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#### **Original Research Article**

# Serum electrolyte imbalances in diabetic vs non-diabetic senile cataract: A comparative study

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#### **Abstract**

**Introduction:** Cataract is one of the leading causes of blindness in developing countries. The most common type of cataract is senile cataract. The exact pathogenesis of cataract is not known, but it is believed that multiple risk factors like age, sex, diabetes mellitus, serum electrolytes, oxidative stress, etc., are responsible for the development of cataract. Rise in serum sodium and potassium levels are responsible for cataract formation.

Aim and Objective: The aim of this study is to estimate serum sodium and potassium in senile diabetic cataract patients, as compared to those without diabetes with cataract and to estimate its relationship in development of cataract.

Materials and Methods: This study consists of 50 senile diabetic cataract patients and age matched 50 non diabetic cataract individuals, for them serum electrolytes like sodium and potassium were measured along with blood glucose and HbA1c.

Results: In our study there is significant rise in serum sodium and mild elevation of potassium levels in cases compare to control group.

Conclusion: We have concluded that serum sodium and potassium are important markers of senile diabetic cataract formation. The diet with low salt and sugar, life style modification may delay the process of cataract formation in diabetics.

Keywords: Cataract, Diabetes mellitus, Electrolytes, Sodium, Potassium, Tertiary care teaching hospital

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#### 1. Introduction

The lens is made up of 65% water and 34% of proteins. The crystalline proteins of the lens are arranged in such a way that it keeps the lens transparent and refracts the light rays to focus it on the light sensitive layer of the eye, the retina. Due to degenerative process, the transparency of crystalline lens is disturbed, leading to opacification of lens fibers, which can obscures the passage of light through the lens causing cataract.

Cataract is one of the leading cause of preventable blindness worldwide and the most common form of cataract is Senile cataract.<sup>1</sup> As per the World Health Organization, in 2010, 285 million people had visual impairment and 51% had blindness due to cataract and in 2014 about 95% of visual impairment was due to cataract worldwide.<sup>2,3</sup> In India 22 million people were blind and nearly 80.1% of blindness was

due to cataract according to the WHO/National Program for Control of Blindness survey.<sup>4</sup>

Various mechanisms proposed for cataract formation are osmotic changes, formation of protein aggregates, post translational modification of proteins, oxidative stress, metabolic diseases and genetics. 4,6 Diabetes mellitus, a wellestablished global health problem, has been recognized as an important catalyst for cataract development.<sup>7,8</sup> In diabetics, cataract develops more frequently, occurs at an earlier age and progression is also more rapid than in the non-diabetic population<sup>9,10</sup> Understanding the mechanism of cataract development in diabetes mellitus has potential implications in clinical management and prevention. Avascular Lens gets nutrients from Aqueous humor which is produced from plasma. The concentration of sodium and potassium is 150 mmol/L and 5mmol/L respectively in the aqueous humor and in the lens the concentration of potassium is 114–130 mmol/L and sodium is 14-26mmol/L approximately. So, the lens has

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high potassium and low sodium compared to aqueous humor. The sodium and potassium cations are kept in balance by Na+-K+ ATPase pump and permeability of the lens capsule.<sup>8</sup> Changes in the electrolyte composition in the plasma alters these cation concentration of aqueous humor, which can ultimately affect the metabolism of lens and leading to cataract formation.

In diabetes, serum sodium levels fluctuates, which disrupts the osmotic equilibrium of lens leading to changes in its hydration and transparency. The study conducted in Diabetic cataract patients by Choudhury et al. showed an elevated serum sodium and potassium.<sup>11</sup> However only few studies are available in this area in Indian settings and as cataract is one among the treatable causes of blindness, we made an attempt to identify a probable link between the electrolyte imbalance and serum the Diabetic cataractogenesis. Also the levels of serum sodium, serum potassium levels were compared between the diabetic senile cataract patients with non-diabetic senile cataract control subjects.

## 2. Objectives

The aim of the study was to estimate and compare serum sodium and potassium levels in diabetic and non-diabetic senile cataract patients and to investigate the relationship between electrolyte imbalance in the pathogenesis of diabetic senile cataract.

