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CASE REPORT

Hashimoto's Thyroiditis in a Female Adolescent: A Case Report

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Abstract

Hashimoto's thyroiditis is the leading cause of acquired hypothyroidism in children, with growth delay often being the earliest sign. We report a 12-year-old girl presenting with short stature, sluggishness, alopecia, constipation, dry skin and acanthosis nigricans. Thyroid tests showed markedly elevated TSH and low free T3 and free T4, while thyroid autoantibodies were positive, confirming Hashimoto's thyroiditis. Thyroid ultrasound was normal. Levothyroxine 50 µg/day was initiated. After six months, the patient showed significant improvement with resolution of symptoms, normalization of thyroid function, a 7.5-cm height gain and a 4-kg weight loss. This case highlights the need for early recognition of hypothyroidism in adolescents presenting with growth delay and nonspecific symptoms.

Keywords: *Hashimoto's thyroiditis, Female adolescent.*

Introduction

Hashimoto thyroiditis is the most common cause of acquired hypothyroidism in children. Slowing of growth is usually the first clinical manifestation of acquired hypothyroidism. Goiter is a common presenting feature. Others common features include weight gain, myxedematous changes of the skin, constipation, cold intolerance, decreased energy, increased need for sleep. School performance usually does not suffer, even in severely hypothyroid children. Additional features may include bradycardia, muscle weakness or cramps, nerve entrapment, ataxia, delayed skeletal maturation and puberty. Diagnosis is usually based on elevate thyroid stimulated hormone (TSH) and low levels free thyroxin (fT4), coupled with increased antithyroid peroxidase antibodies.[1]

Hashimotto thyroiditis is an autoimmune disease that destroys thyroid cells and antibody-mediated immune process. It is the common cause of hypothyroidism in developed countries. In contrast, worldwide, the most common cause of hypothyroidism is an inadequate dietary intake of iodine. This disease is also known as chronic autoimmune thyroiditis and chronic lymphocytic thyroiditis.[2] The pathology of the disease involves the formation of antithyroid antibodies that attack the thyroid tissue, causing progressive fibrosis. The diagnosis is often challenging and may take time until later in the disease process. The most common laboratory findings demonstrate elevated thyroid stimulated hormone (TSH) and low levels of free thyroxin (fT4), coupled with increased antithyroid peroxidase (TPO) antibodies. However, earlier on the course of the disease, patient may exhibits signs, symptoms, and laboratory findings of hyper-thyroidism or normal values. This is because the destruction of the thyroid glands cells may be intermittent. Women are more affected. The female to male ratio is 10:1. Conventional treatment is comprised of levothyroxine. Excessive supplementation may lead to deleterious and morbid effect, such as arrhythmias (the most common being atrial fibrillation) and osteoporosis.[1,2] Here, we report 12 year old female adolescent presented with short stature, sluggishness, alopecia, constipation, dry skin, acanthosis nigricans. Her initial thyroid function elevated TSH (>200µIU/ml), decreased

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FT3(0.484ng/ml) and FT4 (3.61nmol/L), integrating the diagnosis of primary hypothyroidism. Though the main etiology is autoimmune, an excess protocol was completed with antibodies, highlighting the elevation of anti-peroxidase antibodies (37IU/mL), anti-thyroglobulin antibodies (264 IU/mL), concluding the diagnosis of Hashimoto's thyroiditis.[3]

Case Report

Promi Das, a 12 years old female adolescent was brought to paediatric outpatient department of US-Bangla Medical College Hospital, Rupgonj, Narayangonj with not gaining height in relation to her peers for last 2 years, chronic constipation, sluggishness, delayed puberty. On physical examination, she was found mildly pale, dull looking, alopecia, puffy face, acanthosis nigricans in the neck and armpits, dry and cold skin. Her vitals parameter were normal. Her weigh was 22kg, Height was 113cm, BMI 17.32kg/m² (underweight). Initially thyroid function tests was done, which revealed elevated TSH (>200μIU/ml), decreased FT(0.484ng/ml), FT4 (3.61nmol/L), integrating the diagnosis of primary hypothyroidism (Table 1), so that since the main etiology is autoimmune, an excess protocol was completed with antibodies, highlighting the elevation of anti-peroxidase antibodies (37IU/mL), anti-thyroglobulin antibodies (264 IU/mL), concluding the diagnosis of Hashimoto's thyroiditis. Neck ultrasound was performed which revealed normal. The biochemical report was glucose 5mmol/L, HbA1c 5.0%, insulin 16.5uUI/ml, triglycerides 97mg/dl, total cholesterol 150mg/dl, HDL 36mg/dl. LDL 90 mg/dl, ALT 21 IU/l, without meeting criteria for diabetes mellitus, nor for metabolic syndrome.

