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Comparative Study Of Serum And Aqueous Humour Electrolytes In Diabetic And Non-Diabetic Cataract Patients

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Abstract : Intrduction: Of the several etiological factors of cataract, diabetes is one and most common cause. In diabetes, cataract may develop as early as 40 years of age. Several previous studies have shown elevated sodiumlevel in senile cataract cases. Aqueous humour and serum organic and inorganic ions are analogous. Based on this the aim of the study is to establish a relation of serum and aqueous humour electrolytes in the early development of diabetic cataract and reliability of the findings as a possible preventive factor **. Method**:This study was carried out with 100 diabetic and 100 non diabetic cataract cases. Serum and aqueous humourelectrolytes were estimated by system Vitros integrated 5600**. Results**: A highly significant ($p<0.001$) rise in serum and aqueous sodium levels in diabetics as compared to non diabetic. A significant ($p<0.05$) rise in serum potassium levels in diabetic in comparison to non diabetic. Whereas aqueous humour potassium levelswas in significant in diabetic and nondiabetic ($P> 0.05$). Serum and aqueous chloride levels was highly significant ($p<0.001$) in diabetic in comparison to non diabetic**. Conclusion**: From the present study we can conclude that there is difference in the serum and aqueous electrolytes level in diabetic as compared to non diabetic and therefore salt restriction in addition to the prescribed diet for diabetes might delay the onset of cataract.

Keywords: electrolytes ; diabetes ; serum ; aqueous :cataract

I. Introduction

The cataract is used loosely to mean the occurrence of an optical discontinuity in the lens of such magnitude as to cause a noticeable dispersion of light . Though the precise pathogenesis of cataract formation is not clear, the biochemical changes have been studied extensively and certain mechanisms related to cataractogenesis have been suggested[1].Cataract is considered a major cause of visual impairment in diabetic patients as the incidence and progression of cataract is elevated in patients with diabetes mellitus [2,3].

It has been shown that the intracellular accumulation of sorbitol leads to osmotic changes resulting in hydropic lens fibres that degenerates and form sugar cataract[4,5].The enzyme aldose reductase catalyzes the reduction of glucose to sorbitol through the polyl pathway,a process linked to the development of diabetic cataract[6]. In the lens, sorbitol is produced faster than it is converted to fructose by the enzyme sorbitol dehydrogenase. In addition the polar character of sorbitol prevents its intracellular removal through diffusion, which creates a hyperosmotic effect that results in an infusion of fluid to countervail the osmotic gradient. As a result lens swells leading to cataract formation[4,5,7].

Aqueous humour is a clear fluid that fills and helps from the anterior and posterior chambers of the eye. The lens and cornea must remain clear to allow light transmission , and therefore cannot be invested within a vasculature. The aqueous humour is analogous to a blood surrogate for these avascular structures and provides nutrition,removes excretory products from metabolism, transport neurotransmitters,stabilizes the ocular structure and contributes to the regulation of the homeostasis of these ocular tissues[8].

Three mechanisms are involved in aqueous humour formation : diffusion, ultrafiltration and active secretion[9]. This thin fluid is produced from the serum. Therefore serum electrolytes concentration directly affects electrolytes of aqueous humour and in turn regulates lens metabolism[10].It has been proved that, in aqueous humour potassium is replaced with excess sodium and hence there is an alteration in their ratio in cataract patients[11]. Derangement in serum electrolytes may be an risk factor for cataractogenesis.

Aims and objectives

With the previous proposed studies of increased serum electrolytes[12-13,14] and serum and aqueous humour electrolytes level in cataract cases[12], the present study is aimed to establish a relation of serum and

aqueous humour electrolytes in the early development of diabetic cataract and with an objective to probe the reliability of the findings as a preventive factor.

II. Materials And Method

It is a cross sectional observational study conducted in the department of Biochemistry in collaboration with the Regional Institute of Ophthalmology, GMCH Guwahati. The duration of the study was three years. Cataract cases who were scheduled to undergo cataract surgery in the Regional Institute of Ophthalmology were taken as cases. A proforma was used to collect the baseline data and written consent was taken after proper explanation of the need of the study. Detailed history was taken and proper examination of both systemic and ocular examination was performed to fulfil the inclusion and exclusion criteria.

Inclusion criteria

Established cases of cataract of both males and females.

Exclusion criteria

Patients of systemic diseases other than diabetes, on steroid, diuretics are excluded from the study.

Cataract was diagnosed after doing visual acuty, intraocular pressure measurement, slit lamp examination after dilation.

