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Hypothalamic Hamartoma Revealed by Central Precocious Puberty in a Young Girl: A Case Report

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Abstract | **Case Report**

Central precocious puberty (CPP) associated with hypothalamic hamartoma (HH) is a rare but well-established cause of premature activation of the gonadotropic axis. We report the case of a 4 years and 3 months-old girl presenting with rapid onset of pubertal signs, associated with advanced bone age and elevated gonadotropin and oestradiol levels. Brain MRI confirmed the presence of a hypothalamic hamartoma. Treatment with GnRH agonists (triptorelin) led to regression of clinical signs and hormonal normalisation. This case highlights the importance of early diagnosis and multidisciplinary management to prevent complications, particularly reduced growth potential.

Keywords: Central Precocious Puberty (CPP), Hypothalamic Hamartoma (HH), Stature Growth, Early Diagnosis - GnRH Agonists, Multidisciplinary Management, Prevention of Complications.

INTRODUCTION

Precocious puberty is an early onset of secondary sexual characteristics before the normal age of puberty. Central precocious puberty (CPP) results from the premature activation of the hypothalamic–pituitary–gonadal axis. Establishing the diagnosis can be challenging, because this condition can have several etiologies, mostly idiopathic in girls, but congenital or acquired brain lesions cannot be excluded, particularly hypothalamic hamartoma, hence the need for brain imaging. Early recognition, thorough evaluation, and appropriate management are essential, including prompt referral to a pediatric endocrinologist to initiate treatment and prevent potential complications [1-3].

We report a case of precoce puberty to illustrate the diagnostic challenges and therapeutic considerations associated with this condition.

CASE REPORT

A 4 years and 3 months old girl with no particular medical history, who presented an increase in breast size observed by her mother over the past two months, with the appearance of light pubic hair and a moist, secreting vulva without vaginal bleeding (Figure:1).

On clinical examination, vital signs were normal, with no signs of dehydration, acne, or hirsutism. The patient weighed 18.1 kg (+1 standard deviation (SD) and measured 104.5 cm (+1 SD). Tanner stage was S3P2 (Figure:2)

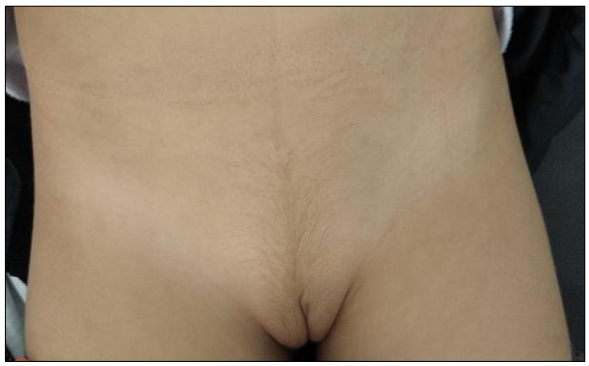

Figure 1: External genitalia with pubic hair stage 2 of Tanner

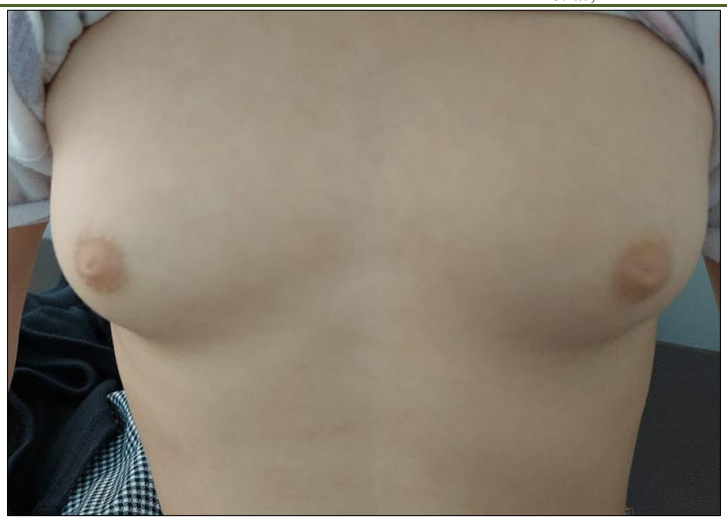

Figure 2: breast Development to Tanner stage 3

Paraclinical investigations revealed elevated levels of FSH (7.80 mIU/mL), LH (3.3 mIU/mL) and a high oestradiol level; the other axes were normal. The bone age was 7 years for a chronological age of 4 years, and the pelvic ultrasound showed a pubertal uterus.

Hypothalamic-pituitary MRI showing a median hypothalamic nodular formation with isosignal to the cerebral parenchyma on all sequences, measuring 9 x 8 mm, suggestive of a hypothalamic hamartoma (Figure:3).

