Case Report

Congenital Hypothyroidism with Precocious Puberty—A Case Report

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Abstract

Congenital hypothyroidism with precocious puberty is a rare condition. In this report a rare case of congenital hypothyroidism with precocious puberty is described. A 10 years old girl presented with features of hypothyroidism together with breast development, vaginal bleeding, lack of pubic hair and delayed bone age. She also had multicystic ovaries. She was treated with L-thyroxine and improved.

Introduction

Congenital hypothyroidism with precocious puberty is a rare condition. It is usually characterized by breast development, vaginal bleeding, lack of pubic hair and delayed bone age. Multicystic ovaries with precocious puberty in hypothyroid patients has been rarely described. Vaginal bleeding in adolescent or younger girls is of clinical importance. It may be caused by local causes such as vulvar or vaginal lesions, or it could be from the endometrium, which is usually a sign of systemic hormonal disturbance.

A convincing explanation of sexual precocity and bilateral ovarian enlargement is that high level of thyroid stimulating hormone (TSH) seen in profound hypothyroidism could act through the follicle stimulating hormone receptor (FSH-r) and cause gonadal stimulation. This causes breast development, uterine bleeding, multicystic ovaries in girls2,3 and macroorchidism without much virilization in boys4.

Case report

Ten years old female patient was admitted into paediatric unit of Rajshahi Medical College Hospital with history of cyclic vaginal bleeding starting at age of five years. The patient did not have a history of convulsions, meningitis, encephalitis, head injury or hormonal therapy. She was born at term pregnancy without any complications in an illiterate family. Her parents noticed that she had slow mental and physical development since birth compared with her brother and sister. There was no previous family history of similar condition.

On examination, patient looked lethargic, pale with coarse features and puffy face. Her temperature was 98 F, R/R 20/min, pulse 65/min, BP 95/55 mm Hg. Height was 112cm (3rd percentile), weight 25 kg (just above 10th percentile), Upper segment – lower segment ratio 1.7:1. No thyroid and lymph node enlargement, heart and chest were normal. Her breast buds were

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developed as tanner stage 4. Abdomen was distended. Umbilical hernia present. Two round masses were found on both sides of lower abdomen occupying iliac, umbilical and hypogastric region, which was firm in consistency, non tender, surface smooth, freely movable, border round. No pitting oedema.

On genital examination no abnormalities were detected. There was an increase in body hair growth, mainly on her back. She was severely intolerant to cold.

Investigation showed- Hb 8.6 gm/ dl, WBC 7000/cu mm, Platelet 250000/cu mm

USG of whole abdomen- Bilateral large ovarian cysts. Right one- 10.3 × 8.9 cm, left one- 9.8 × 6.8 cm. Bulky uterus (8 × 3.2 × 5.1 cm)

USG of thyroid gland- Thyroid gland is not seen in thyroid bed or any other part of the neck.

CT scan of Brain- normal

Thyroid scan- Thyroid gland not visualized, no evidence of functioning ectopic thyroid tissue in the neck.

Endocrinological evaluation revealed –

T4 = 2.8 µgm /dl (normal 4.8 – 11.5 µgm /dl)
T3 = 0.2 ng /ml (normal 0.69 – 2.02ng /dl)
TSH= 35miu/ml (normal 0.3 – 6.2miu/dl)
LH= 0.05 miu/ml (normal 0.6 – 19.0 miu/ml)
FSH= 15 miu/ml (4 – 13 miu/ml)

X-ray wrist – number of ossification centre was 6.

After the establishment of the diagnosis of congenital hypothyroidism and precocious puberty, L-thyroxine was given. After few days of treatment vaginal bleeding was stopped. In addition the patient was improved both physically and mentally. Over the last six months of follow up hormonal assay showed normal T3, T4, TSH, FSH and LH. Pelvic ultrasound revealed normal uterus and ovaries.

Discussion

The cause of vaginal bleeding must be sought when bleeding occurs in young girl. We report a typical case of vaginal bleeding that caused by hypothyroidism and its successful treatment with thyroxine replacement therapy. Findings of delayed bone age in girls with precocious puberty narrows the differential diagnosis to hypothyroidism because other causes of precocious puberty should have an advanced age. Generally children with congenital hypothyroidism present with delayed pubertal development and short stature.

This case presented with precocious pubertal development, short stature and delayed bone development. These findings are consistent with other reports. However, in other reported cases, six isolated menarche occurred in hypothyroidism in the absence of breast development. Interestingly, important clinical presentations that were reported previously, and were detected in our case are bilateral ovarian enlargement with multiple cysts and increased levels of gonadotropins mainly follicle stimulating hormone.

A convincing explanation of sexual precocity and bilateral ovarian enlargement is that high levels of thyroid stimulating hormone seen in profound hypothyroidism could act through the follicle stimulating hormone receptor and cause gonadal stimulation. This causes breast development, uterine bleeding, multicystic ovaries in girls and macroorchidism without excessive virilization in boys.

Conclusion

When vaginal bleeding occurs in young girls, hypothyroidism should be considered specially when vaginal bleeding is associated with other clinical features like short stature, delayed bone age and multicystic ovaries. Thyroxine replacement therapy should lead to complete resolution of such disorder and promote normal physical and mental development in young girls.
References


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