Blood examination for sugar and HbA1c was done in system Vitros integrated 5600 . Depending on the results and history, the patients were divided into two groups

Group 1 Cataract cases without diabetes

Group 2 Cataract cases with diabetes

Each group consists of 100 cases.

2ml of blood was collected with aseptic precaution for estimation of electrolytes.

Collection of aqueous humour

Routine preparation of the case for cataract surgery was done maintaining the required asepsis. Peribulbar block was given. 0.2 c.c of aqueous humour was collected from the anterior chamber by using a 26 gauze needle and an insulin syringe intra operatively. The anterior chamber was reformed with ringers lactate solution through the same needle and the cataract surgery was then continued in its regular way.

Both serum and aqueous humour Na^+, K^+ and Cl^- are estimated by system Vitros integrated 5600.

Ethics

The study is carried out after getting the approval from Institutional Ethical committee of Gauhati Medical College & Hospital.

Statiscs

The data is analyzed by unpaired student's T test and SPSS 20.

III .Results

The study consists of 100 non diabetic cataract cases within the age group of 50 to 82 with 72 females and 28 males and 100 diabetic cataract cases within the age group of 46 to 74 with 64 females and 36 males.

The age wise distribution of patients show maximum no of cataract cases with or without diabetes was in the age group of 61 to 70 . The mean age and standard deviation of cataract cases without diabetic and with diabetes was 65.62±6.07 and 60.44±6.084 respectively, ($P>0.05$) which was insignificant.

The fasting and post prandial blood sugar level of the diabetic cases was 121.49±6.256mg/ml and 173.79±10.638mg/ml respectively with HbA1c level 7.2 ± 2.05% (Table 3)

The reference range for serum sodium was taken as 135-145 meq/l , for potassium was 3.5-5.0meq/l and that for chloride 98-105 meq/l.

The serum sodium level of diabetic cataract cases was highly elevated 148.3234±2.576meq/l in comparison with serum sodium level145.0645±2.243meq/l in non diabetic cataract cases , which was highly significant with $P<0.001$.

The serum potassium level of diabetic was 4.2728±0.4745meq/l in comparison to non diabetic 4.0712±0.4585meq/l was significant with $P<0.05$.

The serum chloride level of diabetic was 102.3554±1.439meq/l in comparison to non diabetic 100.3003±1614.meq/l with $P<0.001$, which was highly significant.

The sodium level of aqueous humour of diabetic was 159.7379±3.236meq/l in comparison to non diabetic 154.5053±3.264meq/l with $P<0.001$, which was highly significant.

The potassium level of aqueous humour of diabetic was 3.9224±0.5128meq/l in comparison to non diabetic 3.9637±0.4960mqe/l with $P>0.05$ which was insignificant.

The chloride level of aqueous humour diabetic was 112.0688±4.222meq/l in comparison to non diabetic 102.4310 ± 1.857.meq/l with $P<0.001$ which was highly significant. Values are depicted in Table 1 and 2.

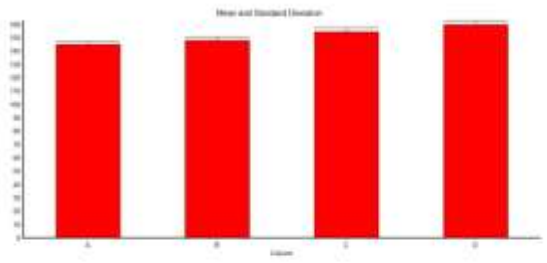
Table-1 Comparison of serum electrolytes in diabetic and non-diabetic cataract cases			
	Diabetic cataract cases(n=100)	Non-diabetic cataract cases(n=100)	P value
Sodium(meq/l)	148.3234±2.576	145.0645±2.243	<0.001***
Potassium(meq/l)	4.2728±0.4745	4.0712±0.4548	<0.05*
Chloride(meq/l)	102.431±1.857	100.3003±1.614	<0.001***
***highly significant * significant			

Table-2 Comparison of aqueous humour electrolytes in diabetic and non-diabetic cataract			
	Diabetic cataract cases(n=100)	Non-diabetic cataract cases(n=100)	P value
Sodium(meq/l)	159.7397±3.236	154.5053±3.264	<0.001***
Potassium(meq/l)	3.9224±0.5128	3.9637±0.4960	> 0.05ns
Chloride(meq/l)	112.0688±4.222	102.3554±1.439	<0.001***
*** highly significant ns= insignificant n= no.of subjects			

