

EXPERIENCE & REASON

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"In Medicine one must pay attention not to plausible theorizing but to experience and reason together. . . . I agree that theorizing is to be approved, provided that it is based on facts, and systematically makes its deductions from what is observed. . . . But conclusions drawn from unaided reason can hardly be serviceable; only those drawn from observed fact."

—Hippocrates, *Precepts*

Pubic Hair of Infancy: Endocrinopathy or Enigma?

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ABSTRACT

Pubic hair of infancy is a rare condition that has not been well-characterized. A retrospective chart review of infants <12 months of age who presented to our pediatric endocrine clinics with isolated pubic hair over the last 5 years was performed. Eleven patients were identified (6 male and 5 female). The average age at diagnosis was 8.3 ± 2.0 months. The majority of patients (73%) had pubic hair in an atypical location. Growth pattern, laboratory evaluation, and bone-age radiographs were unremarkable for all the infants. Of the infants that returned for follow-up, pubic hair resolved by the age of 11.0 ± 1.5 months. From our experience and review of the literature, we suggest that isolated pubic hair of infancy is a benign entity. However, long-term follow-up needs to be done to determine if pubic hair of infancy is an atypical variant of premature adrenarche, which may place these patients at risk for later adult disease.

PREMATURE ADRENARCHE REFERS to the development of isolated pubic hair in children before the age of 8 years in girls and 9 years in boys.¹ Premature adrenarche appears more frequently in girls² and most commonly occurs between the ages of 6 and 8 years. In contrast, children who present with isolated pubic hair during the first 12 months of life are thought to be rare. Furthermore, pubic hair of infancy has not been well-described in the literature.³

Here we report our experience with infants presenting with isolated pubic hair. We sought to determine how often a pathologic process was identified and whether there were any consistent clinical or environmental features that might provide a clue as to the etiology of this unusual finding. We also review the medical literature of infants who present with isolated pubic hair. To our knowledge, this study represents the largest case series of children presenting with pubic hair of infancy that has been reported to date.

CASE SERIES

After institutional review board approval, we identified medical charts of children who presented to our pediatric

endocrine clinics from 1999 to 2004 with pubic hair of infancy. Inclusion criteria for the study were infants <12 months of age with no other signs of premature sexual development. Patients with vellus hair were excluded. Eleven eligible infants (6 male and 5 female) were identified. Patient characteristics and clinical course are shown in Table 1. The ethnic background of these children included 5 black (45%), 3 white (27%), 1 Hispanic (9%), and 2 others (18%). The majority of infants were born term (82%). The mean \pm SD score of length for age was 0.0 ± 1.1 , and the mean \pm SD score of weight for age was 0.1 ± 0.9 . The weight-for-length ratio was >95th percentile in 1 patient (patient 5). One

Key Words: pubic hair, infancy, premature adrenarche, precocious puberty

Abbreviations: 17-OHP, 17-hydroxyprogesterone; DHEA, dehydroepiandrosterone; β -hCG, β -human chorionic gonadotropin

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TABLE 1 Clinical Characteristics of Infants Presenting With Isolated Pubic Hair

Patient	Gender	Birth Weight, g	Maternal Age at Time of Birth, y	Maternal Age at Menses, y	Age at Diagnosis, mo	Age When Hair Resolved, mo	Location of Pubic Hair
1	M	2864	26	NA	7	NA	Scrotum
2	M	3227	26	12.5	5.5	9.5	Scrotum
3	M	4272	28	14	8.5	10	Scrotum
4	M	2727	34	14	6.5	NA	Scrotum
5	M	3551	34	NA	9.5	12.5	Scrotum
6	M	2386 ^a	26	11	6	NA	Scrotum
7	F	3409	18	14	10	NA	Labia
8	F	682 ^a	23	12	10	12	Labia
9	F	3750	22	15	11.5	NA	Labia
10	F	3438	26	NA	7	NA	Mons
11	F	3778	15	NA	10	NA	Mons
Mean (\pm SD)	6 M, 5 F	3099 (\pm 961)	25.3 (\pm 5.8)	13.2 (\pm 1.4)	8.3 (\pm 2.0)	11.0 (\pm 1.5)	

NA indicates not available.

^a Born preterm at 35 (patient 6) and 29 (patient 8) weeks' gestation.

mother noted development of mild hirsutism during pregnancy; however, this resolved before delivery.

