# Premature Sexual Development in Children Following the Use of Estrogen- or Placenta-Containing Hair Products

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**Summary:** Four African-American girls aged 14 months to 93 months developed breast or pubic hair 2 to 24 months after starting the use of estrogen or placenta-containing hair products. Discontinuing the use of the hair products resulted in regression of the breast or pubic hair. Serum gonadotropins and estradiol levels were variable. No other cause for early sexual development was noted in these girls. *Clin Pediatr.* 1998;37:733-740

## Introduction

Premature adrenarche and premature thelarche are common observations. In a report on sexual development among more than 5,000 girls in various parts of the United States, Herman-Giddens et al[1] noted that 37% of 7-year-old and 51.5% of 8-year-old African-American girls show development of breasts or pubic hair, while the corresponding figures for the white girls were 5.5% and 16%, respectively. The cause of racial differences in the age of early sexual development is unknown. Early sexual development in infants and children has been described following unintentional ingestion[2-5] or inhalation[6,7] of estrogen, after its topical application to the breast area,[8] and after intentional topical application of estrogen-containing products to the diaper area to treat diaper rash[9,10] or to the scalp to treat scalp conditions.[11,12] Early development of sexual characteristics in children in association with the use of estrogen-containing cosmetic hair preparations has been reported rarely.[13,14] Only one report described the development of sexual precocity in African-American girls following the use of hair products containing placenta.[14] Rogol and Blizzard[15] suggest that "An extensive investigation regarding possible estrogen ingestion or exposure is essential" for patients with premature thelarche. We describe four patients who showed signs of early sexual maturation after using various cosmetic hair preparations containing estrogenic or human/bovine placental constituents.

## Materials and Methods

The Atlas of Gruelich and Pyle was used for bone age determination.[16] The hormone analysis in the hair preparations was done by Leberco Testing/Inc, Roselle Park, NJ, by previously published methods.[17] The androgens in the hair products were not analyzed because no standard assay method was available to measure these. The hormone analysis of the hair products used by the patients showed that estriol concentration was 19 mg/g in a placenta-containing hair product, 16 mg/g in one estrogen-containing hair

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product, and 20 mg/g in another estrogen-containing hair product. Estradiol (0.04 mg/g) was detected only in the latter estrogen-containing hair product. In the body, estrone is converted to estriol (short-acting estrogen); estrone and estradiol (long acting) are interconvertible. Estriol is biologically active; it binds to immature uterine tissue,[18,19] affects mature vaginal tissue,[20] and alters the plasma progesterone level.[21]

Dehydroepiandrosterone (DHEA), androstenedione, and 17 hydroxypregnenolone were measured by the Endocrine Sciences Lab, CA. All the other serum hormones were measured in the hospital laboratory by standard methods.[22]

For basal control values in children, we measured serum luteinizing hormone (LH), follicle stimulating hormone (FSH), and estradiol in the blood sample taken in the morning from three normal African-American girls, aged 1–6 years, and from several other children not suffering from any endocrine or metabolic disorder or any disease likely to affect the gonadotropin values. The luteinizing hormone-releasing hormone (LHRH) stimulation test was done after IM injection of LHRH (Factrel, Wyeth-Ayrest Laboratories) as described previously.[23] The serum LH, FSH, and estradiol values, and LH/FSH ratios,[24] both basal and after LHRH stimulation in control children and in the patients, are given in Table 1. In our clinical experience after LHRH stimulation, children with precocious puberty show a peak rise in estradiol level of >18.36 pmol/L.

Four African-American girls developed breasts or pubic hair after using estrogen- or placenta-containing hair products for varying periods and showed regression of sexual characteristics after discontinuing the use of such products. The anthropometric data (Table 2), details of use of the hair products (Table 3), and hormonal data of these patients (Table 1) are presented.

All of these patients were born by normal delivery, did not require any resuscitative measures, and had a neonatal course that was unremarkable. Family history of early sexual development was not available for these patients. The physical and mental development were normal in all, and vaginal discharge was absent by history and on examination. None showed thyromegaly or clitoral enlargement. The skeletal system was normal, and no significant skin pigmentary changes were noted. No organic cause for early sexual development was found in any of these four patients.

The thyroid function test results were normal in all patients. Pelvic sonograms done in patients 2, 3, and 4 appeared normal.