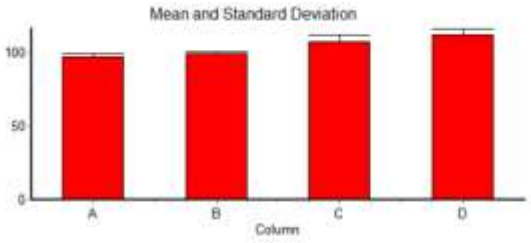
Table-3 Mean and standard deviation of serum sugar in diabetic cataract cases		
Fasting blood sugar(n=100)	Post prandial blood sugar(n=100)	HbA1c
121±6.256 mg/ml	173±10.638mg/ml	7.2±2.05%

Table-4 Odd's Ratio		
	Diabetic	Non-diabetic
Age 40-60yrs	54(a)	17(b)
Age 61-82yrs	14(c)	83(d)
Odd's Ratio =(a)(d)/(b)(c)=(54×83)(17×14)=5		

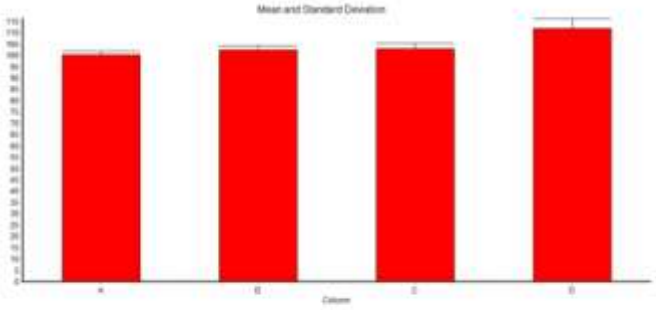
By calculating odd's ratio of age , the present study show the risk of early development of cataract was found to be 5 times higher in diabetic than non diabetic.(Table 4).



Histogram showing mean and standard deviation of serum and aqueous humour sodium level in diabetic and non diabetics.



Histogram showing mean and standard deviation of serum and aqueous potassium level of diabetic and non diabetics.



Histogram showing mean and standard deviation of serum and aqueous chloride level of diabetic and non diabetics.

IV. Discussion

Worldwide more than 285 million people are affected by diabetes mellitus. This number is expected to increase to 439 million by 2030 according to the International Diabetes Federation[15].Several clinical studies have shown that cataract development occurs more frequently and at an earlier age in diabetic compared to nondiabetic patients[27,28].

Data from the Framingham and other eye studies indicate a three to fiur fold increased prevalence of cataract in patients with diabetes under the age of 65 and up to a twofold excess prevalence in patients above 65[27,29]. The risk increased in patients with longer duration of diabetes and those with poor metabolic control[6].Catact may be reversible in young diabetics with improvement in metabolic control. The most frequently seen type of cataract in diabetics is the age –related or senile variety, which tends to occur earlier and progresses more rapidly than in nondiabetics[6].

Diabetic patients develop a constellation of electrolyte disorders. Diabetes mellitus is included among the diseases with increased frequency of electrolyte abnormalities, as impaired renal function, malabsorption syndromes, acid-base disorders and multidrug regimens, the pathophysiological factors are often present in diabetis[16].Diabetes is a well-known cause of dysnatremia via several underlying mechanisms[17.18].Glucose is an osmotically active substance. Hyperglycemia increases serum osmolality , resulting in movement of water out of the cells and subsequently in a reduction of serum sodium levels by dilution[15].

The development of hypernatremia is associated with endrocrine dysfunction. There is some evidence in animals and man that hypernaterimia and hyperosmolarity are associated with impairment of both insulin-mediated glucose metabolism and glucagon-dependent glucose release[19,20]. Moreover hypernaterimia is implicated in the profound inhibition of gonadotrophin release in postmenopausal diabetic women with hyperglycaemic hyperosmolar syndrome[21].

The causes of hypokalemia in diabetics include: redistribution of potassium from the extracellular to the intracellular fluid compartment (shift hypokalemia due to insulin administration); gastrointestinal loss of potassium due to malabsorption syndromes (diabetic induced motility disorders,bacterial overgrowth, chronic diarrheal states); and renal loss of potassium (due to osmotic dieresis and/or coexistent hypomagnesemia)[15].