The average age when pubic hair was first noted by a caregiver was 5.3 ± 2.5 months (range: 2–9 months). The average age when infants were seen in our clinic and diagnosed with pubic hair of infancy was 8.3 ± 2.0 months (range: 5.5–11.5 months). Family history was negative for all patients for early pubic hair development in infancy or childhood. There was no history of exposure to exogenous hormones in any of the patients. None of the infants were on soy formula. The majority of patients (64%) were on no medications and had no medical problems. Those infants who had significant medical problems included reactive airway disease, multiple ear infections, hemoglobinopathy (sickle cell disease), and failure to thrive.

No other signs of virilization or secondary sexual development were noted in any of the infants, specifically no penile or clitoral enlargement or growth acceleration. In all of the boys, the pubic hair was located only on the scrotum. An example of isolated pubic hair in one of the patients is shown in Fig 1. In girls, pubic hair was present on the labia majora in 3 infants and confined to the mons in 2 infants. Bone-age radiographs were obtained for the majority (73%) of patients, and all were equal to the chronologic age. A combination of laboratory tests, including obtaining 17-hydroxyprogesterone (17-OHP), testosterone, estradiol, dehydroepiandrosterone (DHEA)-sulfate, DHEA, androstenedione, and β -human chorionic gonadotropin (β -hCG) levels, were performed in 91% of the patients. Table 2 summarizes the laboratory evaluation that was performed in each infant at diagnosis. All laboratory tests were normal for age except in 2 patients who had significantly elevated 17-OHP levels of 294 ng/dL (patient 6) and 415 ng/dL (patient 8). Patient 6 also had a mildly elevated DHEA level, and patient 8 had a minimally elevated androstenedione level, which both correspond with the elevated 17-OHP levels. These results were not felt to be significant, because the assays

**FIGURE 1**

Example of an infant boy (patient 6) who presented with isolated pubic hair confined to the scrotum.

were performed in a general rather than specialty endocrine laboratory, and the rest of the medical evaluation was entirely normal. It is notable that both of the patients with elevated 17-OHP levels were born preterm, because preterm infants often have falsely elevated 17-OHP levels.⁴

Of the 11 patients, 36% returned for follow-up. All infants who returned were seen by the same pediatric endocrinologist at the initial and all subsequent visits. Among these infants, growth velocity was normal for age at 17.9 ± 1.9 cm/year. From the time it had first been noted in the clinic, pubic hair resolved at an average age of 11.0 ± 1.5 months (range: 10–12.5 months) in all cases.

DISCUSSION

Here we describe 11 infants (6 male and 5 female) who presented to our pediatric endocrine clinics with isolated pubic hair of infancy. We were unable to identify any

TABLE 2 Laboratory Studies for Each Infant at the Time of Initial Evaluation and Diagnosis of Isolated Pubic Hair of Infancy

Patient	Age at Time of Evaluation, mo	17-OHP, ng/dL	Testosterone, ng/dL	DHEA-S, μ g/dL	DHEA, ng/dL	Androstenedione, ng/dL	Other Laboratory Results
1	7	ND	ND	ND	ND	ND	ND
2	5.5	81	<10	<30	ND	ND	ND
3	8.5	52	<4	<30	ND	<10	β -hCG: <1.56 mIU/mL
4	6.5	101	14	<30	ND	10	ND
5	9.5	81	<4	<30	ND	30	ND
6	6	294	13	ND	3.3	30	ND
7	10	91	<4	ND	0.9	<10	ND
8	10	415	<4	<30	ND	80	ND
9	11.5	109	<10	ND	0.8	30	Estradiol: 1.25 ng/dL
10	7	89	<4	<30	ND	10	ND
11	10	67	<4	<30	ND	ND	ND

ND indicates not done. Studies were performed at various laboratories. Normal laboratory values (based on normative data by Lashansky et al²¹): 17-OHP = 11–173 ng/dL (males) and 13–106 ng/dL (females); testosterone = 0.6–500 ng/dL (males) and 0.3–8 ng/dL (females); DHEA-sulfate (DHEA-S) = 2–38 μ g/dL (males) and 4–111 μ g/dL (females); DHEA = 0.3–2.4 ng/mL (males) and 0.3–5.8 ng/mL (females); and androstenedione = 6–54 ng/dL (males) and 12–78 ng/dL (females); estradiol < 1.5 ng/dL; β -hCG < 1.56 mIU/mL.

pathologic process or specific etiology in any of the cases. All of our patients had reassuring growth records, laboratory evaluations, bone-age radiographs, and physical examinations.