**Table 1**

**BASAL AND LHRH-STIMULATED SERUM HORMONAL VALUES**

| Patient No. | Basal Values | | Peak Values | | | Basal Estradiol (pmol/L) |
|---|---|---|---|---|---|---|
| | LH, (IU/L) | FSH, (IU/L) | LH, (IU/L) | FSH, (IU/L) | LH/FSH Ratio | |
| 2 | <0.2 | 3.1 | ND | ND | ND | Normal* |
| 3 | <0.5 | 2.5 | 3.2 | 28 | 0.114 | 20.19 |
| 4 | <0.5 | 2.9 | 2.6 | 30.9 | 0.086 | <18.36† |
| Control values | <0.1–0.5 | 1.7–2.2 | ND | ND | ‡Range 0.75–1.83<br>§Range 0.16–0.41 | <18.36 |

*Normal basal serum estradiol value in prepubertal children is at or below the lower limit of the sensitivity of the assay.

†Serum estradiol (pmol/L) after LHRH injection was 23.13 pmol/L. The value is in the range found in pubertal children.

‡Range of the peak serum LH/FSH ratio in children with precocious puberty (Ref 24).

§Range of the peak serum LH/FSH ratio in children with premature adrenarche (Ref 24).

LH=luteinizing hormone, FSH=follicle stimulating hormone, LHRH=luteinizing hormone-releasing hormone, ND=not done.

**Table 2**

**ANTHROPOMETRIC AND BONE AGE DATA ON THE PATIENTS**

| Patient No. | Height (Centile) | Weight (Centile) | Age in Months: Chronological Age | Age in Months: Bone Age (1 SD) |
|---|---|---|---|---|
| 1 | >50%, <75% | 75% | 14 | 18 ±1.77 |
| 2 | >75%, <90% | >25%, <50% | 20 | 24 ±3.49 |
| 3 | >95% | 95% | 93 | 132 ±10.23 |
| 4 | >95% | >95% | 81 | 94 ±9.64 |

## Patient Reports

*Patient 1*

Patient 1, born on July 12, 1990, was referred at the age of 14 months for evaluation of premature development of pubic hair. The mother started using a placenta-containing hair product on the baby's scalp when the baby was 12 months of age. During a routine visit, a pediatrician observed the presence of pubic hair, referred the patient for evaluation, and advised the mother to discontinue the use of the hair product. Two months after discontinuing the use of the hair product the mother remarked that the pubic hair had decreased. Physical examination at the age of 16 months revealed that the breast tissue was not palpable and the areola was normal. Very fine hair was present in the pubic area and on the labia majora.

Congenital adrenal hyperplasia due to 21 hydroxylase or 3 beta-hydroxysteroid dehydrogenase deficiency was considered and serum 17-hydroxyprogesterone and dehydroepiandrosterone (DHEA) were measured. These metabolite values were normal. The deficiency of either of these enzymes as a cause of premature pubic hair development in this patient is unlikely. In view of the normal findings and decreasing pubic hair after discontinuance of the use of the hair product, no further tests were requested.

*Patient 2*

Patient 2, born on April 18, 1990, was referred at the age of 22 months for evaluation of breast enlargement. The mother began using an estrogen-containing hair preparation on the baby's scalp at 12 months of age. Breast enlargement was noted 6 months later but there was no pubic or axillary hair development. Physical examination showed both breasts were palpable and each breast measured 6 cm by 6 cm. The areola

**Table 3**

**THE DURATION OF USE OF HORMONE- OR PLACENTA-CONTAINING HAIR PRODUCTS AND THEIR EFFECTS ON THE DEVELOPMENT OF SEXUAL CHARACTERISTICS**

| Patient No. | Contents (estrogen or placenta) of the hair product used | Age when the hair product use was started (months) | Onset of sexual development after the start of use of hair product (months) | Total duration of the product use (months) | Type of sexual development |
|---|---|---|---|---|---|
| 1 | Placenta | 10 | 2 | 3 | Pubic hair |
| 2 | Estrogen | 12 | 6 | 10 | Breasts |
| 3 | Estrogen | 60 | 24 | 30 | Breasts and pubic hair |
| 4 | Placenta and estrogen | 48 | 12 | 35 | Pubic hair |

and nipple were immature and no hyperpigmentation was noted. Axillary and pubic hairs were absent, and external genitalia were normal. An endogenous source of estrogen excess was ruled out by normal serum gonadotropins and estradiol values (Table 1), and a normal pelvic sonogram. The mother was advised to discontinue the use of the estrogen-containing preparation. Follow-up at 35 months of age (13 months after discontinuation of the use of the hair preparation) revealed that the breasts had decreased in size and no other sign of sexual development was noted.

