

Androgenetic alopecia in the paediatric population: a retrospective review of 57 patients

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Summary

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Background Hair loss is an unwelcome event at any age, but it can be particularly distressing for adolescents and their families. While androgenetic alopecia (AGA) is the most common form of hair loss in adults, little is known about its prevalence, clinical features and response to treatments in the paediatric population.

Objectives To better characterize the causes of alopecia in a paediatric population.

Methods We performed a retrospective chart review to identify all patients with hair loss seen in an academic paediatric dermatology practice at New York University over a 12-year period to better characterize the causes of alopecia in this population. We review the clinical and histological features, natural progression and associated laboratory abnormalities of AGA in 57 paediatric patients.

Results AGA was identified as the most frequent cause of hair loss in adolescents and the second most common diagnosis overall. The male to female ratio was 2 : 1 and the average age at initial presentation with AGA was 14.8 years. Adolescent girls had diffuse thinning or thinning at the crown, and boys frequently presented with female pattern hair loss. When biopsies were performed, perifollicular inflammation was a common finding. A family history of AGA was reported in 83% of patients. Laboratory evaluation for androgens revealed polycystic ovarian syndrome in three girls and late-onset congenital adrenal hyperplasia in one boy.

Conclusions AGA is the most common form of hair loss in adolescents, and can be the presenting sign of an underlying endocrine disorder. An accurate and timely diagnosis is essential for appropriate medical and psychosocial intervention when warranted.

Androgenetic alopecia (AGA), which encompasses male-pattern hair loss (MPHL) and female-pattern hair loss (FPHL), is a common form of hair loss¹ characterized by progressive thinning of scalp hair in a typical pattern in ageing men and women. In children and adolescents with a genetic predisposition, the first signs of AGA can appear with rising androgens at puberty,^{2,3} and have been observed as early as 6 years of age.⁴

Although AGA poses no direct consequences to physical health, it can lead to significant psychosocial morbidity, particularly if noticeable during adolescence. While numerous studies document the negative effects of AGA on self-image, social perceptions and emotional wellbeing in adults,^{5,6} the psychosocial impact of AGA in adolescents has not been evaluated. Importantly, young age (< 26 years old), earlier onset of hair loss, and a strong reliance on physical appearance as a source of self-esteem have been identified as risk factors for hair loss-related psychological morbidity in adults with AGA.⁷ Satisfaction with physical appearance is important to adoles-

cents,⁸ and discrepancies from an ideal self-image such as hair thinning often lead to feelings of anger, anxiety and embarrassment.⁹ Consequently, adolescents with AGA may be at risk for significant impairment in psychosocial functioning.

Here we present the clinical features, relevant medical and family histories, histopathology, differential diagnoses, laboratory evaluation and treatment of 57 paediatric patients with AGA. Our series corroborates previous reports¹⁰ that AGA in adolescents is not uncommon. It is important to recognize and address this potentially distressing condition with children and their families.

Methods

A retrospective chart review was performed with Institutional Review Board approval to identify all patients younger than 19 years of age with hair loss referred to a paediatric dermatology practice at New York University from 1 July 1997 to

31 December 2008. The patients were identified using ICD-9 billing codes for alopecia (including alopecia, alopecia NOS, alopecia areata, totalis and universalis, telogen effluvium, trichotillomania, and hair disorder NOS), excluding cases of tinea capitis. Epidemiological data for all forms of alopecia were ascertained for the 438 patients identified, including sex, age of onset, age at evaluation and clinical diagnosis. Subjects were then grouped by diagnosis and age (12–19 years or younger than 12 years). For the 57 patients with AGA (52 adolescents and five patients younger than 12 years), additional information on disease course, family and medical history, medications, treatment and response to treatments was recorded and analysed.

Results

Alopecia areata (AA) was the most frequent diagnosis encountered in the 438 paediatric patients evaluated for hair loss in a paediatric dermatology practice (Fig. 1). The second most common diagnosis was AGA. Of the 438 patients with alopecia, 123 (28%) were adolescents (12–19 years old) at the time of evaluation. AGA was the most prevalent diagnosis in adolescents, occurring in 52 of 123 (42%) (Fig. 2), and is discussed below.