#### 3. Materials and Methods

A cross-sectional comparative study was conducted at the Department of Ophthalmology, ESI Medical College, Chennai, after getting approval from the Institutional Ethics Committee, ESIC Medical College & PGIMSR, Chennai, IEC NO-IEC/2019/1/22, dated 15.05.2019. The study was conducted for a period of 1 year from June 2019 to May 2020. A total of 100 senile cataract Patients, who came for ophthalmic evaluation were involved in the study and categorized into two groups (50 diabetic senile cataract patients as case group and 50 non-diabetic senile cataract patients as control group).

- 1. **Group 1 (Case Group):** Diabetic Senile cataract (Nuclear/cortical/posterior subcapsular cataract) patients
- Group 2 (Control Group): Age and sex matched non diabetic individuals with Senile cataract.

## 3.1. Inclusion criteria

Diagnosed cataract patients aged between 44-76 years.

### 3.2. Exclusion criteria

Patients with previous ophthalmic surgeries, other types of cataract like traumatic and complicated cataracts, patients with diarrhea & vomiting, patients on angiotensin converting enzyme inhibitors and angiotensin receptor blockers, those on steroids were excluded.

The included subjects were asked for a detailed history and subjected to detailed ocular examination including slit lamp examination and fundus evaluation by a single experienced ophthalmologist at ESIC. Then blood samples were collected from both case and control group under strict aseptic precautions and sent to the laboratory for analysis. Serum sodium and serum potassium level were measured by using Roche electrolyte analyzer which works on the principle of indirect ion selective electrode method. Fasting and postprandial Blood glucose levels were estimated by hexokinase method and HbA1c was estimated by immunoturbidimetric method by Roche fully automated clinical chemistry analyzer.

Normal serum sodium level - 130-143 meq/L. Normal serum potassium level - 3.5-5.5 meq/L.

# 3.3 Statistical analysis

Statistical analysis was done using SPSS version 21 software. Data were analyzed by independent 't' test. The significance of observed differences among the groups were evaluated and P<0.05 was considered to be statistically significant.

#### 4. Results

The study enrolled 100 subjects, including 50 diabetic senile cataract cases and 50 non diabetic senile cataract controls.

## 4.1. Sex comparison

There was female predominance in our study groups (Table 1). In the case group, 23 were Males and 27 were Females and in control group 22 were Males and 28 were Females.

**Table 1:** Comparision of sex of patients with cataract in diabetic and non-diabetic patients.

Sex * Group Cross tabulation								
		G	roup	Total				
		Control Cases						
C	Male	22	23	45				
Sex	Female	28	27	55				
Total		50	50	100				

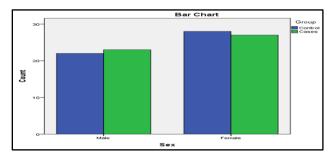


Figure 1: Bar diagram for sex

### 4.2. Demographic distribution and age comparison

Out of 100 subjects, in the age group of 44-54 years, 34 were cases and 26 were controls, totalling 60 participants. In the 55-65 years category, 14 cases and 23 controls were recorded, making up 37 participants. The 66-76 years age group comprised of 2 cases and 1 control, totalling 3 individuals. The mean age  $\pm$  standard deviation (SD) for the cases was  $50.36 \pm 6.08$  years, and for the controls it was  $52.14 \pm 6.75$  years. The comparison of mean ages between cases and controls was not statistically significant (p > 0.05) (**Table 2**)

**Table 2:** Comparision of age of patients with cataract in diabetic and non-diabetic patients.

Group Statistics							
	Grou p	N	Mean	Std. Devi ation	Independent Samples t- test Sig. (2- tailed)		
Ag	Contr	50	52.14	6.75	.864		
e	ol			5			
in	Cases	50	50.36	6.08	.864		
ye				0			
ars							

### 4.3. Blood glucose levels analysis

Diabetic senile cataract cases had elevated fasting blood glucose levels with a mean  $\pm$  SD of 175.84 $\pm$ 45.45 mg/dL. In contrast, the controls, showed a mean  $\pm$  SD of 92.16  $\pm$ 7.4 mg/dL. The difference in fating blood glucose levels between the two groups was statistically significant. (p < 0.001) (**Table 3**).

