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Early Onset Puberty: A Case Study of Precocious Puberty in a 20-Month-Old Girl

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Abstract **Case Report**

Precocious puberty is a medical condition marked by the early onset of sexual maturation. This article presents a rare case of central precocious puberty (CPP) in a 20-month-old girl, highlighting the diagnostic and therapeutic challenges associated with this condition. The case involves advanced bone maturation and elevated levels of LH and estradiol, indicative of premature activation of the hypothalamic-pituitary-gonadal axis. Treatment with GnRH agonists, specifically Triptorelin (Decapeptyl), proved effective in regressing clinical signs and normalizing hormonal levels. This report emphasizes the importance of early detection and individualized treatment strategies to prevent long-term complications such as reduced adult height. A multidisciplinary approach is crucial for accurate diagnosis and effective management of CPP.

Keywords: Severe Hypertriglyceridemia, Central Precocious Puberty (CPP), Hypothalamic-Pituitary-Gonadal Axis, GnRH Agonists, Pediatric Endocrinology, Multidisciplinary Approach.

INTRODUCTION

Precocious puberty is a medical phenomenon characterized by the appearance of signs of sexual maturation before the usual age. It can be classified into two main types: central precocious puberty (CPP), resulting from premature activation of the hypothalamic-pituitary-gonadal axis, and peripheral precocious puberty, caused by autonomous production of sex steroids. CPP is often idiopathic, but can also be associated with structural abnormalities of the central nervous system [1, 2]. This article presents a rare case of CPP in a 20-month-old girl, illustrating the diagnostic and therapeutic challenges of this condition.

CASE REPORT

A 20-month-old girl with no significant medical history was presented with genital bleeding and mild breast development, observed by her mother since the age of 11 months. Persistent regular menstruation, progressive breast development and the appearance of light pubic hair led to a consultation with an endocrinologist.

Initial investigations revealed a bone age of 3.5 years for a chronological age of 1 year and 8 months, indicating advanced bone maturation. Hormonal workup showed high levels of LH (7.33 mIU/mL), FSH was (2.8 U/L) and estradiol (78 pg/mL). Total testosterone levels were below 0.05, and 17-hydroxyprogesterone was 2.0 ng/mL. Cortisolemia at 8h was 270 ng/mL (27 µg/dL).

On clinical examination, the patient was conscious, with no signs of dehydration, acne, hirsutism, pallor or jaundice. Vital signs were normal, with respiratory rate at 20 cycles/min and heart rate at 72 bpm. The patient weighed 13 kg (+1 SD) and measured 85 cm (+1 SD). Tanner stage was S3P2.

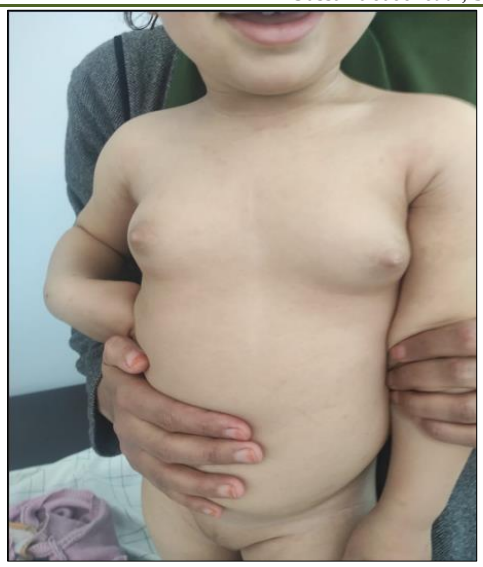

Figure 1: Developed breasts stage 3 of Tanner

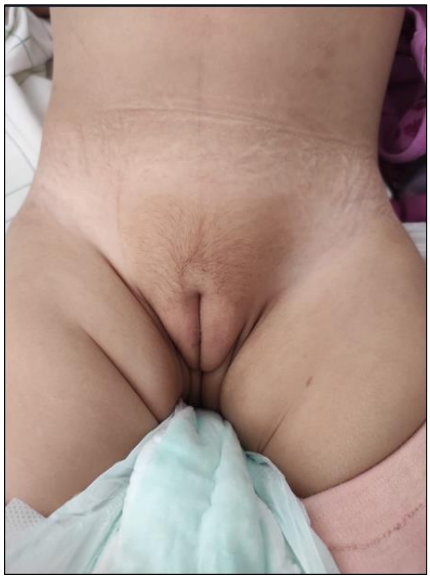

Figure 2: external genitalia with pubic hair stage 2 of Tanner

Radiological examinations during hospitalization included pelvic ultrasound showing diffuse endometrial hypertrophy without mass syndrome, and pituitary MRI revealing a discreetly bulging and enlarged pituitary gland, with an appearance of post-pituitary ectopy.The diagnosis of idiopathic precocious central puberty was retained.

The patient was treated with Triptorelin (Decapeptyl) 11.25 mg administered every three months. After three months of treatment, regression of clinical signs was observed, with FSH levels at 0.3 IU/L and LH at 1.22 mIU/ml.

DISCUSSION

Central precocious puberty (CPP) is a complex condition that requires thorough clinical and hormonal evaluation to differentiate idiopathic cases from those secondary to structural abnormalities of the central nervous system. In the case presented, advanced bone age and elevated LH and estradiol levels suggest premature activation of the hypothalamic-pituitary-gonadal axis, characteristic of CPP [1].

Diagnostic evaluation of central precocious puberty (CPP) usually involves a combination of detailed clinical examinations, hormonal testing and brain imaging. These tools help differentiate idiopathic causes from secondary causes linked to structural abnormalities. Clinical examinations include an assessment of physical characteristics and signs of sexual maturation, while hormonal tests measure levels of gonadotropins and other key hormones to confirm premature activation of the hypothalamic-pituitary-gonadal axis.

Brain imaging, such as MRI, is often used to detect structural abnormalities that could explain CPP.

These abnormalities can sometimes be associated with congenital lesions or malformations that stimulate the pituitary gland abnormally [4]. However, it is important to note that most cases of CPP in girls are idiopathic, meaning that no identifiable cause is found [2]. This distinction is essential to guide the choice of treatment and long-term follow-up of patients with CPP.

Treatment of CPP relies on the use of GnRH agonists, such as Triptorelin (Decapeptyl), which suppress the activity of the HPG axis by reducing pulsatile GnRH secretion. This treatment is effective in slowing bone maturation and halting the premature development of secondary sexual characteristics, thus preserving potential adult height [5].

In this case, after three months of treatment, a regression of clinical signs was observed, accompanied by a significant drop in LH and FSH levels. This confirms the efficacy of the treatment and underlines the importance of early intervention to optimize long-term results [6].

Management of CPP requires a multidisciplinary approach, involving pediatric endocrinologists, radiologists and imaging specialists for accurate assessment. Clinicians should be vigilant for early signs of puberty, such as breast development and vaginal bleeding, especially in very young children [3].

Regular follow-up of these patients is also crucial to adjust treatment according to clinical evolution and hormonal results. Continuous monitoring of growth and bone maturation is necessary to avoid long-term complications, such as reduced adult height due to premature closure of growth plates [7].

CONCLUSION

In conclusion, this case illustrates the diagnostic and therapeutic challenges of CPP in young children. Early detection and appropriate treatment are essential to prevent complications associated with this condition. An individualized approach, based on comprehensive clinical assessment and rigorous follow-up, is crucial to optimize outcomes in patients with CPP [8].

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