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Case Report

Hypothalamic hamartoma presenting as central precocious puberty in a girl: a case report

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ABSTRACT

Central precocious puberty (CPP) is defined as the early onset of secondary sexual characteristics due to premature activation of the hypothalamic pituitary gonadal (HPG) axis. Hypothalamic hamartomas are rare but classical causes of CPP. We report a case of a 2 years 7 months old girl with progressive breast enlargement and pubic hair development, found to have a hypothalamic hamartoma on MRI.

Keywords: Central precocious puberty, GnRH-dependent precocity, Hypothalamic hamartoma

INTRODUCTION

Precocious puberty is the appearance of secondary sexual characteristics before the age of 8 in girls and 9 in boys. It is classified as gonadotropin-dependent (central) or gonadotropin-independent (peripheral). Hypothalamic hamartoma is a congenital, non-neoplastic lesion that can act as an ectopic GnRH pulse generator, causing CPP.[1]

CASE REPORT

A 2 years 7 months old girl, resident of Bihar, presented with bilateral breast enlargement for two years and the development of axillary and pubic hair for the last six months. Her mother noted an increase in growth velocity starting around one year of age. There was no history of vaginal bleeding, headache, seizures, trauma, radiation exposure or signs of systemic illness. She was born at term via normal vaginal delivery, weighing 3 kg and had an unremarkable perinatal period. Developmental milestones were age-appropriate. Dietary history revealed a vegetarian diet with daily intake around 1150 kcal. There was no significant family history of early puberty or endocrine disorders. On examination, the child was alert and afebrile. Height was 103 cm (+3.23 SDS) and weight was 20 kg (+3.24 SDS), with a BMI of 18.8 kg/m² (+2.19 SDS), indicating a growth spurt. Sexual maturity rating (SMR) showed B4 (breast), P2 (pubic hair) and A+ (axillary hair), without signs of menstruation (Figure 1A-C). No skin lesions, bony deformities or abdominal masses were noted. Systemic examination was normal.

On investigations patient had a pubertal basal LH and FSH and normal Thyroid function test, suggesting gonadotropin-dependent precocious puberty (Table 1). Bone age was 5.5 years as per Greulich Pyle atlas (advanced by ~3 years, +6 SDS) (Figure 2). MRI brain revealed a hypothalamic hamartoma in the tuber cinereum region, confirming the central origin (Figure 3). A diagnosis of Gonadotropin-dependent precocious puberty secondary to hypothalamic hamartoma was made and the patient was initiated on inj. leuprolide acetate (GnRH analogue) 11.25 mg intramuscularly every 3 months and a single dose of Medroxyprogesterone acetate 150 mg IM stat to suppress ongoing pubertal progression.

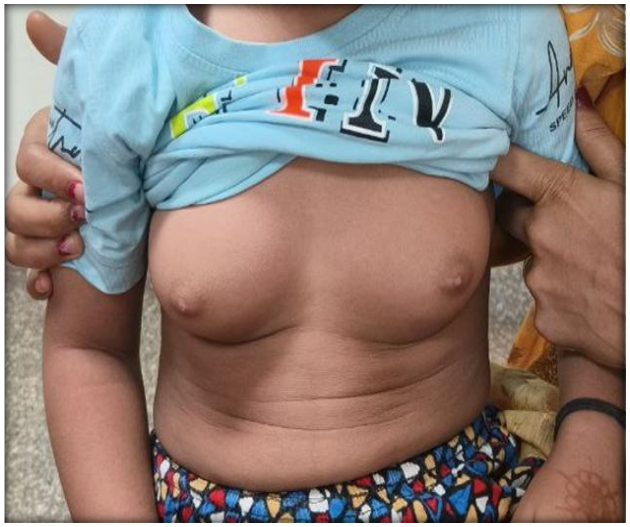

Figure 1 (A): Showing breast stage 4 as per Tanner's sexual maturity rating.

