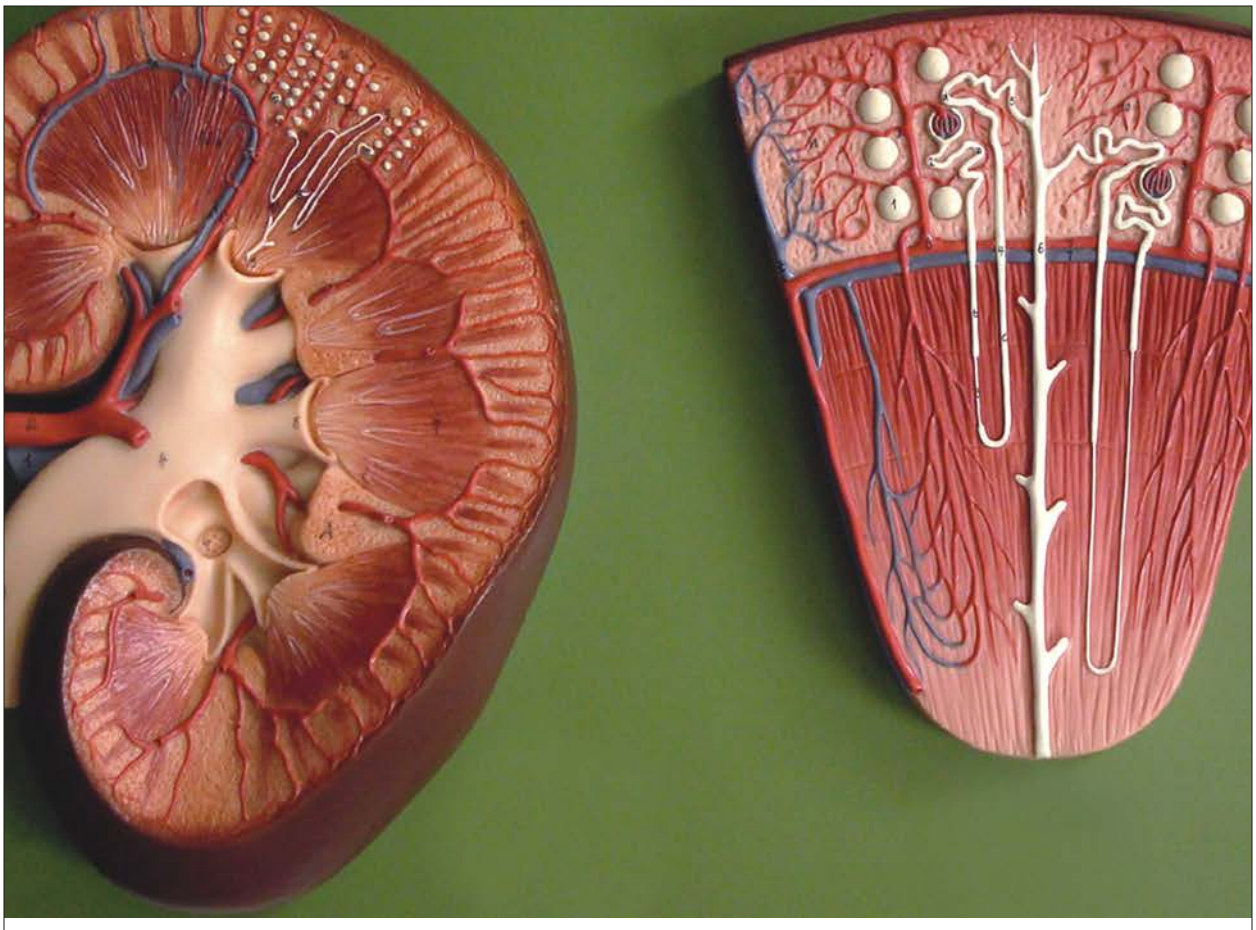

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Research Article
Evaluation of Thyroid Functions, Oxidative Stress and Antioxidants in Egyptian Children with Nephrotic Syndrome