Age of onset

An additional five children (three girls, two boys), 11 years or younger at initial visit, had AGA. Thus, there were a total of 57 patients, 38 boys and 19 girls (M : F, 2 : 1), diagnosed with AGA (Table 1).

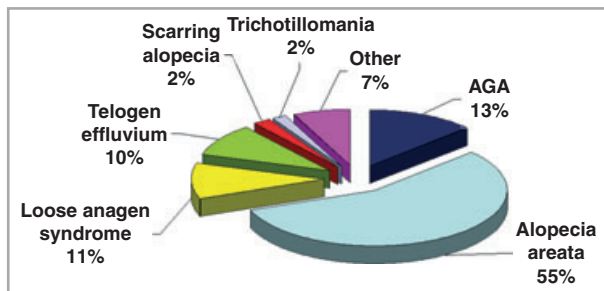


Fig 1. Spectrum of diagnoses in all paediatric patients ($n = 438$) evaluated for hair loss over a 12-year period. AGA, androgenetic alopecia.

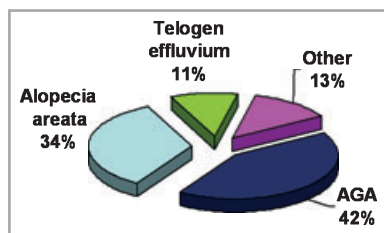


Fig 2. Spectrum of diagnoses in adolescent patients ($n = 123$) aged 12–19 years with alopecia. AGA, androgenetic alopecia.

Family history

A family history was recorded in 35 of 57 patients with AGA (22 boys, 13 girls). Overall, 29 of the 35 (83%) reported a family history of patterned hair loss in either a first- and/or second-degree relative. Of the 13 girls whose family history was recorded, two reported no family history, two girls reported patterned hair loss in both their mother and father, three girls had a father and four girls had a mother with AGA and two girls had paternal second-degree relatives with hair loss. Of the 22 boys, six denied a family history of hair loss, four boys reported patterned hair loss in both their mother and father, eight boys had a father and three boys had a mother with AGA and one boy reported his maternal grandfather had AGA.

Past medical history

Three patients (two boys, one girl) had a long-standing history of hypothyroidism; all were treated and euthyroid at evaluation. One girl had a history of iron-deficiency anaemia and another had fibromyalgia. Two male adolescents were being treated for anxiety and obsessive-compulsive disease. An additional two patients (one girl, one boy) had familial dysautonomia.

Dermatological history

Acne was the most common concurrent dermatological condition, noted in six of 19 (32%) girls and 19 of 38 (50%) boys. Seven of 19 girls (37%) and six of 38 boys (16%) were also found to have seborrhoeic dermatitis of the scalp.

Clinical features

In boys, the most common presentation was thinning of the vertex with varying degrees of bitemporal thinning (Fig. 3a). One-third of boys (13 of 38) presented with diffuse thinning, or prominent thinning at the crown, more consistent with a 'female pattern' (Fig. 4). In girls, the characteristic clinical picture was diffuse thinning of scalp hair or hair loss most pronounced at the crown (Fig. 3b).

Differential diagnosis

Telogen effluvium (TE) and diffuse AA were the most frequent differential diagnoses considered at presentation. In seven girls, AGA and TE were considered as equally likely diagnoses at initial visit. One girl continued to have loss with progressive widening of the central part consistent with AGA. In two of the seven girls, TE was more likely given a history of a crash diet in one and reported recovery of hair loss with weight gain in another. In the remaining four patients, a trichogram revealed changes that could be seen in both TE and AGA, and progression of hair loss was unclear; definitive diagnosis remained uncertain. These four patients were not included in analyses of females with AGA.