The incidence of hyperkalemia is higher in diabetic patients than in the general population[22,23]. Redistribution of potassium from the intracellular to the extracellular compartment (shift hyperkalemia) can induce hyperkalemia with no net total body potassium increase. Examples of shift hyperkalemia in diabetes

mellitus include acidosis , insulin deficiency, hypertonicity, cell lysis and drugs (e.g beta blockers). Reduced glomerular filtration of potassium (due to acute kidney injury and chronic kidney diseases) and many drugs that interfere with potassium excretion are associated with hyperkalemia[15].

Aqueous humour was termed rightly as ocular lymph by Adler[24] Alteration in cation concentration of aqueous humour is attributed to alterations in serum cation concentration[25].

In our study we have found the mean sodium level in the serum and aqueous humour in diabetic cataract cases to be 148.3234 ± 2.257 and 159.7397 ± 3.236 respectively in comparison to non diabetic cases 145.0645 ± 2.243 and 154.5053 ± 3.264 respectively which is highly significant with a $P< 0.001$ in both (serum and aqueous).

Diabetic serum potassium mean level was 4.2728 ± 0.4745 in comparison to non diabetic 4.0712 ± 0.4585 which was within normal limit. However this difference in serum K^+ levels was significant with $P<0.05$. Aqueous humour mean potassium level in diabetic was 3.9637 ± 0.4960 in comparison to non diabetic was 3.9224 ± 0.5128 which was insignificant($P> 0.05$).

Mean serum chloride level in diabetic and nondiabetic was 102.3554± 1.439meq/l in comparison to nondiabetic 100.3003. ± 1.614. meq/l .Although it was within normal limit it however was highly significant with $P<0.001$.

Aqueous humour mean chloride level in diabetic and non diabetic was 112.0688 ± 4.222 meq/l and 102.431 ± 1.857meq/l respectively, which was highly significant with $P<0.001$.

The Na^+ K^+ $2Cl^-$ co transporter defect hampers chloride handling by the lens [26] which might cause raised chloride level in aqueous humour in cataract cases.

The changes of serum sodium, potassium and chloride level in nondiabetic cataract cases are in accordance with the studies by Usha s. Adiga et a[12] ,Tasneem A F et al[14] and Gaurav Mathur et al [13]. The elevation of aqueous humour sodium in non diabetic cataract cases are in accordance with the study by Tasneem A F et al[14].

V. Conclusion

We can conclude from our studies that There are a significant rise in serum and aqueous sodium levels in diabetics. There are increased chloride level in both diabetic and non diabetic aqueous humour, though there is normal serum chloride level in both the cases.Therefore with raised sugar and electrolyte level may be a factor of cataractogenesis in diabetics and salt restricted diet along with advised diabetic diet may delay the onset of diabetic cataract.Though in our study we found no significant relation of raised electrolyte level with early development of diabetic cataract , further study is advisable to propose the elevated level of serum and aqueous electrolyte as a risk factor of early development of diabetic cataract .

Conflicts of interest: – No conflict of interest is associated with this work.
Contribution of Authors :- We declare that this work was done by the authors named in this article and all liabilities pertaining to claims relating to the content of this article will be borne by the authors.

Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Gauhati Medical College and Hospital institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

References

[1]. A K Khurana, Indu Khurana Anatomy and Physiology of Eye, page 105.
[2]. J.J. Harding , M. Egerton, R. Van Heyningen, and R.S. Harding, "Diabetes, glaucoma, sex, and cataract: analysis of combined data from two case control studies," British Journal of Ophthalmology, vol. 77, no. 1 pp. 2-6, 1993.
[3]. H. A. Khan, H.M. Leibowitz, J.P.Ganley, et al, "The Framingham eye study. II. Association of ophthalmic pathology with single variables previously measured in the Framingham heart study," American Journal of Epidemiology, vol.106, no. 1, pp. 33-41, 1977.
[4]. J. H. Kinoshita, "Mechanisms initiating cataract formation. Proctor lecture," Investigative Ophthalmology, vol. 13, no. 10, pp. 713-724, 1974.
[5]. J. H. Kinoshita, S. Fukushi, P.Kador, and L. O. Merola, " Aldose reductase in diabetic complications of the eye," Metabolism, vol. 28, no. 4, pp. 462-469,1979.
[6]. Andreas Pollreisz and Ursula Schmidt-Erfurth, " Diabetic Cataract – Pathogenesis, Epidemiology and Treatment", Journal of Ophthalmology, volume 2010, Article ID 608751, 8 pages.
[7]. P. F. Kador and J. H. Kinoshita, "Diabetic and galactosaemic cataract", Ciba Foundation Symposium, vol. 106, pp. 110-131, 1984.
[8]. Sires B. Orbital and ocular anatomy. In: Wright editor. Textbook of Ophthalmology. Baltimore, MD: Williams and Wilkins; 1997.
[9]. Miller C, Kaufman PL. Aqueous humour: secretion and dynamics, In: TasmanW, Jaeger EA, editiors. Duane's foundations of clinical ophthalmology. Philadelphia: Lippincott-Raven; 1995.
[10]. Abou-Gareeb I, Lewallen S, Bassett K, Coutright P, "Gender and blindness: a meta-analysis of population-based prevalence surveys. Ophthalmic Epidemiol 2001; 8:39-56.
[11]. Luntz MH, Clinical types of cataract. Duane's Clinacal Ophthalmology. Philadelphia: Lippincott-Raven publishers, 2005;5-7.

