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PROTEINURIA SECONDARY TO HYPERTHYROIDISM: A CASE STUDY

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KEYWORDS :

CASE PRESENTATION

A 28-year-old Indian female presented to the endocrinologist with complaints of dryness of mouth, excessive thirst and loss of almost 8 kg of weight in the past 18 months. On further questioning, the patient revealed to have a good appetite, regular periods, no tremors or palpitation or heat intolerance. The patient has no previous history of any chronic diseases and was not any therapy. Clinical examination of the patient revealed tachycardia, mild tremors, and a diffuse thyromegaly however; no lymph node hyperthyroid related symptoms, no bruit, EOM normal or lid lag was detected. Her physical and vital signs at the time of presentation of the case were- weight: 43.0 kg, hip: 84 cm, waist: 78 cm, waist to hip ratio: 0.93, pulse: 90 bpm. The biochemical investigation report of the patient reveals the parameters to be within normal range (random glucose: 105 mg / dL, urea: 42 mg / dL, serum creatinine: 0.6 mg / dL, uric acid: 4.5 mg / dL, sodium: 142 mMol / L, potassium: 4.0 mMol / L, chloride: 110 mMol / L). The hematological examination was found to be normal for most of the parameters (hemoglobin: 13.5 g / dL, packed cell volume: 40.4, WBC: 5.27 mill / cu.mm, mean corpuscular volume: 79 fL, mean corpuscular hemoglobin concentration: 33.4 g / dL, total WBC count: 9200 cells / cu.mm, neutrophils: 60%, lymphocytes: 34%, eosinophils: 03%, monocytes: 3%, basophils: 0%, absolute neutrophil count: 5.52 X 10^3 / µl, absolute lymphocyte count: 3.13 X 10^3 / µl, absolute eosinophil count: 0.28 X 10^3 / µl, absolute monocyte count: 0.28 X 10^3 / µl, absolute basophil count: 0.00 X 10^3 / µl, platelet count: 250 X 10^3 / µl, ESR: 8 mm / hr). However, mean corpuscular hemoglobin was found to be 25.6 pg along with DW-CV: 11.1%, DW-SD: 29.91 fL, SGOT/AST: 33 U/L, SGPT/ALT: 46 U / L, and alkaline phosphatase: 129 U / L. Also, alarming were her thyroid function test results, T3: 5.40 ng / ml while T4 was 28.5 µg / dl and TSH: < 0.010 µlU / ml; proteinuria was also detected. The clinician diagnosed these values as an indication of hyperthyroidism related to Graves diseases/thyroiditis. However, further examination is required to understand the cause of proteinuria and hyperthyroidism. Meanwhile, the patient was immediately instructed to initiate the therapy of non-selective beta-blocker, propranolol 40 mg for symptomatic relief.

To further understand proteinuria, a 24-hour urine protein profile analysis was directed by the doctor. The 24-hour urine analysis for protein/albumin revealed a value of 30.48 mg / 24h as against the normal limit of < 150 mg / 24h, while the urine volume for 24 hours was found to be 1200 ml. At the same time, to reveal the cause of Graves's diseases / thyroiditis, a technetium scan of the thyroid was to be undertaken by the patient on the instructions of the doctor. A thyroid scan was carried out using 3mCi ^{99m}technetium pertechnetate. The scan revealed that the right lobe of the thyroid gland was mildly enlarged and showed increased radiotracer uptake by the parenchyma. The left lobe of the thyroid gland reveals a photon deficient area involving the upper pole, corresponding to the clinically palpable nodule. The rest of the lobe reveals increased radiotracer uptake. At 20 minutes, a 14% uptake of ^{99m}technetium pertechnetate was observed. The presence of a nodule in the left thyroid lobe prompted the investigation for neoplasm via biopsy. Microscopy of the biopsy sample showed a cluster of follicular cells with round nuclei, fine chromatin and moderate cytoplasm arranged in the follicular pattern. Thus, ruling out papillary carcinoma. The patient was put on carbimazole, once-daily therapy along with propranolol, once daily. Further, ultrasound of the thyroid revealed that both the lobes of the thyroid gland are diffusely enlarged with a nodular echotexture and increased vascularity. The left lobe has a well defined hyperchoic nodule occupying the upper and mid pole with a central cystic area of 2.8 X 1.5 cm. Also, the nodule exhibited peripheral vascularity. The isthmus measured 4 mm and was enlarged. No significant cervical lymphadenopathy was observed and the neck vessels were found to be normal. Abdominal ultrasound reveals mild splenomegaly while all the other organs viz., liver, gall bladder, portal vein & splenic veins, pancreas, kidneys, bladder, uterus, and ovaries were found to be normal. The patient continued the propranolol and carbimazole therapy for 45 days before undertaking urine and thyroid function tests. After 45 days, the proteinuria, T3, T4, and TSH significantly improved. An almost negligible amount of albumin was found in urine while T3 reduced to 2.07 ng / ml, T4 reduced to 15.60 µg / dl while TSH was found to be < 0.001 µIU / ml. Similar results were obtained when the urine and thyroid function test was carried out again after 90 days of therapy with propranolol and carbimazole. On completion of almost 4 months of propranolol and carbimazole once-daily therapy, proteinuria was completely absent, with urine microalbumin value of 25.10 µg / mg of creatinine, while T3 reduced to 1.02 ng / ml, T4: 7.30 µg / dl and TSH: 0.04 µIU / ml. Subsequently, propranolol was stopped from the therapy while, a once-daily carbimazole (10 mg) was continued. Urine analysis and thyroid function test after 2 months of therapy were found to be within normal acceptable limits. Eventually, the patient was advised to take on carbimazole 10mg, once daily for another 2 months. After 2 months of the therapy, the urine microalbumin reduced to 16.4 µg / mg creatinine with no proteinuria. Also, the waist to hip ratio improved to 0.95 from the initial value of 0.93, while BMI raised to 18.97, weight increased to 45 kg and pulse reduced to 76 bpm from the initial 100bpm. The patient was advised to continue once daily carbimazole (10 mg) therapy.