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Abstract

Background: Nephrotic Syndrome (NS) is characterized by heavy proteinuria that leads to loss of significant amounts of thyroid hormones. Reactive Oxygen Species (ROS) seem to play an important role in the etiopathogenesis of protienuria in nephrotic syndrome. **Objective:** Evaluation of thyroid functions, oxidative stress and antioxidants in children with nephrotic syndrome. **Materials and Methods:** The present study was carried out at Al-Azhar University Hospital, Damietta during the period from March, 2015 to February, 2016. The study included 30 Egyptian children with steroid responsive nephrotic syndrome in relapsing phase and remission phase (Cases) and other 30 healthy Egyptian children (Control group). Venous blood was collected for the estimation of SOD activity, MDA, glutathione-S-transferase (GST) and estimation of thyroid hormones FT3, FT4 and TSH. **Results:** Serum FT3 and FT4 was significantly lower in patients with relapse (2.84±0.74 and 3.14±1.34, respectively) in comparison to remission (8.1±2.64 and 16.75±3.69, respectively) and control group (8.97±1.95 and 18.34±4.68, respectively). However, there was significant increase of TSH in patients with relapse (6.7±2.4) in comparison to remission (2.6±1.8) and control group (2.4±1.2). The SOD and glutathione-S-transferase levels were significantly decreased in patients with relapse (5.24±2.17 and 2.14±1.27, respectively) in comparison to remission (7.14±1.68 and 3.15±0.96, respectively) and control group (7.69±2.43 and 3.54±1.83, respectively). However, there was significant increase in MDA levels in patients with relapse (8.4±2.74) in comparison to remission (2.1±1.22) and control group (1.8±0.86). **Conclusion:** Nephrotic syndrome can loss significant amounts of thyroid hormones along with protein in urine. Increased Reactive Oxygen Species (ROS) as MDA and decreased antioxidants as SOD and GST may be related to the pathogenesis of proteinuria in NS.

Key words: Nephrotic syndrome, thyroid functions, oxidative stress, antioxidants

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Nephrotic syndrome which is more common in children is characterized by heavy proteinuria, hyperlipidemia, hypoalbuminemia and peripheral edema. Idiopathic Nephrotic Syndrome (INS) is one of the most common renal problems in children[1,2].

Nephrotic syndrome results in loss of plasma proteins and various other macromolecules in the urine leading to their deficiencies. Many of the physiologically important molecules which exist in the plasma, bound to plasma proteins are also carried away and lost in urine. The common abnormalities arising as a result of heavy proteinuria include hypothyroidism, vitamin D deficiency and iron deficiency[3-5].

Thyroid hormones (THs) are essential for normal metabolic functions of the kidneys. Thyroid dysfunction causes remarkable changes in glomerular and tubular functions, electrolytes and water homeostasis. The decrease in thyroid hormones was often attributed to the urinary loss of Thyroid Binding Globulin (TBG) as a result of proteinuria[6,7].

Albumin is a non-enzymatic protein antioxidant that inhibits LDL peroxidation *in vitro*[8]. Oxidative damage has been proposed as one of the possible mechanisms involved in the NS[9].

Oxidative damage by free radicals has been implicated in a number of clinical disorders including renal injury. Reactive Oxygen Species (ROS) promote cell injury by lipid peroxidation which disrupts the structural integrity of the tubular epithelial cells and increase glomerular permeability to proteins along with alteration of glomerular hemodynamics[10,11].

The NS is a consequence of an imbalance between oxidants and antioxidants activity. It was observed that superoxide mediated oxidative injury degrades the glomerular basement membrane and reduces *de novo* synthesis of proteoglycans that affects the glomerular permeability[12].

This study suggests that nephrotic syndrome patients may benefit from antioxidant therapy along with thyroid hormone supplement[13].

Therefore, malondialdehyde (MDA) and nitric oxide indicate the extent of lipid peroxidation caused by reactive oxygen species while, SOD, vitamin E, albumin, uric acid bilirubin and total antioxidant capacity determines antioxidant status of body[14,15].

Cellular defense mechanisms against ROS including enzymatic systems such as superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx) and non-enzymatic antioxidant defense system containing albumin, reduced glutathione, uric acid, vitamin C, vitamin E, selenium and zinc[16]. Antioxidants prevent the production of reactive oxygen substances so, they can play a major role in decreasing the injury in nephrotic syndrome[17].

In general, the molecular mechanisms behind acquired NS remain largely unknown. In addition, there is controversy regarding thyroid function among children with nephrotic syndrome and there is lack of studies among Egyptian children. The present study was designed to evaluate the possible role of oxidative stress and its relation to response to treatment and correlation with thyroid function.

MATERIALS AND METHODS

The present study was carried out at Al-Azhar University Hospital, Damietta from March, 2015 to February, 2016. An informed consent from the parents of children had been performed. The study included 30 children with steroid responsive nephrotic syndrome in relapsing phase and remission phase (Cases) and other 30 healthy children (Control group).

