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Hemolytic Anemia and Thrombocytopenia with Hyperthyroidism : A Case Report

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Abstract

Background: Hemolytic anemia occurs when there are low number of red blood cells due to excess of hemolysis in the body. Thrombocytopenia is a condition that occurs when the platelet count is too low. Hyperthyroidism is when the thyroid gland makes more thyroid hormones than the body needs. Only a few cases of hyperthyroidism associated with hemolytic anemia and thrombocytopenia have been reported. We report a case of a 50 year old female, a case of Type 2 diabetes mellitus who presented with symptoms of hyperthyroidism, anemia and thrombocytopenia. Blood investigations were done which showed findings suggestive of primary hyperthyroidism and hemolytic anemia. She was managed with blood transfusion, insulin and steroid. This case highlights, though rare, hemolytic anemia and thrombocytopenia in a setting of hyperthyroidism.

Key words: Hemolysis, Thrombocytopenia, Hyperthyroidism

Introduction

Hemolysis presents as acute or chronic anemia, reticulocytosis, or jaundice. The diagnosis is established by reticulocytosis, increased unconjugated bilirubin and lactate dehydrogenase, decreased haptoglobin, and peripheral blood smear findings.¹ Hemolysis may occur intravascularly or extravascularly in the reticuloendothelial system, or both. Patients with hemolysis may present with acute anemia, jaundice, hematuria, dyspnea, fatigue, tachycardia, and possibly hypotension.² Thrombocytopenia is defined as a platelet count of less than 150×10^3 per μL . A platelet count from 30 to 50×10^3 per μL rarely manifests as purpura. A count from 10 to 30×10^3 per μL may cause bleeding with minimal trauma.

A platelet count less than 5×10^3 per μL may cause spontaneous bleeding and constitutes a hematologic emergency.³ Few studies have established that thyroid illness have effects on blood cells count and red blood cell indices.⁴ We present a case of a 50 year female with diabetes mellitus and hyperthyroidism complicated by hemolytic anemia and thrombocytopenia.

Case report

A 50 years female, known case of Type II Diabetes Mellitus for four months under medication (Metformin 500 mg twice daily + Glimpiride 1g twice daily) presented with a 12 days history of easy fatigability and shortness of breath not associated with orthopnea and paroxysmal nocturnal dyspnea. She also complains of painless epistaxis since 1 month with no history of nasal picking and history of gum bleeding on and off since seven days. She also gives history of palpitation since 3 months. History of heat intolerance and

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irregular menstruation present. No history of fever, chest pain and cough. No history of sore throat, runny nose, nasal congestion, sneezing. No change in bowel and bladder habit. No history of rash or abnormal discoloration of any body part. She had no other significant medical history in the past. Upon examination, blood pressure was 130/80 mm of mercury, pulse 140 beats per minute, respiratory rate 20 breaths per minute. She was afebrile with an oxygen saturation of 98% in room air. Scleral

icterus and pallor with pale conjunctivae were present. Two firm swellings felt in the bilateral lobes of the thyroid gland with no rise in local temperature or any audible bruit (Figure1). Normal bilateral vesicular breath sound with bilateral equal air entry. Normal heart sounds with no murmurs. No rise in jugular venous pressure. The abdomen was soft, non-tender and non-distended.



Figure 1: Thyroid gland Swelling

Table 1

Hb, g/dl	3.9
Hematocrit %	13.3
WBC, count/ml	11200
DLC	N74L16M10
Platelet	14400
Total Protein , g/l	6.2
Total Serum Bilirubin	5.8
Direct Serum Bilirubin	0.5
ALT/AST	41/50
Thyroid function test	
FT3 (pg/ml)	10.32
FT4 (pg/ml)	46.7
TSH(IU/ml)	0.001

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Peripheral smear for cytology	Anisopoikilocytosis, microcytes, tear drop cells, elliptocytes, hypochromia
Reticulocyte count (%)	1.2
Corrected reticulocyte count (%)	0.35
Iron (µg/ dL)	34
Unsaturated iron binding capacity (µg/ dL)	285
Transferrin saturation (%)	10
Lactate dehydrogenase (IU/L)	506
Direct coomb`s test	Negative
Anti nuclear antibody	Negative
Anti dsDNA	Negative
HbA1c	8.8
Lipid profile	
Total cholesterol (mg/dl)	126
LDL(mg/dl)	45
TAG(mg/dl)	250
HDL(mg/dl)	39
Serology for HIV,HCV,HBsAg	Negative

Ultrasonography of thyroid revealed enlarged lobes of thyroid gland with heterogenous echotexture with multiple hyperechoic and cystic areas within along with increased vascularity .