DISCUSSION

The initial case presentation pointed towards hyperthyroidism related to grave diseases as indicated by the elevated T3, T4, and TSH value along with proteinuria. Hematology report gave a normocytic normochromic blood picture[1]. Hence, to understand the basic cause of the elevated thyroid function and proteinuria, a thyroid scan using ^{99m}Tc pertechnate was carried out along with a 24-hour urine profile. 24-hour urine profile confirmed the proteinuria.

The evaluation of a patient who presents with hyperthyroidism frequently includes a radioisotope uptake scan as well as testing for thyroid function, to make the differential diagnosis between Graves' disease, multi-nodular goiter, toxic adenoma, and thyroiditis. Distinguishing these conditions is desirable, as the treatment of these entities differs. Graves' disease, goiters, and adenomas are treated with thionamide medications (such as methimazole or propylthiouracil), radioactive iodine or surgical resection[2,3]. In contrast, thyroiditis can be managed prospectively, as many patients with this disease return to normal over time or develop hypothyroidism[4].

In ^{99m}Tc pertechnate scan higher uptake of radiotracer by parenchymal cells suggests a toxic goiter or grave's disease pattern[5]. A cold nodule involving the upper pole of the left lobe of the thyroid gland was found in the radioscan. However, the neoplasticity of the nodule needs to be established by biopsy. Microscopic analysis of the biopsy sample ruled out the presence of papillary carcinoma as smear showed a cluster of follicle cells having round nuclei, moderate cytoplasm, and fine chromatin. There was no evidence of malignancy in the biopsy smear. Ultrasound of the thyroid exhibited a diffuse thyromegaly with a nodular echotexture and increased vascularity. A well defined hyperchoic nodule was found to occupy the upper and mid pole of the left thyroid lobe with the central cystic area and peripheral vascularity. The treatment with carbimazole, 10 mg resulted in reduced T3, T4 and

TSH levels to normal. It was found that, with the reduction in thyroid function parameters, there was a reduction in proteinuria as well. There is a renal involvement with thyroid malfunction[6,7]. Once T3, T4 and TSH levels were within the normal range, proteinuria was completely absent[8,9].

In conclusion, in this case, report, we have described the presentation, diagnosis, clinical course and treatment of a patient with hyperthyroidism with proteinuria. There should always be a clinical suspicion of proteinuria secondary to hyperthyroidism. This case proves that when thyroid function is normalized, proteinuria subsided. This avoids unnecessary evaluation of renal cause of proteinuria with radiological or invasive investigation such as renal biopsy.

REFERENCES

1. Gardner LB, Benz EJ, 2005. Anemia of chronic diseases. In: Hoffman R, et al. (eds) Hematology, basic principles and practice, 4th edn, pp. 465–71. Churchill Livingstone, New York.
2. Singer PA, Cooper DS, Levy EG, et al. 1995. Treatment guidelines for patients with hyperthyroidism and hypothyroidism. Standards of Care Committee, American Thyroid Association. JAMA 273:808.
3. Ross DS, Burch HB, Cooper DS, et al. 2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis. Thyroid 26:1343.
4. Hennessey JV, 2018. Subacute Thyroiditis. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279084/
5. DeGroot LJ, 2015. Graves' Disease and the Manifestations of Thyrotoxicosis. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK285567/
6. Singhania G, Singhania N, 2019. Membranous nephropathy associated with profound hypothyroidism. Clinical Case Reports. 8 (1): 120-122.
7. Basu G, Mohapatra A, 2012. Interactions between thyroid disorders and kidney disease. Indian J Endocr Metab 16:204-13.
8. Weetman AP, Tomlinson K, Amos N, Lazarus JH, Hall R, McGregor AM,1985. Proteinuria in autoimmune thyroid disease. European Journal of Endocrinology 109: 341–347.
9. Cakir AD, Canpolat N, Saygili S, Kilicaslan I, Turan H, Ercan O, Evliyaoglu O, 2019. Proteinuria in a patient with Graves' disease: Answers. Pediatric Nephrology 34, 1383–1385.