All children evaluated for:

- Complete clinical examination
- Diagnosis of nephrotic syndrome in relapsing phase by proteinuria (24 h urine protein>150 mg) hypoalbunimia (<2.5 mg dL^{-1}) hyperlipidemia and edema and were carried out by auto-analyzer using commercially available kits. Children were said to be in remission phase when there was no proteinuria or trace (24 h urine protein<150 mg dL^{-1}) for 3 consecutive days and no edema
- Renal function tests as blood urea and serum creatinine
- Thyroid function tests (a) Free tri-iodothyronine (FT3), (b) Free thyroxine (FT4) and (c) Thyroid-stimulating hormone (TSH) using Immulite (REF:030001-04) and laboratory equipment manufactured by Siemens
- Estimation of oxidative stress (Malondialdehyde content) the end product of the free radicals initiating lipid peroxidation was measured by using thiobarbituric acid reactivity as described by Uchiyama and Mihara[18]. Estimation of antioxidants as superoxide dismutase (SOD) was measured by the method of Marklund and Marklund[19]. Glutathione-S-transferase (GST) activity was measured according to the method defined by Habig *et al.*[20] under standard conditions, the amount of enzyme conjugating 1 μmol of 1-chloro-2,4-dinitrobenzene (CDNB) with glutathione

(GSH) in 1 min was defined as 1 unit activity (μmol L^{-1}). All the parameters of oxidative stress were spectrophotometrically analyzed using the biochemical analyzer manufactured by (ERMA INC. Tokyo, JAPAN, model: AE-600N).

About 10 mL of venous blood was collected of which 4-5 mL was poured into sterile bulb containing heparin for the estimation of SOD activity and GST[21]. Remaining blood was taken into sterile plain bulb for estimation of MDA[22], total protein or albumin[23], uric acid and creatinine[24], cholesterol and triglycerides[25].

Exclusion criteria: Patients with steroid resistant with atypical presentation (Age<2 years and >8 years, hematuria, hypertension and abnormal renal function test), patients with obesity and patients on drugs such as vitamins and minerals that alters the oxidative stress parameters.

Statistical analysis: The numerical (quantitative) collected data were presented as Mean±Standard Deviations (SD). On the other hand, categorical data were presented as relative frequency and percent distributions. Unpaired student (t) test was used for comparison between two means and chi-square (χ^2) was used for comparison between two categorical variables. The p-value≤0.05 was considered significant for interpretation of results.

RESULTS

Age and sex distribution of cases and control group were presented in Table 1, there were non-significant difference between cases and control group with regards to the age (p = 0.70) and sex (p = 0.77).

Regarding biochemical parameters of cases and control group were presented in Table 2, the levels of serum total protein and albumin were significantly lower in cases with relapse (3.92±0.90 and 2.10±0.42, respectively) in comparison with cases in remission (6.22±0.71 and 4.63±0.54, respectively) and control group (6.75±1.3 and 4.91±0.58, respectively) while, the levels of cholesterol and triglyceride were significantly higher in cases with relapse (489.23±65.24 and 182.39±49.31, respectively) in comparison with cases in remission (204.28±76.54 and 89.28±31.25, respectively) and control group (158.21±84.46 and 83.14±29.64, respectively). As regarding proteinuria there was significant increase in cases with relapse (3500±1149) in comparison with cases in remission (145±66) and control group (103±29). However, non-significant difference was observed in remission cases and control group regarding total protein, serum albumin, cholesterol, triglyceride and proteinuria. On other hand, non-significant difference was observed between the three groups regarding BUN and creatinine.

Regarding thyroid hormones levels there was significant increase in TSH level in cases with relapse (6.7±2.4) in comparison with cases in remission (2.6±1.8) and control group (2.4±1.2). While, there was significant decrease in FT3 and FT4 levels in cases with relapse (2.84±0.74 and 3.14±1.34, respectively) in comparison with cases in remission (8.1±2.64 and 16.75±3.69, respectively) and control group (8.97±1.95 and 18.34±4.68, respectively). But, there was non-significant difference between cases in remission and control group regarding TSH, FT3 and FT4 (Table 3).