	Male n = 38	Female n = 19	Overall n = 57
Average age of reported onset of hair loss, years (range)	13.9 (9–19)	12.6 (8–16)	13.5 (8–19)
Average age at initial visit for hair loss, years	14.9	14.4	14.8
Average disease duration prior to presentation, years	0.95	1.8	1.2
Onset \leq 11 years, n	5	5	10
Evaluated at \leq 11 years	2	3	5
Premenarchal, n (%)		6 (32)	

Table 1 Total of 57 paediatric patients, including five patients 11 years or younger at initial evaluation, with androgenetic alopecia; the age at initial presentation was similar for boys and girls

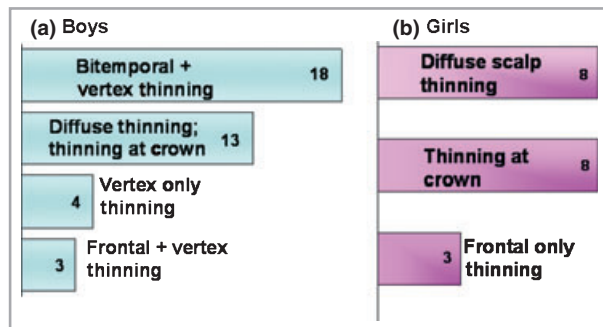


Fig 3. Distribution of clinical patterns of androgenetic alopecia in 38 male (a) and 19 female adolescents (b). One-third of males had diffuse thinning with prominent thinning at the crown, more consistent with a female pattern.



Fig 4. A 15-year-old boy with diffuse hair thinning and preservation of the frontal hair line more consistent with a female pattern.

In two of the 38 males, the diagnosis of TE was considered in addition to AGA. Both boys continued to have patterned hair loss consistent with AGA. In two boys (9 and 12 years)

with female-pattern AGA, diffuse AA was a diagnostic possibility at initial evaluation; in both, a scalp biopsy demonstrated AGA.

Histopathology

Scalp biopsies were performed in five female and nine male patients. All biopsies showed typical features of AGA with increased vellus and telogen hairs, and connective tissue streamers (or follicular stela) below small vellus follicles. Eight of the 14 biopsies (57%), also had varying degrees of a peri-infundibular lymphocytic inflammatory infiltrate and fibrosis (Fig. 5). In four of these, early lichen planopilaris was included in the histological differential diagnosis due to the degree of inflammation and fibrosis.

Laboratory evaluation

Results of laboratory evaluation of thyroid function, iron deficiency and androgens are displayed in Table 2. Testing for hyperandrogenaemia included free and total testosterone, dehydroepiandrosterone sulphate (DHEAS) and androstenedione. Polycystic ovarian syndrome (PCOS) was diagnosed in three girls prior to presentation to our clinic. PCOS became the likely diagnosis in an additional six girls after detection of AGA and other clinical features of hyperandrogenism (acne, hirsutism and/or seborrhoea) and/or hyperandrogenaemia (Table 3).

Treatment

Minoxidil solution was recommended to 16 girls with AGA. Follow-up at least 6 months later is available for six girls, 11–18 years old. Four of six reported stabilization in hair loss with continued use at 1 year. One girl reported increased facial hair with the 5% solution, which resolved when she switched to the 2% solution. Two girls discontinued usage, one after 5 months for lack of efficacy and one after 3 weeks because of headaches and nausea.

Minoxidil 5% solution or foam was recommended to 36 of the 38 boys with AGA. Seven boys, aged 14–17 years, were treated with both minoxidil 5% and finasteride (1 mg daily)

