IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 7 Ver. 9 (July. 2018), PP 62-64
www.iosrjournals.org

Study of Serum Cholinesterase, Lipid Profile and Serum Proteins in Nephrotic Syndrome

[1]U. Gayathri Devi, [2]N. Gayathri Devi,
[1]MSc.(Bio) Tutor, Dr PSIMS & RF, Chinaoutpalli
[2]MD(Bio), Asst. Prof, Dr PSIMS & RF, Chinaoutpalli
Corresponding Author:N.Gayathri Devi

Abstract:
Background: *Nephrotic syndrome is most commonly occurring problem in childhood and others. It is highly morbid even though many advances has evolved.*
Aim*: To study the changes of serum proteins and serum cholinesterase in nephrotic syndrome.*
Materials and methods*: A group of sixty subjects were recruited in this study between the ages of 10 - 20 years. Out of sixty subjects thirty were controls and thirty were age and sex matched subjects. We have estimated serum proteins, serum cholinesterase, serum uric acid, serum cholesterol and serum albumin in the both groups. The parameters cholesterol andcholinesterase showed statistically significantly elevated whereas serum total proteins and serum albumin is statistically significantly decreased.*
Conclusion: *serum cholesterol, serum total proteins and serum albumin are related to the nephrotic syndrome with the elevation of serum cholinesterase activity.*

Date of Submission: 09-07-2018 Date Of Acceptance: 23-07-2018

I. Introduction

Nephrotic syndrome is one of the most common kidney diseases in children than in adults, and is characterized by massive proteinuria, edema, hyperlipidemia and hypoalbuminaemia[1] . The annual incidence and prevalence of nephrotic syndrome in children are 2–7 new cases and 16 cases per 100,000 children, respectively, and in adults the yearly incidence is three new cases per 100,000 adults [2–5]. The primary pathology is due to the damage in podocyte and glomeruli[6,7].Nephrotic syndrome occurs when changes in the permeability of the glomerular capillary wall can no longer restrict the loss of protein to a minimal level, thus resulting in massive protein loss through the urine [8]. Nephrotic syndrome can result in lethal infections, thrombosis, and pulmonary edema as a result of the significant protein loss [8].

The principal biological role of acetylcholinesterase (ACHE, acetylcholine hydrolase, EC 3.1.1.7) is termination of impulse transmission at cholinergic synapses by rapid hydrolysis of the neurotransmitter acetylcholine (ACh) to yield acetic acid and choline[9]. Serum butyrylcholinesterase, commonly known as serum cholinesterase, is an enzyme synthesized by hepatocytes and has the half-life of eleven days[10].

Lipoproteins are major carriers of lipid in the body by different pathways. This metabolism is altered in nephrotic syndrome with or without chronic kidney disease[11]. Magnitude of proteinuria is dependent on altered lipid metabolism. These changes in serum lipids and lipoproteins in patients with nephrotic syndrome are primarily a result of their impaired clearance and, to a lesser extent, their altered biosynthesis. The levels of both IDL and VLDL are increased in patients with nephrotic syndrome owing to defective LPL activity and decreased hepatic lipase activity[12]. The binding of LPL to heparansulfate proteoglycans on endothelial cells occurs by endothelium-derived glycosylphosphatidylinositol-anchored HDL-binding protein 1 (GPIHBP1)[13]. It is downregulated in patients with nephrotic syndrome[14]. The loss of LPL activators is associated with increased glomerular basement membrane permeability, resulting in hyperlipidaemia[15]. In addition to downregulation of LPL activity, nephrotic syndromecauses the down regulation of hepatic lipase activity, which contributes to decreased clearance of IDL and hypertriglyceridaemia[16].

As we find very few studies and there are no recent studies on this, so we like to study on these parameters in nephrotic syndrome..