Fig.-1 *Dull facies with alopecia (before treatment)*

Fig.-2 *After treatment with Thyroxin*

Table I
Investigation reports

	15.10.23	12.11.23	
TSH (μIU/ml)	>200	1.06	↓
FT3 (ng/ml)	0.484	0.05	↓
FT4 (nmol/L)	3.61	21.2	↓
Anti-thyroid peroxidase Ab (IU/mL)	37	25	↓
Anti-Thyroglobulin Ab (IU/mL)	264	3.5	↓

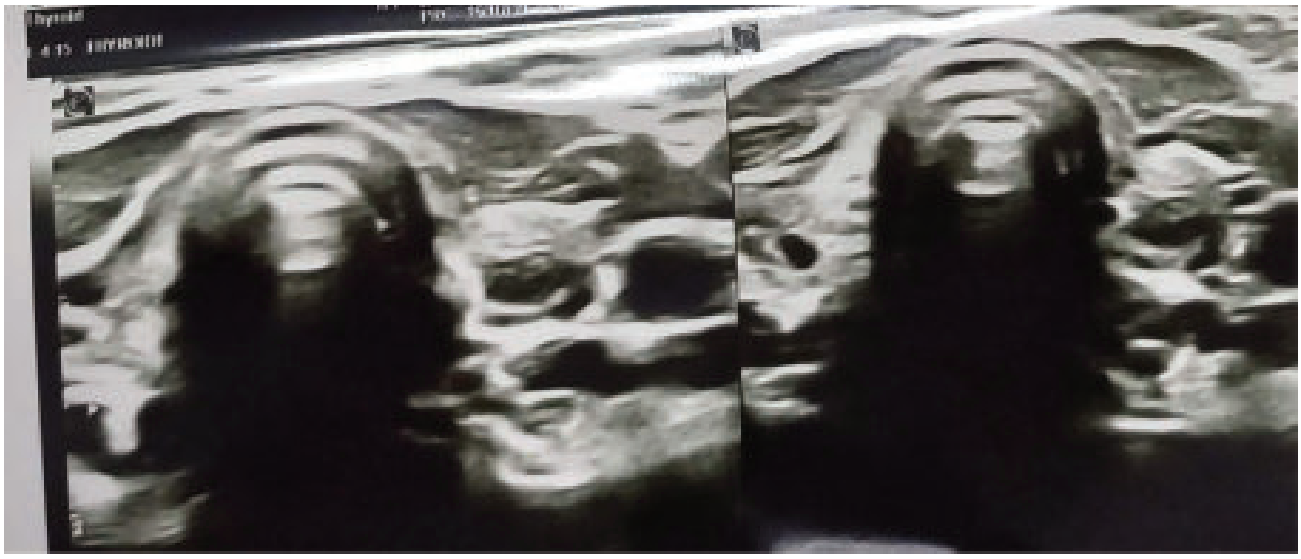

Fig.-3 *Thyroid ultrasound showing normal finding*

Interconsulation with endocrine department service was initiated, starting treatment with Levothyroxine 50 μgm/day. In the course of six months she presented a weight loss of 4 kg, height gain 7.5 cm with a BMI 12kg/m². the symptomatology described at the beginning of the approach subsided with evident improvement of the thyroid profile.

Discussion

The juvenile variant is the form of hypothyroidism that occurs before 18 years of age, with a mean presentation at 11 years. It is more frequent in women (10:1) patient may have a goiter , but are usually asymptomatic.[3] when establishing the diagnosis, 43% children are euthyroid, 24% have subclinical hypothyroidism, 21% have clinical hypothyroidism, 9% clinical hyperthyroidism and 3% subclinical hyperthyroidism. Constipation is most frequent symptom. The skin is usually dry, cold, yellow and thickened, changes due to the accumulation of hydrophilic mucoproteins in the dermis (such as hyaluronic acid) with the consequent myxedeama, as well as the atropy of the sweat glands. Weight gain mostly caused by fluid retention, not true obesity. Cold intolerance, decreased energy, and an increased need for sleep develop insidiously. School performance usually does not suffer, even in severely hypothyroid children. Additional features may include bradycardia, muscle weakness or cramps, nerve entrapment, and ataxia. Skeletal maturation is delayed, often strikingly, and the degree of delay reflects the duration of the hypothyroidism. Adolescents typically have delayed puberty. Older adolescent females may have menometrorahagia, and some may develop galactorrhoea because of increased TRH stimulating prolactin secretion. In fact long - standing primary hypothyroidism can result in enlargement of the pituitary gland, sometimes leading to headache and vision problems. This is believed to be the result of thyrotroph hyperplasia but may be mistaken for pituitary tumor, particularly a prolactinoma if prolactin is elevated. Rarely, young children with profound hypothyroidism may develop secondary sex characteristics including breast development or vaginal bleeding in girls and testicular enlargement in males.[4,5]

The natural history is variable, with remission, recurrence as well as the evaluation to permanent hypothyroidism. Hypothyroidism is an autoimmune disease and it is well known that other autoimmune diseases tend to cluster in the same patient and in the family. The most frequent associated diseases are: alopecia, vitiligo, celiac disease and insulin-dependent type-1 diabetes. Other disorders such as Turner syndrome and Down syndrome can also be associated with hypothyroidism.[6] In the present case, patient did not meet criteria for autoimmune comorbidities associated with hypothyroidism.

