Case Report

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Gynecology & Obstetrics Case Report
ISSN 2471-8165

2017
Vol.3 No.1:44

DOI: 10.21767/2471-8165.1000044

# Severe Hypothyroidism Leading to Life Threatening Menorrhagia

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**Received date:** 26 Dec, 2016; **Accepted date:** 22 Feb, 2017; **Published date:** 24 Feb, 2017

**Citation:** Viau M, Bussieres MC, Bergeron ME. Severe Hypothyroidism Leading to Life Threatening Menorrhagia. Gynecol Obstet Case Rep. 2017, 3:1.

## Abstract

**Background:** In their life-time, 10% to 30% of reproductive-aged women will experience abnormal uterine bleeding. After ruling out a pregnancy, the initial laboratory investigations for these patients should be based on history and clinical examination. Hypothyroidism and hyperthyroidism can be associated with abnormal uterine bleeding.

**Case:** A 49 year-old woman was brought to the emergency department for abnormal uterine bleeding. Her hemoglobin was 26 g/L and her TSH and free thyroxin (T4) levels were >100 mU/L (0.25 to 5.00) and 1 pmol/L (12 to 22), respectively. Oral levothyroxine was then started. High-doses of conjugated estrogen and tranexamic acid were used as needed to control uterine bleeding and the menorrhagia resolved rapidly.

**Conclusion:** Severe hypothyroidism can lead to significant uterine bleeding secondary to ovulation and coagulation disorders.

**Keywords:** Hypothyroidism; Menorrhagia; Abnormal uterine bleeding; Levothyroxine; Hemorrhagic shock

## Abbreviations:

T4: Thyroxin; TSH: Thyrotropin Stimulating Hormone; TRH: Thyrotropin-Releasing Hormone; PFA-100: Platelets Function Assay; SHBG: Sex Hormone-Binding Globulin

## Introduction

In their life-time, 10% to 30% of reproductive-aged women will experience abnormal uterine bleeding. This condition affects health-related quality of life and is a frequent reason for medical consultation [1]. After ruling out a pregnancy, the initial laboratory investigations for these patients should be based on history and clinical examination. In presence of findings suggestive of thyroid disease, thyrotropin-stimulating hormone (TSH) should be measured, as hypothyroidism and hyperthyroidism can be associated with abnormal uterine bleeding [2]. However, these diseases rarely lead to severe menorrhagia.

## Case

A 49 year-old G4T3A1 woman was brought by ambulance to the emergency department of a university hospital for abnormal uterine bleeding. She was pale and hypotensive, but her heart rate was normal. Physical exam revealed profuse uterine bleeding. Resuscitation was started with intravenous crystalloid solution and a blood transfusion. The emergency doctor obtained a telephone consultation with the on-call gynecologist and 25 mg of conjugated estrogens IV and 1500 mg of tranexamic acid IV were prescribed while awaiting for the first blood tests results.

Hemoglobin was 26 g/L (120 to 160) and Hematocrit was 9% (37 to 47). Platelets count was 258 × $10^9$/L (150 to 400). Platelets function assay (PFA-100) was impossible to perform due to bundles of platelets. International normalized ratio and partial thromboplastin time were initially within normal range. Pregnancy test was negative. Serum transaminases were elevated secondary to hepatic ischemia. Serum creatinine was also elevated at 105 U/L (45 to 85) on pre-renal acute renal failure. Lactate level was 3.3 mmol/L (<1.6).

After stabilization and close monitoring, she was transferred by ambulance to our institution to the regional on-call gynecologist. She received two more units of red blood cells. Hypotension resolved. At her arrival, she was pale. Her speech was slow and incoherent. Facial myxoedema was noted. Her thyroid was normal. There was no active vaginal bleeding. At bimanual examination, her uterus was slightly enlarged.

Her past medical history was significant for hypothyroidism since age 23. She delivered three times by Caesarean section. Her last pregnancy was complicated by postpartum haemorrhage due to uterine atony requiring two blood transfusions. However, she denied personal or family history of abnormal bleeding. She had no family history of gynecological cancer.

