



Assessment of serum lipids & proteins in nephrotic syndrome children's

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Abstract

Nephrotic-range proteinuria is the loss of 3 grams or more per day of protein into the urine or, on a single spot urine collection, the presence of 2 g of protein per gram of urine creatinine. Nephrotic syndrome is the combination of nephrotic-range proteinuria with a low serum albumin level and edema. Lipoproteins play an important role in the transport of plasma lipids; their increase or alteration in various fractions may be responsible for hypercholesterolemia, in nephrotic syndrome. Hence based on above literature findings the present study was planned to evaluate the derangement of serum lipids in nephrotic syndrome Children.

The 80 child patients were enrolled From Upgraded Department of Paediatrics in Patna Medical College and Hospital, Patna from Jan 2017 to Oct 2017 in to the present study. The 80 childrens were divided in two groups as control and cases. The group I consist of 40 patients as normal patients and group II patients has 40 patients suffered from nephrotic syndrome.

Hence from above study it can be concluded that the positive relationship between the serum lipid profile and the nephrotic syndrome. Bases on these findings we therefore advocate that lipid profile should be among the biochemical investigations included for any nephritic syndrome patient for better management.

Keywords: cholesterol, lipoprotein, nephrotic syndrome

Introduction

Nephrotic-range proteinuria is the loss of 3 grams or more per day of protein into the urine or, on a single spot urine collection, the presence of 2 g of protein per gram of urine creatinine. Nephrotic syndrome is the combination of nephrotic-range proteinuria with a low serum albumin level and edema.

Nephrotic syndrome has many causes, including primary kidney diseases such as minimal-change disease, focal segmental glomerulosclerosis, and membranous glomerulonephritis. Nephrotic syndrome can also result from systemic diseases that affect other organs in addition to the kidneys, such as diabetes, amyloidosis, and lupus erythematosus^[1].

Nephrotic syndrome is a kidney disorder characterised by high levels of protein in the urine and swelling of body tissue. People of any age can be affected by nephrotic syndrome, although children aged between 18 months and four years are at increased risk. Treatment involves controlling symptoms and treating the underlying condition that is causing the nephrotic syndrome. Long-term nephrotic syndrome can lead to irreparable kidney damage leading to kidney failure, necessitating treatment with dialysis or, eventually, kidney transplant.

Nephrotic syndrome produces a collection of symptoms and signs that occur because tiny blood vessels (the glomeruli) in the kidneys sustain damage and do not function correctly. The syndrome is characterised by abnormally high levels of protein in the urine (particularly one called albumin) and abnormally low protein levels in the blood. This often leads to

fluid retention in the body tissues causing swelling (oedema) and can be associated with high blood cholesterol levels and high blood pressure. The glomeruli act as filters to remove waste products from the blood, which are then excreted from the body in the urine. In nephrotic syndrome these filters become defective and leak, allowing large quantities of protein to be lost in the urine. Protein in the blood acts to prevent water entering the body's cells. Reduced blood protein levels cause water to leak into body tissue, causing swelling.

Treatment will depend on the underlying cause of the condition and is aimed at alleviating symptoms and preventing complications. Medications that reduce the inflammatory effects of the immune system on the glomeruli are the mainstay of treatment. Medications to achieve this include corticosteroids (eg: prednisone) and potent immune-suppressing medications such as cyclosporine, cyclophosphamide, mycophenolate and rituximab. Medications to reduce high blood pressure and high blood cholesterol levels may also be prescribed. Blood thinning medications (anticoagulants) may be prescribed to reduce the risk of developing blood clots^[2].

Proteinuria occurs because of changes to capillary endothelial cells, the glomerular basement membrane (GBM), or podocytes, which normally filter serum protein selectively by size and charge. The mechanism of damage to these structures is unknown in primary and secondary glomerular diseases, but evidence suggests that T cells may up regulate a circulating permeability factor or down regulate an inhibitor of permeability factor in response to unidentified immunogens and cytokines. Other possible factors include hereditary

defects in proteins that are integral to the slit diaphragms of the glomeruli, activation of complement leading to damage of the glomerular epithelial cells and loss of the negatively charged groups attached to proteins of the GBM and glomerular epithelial cells [3].

Hyperlipidemia has been recognized as a common finding in nephrotic patients since 1917, when hypercholesterolemia was described as a feature of nephrotic syndrome. Although pathophysiological aspects of hyperlipidemia have not been completely identified, hypoalbuminemia, increased lipoprotein synthesis and decreased lipoprotein lipase activity are described by various workers. Lipoproteins play an important role in the transport of plasma lipids; their increase or alteration in various fractions may be responsible for hypercholesterolemia, in nephrotic syndrome. There is increased total cholesterol, LDL cholesterol, VLDL cholesterol and triglycerides and normal or low HDL cholesterol. However, in Indian children, the degree of hyperlipidemia is not high as in western children. More recently it has been expressed that hyperlipidemia may contribute to renal injury.