Previous reports of isolated pubic hair of infancy^{3,5–10} are summarized in Table 3. As seen in our patients, these studies indicate that the pubic hair in boys most commonly resolves or decreases over the first few years of life,^{6,7,9,10} which suggests that it may be caused by a transient, undefined phenomenon. The etiology of isolated pubic hair during infancy remains unknown, although some reports have suggested a heightened cellular response¹⁰ or an increased sensitivity⁸ of hair follicles to androgens. Others have proposed that the minipuberty of infancy in boys may account for pubic hair development corresponding to the early physiologic rise in testosterone.^{6,9} Although this explanation would seem logical for pubic hair development in boys, this

theory does not account for the development of pubic hair in girls, who lack an androgen surge and were represented equally in our study sample. However, a number of reports have noted pubic hair development with or without breast enlargement in young children and infants after exposure to estrogens only.^{11–15} Therefore, it may be possible that the estrogen surge experienced by girls during the putative minipuberty of infancy¹⁶ could account for pubic hair development. Infant hair follicles may be responsive to small amounts of estradiol that are not detected by laboratory assays. Researchers have noted extensive individual variability in infant female hormone concentrations, with many values falling below the threshold of detection for various assays.¹⁷ Similar to patient 8, pubic hair regression in young girls has been reported previously,^{11,12} although these cases occurred after a known exposure to estrogen was removed. Despite this intriguing hypothesis, sex-

TABLE 3 Review of Reports in the Medical Literature of Infants Presenting With Isolated Pubic Hair

Authors (Year)	No. of Patients	Gender	Location of Pubic Hair	Outcome
Silverman et al ⁵ (1952)	2	2 girls	Labia (100%); pubic (50%)	Followed for 6–12 mo; outcome not reported.
Diamond et al ⁶ (1989)	6	6 boys	Scrotum (100%)	All spontaneously resolved after 4–12 mo or by 15 mo of age
Rotenstein et al ⁷ (1990)	1	1 boy	Scrotum (100%)	Outcome not reported
Adams et al ⁸ (1992)	3	3 girls	Labial (100%)	All had slow progression of pubic hair with no other signs of virilization over 12–30 mo of observation
Slyper and Esterly ⁹ (1993)	2	2 boys	Scrotum (100%)	Growth and development were normal in both; scrotal hairs diminished and became less prominent after 10 and 19 mo, respectively
Francis and Ruvalcaba ¹⁰ (1993)	1	1 boy	Scrotum (100%)	Spontaneously resolved after 8 mo or by 11 mo of age
Kaplowitz ³ (2004)	8	3 boys, 5 girls	Not specified (“genital area”)	Outcome not reported
Nebesio and Eugster (this article) (2005)	11	6 boys, 5 girls	Scrotum (100%); labia (60%); Mons (40%)	Of the 4 with follow-up, pubic hair resolved by 12 mo of age; pubic hair resolved after 5–10 mo from the time it was first noted by a caregiver (or a mean of 2.6 \pm 1.1 month from diagnosis)

steroid levels were prepubertal or unmeasurable in all patients at the time of initial evaluation.

As first described by Marshall and Tanner, normal pubic hair development in boys begins at the base of the penis¹⁸ and in girls along the medial surface of the labia.¹⁹ It is interesting to note that 73% of the patients in our series had an atypical location of pubic hair, specifically on the scrotum in boys (100%) and on the mons in girls (40%). The explanation for this finding is unclear. Possible reasons, although not proven, include increased local 5- α -reductase activity or a different distribution of androgen receptors in infancy.

The differential diagnosis of pubic hair in prepubertal children includes premature adrenarche and pathologic hyperandrogenism. Although considered a variation of normal development, premature adrenarche has now been established as a frequent forerunner of adult diseases¹ including polycystic ovarian syndrome,²⁰ hyperinsulinism, dyslipidemia, and early cardiovascular disease.²¹ If pubic hair of infancy represents an extreme form of premature adrenarche, these patients may be at higher risk for future adult diseases, which would warrant close monitoring and long-term follow-up. Hyperandrogenism in infants may be caused by precocious puberty,²² classic or nonclassic congenital adrenal hyperplasia,⁴ virilizing tumors,²³ or exogenous exposure.^{11,14,15} However, additional abnormalities including clitoromegaly, increased penile length, advanced bone age, and growth acceleration are invariably present in these pathologic conditions.

One weakness of this study is that it is a retrospective chart review and all patients were referred to the subspecialty clinic. Therefore, the true incidence of early pubic hair development in infants may be even higher. Another flaw of this study is that we have incomplete follow-up. However, it is likely that infants lost to follow-up had a similar clinical course to those who returned to our clinic, because our institution was the only tertiary pediatric referral center in the state at the time that these patients were seen in our clinics.

CONCLUSIONS

Isolated pubic hair of infancy, although considered abnormal, seems to be a benign entity. Prospective long-term studies are needed to determine if children with a history of pubic hair of infancy develop metabolic derangements later on in life.

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