in Cataract Patients with and without Diabetes Mellitus

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Authors' contributions

This work was carried out in collaboration between both authors. Author USA designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Author AH managed the analyses of the study and managed the literature searches. Both authors read and approved the final manuscript.

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ABSTRACT

Introduction: Cataract is the most common cause of blindness which is treatable. Though there are multiple risk factors involved, exact pathogenesis of cataract is yet to be established. Diabetics are known to be associated with electrolyte disturbances, like hypo/hypernatremia, hyperkalemia. We hypothesize that serum electrolytes may be altered in diabetic cataract patients. The aim of the study was to compare serum electrolytes in diabetic and non-diabetic cataract patients as well as to assess the correlation of duration of diabetes with electrolytes as well as risk of cataractogenesis in diabetics.
Methods: The cross sectional prospective study was conducted in Clinical Biochemistry, IGMCRI, Puducherry. Blood samples of fifty each diabetic and non-diabetic cataract patients were analyzed for electrolytes using Roche electrolyte analyzer which works on the principle of ISE. Statistical analysis was done using SPSS version 21.Student's unpaired 't' test was used to compare the means of electrolytes, Pearson's correlation coefficient was calculated to find the correlation between duration of diabetes and electrolytes. Odd's ratio was calculated to study the association between electrolytes and the risk of cataractogenesis.
Results: We observed a significantly higher serum sodium (3.3%), potassium (10.2%) and chloride

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levels (2.8%) [(P<0.001), (P<0.05) and (P<0.01) respectively] in diabetic cataract patients as compared to non-diabetics. Elevation of sodium, potassium and chloride pose 3.45, 1.76, 1.36 times respectively higher risk of cataractogenesis in diabetics. Sodium and potassium levels were positively correlated with duration of diabetes (r= 0.452, p<0.01 and r= 0.349, P<0.05 respectively).
Conclusion: Alterations in electrolytes pose an added risk of cataract formation in diabetics in addition to hyperglycemia. Along with carbohydrate restricted diet, salt restriction may help in prolonging cataract formation as well as progression.

Keywords: Electrolytes; cataract; T2DM.

1. INTRODUCTION

Cataract is the major cause of visual impairment in diabetes mellitus. Studies have reported an increased incidence and faster progression of cataract in diabetics. However exact mechanism is yet to be established. There are various factors that influence the transparency of lens. Aqueous humor which is the main source of nutrition to lens and is formed from serum. Therefore any change in the composition of blood may directly affect its composition. As lens lies bathed in aqueous humor and derives its nutrition from it, any altered composition of this fluid may affect lens metabolism.

Diabetes mellitus is known to be associated with dysnatremias by various mechanisms. Both hypo and hypernatremia are reported in diabetics. We had observed hyponatremia in diabetics in our previous study [1]. Serum sodium is an equilibrium between two antagonizing forces, namely osmotically active glucose moving water out of the cells and thereby causing dilutional hyponatremia and glucosuria induced osmotic diuresis which tends to cause hyperrnatremia. These alterations in serum electrolytes may alter the composition of aqueous humor which is the main source of nourishment to lens. Lens metabolism and hence the transparency of lens may get affected. Disturbance of intra and extracellular equilibrium of water and electrolytes disturbs colloid system within lens fibers leading to opacification of lens.

Study by Choudhury et al. reported an elevated serum and aqueous humor sodium and chloride as well as hyperkalemia in diabetic cataract patients [2]. However there are a few studies in this area in Indian settings to the best of our knowledge. As cataract is one among the treatable causes of blindness, it is justifiable to make an attempt to identify a probable risk factor for the cataractogenesis.

1.1 Objectives

The aims of the study were to

1. Compare serum electrolytes in diabetic and non-diabetic cataract patients.
2. Assess the correlation of duration of diabetes with electrolytes as well as risk of cataractogenesis in diabetics.

2. METHODOLOGY

2.1 Study Design

A prospective case control study was carried out in the Department of Clinical Biochemistry, IGMCRI, Pondicherry in collaboration with the Dept of Ophthalmology from January 2014 to May 2014. Institutional ethics committee approval was sought.

Blood samples of fifty diabetic cataract and fifty non-diabetic cataract individuals were analyzed for electrolytes.

Inclusion criteria: Diabetic cataracts with a mean age of 67.3±3.7 years.