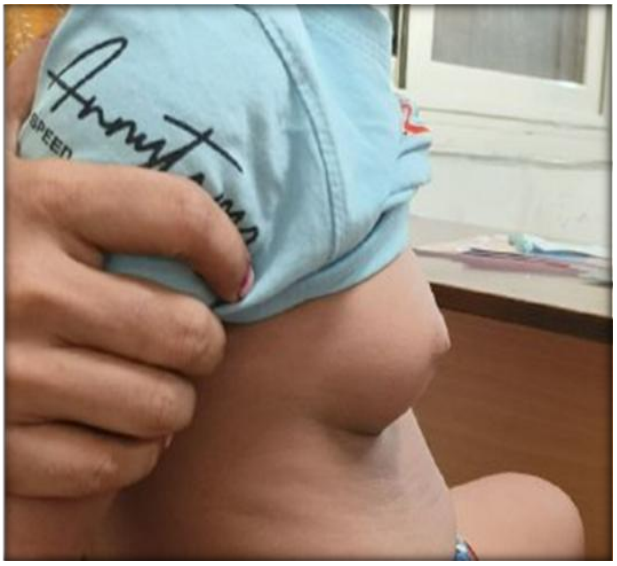

Figure 1 (B): showing breast stage 4 as per Tanner's sexual maturity rating.

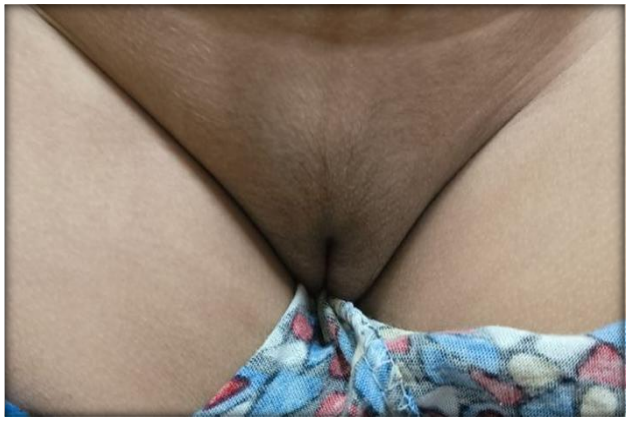

Figure 1 (C): Showing pubic hair stage as per Tanner's sexual maturity rating.

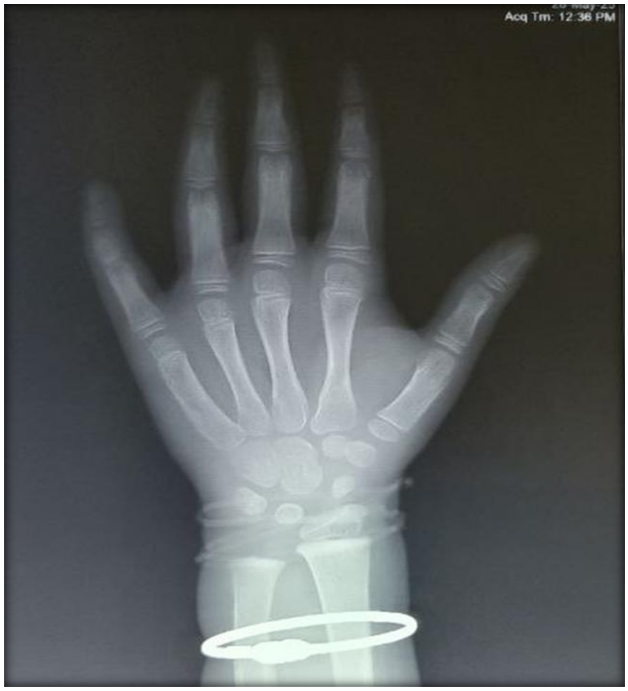

Figure 2: X-ray left wrist (AP view) showing bone age 5.5 years as per Greulich-Pyle atlas.

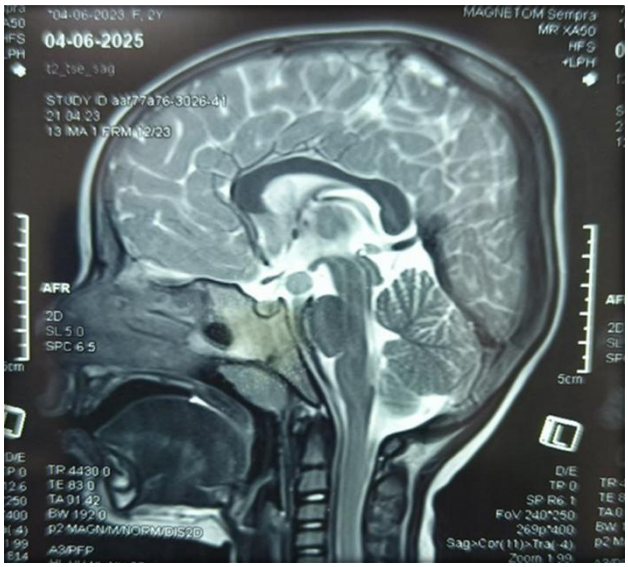

Figure 3: T2 Weighted MRI pituitary (sagittal section) showing a sessile homogenous rounded mass of 1.1×0.8 cm, isointense to the gray matter in the tuber cinereum region, suggestive of hypothalamic hamartoma.