**Table 3:** Comparision of fasting blood glucose of patients with cataract in diabetic and non-diabetic patients.

Group Statistics							
	Gro up	N	Mean	Std. Deviati on	Independent Samples t- test Sig. (2- tailed)		
Fastin g	Cont rol	50	92.16	7.416	.000		
Blood Sugar	Case	50	175.84	45.449	.000		

All the diabetic senile cataract cases had elevated post prandial blood glucose levels with a mean  $\pm$  SD of 279.82 $\pm$ 82.04 mg/dL. In contrast, the controls subjects, who were non diabetic, showed a mean  $\pm$  SD of 119.68  $\pm$  11.61 mg/dL. The difference in post prandial blood glucose levels between the two groups were statistically significant (p < 0.001) (**Table 4**).

**Table 4:** Comparision of post prandial blood glucose of patients with cataract in diabetic and non-diabetic patients.

Group Statistics							
	Gro up	N	Me an	Std. Deviatio	Independent Samples t-test		
				n			
					Sig. (2-tailed)		
Postpra	Con	50	119	11.610	.000		
ndial	trol		.68				
Blood	Cas	50	279	82.046	.000		
Sugar	es		.82				

# 4.3. HbA1c level analysis

All the diabetic senile cataract cases had elevated HbA1c levels with a mean  $\pm$  SD of 8.108 $\pm$ 1.48%. In contrast, the non-diabetic controls subjects, showed a mean  $\pm$  SD of 5.29 $\pm$ 0.29 %. The difference in HbA1c levels between the two groups were statistically significant (p < 0.001) (**Table 5**).

**Table 5**: Comparision of hba1c of patients with cataract in diabetic and non-diabetic patients.

Group Statistics							
	Group			Deviation	Independent Samples t-test Sig. (2-tailed)		
	Contro l	50	5.298	.2986	.000		
С	Cases	50	8.108	1.4876	.000		

#### 4.4. Distribution of serum sodium levels

The diabetic senile cataract group had mean serum sodium levels of  $144.16\pm2.25$ meq/L, which was notably higher than the  $140.26\pm3.60$ meq/L observed in the non-diabetic control group. This variation was statistically significant, as indicated by a P<0.001(**Table 6**).

**Table 6:** Comparision of serum sodium of patients with cataract in diabetic and non-diabetic patients.

Group Statistics							
	Group	N	Mean	Std. Deviati	Independent Samples t-test		
				on	Sig. (2-tailed)		
Sod	Contro	5	140.2	3.607	.000		
ium	1	0	6				
	Cases	5	144.1	2.235	.000		
		0	6				

## 4.5. Distribution of serum potassium levels

The mean serum potassium  $\pm$  SD level was  $4.43\pm$  .38mEq/L in cases which was slightly elevated than the controls  $4.41\pm$  .29 mEq/L, with this difference being statistically NOT significant (p >0.05) (**Table 7**).

**Table 7:** Comparision of serum potassium of patients with cataract in diabetic and non-diabetic patients

Group Statistics								
	Group	N	Mean	Std.	Independent			
	_			Deviation	Samples t-test			
					Sig. (2-tailed)			
Potass	Control	50	4.4120	.29696	.770			
ium	Cases	50	4.4320	.38129	.770			

### 5. Discussion

Cataract is the leading cause of visual impairment contributing to about 80% of treatable cause of blindness in India. WHO predicts diabetic cases in India would be around 80 million in 2030. 12,13 The risk factors for the development of senile cataracts as per the previous studies includes advancement of age, obesity, genetics, Diabetes mellitus, changes in serum electrolytes, radiation, smoking and socioeconomic status. 14,15 The relationship between Diabetes mellitus and cataract development was confirmed by previous studies. 16,17 we observed that the senile cataract tend to develop earlier in diabetic patients than non-diabetic individuals and a higher prevalence of diabetic cataract among females than males, which is consistent with previous studies. 18,19 The most likely cause of female gender prevalence is changes in estrogen levels due to menopause. 20