Exclusion criteria: Previous ophthalmic surgeries, those on steroids, traumatic and complicated cataracts, diarrhea, vomiting, patients on angiotensin converting enzyme inhibitors and angiotensin receptor blockers.

Inclusion criteria for controls: Non diabetic individuals in the age group of 65.4± 3.2 yrs.

Table 1. Demographic profile of cataract patients

	Diabetic cataract (n=50)	Non diabetic cataract (n=50)
Male: Females	30:20	25:25
Age of patients in years	67.3±3.7	65.4± 3.2
Average duration of diabetes in years	11.85±1.14	-

2.2 Data Collection and Analysis

Five ml of venous blood sample was collected by puncturing antecubital vein with aseptic precautions. Samples were centrifuged at 3000 rpm for 15 minutes. Sample was analyzed for electrolytes with Roche electrolyte analyzer which works on the principle of indirect ion selective electrodes. Blood glucose and HbA1c were estimated by Glucose oxidase-peroxidase method and immunoturbidimetric method respectively.

2.3 Statistical Analysis

Statistical analysis was done using SPSS version 21 software. Data was analyzed by Student's unpaired 't' test. P< 0.05 was taken as significant. Pearson's correlation coefficient, r (between -1 and +1) was calculated for the correlation studies. Odd's ratio is calculated to find the relative risk.

3. RESULTS

The results were expressed as mean ± standard error of mean (SEM) and are represented in the Table 2. We observed a significantly higher serum sodium (3.3%), potassium (10.2%) and chloride levels (2.8%) [(P<0.001), (P<0.05) and (P<0.01) respectively] in diabetic cataract patients as compared to non-diabetics (Table 2). Elevation of sodium, potassium and chloride pose 3.45, 1.76, 1.36 times respectively higher risk of cataractogenesis in diabetics (Tables 3, 4, 5).

Sodium and potassium levels were positively correlated with duration of diabetes (r= 0.452, p<0.01 and r= 0.349, P<0.05 respectively).

4. DISCUSSION

Elevation of serum electrolytes observed in the study (Table 2) suggests a strong etiological role of electrolytes disturbances in the pathogenesis of diabetic cataract. Our results are in accordance with the study by Choudhury et al. [2]. Studies by Klein et al. and Ederer et al. reported two to four fold increase risk of cataract in diabetes patients as compared to non diabetics [3,4]. Positive correlation of sodium and potassium levels with the duration of diabetes is supported by a study by Pollreisz et al. This study also suggested increased risk of cataract formation with the longer duration of diabetes [5].

Elevation of sodium, potassium and electrolytes pose 3.45, 1.76, 1.36 times higher risk of cataractogenesis respectively in diabetics. Choudhury et al. reports that the risk of early development of cataract is 5 times higher in diabetics as compared to non diabetics [2].

Table 2. Comparison of parameters between diabetic and non –diabetic cataract patients

	Diabetic cataract	Non-diabetic cataract	P value
Fasting blood glucose (mg/dl)	138.49±5.4	102.57±4.2	<0.001
Sodium (mEq/L)	148.8±2.32	144.08±2.0	<0.01
Potassium (mEq/L)	4.42±0.74	4.01±0.32	<0.05
Chloride (mEq/L)	102.12±1.02	99.31±1.57	<0.01
HbA1C	9.3±0.56	4.8± 0.87	<0.05

P<0.05 – significant
P<0.01 – highly significant
P<0.001-very highly significant

Table 3. Odds ratio for sodium

	Diabetic cataract	Non-diabetic cataract
Higher sodium	33(a)	18(b)
Normal sodium	17(c)	32(d)

Odd's ratio =ad/bc = 3.45

Table 4. Odds ratio for potassium

	Diabetic cataract	Non-diabetic cataract
Higher potassium	27 (a)	20 (b)
Normal potassium	23(c)	30(d)

Odd's ratio =ad/bc = 1.76

Table 5. Odds ratio for chloride

	Diabetic cataract	Non-diabetic cataract
Higher chloride	26(a)	22(b)
Normal chloride	24(c)	28(d)

Odd's ratio =ad/bc = 1.36

There are various theories which explain the mechanism of cataract formation in diabetics.

The permeability of the lens cell membrane and the activity of sodium pump within the cell membranes of the lens determine the cation balance between inside and outside of the lens. Adler et al reported a diminished potassium and rise in sodium content of lens [6]. The rise in the sodium levels in aqueous humor could be attributed to raised sodium in the serum leading to cationic imbalance, thus leading to cataract formation.