*Patient 3*

Patient 3, born on September 20, 1984, was referred at the age of 93 months for evaluation of bilateral breast enlargement. Bilateral breast enlargement (3 cm by 3 cm) without the presence of pubic or axillary hair was first noted at the age of 83 months during a school physical examination. She suffered an injury to the right breast at the age of 92 months and presented to the clinic 1 month later because of painful right breast. Bilateral breast enlargement (right breast greater than the left) was noted. Further history revealed that the girl had used an estrogen-containing hair cream for 2 weeks of each month for the last 3 years. Breast development was noted 2 years after initiation of the hair product use. The girl was advised to discontinue the use of the estrogen-containing hair preparation, and 1 month later a decrease in the breast size was noted. Physical examination at the age of 95 months revealed that the right and left areola each measured 1.5 cm in diameter and were not hyperpigmented. The right breast measured 7.5 by 7.5 cm and the left breast measured 4.5 by 4.0 cm. Fine downy pubic hair was noted and the external genitalia were normal. Follow-up at the age of 101 months revealed that both breasts had decreased in size; the right breast had regressed to 4 by 5.5 cm and the left breast to 4 by 4 cm; the areola remained prepubertal and measured 1.5 cm in diameter. Fine downy pubic hair was present on the mons pubis and labia majora (no spread or increase in length and quantity of the hairs were noted). Her growth velocity, which had been tracking between the 90th and 95th percentile since the age of 3 years, continued to follow the same trend. Congenital adrenal hyperplasia was ruled out by a normal result from a synthetic ACTH (Cortrosyn) stimulation test.

*Patient 4*

Patient 4, born on July 20, 1985, was referred at 83 months of age for evaluation of premature progressive development of pubic hair. History revealed the use of hair preparations containing placental constituents or estrogen since the age of 48 months. The pubic hair, noted 1 year after initiation of the use of the hair products, continued to spread and increase in amount. Physical examination revealed the presence of acne on the forehead and pubic hair on the mons pubis and on the labia majora. Axillary hair was also noted. No breast tissue was palpable and nipple and areola were immature. She was advised to stop the use of hair products containing estrogen or placenta. After she discontinued the use of the hair preparations, the pubic hair growth decreased. She was followed up at 5–6 month intervals until the age of 94 months. During the follow-up, the nipple and areola remained immature and breast tissue was not palpable. About 1 cm long, sparse pubic hair, which had not increased since the previous examination, was noted on the mons pubis but not on the labia majora. Acanthosis nigricans was noted on the back of the neck and axillae. The importance of a balanced diet, exercise, and a healthy life style was emphasized on each follow-up visit. She was advised to continue to refrain from using the hormone-containing hair preparations.

The basal values of cortisol, 17-hydroxypregnenolone, 17-hydroxy progesterone, and testosterone were within the normal range, and synthetic ACTH (Cortrosyn)-stimulated responses for cortisol and 17-hydroxypregnenolone were normal. The serum dehydroepiandrosteronesulfate (DHEA-S) and the androstenedione levels, which were initially raised for age but normal for Tanner stage, were within normal limits for age and for the Tanner stage when measured at a later date. The serum ACTH value was also normal. Congenital adrenal hyperplasia as a cause of premature pubic hair development was unlikely. The serum gonadotropin and estradiol values are given in Table 1. The peak serum LH/FSH ratio of 0.086 found in this patient after the LHRH injection was not in the range noted in children with premature adrenarche or in those with precocious puberty,[24] although the estradiol responses to LHRH stimulation was in the range seen in pubertal girls (Table 1).

## Discussion

Among all four patients, secondary sexual characteristics de-

veloped following the use of a hormone- or placenta-containing hair product and regressed after they stopped the use of the hair products. No other cause for premature sexual development was found. Taken together these observations suggest a causal role for these hair products in premature development of sexual characteristics.

The early age of onset, accelerated bone age, and normal serum DHEA level argue against the diagnosis of idiopathic premature adrenarche in patient 1.[15] Similarly, accelerated linear growth and advanced bone age, noted in patient 2, are not features of premature thelarche.[15] The basal and LHRH-stimulated gonadotropin values, basal and Cortrosyn-stimulated adrenal hormone values, and appearance of the pelvic sonogram were normal in patients 3 and 4. Thus ovarian and adrenal pathology were ruled out. Intracranial lesions, although possible, are unlikely in view of regression of sexual characteristics. Additionally, the peak serum LH/FSH ratio (Table 1) is less in these patients than reported in children with precocious puberty.[24] In patient 4 the presence of acne, the absence of further progression of pubic hair during the 10-month follow-up,[15] and the elevated estradiol response to LHRH stimulation make the diagnosis of idiopathic premature adrenarche unlikely. Variable estrogen levels, from normal to raised, noted in our patients have been observed by others[4,5,8,10,13,25] and may be due to irregular use of the product (Table 4). However, biological effects of estrogens may persist long after the estrogen levels return to normal.[8]

During the period of follow-up, our patients showed regression but not complete resolution of breasts. Varying degrees of breast regression have been described after the withdrawal of the estrogen-containing products.[5,6,8,9,10,25] Gabrilove et al[25] observed that prolonged use of an estrogen-containing product caused fibrous changes in the breasts that slowed or stopped the regression of breasts in an adult male even though the use of hair product ceased. We noted a decrease in pubic hair in our patients 1 and 4. Cook et al[2] and Weber et al[5] observed a decrease in pubic hair after exposure to estrogen ceased.