As per the previous studies there is a significant relationship between increased serum glucose levels and incidence of cataracts.<sup>21</sup> The duration of diabetes and poor control of serum glucose levels in diabetes can increase the risk of development of cataracts.<sup>22</sup> Chronic hyperglycemia in diabetics can lead to systemic complications like neuropathy, vasculopathy and ocular complications like retinopathy and cataract. Hyperglycemia in diabetic cataract is implicated in the glycation of lens proteins and activation of polyol pathway. In the lens, glucose is reduced to sorbitol via Polyol pathway by the enzyme aldose reductase, which gets accumulated leading to a hyperosmotic effect, resulting in hydrobic lens fibers, that degenerate and losses its transparency leading to cataract.<sup>23,24</sup> Aqueous humor hyperglycemia induces glycation of lens proteins and the formation of advanced glycation end products (AGE) with generation of free radicals and its accumulation can lead to oxidative stress and cataract formation.<sup>25</sup>

As per the American Diabetes Association (ADA) recommendation the glycated hemoglobin (HbA1c) is the measure of glycemic control for the last 6–8 weeks. <sup>26</sup> HbA1c of 6.5% is the cut-off value to diagnose diabetes. <sup>27</sup> In this study, the glycated hemoglobin level was taken as a marker of glycemic control in the two groups. The diabetic senile cataract group showed an elevated mean HbA1c levels as compared to the non-diabetic senile cataract group, which was consistent with the results of previous large cohort Beaver Dam Eye Study. <sup>28</sup>

In this study we studied the possible biochemical changes linked to diabetic senile cataract development, particularly the connection between serum sodium and potassium imbalances and cataract formation in diabetic individuals. Our study findings showed that serum electrolytes were increased in the diabetic senile cataract group suggesting a strong etiological role of electrolytes disturbances in the pathogenesis of diabetic senile cataract. It was also noted that there was significant differences in serum sodium levels between diabetic senile cataract patients and control subjects, with elevated levels in the diabetic cataract, highlighting the role of raised sodium levels on the progression of cataract in diabetic patients. Disturbances in serum electrolyte concentrations directly affect aqueous humor electrolyte composition impacting the lens metabolism. Thus electrolyte imbalance is an added risk factor for the cataract formation in diabetics as they are already at a higher risk of cataract due to hyperglycemia. The electrolyte imbalance in diabetic individuals may be related to renal function changes or diabetic nephropathy, malabsorption syndromes, acid-base disorders, multidrug regimens and other pathophysiological factors present in diabetics.<sup>29</sup> In Diabetes dysnatremia is due to several underlying mechanisms like endocrine dysfunction associated with impairment of both insulin mediated glucose metabolism and glucagon-dependent glucose release. 30,31,32 The lens maintains a delicate balance of high potassium and low sodium concentrations, achieved through the permeability of lens membrane and the activity of Na-K ATPase pump.<sup>33</sup> When there is increased concentrations of sodium in aqueous humor, it is difficult for the sodium pumps of lens membrane to maintain a low intracellular sodium ion level. In turn, a higher sodium ion concentration of the aqueous humor, coupled with an altered membrane permeability of lens, increases the intracellular sodium ion concentration leading to osmotic effect and hydration of the lens fibers, thereby resulting in loss of its transparency and development of cataract.34

Our study also revealed elevated serum potassium levels in diabetic senile cataract patients. Hyperkalemia in diabetes is attributed to the redistribution of potassium from intracellular to extracellular compartments.<sup>35</sup>

In our study we have found the mean sodium and potassium levels in the serum were significantly high with p value of sodium <0.001, and p value of potassium >0.00 in controls when compared with cases. As per our observation the raise of Na+in serum would increase the risk of cataract formation up to the levels which is statistically significant (p<0.001). The elevated serum sodium and potassium levels affect the concentration of these electrolytes in the aqueous humor. This may overwhelm the ability of lens to regulate these ionic homeostasis and osmatic effect exerted by electrolytes and glucose leads Diabetic Cataract genesis.

### 6. Limitations of the Study

The study was observational and done for a brief duration with small sample size, hence a causal relationship could not be established. The subjects were not randomized. The blood biochemistry did not encompass all components of the serum electrolyte study, such as Chloride, Calcium, Magnesium, Bicarbonate, Phosphate and was confined to Sodium and Potassium. The study did not consider the duration of Diabetes, dietary profile and treatment of Diabetes and hypertension, which could have influenced the development of cataract. The study was unable to sample individuals with senile cataracts from various socio-economic status. However, in line with previous research, our findings highlight the intricate relationship between diabetes, electrolyte imbalances, and cataract genesis.