On follow-up after 3 months of inj. leuprolide, the child had regression of breast development to a B3 stage from a B4 stage, pubic hair remained at a P2 stage and height was 103.3 cm. As the patient had a regression of pubertal development, she was continued on the same dosage of injection leuprolide. We also performed a stimulated LH test 4 hours after the subsequent dose of leuprolide, which yielded a result of 5.3 IU/l. However, as there was an adequate clinical response, we decided to continue the

same dose and frequency of leuprolide and to follow up after 3 months.

Table 1: Hormonal investigations.

Parameter	Patient value	Reference range
Serum LH	3.2 IU/l	0.8-7.6 IU/l
Serum FSH	7.3 IU/l	0.7-11.1 IU/l
T3	1.48 ng/ml	0.87- 1.78 ng/ml
T4	7.02 mcg/dl	4.82-15.65 mcg/dl
TSH	1.46 µIU/ml	0.38-533 µIU/ml

DISCUSSION

Puberty is a phase of life characterized by the development of secondary sex characteristics associated with growth acceleration. This phase also causes social and psychosocial development in a child. Precocious puberty means the early appearance of puberty before the usual onset of puberty. The cutoff to consider precocious puberty has been taken as the appearance of puberty before 8 years in girls and before 9 years in boys. Pathophysiologically, it can occur secondary to premature activation of the hypothalamic-pituitary-gonadal axis, known as central precococious puberty or without activation of the hypothalamic-pituitary-gonadal axis, known as peripheral precococious puberty.[2] The prevalence of precocious puberty is around 10 times higher in girls compared to boys.[3]

Among girls, central precocious puberty is most common and more than 90% of cases are idiopathic.[4] Hypothalamic hamartomas are rare but well-recognized causes of central precocious puberty. These benign lesions secrete GnRH in a pulsatile fashion, leading to early activation of the pituitary-gonadal axis.[5] Apart from central precocious puberty, various types of seizures are also found in up to 45% of patients. Among seizures, gelastic seizures, characterized by inappropriate laughter episodes are most common, which are often resistant to antiepileptics.[6] However, fortunately, our patient did not have any seizures. Biochemically, central precocious puberty can be diagnosed if basal LH >= 0.3 IU/l.[7] MRI is the imaging modality of choice for hypothalamic hamartoma diagnosis.[8] On MRI, hypothalamic hamartomas are hyperintense on T2-weighted images and hypointense on T1-weighted images, compared with normal gray matter and have reduced N-acetylaspartate and increased myoinositol on MR spectroscopy.[9]

Management typically includes long-acting GnRH analogs like leuprolide, which suppress the HPG axis and delay further pubertal progression.[10] Adjunctive use of medroxyprogesterone acetate can provide rapid suppression of sex steroid effects. Early diagnosis and treatment are crucial to prevent early epiphyseal closure and compromised adult height.[11] Follow-up should be done in these cases every 3-6 months. Sexual maturity rating and growth velocity should be checked on follow-up. It is expected to regress the growth velocity and breast stage in females. Biochemically, the patient should be monitored with basal LH and leuprolide-stimulated LH. The cutoff to define biochemical control is <0.6 for basal LH and <4 IU/l for stimulated LH. However, if the patient is clinically responsive, then the results of biochemical tests need not indicate treatment failure.[12] So, in our case we have decided to continue with the same dose and frequency of leuprolide acetate and follow after 3 months.

CONCLUSION

This case highlights the importance of recognizing central precocious puberty in very young children and the necessity of neuroimaging in all cases of suspected CPP to rule out hypothalamic lesions. Prompt intervention with GnRH analogs can significantly alter the course and prognosis.

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Ethical approval: Not required

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