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Case Report

Severe primary hypothyroidism leading to life threatening heavy menstrual bleeding: a case report

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ABSTRACT

Thyroid disorders are one of the leading causes of abnormal uterine bleeding in women of all age groups and in India its prevalence in women is about 26%. Sequelae of thyroid disorders may vary from infrequent menstrual cycle, light menstrual bleeding to even a very severe life threatening heavy menstrual bleeding leading to anemia & shock. It occurs due to anovulation, endometrial hyperplasia and coagulation defects. Thyroid screening is important while investigating all cases of AUB. A 18 year old girl was brought to Dr. Bhim Rao Ambedkar Memorial Hospital Raipur with very severe anemia (Hb: 1.1gm/dl) and grade IV hemorrhagic shock (BP 50/30mm of Hg) but surprisingly pulse rate was normal (80bpm). Her peripheries were cold and clammy. SpO₂ -80% on room air, she had facial puffiness and grade III pitting edema over her hands and feet. Her TSH was very high >100 µIU/ml with decreased (T₄ - 0.678µg/dl, T₃ - 0.359 µg/ml) suggestive of severe primary Hypothyroidism. USG was suggestive of bulky uterus with 14 mm endometrial thickness. Her shock was managed and tablet norethisterone, tranexamic acid, levothyroxine and iron supplements started. Severe hypothyroidism can cause life threatening uterine bleeding. This case is of peculiar interest because of profound hypothyroidism associated with hemorrhagic shock. Early recognition and proper management is important to prevent hazardous complications.

Keywords: AUB, HMB, Hypothyroidism, Shock

INTRODUCTION

Abnormal uterine bleeding (AUB) is defined as any deviation from normal menstruation or normal menstrual cycle pattern. 10-20% of women in their reproductive life experience AUB and 20-30% of Indian women presents to OPD with this complaint.¹

Prevalence of menstrual irregularities in patients with untreated hypothyroidism was reported to be 23.4%.

An Indian study reported 68.2% of hypothyroid women had menstrual abnormalities compared to 12.2% of healthy controls.²

CASE REPORT

A 18 year old unmarried girl was admitted in our hospital by her parents. She had complaints of heavy menstrual bleeding, generalized weakness and breathlessness. She became so weak that was unable to move or talk to anyone in the family and almost gone into subconscious state. On admission she was very pale and dehydrated. Edema was present over face, hands and feet, peripheries were cold and clammy. Skin was dry and scaly; nails were brittle and with short neck. Although BP was 50/30 mmHg but her pulse rate was 82 bpm. She was tachypneic and SpO₂ was 80% on room air. Her height was 126 cm (less than 5th centile according to WHO growth chart) As per history given by her parents, she had her menses 2days back and

bleeding was very heavy with passage of clots. Her previous menstrual cycles were irregular (3-5 months).

Since childhood her milestones were slightly delayed and was always lethargic. Her appetite was less and voice was hoarse. Her height and weight were lesser as compared to children of the same age group. She had problems in correctly recalling the past events.



Figure 1: The 18 year old unmarried girl with heavy menstrual bleeding.

Management

Resuscitation done immediately and was supported by crystalloids, colloid (while waiting for blood) and blood transfusion. Pregnancy was ruled out. Our next aim was to arrest bleeding for which Inj Tranexamic acid 500mg stat, Inj Ethamsylate 500mg 6 hourly and tablet norethisterone 10mg TDS was started.

Her reports showed – Hb- 1.1gm/dl (↓↓) with normal TLC and platelet. Bleeding and clotting time were within normal limit, and normal coagulation studies (PT-17 seconds and INR-1.49 seconds). Liver, renal function and serum electrolytes were on normal range. USG showed endometrial thickness of 14 mm in spite of heavy bleeding since 2days. Increased echogenicity of bilateral renal cortex was found. TSH was very high 100 μ IU/ml (↑↑) with decreased T4-0.678 μ g/dl and T3-0.359 ng/ml suggestive of severe primary Hypothyroidism. Serum prolactin was slightly raised (37.94 ng/ml) but no galactorrhea found. LH and FSH were also decreased.

Endocrinologist was consulted and tablet Levothyroxine 100 μ g OD started. After 5 units of blood transfusion her hemoglobin improved to 8.35 g/dl. Oral treatment for anemia started and dose of tablet norethisterone was tapered as bleeding controlled and continued for a month. She was discharged after 10 days on tablet norethisterone

and iron supplementation. Tablet tranexamic acid 500 mg TDS was advised during her next menses.

She is on tablet thyroxin and having normal menstrual cycle. During her follow up visits after one year, patient was having normal menstrual cycle with her TSH value decreased (thyroxine was tapered) and endometrial thickness of about 8.4 mm was found.

DISCUSSION

AUB is one of the most common, yet complicated clinical presentation. FIGO has categorized AUB depending upon the etiology into 9 main categories in an acronym as PALM-COEIN. Among all the etiologies, ovulatory disorder is one of the most common causes which usually occurs secondary to thyroid dysfunction.³ Thyroid dysfunction is the systemic disease most often associated with AUB. Various studies have stated that any menstrual abnormality in women justifies screening for thyroid disorders.⁴ activity of thyroid is regulated by hypothalamus and pituitary glands with the help of its secreted hormones, thyrotropin releasing hormones (TRH) and TSH respectively. TRH regulates the release of TSH from anterior pituitary. Hypothalamic-pituitary-ovarian axis is physiologically related to hypothalamic-pituitary-thyroid (HPT) axis. TSH shares common alpha subunit with LH and FSH, the mid cycle LH, FSH surge may thus be blunted in hypothyroidism. Moreover, thyroid hormones synergize with follicle stimulating hormone and responsible for ovulation and corpus luteum formation.

HMB is a frequent complication in hypothyroidism and is probably due to estrogen breakthrough bleeding secondary to anovulation. But it may first present late in adolescence or in adult life after HPO axis matures, like in our case. Other factors that may contribute to excess bleeding are defective intrinsic clotting mechanism due to decreased plasma concentration of intrinsic clotting factors VIII and IX, Von Willebrand's disease, increased capillary fragility and the decrease in platelet adhesiveness, all secondary to hypothyroidism. Notably both symptoms and laboratory findings tend to normalize after treatment with thyroxine.⁵

The increased TRH levels cause rise in prolactin levels and therefore galactorrhea may be seen in some cases.⁵ Thyroid hormone mediates growth and development of skeleton and constitutes one of the major treatable causes of short stature (less than 5th centile according to WHO). Patients with hypothyroidism have decreased spontaneous GH secretions and blunted response to GH provocative tests.⁶ Hypothyroidism causes decrease in heart rate, cardiac contractility and thereby decreases cardiac output. As seen in our case she came with grade IV hemorrhagic shock but normal pulse rate.

The overall effect of hypothyroidism is decrease in intelligence but in our case, though hypothyroidism was severe but intelligence was minimally affected. It is general consensus in a developing country like India that

even grossly apparent cases of hypothyroidism are often neglected until it leads to significant morbidities. Consequences of prolonged untreated hypothyroidism during adolescence are retarded growth and development of children causing them lifelong stigma and affecting quality of life.

CONCLUSION

Hypothyroidism causes menstrual irregularities but causing extreme degree of HMB leading to very severe anemia and shock which is hazardous for life is very rare. And this makes our case an interesting one. Every girl and women presenting with AUB should be timely evaluated and treated for thyroid dysfunction. Early recognition and prompt management can prevent many morbidities, ICU admissions and mortalities.

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