#### 7. Conclusion

We concluded from our study that there is a significant elevation of serum Sodium and Potassium levels in diabetic senile cataract patients when compared to non-diabetic cataract patients. This electrolyte imbalance along with hyperglycemia may be a significant risk factor for the development of cataract at an early age and its rapid progression in Diabetic patients. Elevation of serum sodium may be one of the most important factor. Raised blood glucose and electrolytes are modifiable risk factors. To prevent the further progression of cataract, salt restriction along with diabetic diet, water intake, other pharmacological interventions, lifestyle modification, etc. may be strictly advised. This proactive approach could aid in preventing visual disabilities and other diabetic complications, thereby enhancing the overall quality of life. Addressing the electrolyte imbalances in diabetic cataracts along with the existing approaches can help in diabetic cataract management.

# 8. Source of Funding

None.

#### 9. Conflict of Interest

None.

## References

- Donnelly CA, Seth J, Clayton RM, Phillips CI, Cuthbert J. Some plasma constituents correlate with human cataract location and nuclear colour. Ophth Res. 1999;29(4):207-17.
- Sreelakshmi V, Abraham A. Age related or senile cataract: pathology, mechanism and management. Aust J Clin Ophth. 2016;3(2):1067.
- Alshamrani AZ. Cataracts pathophysiology and managements. Egyptian J Hosp Med. 2018;70(1):151-4.
- Government of India. National survey on blindness and visual outcomes after cataract surgery. Dr. Rajendra Prasad Centre for Ophthalmic Sciences. New Delhi: All India Institute of Medical Sciences; 2001-2002
- 5. Duncan G, Bushell AR. Ion analyses of human cataractous lenses. *Exper Eye Res.* 1975;20(3):223-30.