[12]. Usha S. Adiga, Adline Harris, T.N. Ezhilvathani and Sharbari Basu, "Serum electrolytes in senile cataract patients". Al Ameen J Med Sci 2014; 7(2): 164-168.
[13]. Mathur G, Pai V, "Comparision of serum sodium and potassium levels in patients with senile cataract and age-matched individuals without cataract" , Indian Journal of Ophthalmology.
[14]. Tasneem A F, Shwetha B A, Mamata N, "Comparative study of serum and aqueous humour electrolyte levels in cataract patients", International Journal of Recent Trends in Science and Technology. October 2014; 12(3): 507-513.
[15]. George Liamis, Evangelos Liberopoulos, Fortios Barkas, and Moses Elisaf, "Diabetes mellitus and electrolyte disorders" World J Clin Cases. 2014 Oct 16; 2(10): 488-496.
[16]. Elisaf MS, Tsatsoulis AA, Katopodis KP, Siamopoulos KC, Acid-base and electrolyte disturbances in patients with diabetic ketoacidosis. Diabetes Res Clin. Pract. 1996; 34:23-27. [PubMed]
[17]. Liamis G, Rodenburg EM, Hofman A, Zietse R, Stricker BH, Hoon EJ. Electrolyte disorders in community subjects: prevalence and risk factors. Am J Med. 2013; 126:256-263.[PubMed]
[18]. Liamis G, Tsimihidimos V, Doumas M, Spyrou A, Bairaktari E, Elisaf M. Clinical and laboratory characteristics of hypernatraemia in an internal medicine clinic. Nephrol Dial Transplant, 2008;23:136-143.[PubMed]
[19]. 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. Biochem Med Metab Biol. 1988;39:312-318.[PubMed]
[20]. Liamis G, Milionis H, Elisaf M. Blood pressure drug therapy and electrolyte disturbances. Int J Clin Pract. 2008;62:1572-1580.[PubMed]
[21]. Lado-Abeal J, Lorenzo-Solar M, Lago-Lestón R, Palos-Paz F, Domingez-Gerpe L. Hyperglycaemic hyperosmolar nonketotic state as a cause of low gonadotrophin levels in postmenopausal diabetic women: a role forhypernatraemia. J Neuroendocrinol. 2007;19:983-987.[PubMed]
[22]. Palmer BF. Mcanaging hyperkalemia caused by inhibitors of the rennin-angiotensin-aldosterone system. N Engl J Med. 2004;351:585-592.[PubMed]
[23]. Uribarri J, Oh MS, Carroll HJ. Hyperkalemia in diabetes mellitus.J Diabet Complications.1990;4:3-7. [PubMed]
[24]. ADLER, F.H.: The 'Aqueous Humour' in physiology of the eye, Clinical application.4th Ed. P. 96-143 C.V. & Co. ; St. Louis (1995).
[25]. Clayton RM, Cuthbert J, Phillips CI et al. Analysis of individual cataract patients and their lenses: A progress report. Exp Eye Res 1980; 31:533-536.
[26]. Zhang JJ, Jacob TJC, "The role of chloride in the lens of the eye," Experimental Physiology 1997;82:245-259.
[27]. B.E. K. Klein, R. Klein, and S.E. Moss, "Prevalence of cataracts in a population-based study of persons with diabetes mellitus", Ophthalmology, vol.92,no. 9, pp. 1191-1196,1985.
[28]. W. E. Benson, "Cataract surgery and diabetic retinopathy", Current Opinion in Ophthalmology, vol. 3, no. 3, pp. 396-400, 1992.
[29]. F. Ederer, R. Hiller, and H.R. Taylor, "Senile lens changes and diabetes in two population studies", American Journal of Ophthalmology," vol. 91, no. 3, pp. 381-395.1981.