Na-K ATPase dysfunction , impairment of Ca-Mg ATPase, Na -Ca exchanger, Ca pump which are located in cell membrane, mitochondria or endoplasmic reticulum have been reported to be responsible for alterations in electrolytes in type 2 diabetes mellitus [7].

This mechanism of electrolyte imbalance is an added risk of cataract formation in diabetics as they are already at a higher risk of cataract due to hyperglycemia. The mechanism is explained as below;

Polyol pathway has been explained to have a role in the pathogenesis of diabetic cataract. Intracellular accumulation of sorbitol causes osmotic changes resulting in hydropic lens fibers which lead to degeneration and cataract formation [8,9]. Animal studies have shown that the intracellular accumulation of polyols leads to a collapse and liquefaction of lens fibers, which ultimately results in the formation of lens opacities [10]. Furthermore, studies have shown that osmotic stress in the lens caused by sorbitol accumulation [11] induces apoptosis in lens epithelial cells [12] leading to the development of cataract [13]. Polyol pathway leads to the generation of free radicals and causes oxidative stress damage to lens fibers [14]. Hyperglycemia in the aqueous humor may induce glycation of lens proteins and formation of advanced glycation end products (AGE) which further generate free radicals [15]. In addition to increased levels of free radicals, diabetic lenses show an impaired antioxidant capacity, increasing their susceptibility to oxidative stress.

The possible mechanism of cataract formation is depicted as in Fig. 1.

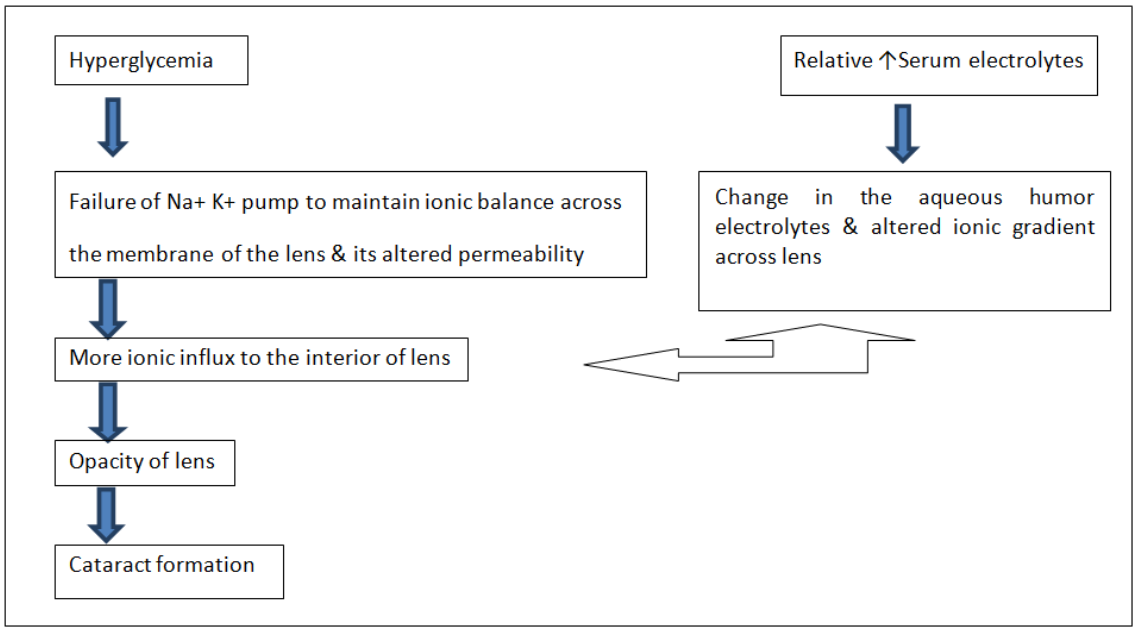

Fig. 1. Possible mechanism of cataract formation of in diabetes mellitus

5. CONCLUSION AND SIGNIFICANCE OF THE STUDY

Comparatively higher serum electrolytes observed and higher risk of cataract formation in diabetics suggest an etiological role of electrolytes in cataract formation.

This study may be useful in patient care in the following way:

The derangement of electrolytes can be corrected which may delay the progression of cataract.

Dietary restriction of salt may prolong cataract formation, which can be a preventive measure useful to the community.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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