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(CASE REPORT)

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Association of severe autoimmune hypothyroidism with reversible proteinuria: A case report

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Abstract

Hypothyroidism is a prevalent medical condition characterized by an underactive thyroid gland, resulting in insufficient thyroid hormone production. This hormone plays an important role in regulating the body's metabolism, so when its levels are low, it can lead to various metabolic disturbances. In this article, we present a case report of a 50-year-old female with severe hypothyroidism who exhibited several symptoms of renal impairment and hypothyroidism. She had proteinuria along with elevated lipid levels. Following a diagnosis of Hashimoto's thyroiditis, the patient was treated with levothyroxine, resulting in a remarkable reversal of her proteinuria and other symptoms. The lipid profile also showed significant improvement without the need for cholesterol-lowering medications. This case report highlights the importance of recognizing and managing thyroid dysfunction in patients with renal abnormalities. It provides valuable insights into the complex interplay between thyroid function, renal abnormalities, and lipid levels.

Keywords: Hypothyroidism; Proteinuria; Dyslipidemia; Hashimoto's thyroiditis

1. Introduction

Hypothyroidism, a common medical condition, has various metabolic effects, such as dyslipidemia, hypertension, atherosclerosis, and coronary artery disease. It also affects renal function, reducing estimated glomerular filtration rate (GFR), proteinuria, and potentially severe complications like rhabdomyolysis and acute renal insufficiency. Proteinuria severity is directly linked to thyroid-stimulating hormone levels. Moreover, dyslipidemia has also been associated with hypothyroidism. While evidence on the reversibility of proteinuria in hypothyroidism is limited, we report the case of successful proteinuria reversal along with lowering lipid levels with the treatment of severe hypothyroidism.

2. Case Presentation

A 50-year-old female with no past medical history presented to the outpatient department with complaints of paresthesia in bilateral hands and feet, foamy urine, swelling in her lower extremities (LE), generalized body pain,

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swallowing difficulty, and orthopnea. She was taking no medications. On examination, she had thyromegaly, narrowing of the posterior oropharynx due to enlarged tonsils,+3 pitting edema of bilateral LE, and severe xerosis cutis. Lab results revealed severe hypothyroidism with thyroid stimulating hormone (TSH) level of 91.86 mIU/L and a free T4 level of 0.1 ng/dL. Her renal function was mildly elevated with creatinine (Cr) of 1.08 mg/dl and reduced eGFR of 63 mL/min. Urinalysis revealed +1 occult blood and +1 proteinuria. CBC was within normal limits. A serum lipid profile revealed 364 mg/dL of total cholesterol with elevated LDL and triglycerides at 279 mg/dL and 149 mg/dL, respectively. The patient's antibody to thyroid peroxidase (TPO) was positive, confirming the diagnosis of Hashimoto's thyroiditis.

The patient was treated with a body weight-adjusted levothyroxine dose of 200 mcg. At a 2-month follow-up, the patient reported significant improvement in her symptoms. Lab tests revealed that TSH normalized to 0.94 mIU/L. CMP showed Cr and eGFR within normal limits. Urinalysis showed complete resolution of proteinuria and occult hematuria. Her serum cholesterol panel also improved with a total cholesterol level of 248 mg/dL, LDL 162 mg/dL, and triglycerides 218 mg/dL without medications.

3. Discussion

The impact of reduced thyroid function on kidney function has been a subject of study since the 1940s and 1950s. In 1947, Corcoran et al. documented two myxedema patients who were treated with desiccated thyroid extract. Before and after treatment, their glomerular filtration rate (GFR) and renal plasma flow (RPF) were measured, revealing an increase of +138% and +38%, respectively. Additionally, treatment led to a reduction in renal vascular resistance [1]. Hypothyroidism is a prevalent medical condition characterized by elevated TSH levels exceeding 4.0 mlU/L. Depending on the levels of free triiodothyronine and thyroxine, it is classified into either overt hypothyroidism or subclinical hypothyroidism. The discovery of proteinuria in hypothyroidism is a recent development [2], and there has not been prior research on whether it can be reversed [3]. Numerous studies have demonstrated a connection between hypothyroidism and increased serum creatinine levels, likely attributed to myopathy. These studies have also indicated that treatment with thyroxine can reverse this effect [4]. The precise causes of myopathy in this context remain uncertain, although hypotheses have suggested alterations in intramuscular glycogen metabolism and shifts in mitochondrial physiology as potential contributing factors.