Initial treatment comprised 1 pint of platelet-rich plasma and 1 pint of whole blood

transfusion. She was started on empirical IV methylprednisolone 1g once daily in emergency, which was stopped the next day and switched to oral prednisolone 40mg once daily. Tablet propranolol 20mg twice daily was added the next day. Thionamides were not started in view of low count .

	D1	D3	D4	D5	D6	D7	D8
Hb	3.9	6.8	7.9	8.1	8.6	8.2	8.4
Platelet	14000	50000	158000	20000	22000	40000	150000
TLC	11200	6500	6200	7500	8000	8200	8800

The patient improved clinically and was discharged on oral medications propranolol, prednisolone, iron folate and injection mixtard insulin.

The blood reports were normal during 1 week follow up.

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Discussion

The common causes of an excessive production of thyroid hormones are Graves disease, toxic multinodular goiter, and toxic adenoma. The most common cause of an excessive passive release of thyroid hormone is painless (silent) thyroiditis, although its clinical presentation is the same as with other causes. Hyperthyroidism caused by overproduction of thyroid hormones can be treated with antithyroid medications (methimazole and propylthiouracil), radioactive iodine ablation of the thyroid gland, or surgical thyroidectomy.⁵ β blockers are used in symptomatic thyrotoxicosis, and might be the only treatment needed for thyrotoxicosis not caused by excessive production and release of the thyroid hormones.⁶ The normal life span of human red blood cells is 120 days after their release from the bone marrow as reticulocytes. Premature destruction of red blood cells is referred to as hemolysis. This can occur intravascular (red cells lyse in circulation) or extravascular (ingestion of red cells by macrophages in the spleen and liver). Extravascular hemolysis which is more common requires the presence of red cell surface abnormalities such as bound immunoglobulin, for which macrophages have specific receptors, or the presence of certain physical factors that restrict the red cell deformability, thereby preventing its exit from the spleen.⁷ Thrombocytopenia is defined as a platelet count of less than 150×10^3 per μL .³ Establishing the cause of thrombocytopenia has obvious clinical repercussions, but is sometimes quite challenging. Some common causes of thrombocytopenia include Immune thrombocytopenic purpura, infections, Connective tissue disorders, liver disease, myelodysplastic syndromes.⁸ Since 1931, sporadic reports have appeared noting an apparent association between hyperthyroidism

and a low platelet count.⁹ In a cohort of patients with hyperthyroidism, a high prevalence of anemia was found, which returned to normal following antithyroid therapy.¹⁰ In our case, the diagnosis of primary hyperthyroidism was made on the basis of an abnormal thyroid profile (elevated thyroid hormones with a low TSH). Elevated unconjugated bilirubin, elevated LDH and peripheral smear findings were suggestive of hemolysis. A low platelet count was observed. It is not well established whether hyperthyroidism and abnormal blood cells count share some common pathogenic mechanisms. There are limited studies related to hyperthyroidism with anemia and thrombocytopenia. In our case there was new onset anemia and thrombocytopenia in the background of hyperthyroidism. The patient responded well to treatment administered.

Conclusion

In conclusion, hyperthyroid patients who present with symptoms of anemia and low platelet count should be evaluated for the same, even though it's a rare association. Our case report emphasizes how timely diagnosis and effective treatment correct all the blood parameters and reduce further complications.

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Availability of data and materials.

The datasets supporting the conclusions of this article are included within the article.

Competing interests.

The authors declare that they have no competing interests.

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Consent for publication.

Informed consent was taken from the patient to publish this case report.

Ethics approval and consent to participate.

Not applicable.

Author's contribution.

NMJ provided the data and materials from the archive and the notes. NMJ, BA, AA, AM wrote the manuscript, collected the images, and put them in perspective according to the timeline of the case. RM reviewed the manuscript and provided us the endocrinological expertise. All the authors read the final manuscript and approved the case.

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