For many years, she had prolonged heavy menstrual bleeding. Her menstrual periods were irregular, occurring every 15 to 30 days. Her menses lasted 6 to 15 days. She reported changing pads every hour and passing clots 3 cm in

 

diameter. However, in the last three months, she noted an increase in the frequency and duration of her menses.

Without any medical follow-up, she stopped using levothyroxine four months before presenting to the emergency department. She developed symptoms of fatigue, cold intolerance, constipation and dry skin. More recently, she became apathetic and asthenic; she could not even walk out of bed because of extreme tiredness. Despite the recommendations of her family members, she did not seek medical attention for her symptoms.

Transabdominal ultrasounds revealed an increased size uterus with a 1.0 * 0.7 cm intramural leiomyoma not protruding into the endometrial cavity. Myometrium was described as heterogeneous. The uterus was empty. There was no evidence of arteriovenous malformations. Both ovaries were normal. There was no free fluid.

Volume repletion was achieved with crystalloid solution and a total of five units of red blood cells. She presented bradycardia. ECG was otherwise normal, and cardiac biomarkers were not elevated. Serum cortisol was drawn, but results were not available. She received IV hydrocortisone because adrenal insufficiency was suspected. However, cortisol levels returned appropriately elevated. Also, she developed disseminated intravascular coagulation and hypocalcaemia due to multiple blood transfusions, for which she received fresh frozen plasma and IV calcium gluconate, respectively. TSH and free thyroxin (T4) levels became available and were >100 mU/L (0.25 to 5.00) and 1 pmol/L (12 to 22), respectively. Oral levothyroxine was then started.

High-doses of conjugated estrogens and tranexamic acid were used as needed to control uterine bleeding and the menorrhagia resolved rapidly.

She was admitted to ICU. The next day, diuresis and creatinine returned to normal. She experienced diffuse myalgia secondary to rhabdomyolysis. She had episodes of retrosternal pain. However, cardiac investigations remained normal. Vaginal bleeding stopped the day after her admission. Her hemoglobin remained stable. Serum transaminases returned to normal.

Endometrial biopsy was performed before her discharge and showed an inactive endometrium. After discussion and counselling about available options for the treatment of abnormal menstrual bleeding and suspicion of adenomyosis, the patient received depot medroxyprogesterone acetate. She returned home four days after her admission with iron supplementation and tranexamic acid to use in case of significant bleeding. Her hemoglobin level at discharge was 88 g/L.

Levothyroxine was adjusted, and TSH returned to normal reference range. Last follow-up was 18 months after her discharge from our hospital. She is still using depot medroxyprogesterone acetate and has been amenorrheic except for a recent episode of mild bleeding where an endometrial biopsy was performed and came back normal.

## Discussion

Thyroid dysfunction is the systemic disease most often associated with abnormal uterine bleeding [3]. Prevalence of menstrual irregularities in patients with untreated hypothyroidism was reported by Krassas to be 23.4% [4]. This is much less than that reported in previous studies, which showed that 50% to 70% of hypothyroid female patients had menstrual abnormalities [4]. In an Indian study, 68.2% of hypothyroid women had menstrual abnormalities, compared to 12.2% of healthy controls [5]. These differences can be explained by a more severe disease at diagnostic. Indeed, patients with severe hypothyroidism have a higher prevalence (34.8%) of menstrual disturbances than mild-moderate cases (10.2%) as reported in a study from Japan [6].

Hypothyroidism can result in menorrhagia, metrorrhagia, polymenorrhea, oligomenorrhea and amenorrhea [4,7]. Oligomenorrhea, polymenorrhea and menorrhagia are the menstrual disorders most commonly reported, while amenorrhea is rare [4,5,8,9].

There are many ways in which hypothyroidism can cause abnormal uterine bleeding, by disturbing both the menstrual cycle and hemostasis.

Thyrotropin-releasing hormone (TRH) increases the secretion of both TSH and PRL [4]. Serum prolactin level may be increased in hypothyroidism [9]. Hyperprolactinemia resulting from long-standing primary hypothyroidism has been implicated in ovulatory dysfunctions ranging from inadequate corpus luteal progesterone secretion when mildly elevated to oligomenorrhea or amenorrhea when circulating PRL levels are high [4].