The causal relationship between hypothyroidism and proteinuria remains uncertain. In individuals with nephrotic syndrome, there is substantial urinary loss of thyroid hormone-binding proteins, including thyroxine-binding globulin, transthyretin, and albumin, which decreases total T4 levels. However, the thyroid gland can compensate for this loss, leading to the majority of patients maintaining a euthyroid state since their serum-free T4 or free T3 levels remain within the normal range. In a study involving 159 patients with nephrotic syndrome and 900 controls, it was observed that patients with nephrotic syndrome had slightly elevated TSH levels within the normal range (1.81 vs. 1.34 mIU/L, $P < 0.001$) and similar free T4 levels (13.1 vs. 13.1 pmol/L) compared to the control group [5]. On the other side, hypothyroidism has been linked to various forms of glomerulopathy, including membranous glomerulopathy, minimal change nephritic syndrome, and membranoproliferative glomerulonephritis. The detection of immunoactive but biologically inactive TSH can occur in nonthyroidal illness due to factors such as reduced levels of thyrotropin-releasing hormone and alterations in the glycosylation of TSH. This is particularly relevant from a clinical perspective, considering recent guidelines recommending treatment for subclinical hypothyroidism when TSH levels are at or below 10 mIU/L, especially in individuals with atherosclerotic cardiovascular disease, heart failure, or risk factors for these conditions, which are commonly found in patients with chronic kidney disease.

Furthermore, when measuring free T4 using immunoassay methods, it is crucial to be aware that these measurements can yield artificially low values, especially in cases of hypoalbuminemia, and may not be reliable in patients with nonthyroidal illness. In individuals with chronic kidney disease, total T3 levels may be low due to factors like reduced conversion of T4 to T3 in peripheral tissues, changes in protein levels affecting hormone binding, and the influence of chronic metabolic acidosis and increased levels of inflammatory cytokines impacting these processes. Notably, a strong inverse relationship exists between decreased free T3 levels and elevated interleukin-6 levels in chronic kidney disease. Free T4 levels tend to vary in patients with chronic kidney disease, generally falling within the normal to low range, and there is no significant increase in the conversion of free T4 to reverse T3. Typical diagnostic indicators include elevated TSH and organic iodine levels and a normal radioactive iodine uptake. In such cases, hypothyroidism triggered by excess iodine intake can be resolved through dietary iodine restriction, and there may be no need for thyroid hormone supplementation [6]. However, in our patient, the reason for hypothyroidism was Hashimoto thyroiditis, autoimmunity leading to the decreased formation of thyroid hormone. This variant of hypothyroidism needs treatment with thyroxine. The treatment resolves the renal abnormalities and lowers the cholesterol abnormalities as well.

4. Conclusion

In conclusion, hypothyroidism is a prevalent medical condition with far-reaching metabolic consequences, affecting not only the endocrine system but also renal function and lipid metabolism. This case study of severe hypothyroidism is a prime example of this condition's profound impact on various physiological processes. The successful reversal of proteinuria, improvement in renal function, and favorable changes in lipid profiles following levothyroxine treatment emphasize the need for heightened awareness of thyroid dysfunction in patients presenting with renal abnormalities. Moreover, this case underscores the intricate interplay between thyroid hormones, renal health, and lipid levels, calling for careful consideration of these conditions while managing patients with severe hypothyroidism.

Compliance with ethical standards

Disclosure of conflict of interest

There is no conflict of interest of any kind.

Statement of informed consent

The signed consent from the patient was obtained. We retain the informed signed consent form.

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