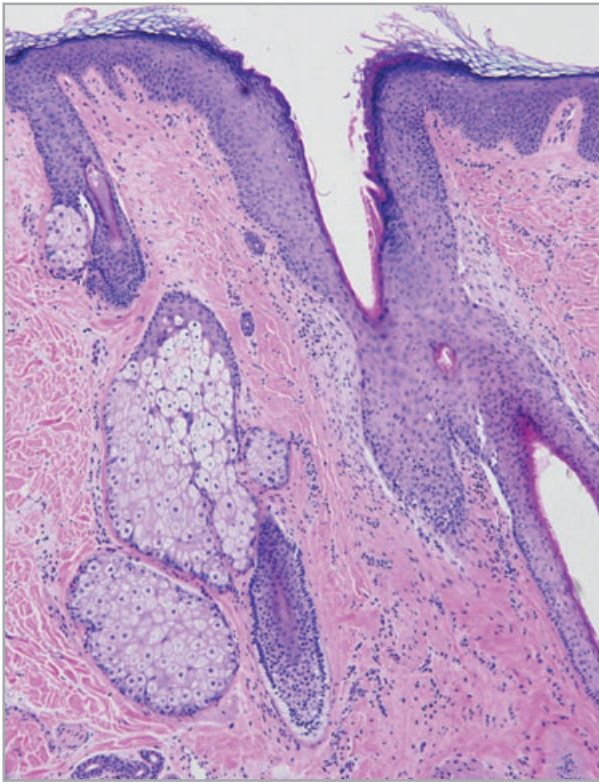


Fig 5. High-power view of a scalp biopsy in a 14-year-old boy with androgenetic alopecia. Vellus hairs and a peri-infundibular lymphocytic inflammatory infiltrate are evident.

Table 2 Laboratory findings in patients with androgenetic alopecia

Laboratory test	Boys (%)	Girls (%)
Abnormal thyroid function ^a	0/25 (0)	0/19 (0)
Antithyroid antibodies	1/7 (14)	1/5 (20)
Low serum iron	0/21 (0)	3/14 (21)
Iron-deficiency anaemia	0/21 (0)	0/14 (0)
Hyperandrogenaemia	2/14 ^b (14)	6/16 ^c (38)
Low testosterone	3/14 (21)	0/16 (0)

^aIncludes three patients with known hypothyroidism on thyroid hormone replacement. ^bBoth had elevated dehydroepiandrosterone sulphate (DHEAS); late-onset congenital adrenal hyperplasia was diagnosed in one boy (hair loss began at age 11). ^cThree girls with elevated androgens had pre-existing polycystic ovarian syndrome (PCOS) and in an additional three girls, the detection of elevated serum androgens made the diagnosis of PCOS likely (see Table 3).

and two were treated with finasteride alone (18 and 19 years old). Follow-up at least 6 months later (range 6 months to 8 years) was available for 23 of the 29 boys treated with minoxidil 5% alone and 18 of the 23 reported stabilization in hair loss and/or fuller hair. Two never started treatment and two discontinued usage, one at 4 months for lack of efficacy and one after 3 weeks due to development of an acneiform scalp eruption. One patient reported progression of hair loss

at 2-year follow-up despite using minoxidil. Follow-up is available for the six of nine patients treated with finasteride; all six boys (four also using minoxidil 5%) reported increased hair density with no progression in hair loss. One experienced decreased sexual function that resolved despite continued finasteride use.

Discussion

While AGA is the most common and widely studied form of hair loss in adults, little is known about its prevalence, characteristics and natural history in the paediatric population. Limited data from the U.S. suggest that 14.1–15.5% of male adolescents, aged 15–17 years, have early evidence of male-patterned baldness.¹⁰ In our series, AGA was the second most common diagnosis overall and the most common among adolescents with hair loss as the presenting complaint. Onset of adolescence has historically been linked to the start of puberty. Due to the retrospective nature of this review, we were unable to determine pubertal timing in all patients and thus we defined an adolescent by age older than 12 years because it represents the average age of testicular enlargement in boys and of menarche in girls,¹¹ both markers of early puberty. In addition, 12 years was the cut-off age used in a previous series of 43 adolescents (12–18 years) with AGA.³ In this report, Kim *et al.*³ also noted a male predominance, but reported a slightly higher age of onset (14.8 years) and an older age at initial presentation (16.8 years). In their series, girls, rather than boys, as in our series, had a shorter delay to diagnosis.¹²