- Kupfer C. Bowman lecture. The conquest of cataract: a global challenge. Trans Ophthalmol Soc. 1984;104:1-0.
- López-Contreras AK, Martínez-Ruiz MG, Olvera-Montaño C, Robles-Rivera RR, Arévalo-Simental DE, Castellanos-González JA, Hernández-Chávez A, Huerta-Olvera SG, Cardona-Muñoz EG, Rodríguez-Carrizalez AD. Importance of the use of oxidative stress biomarkers and inflammatory profile in aqueous and vitreous humor in diabetic retinopathy. *Antioxidants*. 2020;9(9):891.
- Tiwari BK, Pandey KB, Abidi AB, Rizvi SI. Markers of oxidative stress during diabetes mellitus. *J Biomark*. 2013; 2013(1):378790.
- Böhm EW, Buonfiglio F, Voigt AM, Bachmann P, Safi T, Pfeiffer N, Gericke A. Oxidative stress in the eye and its role in the pathophysiology of ocular diseases. *Redox Biol.* 2023:102967.
- Harding JJ. Physiology, biochemistry, pathogenesis, and epidemiology of cataract. Curr Opin Ophthalmol. 1991;2(1):3-15.
- Choudhury RB, ParthaSarathi G, Das B. Comparative study of serum and aqueous humour electrolytes in diabetic and nondiabetic cataract patients. *IOSR JDMS*. 2016;15(4):1-6.
- 12. Ae K. Hyperglycemic crisis in adult patients with diabetes. *Diab Care*. 2006;32(7):1335-43.
- Bae JH, Shin DS, Lee SC, Hwang IC. Sodium intake and socioeconomic status as risk factors for development of age-related cataracts: the Korea National Health and Nutrition Examination Survey. PLoS One. 2015;10(8):e0136218.
- Meyer CH, Sekundo W. Nutritional supplementation to prevent cataract formation. *Nutr Eye*. 2005;38:103-19.
- Kitabchi AE, Umpierrez GE, M Miles JM, Fisher JN, Hyperglycemic crisis in adult patients with diabetes. *Diab Care*. 2006; 32:(7):1335-43
- Brian G, Taylor H. Cataract blindness: challenges for the 21st century. Bulletin of the World Health Organization. 2001;79:249-
- Delcourt C, Carrière I, Ponton-Sanchez A, Lacroux A, Covacho MJ, Papoz L, POLA Study Group. Light exposure and the risk of cortical, nuclear, and posterior subcapsular cataracts: the Pathologies Oculaires Liesal' Age (POLA) study. Arch ophthalmol. 2000;118(3):385-92.
- JadavPrashantkumar M, Sharma Hariom M, ManiarMegha A, ChaudharyNitinkumar G, MaheshwariAmit V, Javia H. Relationship between altered level of serum electrolytes and risk of senile cortical cataract—A Case Control Study.2013;
- Harding JJ, Egerton M, Van Heyningen R, Harding RS. Diabetes, glaucoma, sex, and cataract: analysis of combined data from two case control studies. *Brit J Ophthalmol*. 1993;77(1):2-6.
- Zetterberg M, Celojevic D. Gender and cataract—the role of estrogen. Curr Eye Res. 2015;40(2):176-90.
- Harahap J, Rania R. Cataracts Risk Factors and Comparison of Blood Glucose Levels in Diabetic and Non-Diabetic Patients towards the Occurrence of Cataracts. *Open Access Macedonian J Med Sci.* 2019;7(20):3359.
- Kahn Ha, Leibowitz Hm, Ganley Jp, Kini Mm, Colton T, Nickerson Rs, Dawber Tr. The Framingham Eye Study: II. Association of ophthalmic pathology with single variables previously measured in the Framingham Heart Study. Am J Epidemiol. 1977;106(1):33-41
- Kador PF, Wyman M, Oates PJ. Aldose reductase, ocular diabetic complications and the development of topical Kinostat®. Progress in Ret Eye Res. 2016;54:1-29.
- 24. Kinoshita JH. Mechanisms initiating cataract formation proctor lecture. *Invest Ophthal Visual Sci.* 1974 Oct 1;13(10):713-24.
- Stitt AW. The maillard reaction in eye diseases. Ann the New York Aca Sci. 2005;1043(1):582-97.
- Sherwani SI., Khan HA., Ekhzaimy A., Masood A., Sakharkar MK. Significance of HbA1c test in diagnosis and prognosis of diabetic patients. *Biomarker Insights*. 2016:BMI-S38440.
- Gillett MJ. International expert committee report on the role of the A1c assay in the diagnosis of diabetes: *Diab Care* 2009;32(7): 1327–34.
- Klein BE, Klein R, Wang Q, Moss SE. Older-onset diabetes and lens opacities. The Beaver Dam Eye study. *Ophthal Epidemiol*. 1995;2(1):49-55.
- Elisaf MS, Tsatsoulis AA, Katopodis KP, Siamopoulos KC. Acidbase and electrolyte disturbances in patients with diabetic ketoacidosis. *Diab Res Clin Pract*. 1996;34(1):23-7.
- Liamis G, Rodenburg EM, Hofman A, Zietse R, Stricker BH, Hoorn EJ. Electrolyte disorders in community subjects: prevalence and risk factors. Am J Med. 2013;126(3):256-63.

- 31. Liamis G, Tsimihodimos V, Doumas M, Spyrou A, Bairaktari E, Elisaf M. Clin Labor Character Hypernatraemia in an internal medicine clinic. *Nephrol Dial Transp.* 2008; 23(1):136-43.
- Komjati M, Kastner G, Waldhäusl W, Bratusch-Marrain P. Detrimental effect of hyperosmolality on insulin-stimulated glucose metabolism in adipose and muscle tissue in vitro. *Bioch Med Metab Biol.* 1988; 39(3):312-8.
- Uribarri J, Oh MS, Carroll HJ. Hyperkalemia in diabetes mellitus. J Diab Compl. 1990;4(1):3-7.
- Rajakrishnan pd. An analysis of the levels of serum sodium and potassium ions in senile Cataract Patients. *Univer J Pre Paraclin Sci.* 2016;2(2).1-7
- Palmer BF. Managing hyperkalemia caused by inhibitors of the renin–angiotensin–aldosterone system. New England J Med. 2004;351(6):585-92.

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