disease. Third stage larva is ideal for species identification [5].

*Chrysomya megacephala*, more commonly known as the Oriental Latrine Fly, is known to breed in human feces, meat and fish. In the rural Indian population, defecating in open air is a common practice. The fly is attracted by feces and lays eggs on them. After landing on feces it lands commonly on human foods and on very rare occasion on open human wounds or on umbilicus of a newborn [6]. These may be the events that lead to umbilical myiasis in the two cases that we have reported.

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*Contributors:* All were responsible for patient care and investigation. TG & NG prepared the manuscript. KN organized the management and follow up of the case. MKG was

responsible for radiological examination of the babies. All authors read and approved the final manuscript. *Funding*: None.

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# Hormone Dust Exposure – A Reversible Cause of Precocious Pseudopuberty in Siblings

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Correspondence to: Dr Hari Anupama, Professor of Obstetrics and Gynaecology, C1-Vora Towers, Madhura Nagar, Hyderabad, AP 500 038, India. hradhakrishna2020@rediffmail.com Received: July 20, 2009; Initial review: August 3, 2009; Accepted: January 4, 2010. A seven year old female child presented with two episodes of vaginal bleding and bilateral breast development. Examination showed normal anthropometric measurements and external genitalia of prepubertal type. Her 5-year old younger brother and her father had gynaecomastia. Search for a structural cause for precocious puberty was negative. Occupational exposure of her father to hormonal dust was identified as the cause. All pubertal changes reverted to normal after observing suitable precautions.

Key words: Hormonal dust exposure, precocious pseudopuberty.

Precocious puberty can occur due to exogenous hormone administration in the form of oral hormone pills, estrogen containing creams, cosmetics, as well as estrogen contaminated food. We report an unusual estrogen exposure leading to precocious pseudopuberty in two siblings and gynecomastia in their father.

### **CASE REPORT**

A seven year old female child was brought by her mother with a history of two episodes of vaginal

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bleeding and bilateral breast development. She had menstruated twice, each time lasting for 3 days, with moderate flow and the interval between the two cycles was 22 days.

There was no history of recent growth spurt, headache, visual disturbances or café-au-lait spots on the skin. On examination, her height was 92.5 cm and weight 21 kg. She had sparse axillary and pubic hair. Breasts were of Tanner stage III. Nipples and areolae were hyperpigmented. External genitalia were of prepubertal type and there was no nipple discharge. Ocular fundus examination was normal.

A detailed family history was elicited. Her mother attained menarche at the age of 14 years. The girl had one younger brother of 5 years age. He was found to be having gynecomastia with no other abnormality. At request, the father also consented for examination and was found to be having bilateral gynecomastia without any galactorrhea. The father was employed in a pharmaceutical company where oral contraceptives were manufactured. In that company, his job was to supervise the site where tablets were made from the hormone powder. After returning from his duties, he used to play with the children for 2-3 hours daily and then only he used to change from the office clothes. This was his routine for about previous 2 years.

Investigations of the girl child revealed uterine size of  $5.4 \times 4.0 \times 2.1$  cm with endometrial thickening of 6 mm on ultrasound. Right ovary measured  $1.3 \times 1.5$  cm and left ovary measured  $1.3 \times 1.4$  cm without any follicles. Adrenals were not enlarged. Magnetic resonance imaging scan of the brain was normal.

Hormonal investigations included: follicular stimulating hormone (FSH) -3.2 IU/mL; leutinizing hormone (LH) - 2.7 IU/mL; and 17  $\beta$ -estradiol - 64 pg/mL (increased); thyroid stimulating hormone were normal. 17-OH progesterone - 2.5 nmol/L, dihydroepiandrostenedione (DHEAS) - 2.2 nmol/L and serum testosterone level - 0.3 ng/mL. Bone age and estimated by studying the *X*-ray of the nondominant hand using Greulich Pyle Atlas, was 7 years.

The entire family was given reassurance and explained about the etiopathology of precocious

puberty in the girl. They were also counselled in detail about the reversibility of the condition. The father was advised change of occupation. If it was not possible, he was advised to use face masks to avoid inhalation of hormonal dust. He was also asked to change his clothing and to take bath immediately after returning home from his work place.

The girl was followed up on monthly basis for eight months. All the changes regressed completely within six months. Interestingly, the boy also showed regression of gynecomastia. The father could not find an alternate job and continued to have gynaecomastia even after eight months.

# DISCUSSION

In the present case, the possible mechanism for premature thelarche and menarche appear to be due to the estrogen dust which the father was carrying on his clothes from his work place which was an oral contraceptive manufacturing industry. Estrogens can be readily absorbed through skin. In the present case, estrogens might have entered the systemic circulation not only through absorption from the skin but also through inhalation (in the case of the father). Prepubertal girls are extremely sensitive to exogenous estrogens. In the present case, it manifested in the form of premature menarche and premature thelarche without affecting the general growth. Similar mechanism might have played its part in the development of gynecomastia in her brother and father.

It is now understood that nearly every case of precocious pseudo puberty can secondarily activate the hypothalamo-pituitary-gonadal axis with development and superimposition of a central GnRh dependent true precocity process [1]. Because of this reason, the presence of detectable levels of FSH and LH is not surprising in the present case.

The linear growth of the body was not affected in the present case as evidenced by the bone age. Estrogen in low doses stimulates growth hormone induced Insulin Growth Factor 1 (IGF-1) secretion of which is responsible for the linear growth. High doses of estrogen suppresses IGF-1 levels. Another possible mechanism by which the linear growth is

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unaffected is that, feedback inhibition of GnRH by large doses of estrogen. The GnRH plays an important role in pubertal growth spurt. A similar finding was noted in a previous study wherein *Rhesus* monkeys were treated with exogenous estrogen combination. Premature thelarche and menarche were observed without any change in the linear bone growth [2].

The highest known incidence of premature thelarche was observed in Puerto Rico where significant serum levels of 2 ethylhexyl phthalates and plasticizers were identified as endocrine disrupting chemicals of estrogenic nature [3]. There are reports where use of hormone containing hair products like shampoos and oils had contributed to earlier onset of puberty in the African American population [4]. Tiwary [5] analyzed that hair products contained up to 4mg of estradiol per 100gms and he also noted that the pubertal changes regressed on discontinuing the use of these products.

A detailed history, examination and investigations to elicit the etiological factors are of the utmost importance. Drug ingestion should be suspected in all unexplained cases of pubertal precocity.

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# MRI Abnormalities of the Anterior Temporal Lobe: A New Indicator of Congenital Cytomegalovirus Infection

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Correspondence to: Dr Mahesh Kamate, Assistant Professor of Paediatrics, KLE University's J N Medical College, Belgaum 590 010, Karnataka State, India. drmaheshkamate@gmail.com Received: August 07, 2009; Initial review: November 23, 2009; Accepted: January 4, 2010. Abnormalities of the anterior part of the temporal lobe (abnormal and swollen white matter, cysts, and focal enlargement of the anterior part of the inferior horn- either alone or more often in combination) suggest congenital cytomegalovirus (CMV) infection. This is not widely known. These can be seen in neonatal period and they continue to persist in later life.

Key words: Cytomegalovirus, Neonate, Temporal lobe.

ytomegalovirus (CMV) is the leading cause of congenital infections and in the West, it affects about 1% of all live births [1]. Intrauterine CMV infection presents in the neonatal period as jaundice, hepatosplenomegaly, petechiae, microcephaly, and chorioretinitis. At the same time, it is also known that about 90% of infants affected by intrauterine CMV

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