Hence based on above literature findings the present study was planned to evaluate the derangement of serum lipids in nephrotic syndrome Children.

Materials and methods

The 80 child patients were enrolled From Upgraded Department of Paediatrics in Patna Medical College and Hospital, Patna from Jan 2017 to Oct 2017 in to the present study. The 80 children were divided in two groups as control and cases. The group I consist of 40 patients as normal patients and group II patients has 40 patients suffered from nephrotic syndrome.

The approval of the institutional ethics committee was taken before starting the study. All the patients and their parents were informed consents. The aim and the objective of the present study were conveyed to them.

A diagnosis of nephrotic syndrome was confirmed in patients in the presence of Massive proteinuria, Hypoalbuminaemia, Oedema and Hypercholesterolaemia.

The inclusion criterion for present study is infants and children between 0-15 years of age suffering from nephrotic syndrome. The children with liver disorders, oedema due to Kwashiorkor were excluded from the present study.

Fasting venous blood samples were collected with minimum stasis into plain container. This was allowed to clot and spun in a centrifuge for 10 minutes. The serum was separated and kept frozen until required for analysis. The samples were analysed for Protein profile (Serum Total protein, serum albumin, serum globulin, A: Gratio, urinary proteins, Blood urea & serum creatinine), Lipid Profile (Total cholesterol, HDL-C, LDLC, VLDL, Non-HDLC, serum phospholipids and triglycerides).

Results & Discussion

The data from 80 children were collected. The 80 children were divided in two groups as control and cases. The group I consist of 40 patients as normal patients and group II patients has 40 patients suffered from nephrotic syndrome. The data were summarized and presented as below.

Table 1: Age, Sex & No. of Cases

Group	Group I	Group II
Age in years	Normal patients	Nephrotic syndrome patients
Below 5 years	22	25
5 to 10 years	15	9
Above 10 years	3	6
Total	40	40
Sex	No of cases	No of cases
Males	22	25
Females	18	15
Total	40	40

Table 2: Observed Serum Levels of Lipid Profile

Group	Group I	Group II
Type of Patients	Normal patients	Nephrotic syndrome patients
Type of Cases	Normal patients	Nephrotic syndrome patients
Total Cholesterol	158.5-223.6mg/dl	285.5-510.5mg/dl
High Density Lipids	42.6-55.9mg/dl	44.3-57.6mg/dl
Low Density Lipids	112.3-146.9mg/dl	253.4-349.8mg/dl
Very Low Density Lipid	41.5-53.5mg/dl	46.2-61.5mg/dl
Triglycerides	78.9-112.5mg/dl	266.7-289.6mg/dl

Table 2: Observed serum levels of serum proteins

Group	Group I	Group II
Type of Patients	Normal patients	Nephrotic syndrome patients
Serum Total Protein	6.85-7.93 g/dl	3.37-4.25 g/dl
Serum Albumin	3.97-4.43 g/dl	1.55-2.08 g/dl
Serum Globulin	3.07-3.63 g/dl	2.19-2.59 g/dl

Our study shows that in nephrotic syndrome, there is generalized hyperlipidemia and hypoalbuminemia. Although hyperlipidemia is most marked when serum albumin is low, yet no definite correlation can be established between the degree of hypoalbuminemia and rise of lipids.

Arije *et al* also observed persistent rise in serum lipids in frequent relapse cases [4]. Milner ported that the total cholesterol in nephrotic syndrome may be higher than 1000 mg% [5] we observed low serum lipids in Indian children.

Merouani *et al* observed hyperlipidemia during the active phase of the disease and disappeared with resolution of the proteinuria and was persistently abnormal in frequently relapsing children [6]. Tsukahara *et al* observed that children with frequently relapsing nephrotic syndrome have prolonged periods of hypercholesterolemia [7]. Queried used statins in his study and observed 30-40% reduction in the total cholesterol [8]. Buyokcelik *et al* observed significant reduction in the total cholesterol with statins in adult patients with nephrotic syndrome [9].

Thomas *et al* found correlation between serum cholesterol and albumin and did not find correlation between serum cholesterol and globulin and total protein [10-11]. Friedman and Byers postulated that hypoalbuminemia causes hyperlipidemia [12].

The underlying cause of hyperlipidemia in nephrotic syndrome was related by previous investigators to proteinuria and altered lipoprotein metabolism. It has been proposed that

hypoalbuminemia causes reduced serum oncotic pressure, which in turn stimulates hepatic synthesis of albumin another liver-derived proteins, including Apo lipoproteins.

Conclusion

Hence from above study it can be concluded that the positive relationship between the serum lipid profile and the nephrotic syndrome. Bases on these findings we therefore advocate that lipid profile should be among the assurances of the biochemical investigation including these indices for any nephrotic syndrome patient for better management.

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