The exact pathogenesis of AGA is unclear and several distinct mechanisms may contribute. In men and in a subset of women, AGA is the result of the action of dihydrotestosterone, the 5- α reduced metabolite of testosterone, on a genetically susceptible hair follicle leading to progressive follicular miniaturization.¹² In women with AGA, the role of androgens is less clear and most experts prefer the term FPHL.¹³ There is undoubtedly a subset of women in whom hair loss is associated with hyperandrogenism, such as those with PCOS¹⁴ or androgen-secreting tumours. The observation of AGA in children with prepubertal testosterone levels and in women with normal hormonal profiles has led some to believe that FPHL in children, and in some women, may not be androgen-dependent,¹⁵ or that adrenal androgens may have a direct role.⁴

Although the majority of boys in our series had age-appropriate levels of androgens, adrenal androgens may have played a role in three who presented with female-patterned AGA. All three had prepubertal levels of testosterone; two also had elevated DHEAS. Further testing in one 11-year-old boy with significant gynecomastia revealed late-onset congenital adrenal hyperplasia. Thus, early-onset AGA in boys, especially if in a female pattern, and/or aberrant signs of pubertal development should prompt a full endocrine evaluation.

Likewise, in an adolescent girl, AGA can be the first sign of an androgen excess disorder such as PCOS; therefore, full hormonal evaluation is worthwhile. The most recent diagnostic

Table 3 Nine girls with androgenetic alopecia (AGA) and polycystic ovarian syndrome

Patient	Age (years)	Hyperandrogenism				Biochemical	Oligo- or anovulation	Polycystic ovaries
		AGA	Hirs	Acne	Seb			
1 ^a	12	+	+	+	+	↑ Free and total testosterone	Oligo	+
2 ^a	15	+		+		↑ Free testosterone	Oligo	NA
3 ^a	16	+	+	+		↑ Total testosterone	Oligo	NA
4	11	+		+		↑ Free and total testosterone	An (premenarche)	NA
5	15	+		+		↑ Free and total testosterone, and ↑ DHEAS	Oligo	–
6	15	+			+	↑ Free and total testosterone	Oligo	NA
7	15	+	+			N/A	An (premenarche)	NA
8	15	+				None	Oligo	NA
9	18	+	+	+		None	Oligo	NA

^aPolycystic ovarian syndrome was a known diagnosis in three girls. It was a likely diagnosis in six additional girls supported by detection of other clinical features of hyperandrogenism [acne, hirsutism (Hirs) ± seborrhoea (Seb)] and/or hyperandrogenaemia, oligo- or anovulation (An) and/or polycystic ovaries.
DHEAS, dehydroepiandrosterone sulphate; NA, not assessed.

criteria for PCOS require at least two of the following to be present: (i) oligo- and/or anovulation; (ii) clinical and/or biochemical evidence of hyperandrogenism; and (iii) polycystic ovaries.¹⁶ Thus, clinical evidence of hyperandrogenism such as AGA, seborrhoea, acne or hirsutism can help make the diagnosis of PCOS. Although a recent review found hirsutism to be the strongest clinical predictor of PCOS, AGA alone can be seen in women with PCOS.¹⁷ Overall, 37.5% of adolescent girls with AGA in our series had elevated androgen levels and 47% had an additional clinical feature of hyperandrogenism (seborrhoea, hirsutism or acne). Detection of AGA and other signs of androgen excess and the resultant laboratory workup identified six additional patients with PCOS (Table 3). Similar to a previous report of patients with PCOS, the clinical pattern and severity of AGA in our patients varied.¹⁸ PCOS has serious health implications including an increased risk for infertility, endometrial carcinoma, obesity, type 2 diabetes, dyslipidaemia, hypertension and possibly cardiovascular disease.¹⁷ Early diagnosis is essential for proper treatment and surveillance.