II. Materials and Methods

Total of Sixty subjects were recruited in this study between the ages of 10 - 20 years. Out of sixty subjects thirty were controls and thirty were age and sex matched subjects. All subjects agreed to provide their personal information regarding the purpose and the procedures of our study and written informed consent. This study was approved by local ethics committee. This study was conducted in department of Biochemistry, Dr

PSIMS & RF Chinnaoutpalli. The study was approved by Institutional Ethical Committee (IEC). Written Informed consent of participants was taken prior to study. Fasting samples were collected from all the sixty subjects. Serum uric acid was estimated by the uricase method, triglycerides were estimated by , Total cholesterol by CHOD-PAP, Albumin by Bromocresol Green Method, Total proteins byBiuret Method and Serum Cholinesterase by knedel and Klin Method using standard enzymatic methods by Randox Daytona autoanalyser.

Inclusion criteria:
Patients were cooperative and willing to give the required information.
Patients from the age of 10 – 20 years of both sexes were included in our study.
Proteinuria greater than 3-3.5 g/24 hour or spot urine
Protein:creatinine ratio of >300-350 mg/mmol
Serum albumin <2.5 g/l
Clinical evidence of peripheral edema
Severe hyperlipidaemia (total cholesterol often>10 mmol/l) is often present

Exclusion criteria:
Patients who were not cooperative for the study.
Patients having diagnosed for renal failure and liver diseases were excluded in our study
Patients of age more than 20 years were excluded.

III. Results and Discussion

S. No.	Investigations	Control		cases	P – Value
		Range	Mean(±SD)	Mean(±SD)	
1.	Total proteins(gm/dl)	6.2 – 7.5	6.85(±1.61)	4.25(±0.58)	P<0.001
2.	Serum Albumin(gm/dl)	3.2 -4.9	4.05(±0.56)	1.65(±0.08)	P<0.001
3.	Serum Globulins(gm/dl)	2.6 – 3.2	2.9(±0.29)	2.59(±0.20)	P<0.001
4.	Serum Cholinesterase(IU/L)	9000-12000	9953.7(±1949.1)	30597(±8723.1)	P<0.001
5.	Serum Cholesterol (mg/dl)	120 – 220	170(±26.3)	366.6(±135)	P<0.001
6.	Serum triglycerides(mg/dl)	40 – 150	137.5(±18.68)	314.8(±190.9)	P<0.001

In our study Total cholesterol levels were statistically significantly increased (p<0.001) in nephrotic cases compared to controls. In our study Triglycerides levels were statistically significantly increased (p<0.001) in nephrotic cases compared to controls.Vaziri[11] in his work attributed this hypercholesterolemia to a defective regulatory response of 3- hydroxy-3-methylglutararyl-coenzyme A (HMG-COA) reductase and hepatic cholesterol 7α-hydroxylase in nephrotics. These enzymes are rate–limiting enzymes in cholesterol biosynthesis and catabolism to bile acids in humans. There was also an increased level of triglycerides in nephrotics when compared with control. This is in agreement with the previous reports[17-19] This hypertriglyceridaemia observed in nephrotics is attributed to downregulation of lipoprotein lipase as found in nephrotics skeletal muscle, myocardium and adipose tissue, which is the principle sites of fatty acids consumption and storage[11].In nephrotic syndrome the total cholesterol and triglycerides are elevated due to hypoproteinemia inducing increase in protein synthesis in liver leading to hyperlipidemia[20].

In our study serum total proteins and albumin were statistically decreased (p<0.001) in nephrotic cases compared to controls. Nephrotic syndrome is characterized by increased urinary excretion of albumin and other proteins leading to development of hypoprotienemia. The rate of albumin synthesis may be increased, but not sufficiently to maintain normal serum albumin concentration or albumin pools. Augmentation of dietary protein in nephrotic rats directly stimulates albumin synthesis by increasing albumin mRNA content in the liver, but also causes an increase in glomerular permeability to macromolecules so that much if not all of the excess albumin synthesized is lost in the urine[21].

In our study serum cholinesterase were statistically increased (p<0.001) in nephrotic syndrome cases compared to controls. Our study Is coordinating with the studies of Kunkel and Ward[22], Maier[23], Pietschmann[24], Stefenelli[25], et al. . The nephrotic syndrome is the only condition in which hypo-albuminemia and a high serum cholinesterase regularly co-exist.

IV. Conclusion

Dyslipidemia and hypoproteinemia are strongly associated with the development of nephrotic syndrome and on cholinesterase, still more studies are required to establish its importance in nephrotic syndrome or kidney disorders.

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