Regarding parameters of oxidative stress and antioxidant there was significant increase in (MDA) in cases with relapse (8.4±2.74) in comparison with cases in remission (2.1±1.22) and control group (1.8±0.86). On other hand, there was significant decrease in SOD and GST in cases with relapse (5.24±2.17 and 2.14±1.27, respectively) in comparison with cases in remission (7.14±1.68 and 3.15±0.96, respectively) and control group (7.69±2.43 and 3.54±1.83, respectively).

Table 1: Age and sex distribution of cases and control groups

Group variable	Cases (N = 30)	Control (N = 30)	p-value
Age (Mean±SD)	5.34±3.67	5.67±2.93	0.70
Sex (Boys/Girls)	17/13	16/14	0.77

Independent sample t-test was performed at 5% level of significance

Table 2: Biochemical parameters of cases and control groups

	Cases		
Group variable (Mean±SD)	Relapsing	Remission	Control
Total serum protein (g dL^{-1})	3.92±0.90	6.22±0.71*	6.75±1.3*
Serum albumin (g dL^{-1})	2.10±0.42	4.63±0.54*	4.91±0.58*
Serum cholesterol (mg dL^{-1})	489.23±65.24	204.28±76.54*	158.21±84.46*
Serum triglyceride (mg dL^{-1})	182.39±49.31	89.28±31.25*	83.14±29.64*
Proteinuria (mg/24 h)	3500±1149	145.00±66*	103.00±29*
BUN (mg dL^{-1})	13.23±2.43	12.37±2.51	11.84±2.97
Serum creatinine (mg dL^{-1})	0.56±0.41	0.51±0.52	0.48±0.39

ANOVA test was performed at 5% level of significance and *Significant difference from relapsing group

Table 3: Thyroid hormone levels in cases and control groups

	Case		
Group variable	Relapsing	Remission	Control
TSH (μIU mL^{-1})	6.70±2.4	2.60±1.8*	2.40±1.2*
FT_3 (pg mL^{-1})	2.84±0.74	8.10±2.64*	8.97±1.95*
FT_4 (pg mL^{-1})	3.14±1.34	16.75±3.69*	18.34±4.68*

ANOVA test was performed at 5% level of significance and *Significant difference from relapsing group

Table 4: Parameters of oxidative stress and antioxidants in cases and control groups

	Cases		
	Relapsing	Remission	Control
Malondialdehyde (MD) (nm mL^{-1})	8.40±2.74	2.10±1.22*	1.80±0.86*
Superoxide dismutase (SOD) (IU mL^{-1})	5.24±2.17	7.14±1.68*	7.69±2.43*
GST (μmol L^{-1})	2.14±1.27	3.15±0.96*	3.54±1.83*

ANOVA test was performed at 5% level of significance and *Significant difference from relapsing group

Table 5: Correlation between thyroid function tests and parameters of oxidative stress

Thyroid functions		TSH	FT3	FT4
MDA	r	0.26	-0.37	-0.46
	p	0.043	0.017	0.011
SOD	r	-0.19	0.27	0.38
	p	0.042	0.006	0.003
GST	r	0.30-	0.22	0.16
	P	0.007	0.021	0.047

Pearson's correlation coefficient was performed, MDA: Malondialdehyde, SOD: Superoxide dimutase and GST: Glutathione-S-trandferase

But there was non-significant difference between cases in remission and control group regarding MDA, SOD and GST levels (Table 4).

Table 5 shows MDA levels have significantly negative correlation with FT4 and FT3 but have significantly positive correlation with TSH. On other hand, SOD and GST levels have significantly positive correlation with FT4 and FT3 but have significantly negative correlation with TSH.

DISCUSSION

Nephrotic Syndrome (NS) is a common disorder characterized by alteration of permeability of the glomerular capillary wall, resulting in its inability to restrict the urinary loss of proteins with hypoalbuminemia and hyperlipidemia associated with peripheral edema[26,27].

Hypoalbuminemia in NS is due to increased glomerular permeability leading to proteinuria. Infact, this molecule may represent the major circulating antioxidant in plasma known to be exposed to continuous oxidative stress[28,29].

The present study was designed to evaluate thyroid function, oxidative stress and antioxidant in children with nephrotic syndrome. The results of the study revealed that the levels of serum total protein and albumin were significantly lower in cases with relapse in comparison with cases in remission and control group while, the levels of cholesterol and triglyceride were significantly higher in cases with relapse in comparison with cases in remission and control group such as regarding proteinuria there was significant increase in cases with relapse in comparison with cases in remission and control group. However, non-significant difference was observed in remission cases and control group regarding serum total protein, albumin, cholesterol, triglyceride and proteinuria. On other hand, non-significant difference was observed between the three groups regarding BUN and creatinine. This was in agreement with study inducted by Kharb[14] who shows that cholesterol and triglyceride levels were significantly increase in study group of nephrotic syndrome.