In hypothyroidism, gonadotropin levels are usually normal [4,5]. TSH, which is markedly increased in hypothyroidism, has a small FSH- and LH-like effect because of the shared alpha-subunit [9,10]. The midcycle FSH and LH surge may thus be blunted or absent [4,5].

Moreover, the binding activity of sex hormone-binding globulin (SHBG) in plasma is decreased, with the result that the plasma concentrations of unbound testosterone and estradiol are increased despite normal total levels [4,10].

All these changes in menstrual regulation can result in anovulation. Menorrhagia is a frequent complaint and is probably due to estrogen breakthrough bleeding secondary to anovulation, which is frequent in severe hypothyroidism [4,5,9]. The anovulation is reflected in the frequent finding of a proliferative endometrium on endometrial biopsy [5].

Defects in hemostasis, such as the decreased levels of factors VII, VIII, IX, and XI that have been demonstrated in hypothyroidism, may also contribute to polymenorrhea and menorrhagia [4,5,9].

It is known that with thyroid replacement therapy, TSH levels normalize and the abnormal uterine bleeding usually resolves within 3 to 6 months [7].

With profound hypothyroidism, memory and concentration are impaired. In the present case, it surely contributed to the fact that the patient didn't seek medical treatment earlier. Moreover, myocardial contractility and pulse rate are reduced, leading to a reduced stroke volume and bradycardia. This phenomenon explains the absence of the reflex tachycardia expected in the setting of severe anaemia. Adrenergic shock can also blunt sympathetic response, and therefore must be part of the differential diagnosis as well.

## Conclusion

In conclusion, severe hypothyroidism can lead to significant uterine bleeding secondary to ovulation and coagulation disorders. This case is of particular interest because of the profound hypothyroidism and associated hemorrhagic shock. Recognition and proper aggressive management of this condition are important, as this condition can be life-threatening [11].

## Acknowledgements:

None

## References

1. Liu Z, Doan QV, Blumenthal P, Dubois RW (2007) A systematic review evaluating health-related quality of life, work impairment, and health-care costs and utilization in abnormal uterine bleeding. Value Health 10: 183-194.
2. Singh S, Best C, Dunn S, Leyland N, Wolfman WL, et al. (2013) Abnormal uterine bleeding in pre-menopausal women. J Obstet Gynaecol Can 35: 473-479.
3. Cowan BD, Morrison JC (1991) Management of abnormal genital bleeding in girls and women. N Engl J Med 324: 1710-1715.
4. Krassas GE (2000) Thyroid disease and female reproduction. Fertil Steril 74: 1063-1070.
5. Doufas AG, Mastorakos G (2000) The hypothalamic-pituitary-thyroid axis and the female reproductive system. Ann N Y Acad Sci 900: 65-76.
6. Kakuno Y, Amino N, Kanoh M, Kawai M, Fujiwara M, et al. (2010) Menstrual disturbances in various thyroid diseases. Endocr J 57: 1017-1022.
7. Brenner PF (1996) Differential diagnosis of abnormal uterine bleeding. Am J Obstet Gynecol 175: 766-769.
8. Krassas GE, Pontikides N, Kaltsas T, Papadopoulou P, Paunkovic J, et al. (1999) Disturbances of menstruation in hypothyroidism. Clin Endocrinol (Oxf) 50: 655-659.
9. Koutras DA (1997) Disturbances of menstruation in thyroid disease. Ann N Y Acad Sci 816: 280-284.
10. Moragianni VA, Somkuti SG (2007) Profound hypothyroidism-induced acute menorrhagia resulting in life-threatening anemia. Obstet Gynecol 110: 515-517.
11. Jameson J, Mandel SJ, Weetman AP (2015) Disorders of the thyroid gland. In: Kasper D (Eds). Harrison's Principles of Internal Medicine (19thedn) New York, NY: McGraw-Hill.