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Case Report

Clinical Hypothyroidism in Steroid Sensitive Nephrotic Syndrome in a Young Boy

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ABSTRACT

Background: Nephrotic Syndrome is damage to kidney that leads to the hyperfiltration and excretion of protein in urine. Persistent proteinuria causes progressive oedema, Hypoalbuminemia and Hypercholesteremia. Many associated manifestations can be possible with the disease either due to genetic susceptibility or complications secondary to proteinuria including transient hypothyroidism due to loss of thyroxine binding globulin, transthyretin and albumin in urine.

Case Presentation: We report a case of 10 years old male patient previously well, presented with generalized body swelling for 1 month, constipation, lethargy and somnolence for 3 weeks. Clinically he had Anasarca, massive ascites and signs of pleural effusion. Labs revealed Hypoalbuminemia, Hypercholesterolemia and Protein+3 in dipstick with Urine PCR of 3.8. TSH= 21 significantly high. Secondary causes excluded and Paediatrics nephrology consult taken, advised to give steroid and Thyroxine for symptomatic Hypothyroidism.

Conclusion: As patient achieved complete remission after 4 weeks of daily methylprednisolone, Thyroxine also tapered with tapering dose of steroids and stopped at 12 weeks of treatment. Patients showed complete response with treatment and no relapses till 1 year of follow up.

Keywords: Clinical hypothyroidism, Steroid-sensitive nephrotic syndrome, Paediatric nephrology

INTRODUCTION

Idiopathic nephrotic syndrome is the most prevalent glomerular disease in the paediatric population, impacting 1 to 17 out of every 100,000 children with a particularly high incidence among those of South Asian ancestry[1,2]. It is characterized by massive proteinuria (≥3+ protein corresponding to ≥300 mg/dL by dipstick examination), hypoalbuminemia (less than 2.5 gm/dL), generalized oedema and hyperlipidemia[3]. Although a kidney biopsy is not commonly performed for diagnosing this condition, in cases where it is done, it reveals injury patterns that align with minimal change disease or focal segmental glomerulosclerosis (FSGS) commonly[4]. These two conditions are classified as "podocytopathies" due to the damage they cause to podocytes, which makes up an important component of the glomerular filtration barrier. Idiopathic nephrotic syndrome is associated with a wide array of complications, largely stemming from the leakage of important proteins from the body, of which hormonal alterations make up a significant proportion[5]. Numerous studies report the presence of hypothyroidism in children[6-8]; however, more often, this hypothyroidism is subclinical, with no evidence of classic symptoms such as fatigue, weight gain, and constipation. There is limited data reporting evidence of overt hypothyroidism secondary to nephrotic syndrome in children.

This case report presents a case of a 10-year-old male with a one-month history of nephrotic syndrome together with clinical symptoms of hypothyroidism.

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CASE PRESENTATION

We report a case of 10 years old male patient, presented with generalized body swelling followed by constipation and somnolence. The patient and his direct family had no notable history of arthralgia, photosensitivity, oral ulcers, tuberculosis or hepatitis. Clinical examination revealed normotension, ascites and decreased air entry in lungs, characterized by stony dullness to percussion at basal lung fields. Ultrasound KUB and thyroid were normal. Summarized laboratory findings of the patients are indicated in **Table I.**

The weight of the patient had notable increase from 30kg to 34kg. The patient had respiratory symptoms due to pleural effusion for which 1g/kg Albumin injection along with furosemide for diuresis were administered, which otherwise is not routinely recommended. This was followed by consultation from paediatrics nephrologists which advised screening for anti-nuclear antibody (ANA) and C3 levels that came out negative. Transient hypothyroidism was attributed to loss of thyroid binding globulin (TBG) in urine; rarely are such high levels associated with clinical symptoms.

Renal biopsy was deferred due to patient's age. Trial steroids are given to all patients with nephrotic syndrome due to which upon discharge, the patient was placed on methylprednisolone at 60 mg orally once daily and thyroxin at 50 μg orally once daily.

At week 4 of treatment, the patient had complete remission due to which methyl prednisolone was tapered to 40mg every alternative day (1.5mg/kg) and on reduced weight to 27kg, Thyroxine was adjusted to 25 μg daily.

During the follow-up at 12th week, patient maintained complete remission. TSH and FT4 levels were reduced to 8 m IU/L and 0.67 ng/dl and at 1 year after treatment, hormonal levels recorded as 4.8 m IU/L and 1.2 ng/dL respectively.

Table I: Diagnostic Reports at Presentation				
Laboratory Tests	**Results**	**At 12 weeks follow up**	**1 year follow up**	**Reference Range**
Serum Cholesterol	760mg/dL	---	---	<200mg/dl
Serum Albumin	1.3g/dL	3.1	---	>3.5 g/dl
Urine Dipstick	(+4)	Negative	Negative	Negative
Urine Protein-Creatinine Ratio	3.8	0.1	---	<0.2
TSH	21	08	4.8	0.5 to 5 mIU/L
FT4	0.2	0.67	1.2	0.8 to 1.8 ng/dL

DISCUSSION

Thyroid hormones are crucial for normal development and growth of many target tissues in adolescents, including the brain and the skeleton. They exist in the circulation mainly bound to thyroglobulin binding protein, transthyretin and albumin. In individuals with normal thyroid function, only 0.03% of T4 and 0.3% of T3 is unbound or 'free' and immediately available to enter cells for downstream effects[9]. Owing to the proteinuria, which is a hallmark feature of idiopathic nephrotic syndrome, there is subsequent loss of these carrier proteins that contributes to the hypothyroidism observed. This not only has profound implications for the metabolic system but also significantly impairs cognitive function, as demonstrated in our case, where the child exhibited somnolence, lethargy, and persistent fatigue. The typical pattern of thyroid function alteration reveals a high TSH and low total T4, as seen in our patient. Idiopathic nephrotic syndrome can follow two courses; it may be steroid sensitive (SSNS) or steroid resistant (SRNS), both of which are associated with hypothyroidism as a potential complication with SRNS being more strongly associated with the development of clinical hypothyroidism[10].Our patient showed improvement with steroids and did not report any relapse up to 1 year of follow up, aligning with SSNS. It is important to note that future relapses may occur, and there is a risk of progression to steroid-resistant nephrotic syndrome (SRNS), which is associated with worse outcomes[11].The degree of proteinuria and serum albumin levels correlate positively with the severity of thyroid dysfunction thus explaining the worsening of thyroid function in some while others remain euthyroid[12]. Presence of preexisting kidney disease or autoimmune thyroid disorder may also be contributing factors for overt symptoms; however, these were ruled out in our case. A key limitation in our case report is a lack of known histological variant of nephrotic syndrome as thyroid function abnormalities may vary greatly across different subtypes. In resource limited countries like Pakistan, patients presenting in tertiary care hospitals have more fulminant symptoms[13]. In general, and thus the degree of proteinuria may be high as is evident from the lab findings in our patient. It is therefore essential to promptly recognize these symptoms through thorough history taking and thyroid function tests must be reviewed in any patient presenting with high grade proteinuria.

CONCLUSION

Subclinical hypothyroidism is a recognized complication of nephrotic syndrome, while overt clinical presentation is uncommon. In the present case, remission was achieved with steroid therapy, allowing successful tapering and discontinuation of thyroxine, with sustained clinical recovery and no relapse at one year of follow-up.

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