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Multicystic ovaries in uncontrolled congenital hypothyroidism

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Abstract: We report on a 12-year-old Saudi girl with uncontrolled congenital hypothyroidism, due to thyroid gland aplasia. She was found to have bilateral multicystic ovaries and menstrual dysfunction. This association is not widely recognized.

Keywords: Aplastic gland, congenital, hypothyroidism, multicystic, ovaries.

INTRODUCTION

Congenital primary hypothyroidism is one of the most common endocrine disorders encountered by the practitioner. It may be due to an absent or hypoplastic gland, an ectopic gland or an inborn error of thyroid hormone metabolism [1].

The association of cystic ovarian enlargement with the primary hypothyroidism is not widely recognized in the medical literature [2-4]. Clinical features include menstrual dysfunction [5-7].

We report, a 12-year-old Saudi girl with aplastic congenital hypothyroid, with bilateral multi cystic ovaries, and menstrual dysfunction.

CASE REPORT

A 12-year-old Saudi female child, was diagnosed with congenital primary hypothyroidism secondary to athyrotic thyroid gland in the neonatal period on screening and was started on Thyroxine therapy, she continued to be clinically and biochemically euthyroid. Her mother died when she was 7 years, and therefore she was cared for by her older sister. In spite of repeated counselling, she was not regular in her follow up and tended to skip medication. Her menarche was at 10 years. At 12 years she reappeared in the endocrine clinic with overweight and complained of menstrual irregularity. Physical examination revealed a puffy face, slightly obese girl, with weight of 58 Kg (>90th %) and had stunted growth for her age, the height of 146 cm (10th-24th %), with breast at Tanner stage 5, and completely developed pubic hair. A poorly controlled thyroid function which showed, thyroid stimulating hormone (TSH) of 186 mU/L (normal,<5), and free thyroxine (FT4) of 8.5 Pmol/L (normal ;10-25), and normal IGF-1. An abdominal sonography showed enlarged bilateral ovaries, with multiple cysts, serum prolactin was elevated at 990 Mu/L, serum Luteinizing hormone (LH) 1.2 U/L (normal 0.5-2.2), follicle stimulating hormone (FSH) was 6 U/L (normal; 0.2-6.0), oestradiol of <50 Pmol/L (normal 45.9-650), with an elevated serum testosterone at 65 ng/dL (normal 10-80) and elevated dehydroepiandosterone-s (DHEA-S) OF 476 mg/Dl (normal 65-360).

DISCUSSION

This case represents the importance of compliance with the therapy at any stage of management. The association of cystic ovaries enlargement with the primary hypothyroidism is not widely recognized in the medical literature [2-4]. The pathophysiology of this entity is unclear. Various mechanisms have been proposed as to the cause, these include altered estrogen metabolism, hypothalamic pituitary dysfunction, a direct effect on the ovaries or an altered prolactin metabolism [8]. Thyroid stimulating hormone (TSH), follicle stimulating hormone (FSH), luteinizing hormone (LH) have in common alpha chain and it is their chain that confer specifity. Cross-reaction of very high TSH could produce FSH and LH like activity responsible for the luteinized ovarian cysts [9]. In some cases reports the FSH levels have been high, therefore, there may be some action of thyroid releasing hormone on the pituitary cells to stimulate the gonadotropin release and, hence, FSH and LH. Other mechanism which may explain these changes are; an increased ovarian sensitivity to gonadotropin. An increase aromatization of androstenedione to estrogen [10]. On the other hand hyperprolactinemia reduced the gonadotropin clearance and decreased the dopaminergic and opioid tone at the hypothalamic pituitary axis [10]. However, the presize mechanism and many un answered question await further studies of this complex endocrine interaction involved.

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