In the present study, the findings of low levels of thyroxin with high levels of TSH in cases with relapsing suggest that a state of primary biochemical hypothyroidism exists in relapsing nephrotic children. This was in agreement with the study conducted by Feinstein *et al.*[30] who showed that the reduced serum levels of FT4 and FT3 in patients with NS may be due to decreased binding to and concentration of serum carrier proteins. The NS in relapse results in loss of plasma proteins and various other macromolecules in the urine leading to their deficiencies.

Gilles *et al.*[5] reported that abnormalities in thyroid function are seen in patients with proteinuria. Specifically, TSH levels were higher in patients with proteinuric renal diseases when compared with controls. These results indicating that nephrotic syndrome increases L-thyroxine requirements because of urinary loss of free and protein-bound thyroid hormones which might leads to clinical hypothyroid state and need for L-thyroxine supplementation. Thus, it is important to monitor clinical symptoms and signs of thyroid dysfunction in children with NS.

Antioxidants may play an important preventive role in nephrotic syndrome and its progression by decreasing the free oxygen radicals. Consequently, hyperlipidemia that increases the lipid oxidation reactions and decreases the antioxidant status may lead to glomerulosclerosis and progression of glomerular damage in nephrotic syndrome[31].

In the present study, there was significantly increase in MDA in cases with relapse in comparison with cases in remission and control group which agree with studies of other researchers, where there has been an increase in the concentrations of MDA in nephrotic syndrome[8].

There was a significant elevation in the levels of serum MDA in NS. Since, reactive oxygen species can be involved in

many degradative processes including lipid peroxidation and increased generation of reactive oxygen species in glomerular basement membrane[9].

Lipids are the target molecules for free radicals and this is probably a result of increased consumption of antioxidant components such as erythrocyte-SOD. In the present study, reduction in erythrocyte-SOD activity reflects increased susceptibility of RBC membrane to lipid peroxidation[32].

In the present study, there was significant decrease in SOD in cases with relapse in comparison with cases in remission and control group and this was in agreement with the study conducted by Noyan *et al.*[33] who showed that the decreased SOD concentrations have also been observed in nephrotic syndrome.

Zachwieja *et al.*[34] studied the total antioxidant status and mean antioxidant activity in 82 children with nephrotic syndrome of 4-16 years. The study suggested that reduced antioxidant activity in nephrotic syndrome may be related to lipid abnormalities[34].

The GSH is a substrate for antioxidant enzymes like glutathione peroxidase (GPx), glutathione-S-transferase (GST) and glutathione reductase (GRx). The decrease in the concentrations may be due to the increased turnover of GSH in preventing oxidative damage in these cases[35].

In the present study, there was significant decrease in GST in cases with relapse in comparison in cases in remission and control group, similar studies of lowered GSH concentrations in nephrotic syndrome have been reported earlier[36,37] suggesting increased oxidative stress in nephrotic syndrome.

Another study estimated the antioxidant status and reliable factor involved in antioxidant protection in children with nephrotic syndrome. The study suggested an increase in lipid peroxidation and insufficient antioxidant defense in nephrotic syndrome[9].

The MDA levels have significantly negative correlation with FT4 and FT3 but have significantly positive correlation with TSH. On other hand, SOD and GST levels have significantly positive correlation with FT4 and FT3 but have significantly negative correlation with TSH and similar study conducted by Sawant *et al.*[38].

The present study confirmed the previous reports regarding the decline of thyroid function in children with NS among Egyptian children. In addition, it highlighted the possible role of oxidative stress and deficiency of antioxidants in the etio-pathogenesis of NS. Furthermore, studies evaluating the effect of antioxidant supplementation is necessary to evaluate the effect on the course and prognosis of NS.

CONCLUSION

Nephrotic patients can loss significant amounts of thyroid hormones along with protein in urine. Increased Reactive Oxygen Species (ROS) and decreased antioxidants defense may be related to the pathogenesis of proteinuria in NS.

The NS is a consequence of an imbalance between oxidants as MDA and antioxidants activity as SOD and GST.

The findings of this study suggests that nephrotic syndrome patients may benefit from antioxidant therapy along with thyroid hormones supplement.

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