Estrogens present in various dermatologic formulations can be absorbed from the intact human skin.[9-11,26-28] Topical skin and hair products containing even small amounts of estrogen may cause precocious puberty in infants and children.[11]

The phenomenon of pubic hair development, usually an androgenic effect, after exposure to estrogen-containing hair products observed by other authors[3,4,8-10] and by us remains unexplained. Two of our patients (1 and 4) showed development of pubic hair only and these patients had used a placenta-containing preparation. Placenta contains progesterone, which has some androgenic properties and may be related to pubic hair development.

The hair products used by our patients were: (1) used for cosmetic rather than for medicinal purposes, (2) available freely over the counter, and (3) labeled with the words "hormone," "placenta," or "estrogen."

All of our patients were African-American girls. Greater use of hormone- or placenta-containing hair preparations by many African-Americans (approximately 64%) as compared with whites (approximately 7%) has been reported.[29] The greater use of these hair products by African-Americans may explain the predominance of early sexual development among African-Americans reported here and elsewhere.[14]

## Conclusion

A history regarding the use of an estrogen- or placenta-containing hair preparation should be sought in all children presenting with early sexual development. Our experience as well as that of others[6,14] suggest that initial attempts to obtain an affirmative response regarding the use of hormone- or placenta-containing products may fail, and repeated questioning and reading the labels of the hair products used for ingredients may be necessary. Some cases of early sexual development may be due to the use of hormone- or placenta-containing hair products, and a follow-up of such patients is recommended to observe whether sexual characteristics have regressed after they have stopped the use of such hair products. Even after stopping the use of an estrogen- or placenta-containing preparation, the sexual development may persist up to 36 months.[8]

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**Table 4**

**SEXUAL CHARACTERISTICS AND HORMONE VALUES IN CHILDREN EXPOSED TO ESTROGEN: DATA FROM THE LITERATURE**

| Reference Number | Patients No./Sex | Patients Age Range (Months) | Patients Clinical Features | Endocrine Studies |
|---|---|---|---|---|
| 2 | 2 F | 51–84 | B, VB | FSH, and urinary 11-oxysteroids &17-ketosteroids were not increased in the younger girl. Urinary gonadotropin assay was positive in the older girl |
| 3 | 4 M<br>1 F | 60–120<br>84 | B, PH<br>B, PH | Not reported |
| 4 | 1 F | 39 | B, PH | Urinary estrogen was well below adult lower limit of normal, and near the minimum sensitivity of the method |
| 5 | 3 M<br>4 F | 20–101<br>20–103 | B, PH‡ | Urinary 17-ketosteroids, measured only in two girls (42 & 103 months old) and two boys (20 & 46 months old), were normal. Urinary estrogen, raised in two boys (20 & 46 months old) & one girl (42 months old), was normal in all others. Urinary FSH was normal in the 42-month-old girl |
| 6 | 1 M<br>1 F | 44<br>23 | B, PH<br>B, PH | Not reported |
| 7 | 1 M | 48 | B, PH | Gonadotropins were normal and urinary 17-ketosteroids were raised |
| 8 | 1 M<br>1 F | 78<br>120 | B<br>B | Basal and LHRH stimulated serum gonadotropins were normal in both patients. Total serum estrogen was normal in the girl but raised in the boy |
| 9 | 6 M<br>1 F | 4–8<br>24 | B, PH<br>B, PH | Urinary gonadotropins and pregnenatriol were normal in all |
| 10 | 1 F | 8 | B, PH | Serum LH, FSH, estradiol, and 17-hydroxyprogesterone were normal |
| 12 | 1 F | 60 | B, VB | Urinary 17-ketosteroids were normal |
| 13 | 1 M | 60 | B | Serum estrogen was raised; urinary 17-ketosteroids and 17-hydroxysteroids were normal |
| 25 | 1 M | Adult | B | Urinary gonadotropin was 5 mouse uterine units, serum estrogen was raised and the urinary 17-ketosteroids were normal |

M=Male, F=Female, B=Breasts present, PH=Pubic hair present, VB=Vaginal bleeding, LH=luteinizing hormone, FSH=follicle stimulating hormone, LHRH=luteinizing hormone-releasing hormone.

‡=Breasts were present in all patients and pubic hair was present in three patients—one girl and two boys. Vaginal discharge was noted in three and vaginal bleeding in one patient.

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