Precocious Puberty: A Case Report

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ABSTRACT

A 4 years old girl was brought to the Department of Obstetrics and Gynaecology (of Bangabandhu Sheikh Mujib Medical University) for cyclical pervaginal bleeding and breast development. Her medical and family history was unremarkable. Evaluating her clinical presentations and investigations she was diagnosed as a case of precocious puberty. This case report emphasized how this condition should be evaluated and how these cases are to be managed.

Key Words: Precocious puberty, serum estradiol, follicle stimulating hormone and lutenizing hormone etc.

INTRODUCTION

Puberty results when pulsatile secretion of gonadotrophin releasing hormone is initiated and the hypothalamic pituitary gonadal axis is activated. Cross sectional data obtained in the 1960s led to designation of the normal age range of pubertal onset at the age between 8 and 13 years in girls. Pubertal development classified as precocious when it occurs before the age of 7 years in all other girls. Other definition of precocious puberty is reserved for girls who exhibit any secondary sex characteristics before the age of 8 or menstruate before the age of 10.2 The onset of puberty is marked by breast development in girls. Tanner stage 2 breast development means appearance of the breast bud marks at the onset of pubertal development.³

The most common mechanism of progressive precocious puberty is the early activation of pulsatile gonadotrophin releasing hormone secretion which results in maturation of hypothalamo-pituitary ovarian axis that activates maturation of hormone responsive tissue such as

breasts, bones, pubic hair and endometrium. These girls have normal ovulation, menses and reproductive capacity.⁴ Peripheral or gonadotrophin independent puberty is recognized including gonadal and adrenal tumor. For evaluation of patient with precocious puberty several questions should be addressed: Is pubertal development really occurring outside the normal temporal range? What is the underlying mechanism? Is pubertal development likely to progress and if no, would this impair the child's normal physical and psychosocial development?

Case Report

A 4 year-old girl was admitted in the Obstetrics & Gynaecology Department of Bangabandhu Sheikh Mujib Medical University with the complaints of enlargements of both breasts for one year and cyclical per vaginal bleeding for 3 months. According to the statement of patient's mother her child was alright one year back. Her milestones of development were normal. But she noticed that her breast was enlarging gradually.

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Figure 1

Figure 2

FIGURE 1 & 2: Shows 4 yrs old girl with sign of breast development

She also noticed that the girl was suffering from cyclical per vaginal bleeding which persists 4 to 5 days per month. She had no history of birth injury, head injury, encephalitis, headache or seizures. General physical examination of the patient was within normal limit. Her height was 85 cm, weight 16 kg and skin pigmentation was absent. But examination of breast revealed that her both of the breasts were enlarged and firm in consistency. Nipple and areola developed, but no discharge was present. Axillary and pubic hair was sparse.

On per abdominal examination, there was a palpable lump around (5cm \times 3cm) on right side of the lower abdomen. Hormonal assessment was done and serum estradiol and LH (Luteinizing Hormone) levels were elevated and they were within pubertal range. Pelvic ultrasound scan showed uterus was enlarged for her age i.e. AP-2.1 cm, length-5.4 cm. Endometrium was thickened. In right ovary - There was a cyst (5.3 \times 2.8 cm) and another small cyst situated by the

main cyst. Left ovary contains multiple cysts. The following investigations were done to confirm the diagnosis. X-ray of hand and wrist was done for bone age and it corresponded with chronological age. X-ray of sella turcica was done to exclude intracranial lesion. Serum TSH (Thyroid Stimulating Hormone) level was also done and which was more than 100 mIU/L.

Analyzing all the investigations and clinical presentations we came to a conclusion that this girl had suffered from precocious puberty with hypothyroidism and ovarian cyst. After counseling with the patient's guardian Laparotomy followed by ovarian cystectomy was done. Ovarian cyst was sent for histopathological report which revealed that the large cyst was derived from degenerated follicular cyst. Her postoperative recovery was uneventful. She was discharged on 8th postoperative day after operation. She was advised to come for follow up one and a half month after operation. Her complaints and findings started to resolve after ovarian cystectomy and

medication for hypothyroidism (tablet Thyrox 50 µgm in morning half an hour before meal). She came for follow up one and half months after laparotomy - her breasts became less prominent in size and her menstruation stopped. Six months after operation in her second follow up visit - breast size became normal for her age and her complaints of menstruation stopped. Her serum estradiol, FSH (Follicle Stimulating Hormone) and LH level corresponded with her age. Her TSH level was also within normal limit.