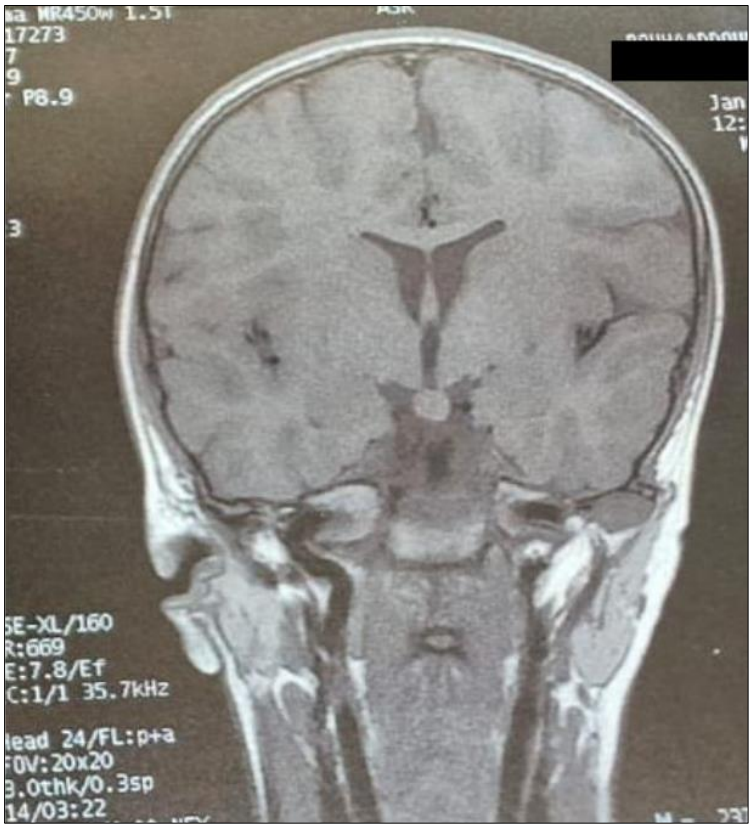

Figure 3: Coronal section of a hypothalamic-pituitary MRI in T1 sequence, showing a median hypothalamic nodular formation with iso-signal intensity in the cerebral parenchyma, measuring 9 x 8 mm, suggesting a hypothalamic hamartoma

In light of these results, a diagnosis of central precocious puberty due to a hypothalamic hamartoma was made.

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 1.04 IU/L and LH at 1.3 mIU/ml.

DISCUSSION

Hypothalamic hamartoma (HH) is a rare but well-characterised cause of central precocious puberty (CPP), accounting for less than 5% of identified aetiologies. Despite its rarity, it occupies a special place in the pathophysiology of CPP due to its ability to induce premature autonomic activation of the hypothalamic-pituitary-gonadal axis. The case presented illustrates several characteristic features of this pathology and highlights the diagnostic and therapeutic challenges specific to it [4].

From a pathophysiological perspective, HH acts as an ectopic generator of GnRH pulses, independent of the central neuroendocrine system. This chaotic pulsatile activity explains the often very early onset of pubertal signs, sometimes as early as infancy, as well as the significant advancement in bone age observed in these patients. Unlike idiopathic CPP, which is more common in girls and generally progressive, HH induces earlier onset of puberty, which progresses more rapidly and is generally associated with clearly pubertal gonadotropin levels [5].

The diagnostic evaluation of CPP is based on an integrated approach combining clinical examination, hormonal testing and brain imaging. Clinical examination allows the stage of pubertal maturation to be assessed, while hormone assays, particularly for gonadotropins and sex steroids, confirm premature activation of the hypothalamic-pituitary-gonadal axis [6].

However, diagnosing hypothalamic hamartoma is not particularly difficult compared to other masses in the region, as it is based on brain MRI scans, which have a characteristic appearance [7].

Due to the difficulty of access and the benign nature of the lesion, treatment is essentially medical, involving the administration of GnRH agonists, such as triptorelin (Decapeptyl), which suppress the hypothalamic-pituitary-gonadal axis by blocking the pulsatile secretion of GnRH. This approach slows bone maturation, interrupts the premature development of secondary sexual characteristics and preserves final height potential [8]. Surgical resection is indicated only in cases of progressive neurological deficit, hydrocephalus or frequent epileptic seizures that are refractory to medical treatment [9].

In the case presented, a clear clinical improvement was observed after six months of treatment, with regression of pubertal signs and a marked decrease in LH and FSH levels. This favourable evolution confirms the efficacy of the treatment and highlights the importance of early management to optimise the long-term prognosis [10].

The management of CPP due to a HH requires a multidisciplinary approach integrating the expertise of paediatric endocrinologists, radiologists and imaging specialists to ensure optimal diagnostic evaluation. However, clinicians must be vigilant for signs of precocious puberty, particularly in younger children, such as breast development or metrorrhagia, which may reveal an underlying organic aetiology [11].

Long-term follow-up is an essential part of management. It is based on regular clinical reassessment, periodic monitoring of hormonal parameters, and monitoring of stature growth and bone maturation. This approach allows for early adjustment of treatment based on therapeutic response and prevents late complications, particularly reduced stature potential due to premature closure of the growth plates [12].

CONCLUSION

Central precocious puberty secondary to a hypothalamic hamartoma, as rare as it is, must be identified quickly due to its major clinical implications. This case highlights the importance of early diagnosis based on careful clinical evaluation, appropriate hormone testing, and systematic brain imaging to rule out organic causes.

Early, individualised management, combined with regular clinical and radiological follow-up, remains essential to prevent long-term complications and optimise the prognosis for patients with CPP associated with hypothalamic hamartoma [13].

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