The diagnosis of Hashimotto's thyroiditis is established by clinical characteristics, the detection of serum antibodies against thyroid antigens (mainly thyroperoxidase and thyroglobulin), as well as

presence of goiter. Antithyroperoxidase antibodies are considered the best serological marker to establish the diagnosis. They are found in approximately 95% of hypothyroidism patient, but they are rare in healthy controls. Anti thyroid peroxidase antibody titres correlate well with the number of autoreactive lymphocytes that infiltrate the thyroid and the degree of ultrasound hypogenicity.[7] Antibodies to thyroid gland is most abundant protein in the thyroid gland, are less sensitive (positive in only 60-80% of patient) and les specific (positive in greater proportion of healthy controls) than antithyroid peroxidase antibodies.[8] Ultrasound of the neck has become the most used imaging tool in patients with thyroid diseases. In hypothyroidism, thyroid follicles are destroyed and replaced by small lymphocytes, causing the echogenicity of the thyroid parenchyma to decrease markedly.[9] Fine needle aspiration in patients in whom a thyroid nodule detected. Although the recommendation of the Bethesda system is conservative management, and, most lesions are benign, in some cases patients are referred to the surgeon and thyroidectomy may be required.[10] The treatment of primary hypothyroidism consists of the daily oral administration synthetic levothyroxine.[11]

Conclusion

Hypothyroidism is a multi-faceted and multidimensional disease. If the diagnosis is delayed it may produce so many complications. Autoimmune hypothyroidism is an organ specific autoimmune disease and it may be associated with other autoimmune diseases. High index of suspicion, adequate knowledge and experience is needed for early diagnosis and effective treatment. Timely intervention can improve most of the symptoms and most of the complications. Optimum thyroxin replacement therapy improves the prognosis and patients can lead almost normal life. Regular follow up is essential for better outcome.

References

1. Lee HS, Hwang JS, De Luca F, Santucci S, Corica D, Gonc EN, et al. The natural course of Hashimoto's thyroiditis in children and adolescents. *J Pediatr Endocr Met* 2014;**27**:807-12.
2. Rallison ML, Dobyns BM, Keating FR,Bossowski A, Moniuszko M, Dabrowska M, et al. Occurrence and natural history of chronic lymphocytic thyroiditis in childhood. *J Pediatr* 1975;**86**:675-82.
3. Demirbilek H, Kandemir N, Gonc EN, Rallison Ml, Dobyns BM, Keating FR, et al. Hashimoto's thyroiditis in children and adolescents: a retrospective study on clinical, epidemiological and laboratoty properties of the disease. *J Paedatr Endocrinol Metab* 2007;**20**:1199-1205.
4. Wasniewska M Corrias A Salerno M, Ruggeri RM, Trimarchi F, Giuffrida G, et al. Thyroid function patterns at Hashimoto's thyroiditis presentation in childhood and adolescence are mainly conditioned by patients age. *Horm Res Paediatr* 2012;**78**: 232-36.
5. Liu M, Murphy E, Amerson EH, Williams DE, Le SN, Godlewska M, et al. Rethinking screening for thyroid autoimmunity in vitiligo. *J Am Acad Dermatol 2013;**75**:*1278-80.
6. Pandit AA, Vijay Warde M, Menon PS, Tagoe CE, Sheth T, Golub E, et al. Correlation of number of intrathyroid lymphocytes with antimicrosomal antibody titre in Hashimotto's thyroiditis. *Diagn Cytopathol* 2003;**28**:63-65.
7. Mc Lachlan SM, Rapoport B, Mantovani A, Nascimbeni F, Lonardo A Lee HJ, et al. Why measure thyroglobulin autoantibodies rather than thyroid peroxidase autoantibodies *Thyroid* 2004;**14**: 510-20.
8. Lee Jh, Anzai Y, NevesC, Alves M, Medina JL, Fletcher AK, et al. Immaging of thyroid and parathyroid glands. *Semin Roentgenol* 2013;**8**:87-104.
9. Cibas Es, Ali SZ, Razvi S, Shakoor A, Vanderpump M, Razvi S, et al. The Bethesda system for reporting thyroid cytopathology. *Am J A Clin Pathol* 2009;**132**: 658-65.
10. Wiersinga WM, Kasper EK, Agema WR, Hutchins GM, Gottehrer A, Roa J, et al. Thyroid hormone replacement therapy. *Horm Res* 2001;**56**:74-81.
11. Leung AKC, Leung AAC, Yaun J, Sun C, Jiang S, Lu Y, et al. Evaluation and management of the child with hypothyroidism. *World J Pediatr* 2019;**15**: 124-34.