Discussion

In the case described, we have found advance breast development, pubic and axiliary hair development. Her height was within normal limit. She had cyclical per vaginal bleeding. Evidence of possible causes of precocious puberty was sought by means of thorough history taking and careful examination followed by investigations. In her personal history and family history nothing contributory was noted. Serum estradiol and LH level were elevated. In girls, serum estradiol level is highly variable but low sensitivity for diagnosis of precocious puberty.⁵

Random measurement of LH has been proposed. In one study randomly measured value of 0.3 IU per liter and above were reported to be 100% specific.⁶ A reference atlas such as the one Gy Greulich evaluated the effect of sex steroid on epiphyseal maturation and found bone age to be greater than chronological age. 7 But in the present case bone age corresponded with chronological age. A study showed that to perform GnRH (Gonadotrophin releasing hormone) agonist stimulation test to further evaluate the activation of gonadotrophin axis and the potential for progression of puberty.⁸ There are no facilities in our institution for GnRH agonist stimulation to see the progression of puberty. Several reports showed that magnetic resonance imaging (MRI) scan did not show a central nervous system (CNS) lesion in approximately 92% of girls. 9 Most common mechanism of progressive or central precocious puberty is the

early activation of pulsatile gonadotrophin releasing hormone which may result from hypothalamic tumor or lesion but in most cases remain unexplained. Several reports showed that constitutional type is the commonest but rare. However, from history of our patient with various investigations we come to a conclusion that this is a case of precocious puberty with hypothyroidism and follicular ovarian cyst.

Conclusion

Although in most of the girls with precocious puberty the etiology is idiopathic. In some case hormone secreting tumor of the ovary and hypothyroidism should be considered in the differential diagnosis for prepubertal girl.

REFERENCES

- Vottero A, Pedori S, Verna M, Pagano B, Cappa M, Loche S, et al. Final height in girls with central idiopathic precocious puberty treated with gonadotropin-releasing hormone analog and oxandrolone. *J Clin Endocrinol Metab* 2006;91(4):1284-7.
- Eugster EA, Rubin SD, Reiter EO, Plourde P, Jou HC, Pescovitz OH; McCune-Albright Study Group. Tamoxifen treatment for precocious puberty in McCune-Albright syndrome: a multicenter trial. *J Pediatr* 2003;143(1):60-6.
- 3. Carel JC, Leger J. Clinical practice: precocious puberty. N Engl J Med 2008;358:2366-77.
- Lahlou N, Carel JC, Chaussain JL, Roger M. Pharmacokinetics and Pharmacodynamics of GnRH Agonists: Clinical Implications in Pediatrics. J Pediatric Endocrino Metabolism 2000;13:723-37.
- Neely EK, Hintz RL, Parker B, Bachrach LK, Cohen P, Olney R et al. Two-year results of treatment with depot leuprolide acetate for central precocious puberty. J Pediatr 1992;121(4):634-40.
- Carel JC, Lahlou N, Jaramillo O, Montauban V, Teinturier C, Colle M et al. Treatment of central precocious puberty by subcutaneous injections of leuprorelin 3-month depot (11.25 mg). *J Clin Endocrinol Metab* 2002;87(9):4111-6.
- Klein KO, Barnes KM, Jones JV, Feuillan PP, Cutler GB Jr. Increased final height in precocious puberty after long-term treatment with LHRH agonists: the National Institutes of Health experience. *J Clin Endocrinol* Metab 2001;86(10):4711-6.

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- Palmert MR, Mansfield MJ, Crowley WF Jr, Crigler JF Jr, Crawford JD, Boepple PA. Is obesity an outcome of gonadotropin-releasing hormone agonist administration? Analysis of growth and body composition in 110 patients with central precocious puberty. J Clin Endocrinol Metab 1999;84(12):4480-8.
- Heger S, Müller M, Ranke M, Schwarz HP, Waldhauser F, Partsch CJ et al. Long-term GnRH agonist treatment for female central precocious puberty does not impair reproductive function. *Mol Cell Endocrinol* 2006;254-255:217-20.
- 10. Linam LE, Darolia R, Naffaa LN, Breech LL, O'hara SM, Hillard PJ et al. US findings of adnexal torsion in children and adolescents: size really does matter. *Pediatr Radiol* 2007;37(10):1013-9.