It is accepted that AGA is a hereditary condition. Individuals without a family history of AGA are at low risk for baldness while men with a history of hair loss in either parent are 2.5 times more likely to have hair loss.¹⁹ Children and adolescents with AGA have a strong genetic predisposition, as observed in our series and previous reports.^{3,4} Younger children are more likely to have a family history.⁴ AGA is likely a polygenic disorder and mutations in several candidate genes have been identified;^{20,21} however, these are not sufficient to explain the disease in all men.¹⁸

As in adults, the diagnosis of AGA in children and adolescents is made by recognizing the pattern and progression of hair loss in the context of the family history. In women, the most common patterns of hair loss are centrifugal loss at the crown with preservation of the frontal hair line as graded by Ludwig²² and the frontal accentuation or 'Christmas tree'

pattern as described by Olsen.²³ In our adolescent girls, a large proportion had diffuse thinning of scalp hair, in addition to the more characteristic adult female pattern described by Ludwig. Men typically experience progressive bitemporal recession and thinning in the frontal and vertex scalp as graded by the widely used Norwood and Hamilton scale.²⁴ While this pattern was seen in the majority of our adolescent boys, one-third presented with FPHL. Female pattern in adult men with AGA has been reported, although it is far less frequent than other presentations.^{25,26} The female pattern appears to be more common in male children and adolescents and was recorded in 20% of adolescent boys in one report,³ and in all boys in the Tosti *et al.*⁴ series.

Diffuse thinning in a young girl or thinning in a female pattern in a male adolescent can make the diagnosis of AGA challenging at first, but progression of hair loss and family history usually provide sufficient information to correctly identify AGA. The main mimics of AGA in an adolescent are acute TE and diffuse AA (Table 4). In our experience, a trichogram does not reliably distinguish AGA from other causes of hair loss. Recently, dermoscopy has been used to help distinguish the various forms of nonscarring alopecia (Table 4).

A scalp biopsy is rarely necessary to make the diagnosis of AGA, but it can be helpful if the clinical picture is atypical and for diagnostic confirmation for an anxious family. Horizontal sectioning is most helpful as it allows for determination of a terminal to vellus hair ratio (T : V). A T : V ratio < 4 : 1 is considered diagnostic of AGA, while 7 : 1 is normal.²⁷ All 14 biopsies in our series had increased vellus hairs consistent with AGA. Eight of the 14 biopsies additionally had varying degrees of perifollicular inflammation and fibrosis in the upper hair follicle. Increasing evidence indicates that this is a common histological feature in AGA.^{28,29} Several large studies of male-pattern alopecia have documented variable peri-infundibular inflammation and fibrosis.^{30,31} In 1017 AGA

Table 4 Differential diagnoses of androgenetic alopecia in adolescents

	Androgenetic alopecia	Acute telogen effluvium	Diffuse alopecia areata
Clinical presentation	Gradual onset, patterned thinning (see Fig. 3a,b)	Abrupt onset of increased (can be excessive) hair shedding, involves entire scalp	Acute onset, marked shedding, widespread or may predominantly affect the top of the head and pigmented hairs
Triggering factor	No	Yes	Sometimes
Natural history	Slowly progressive	Self-limited, total recovery usually in 3–6 months	Rapidly progressive (months)
Family history	Usually positive	Negative	One of five patients has a positive family history ⁴⁷
Hair pull	Negative (usually)	Positive	Positive
Trichogram (hair mount of forcible hair pluck) ²	Normal anagen : telogen ratio, increased telogen hairs in early cases	Decreased anagen : telogen ratio; (> 20% telogen hairs)	Decreased anagen and slightly increased telogen hairs; increased dysplastic hairs ⁴⁸
Dermoscopy	Brown halo at follicular ostium (peripilar signs) and > 20% hair shaft diameter variability in hair size ^{32–49}	Numerous short-tip pointed regrowing hairs, no hair diameter variability ^{50,51}	Perifollicular yellow dots (95% of patients), black dots, tapering hairs (exclamation point hairs), broken hairs and short regrowing hairs ^{50,51}
Histology	Normal number of total follicles, increased percentage of miniaturized terminal and vellus follicles, numerous connective tissue streamers, reduced terminal to vellus hair ratio (< 4 : 1 is diagnostic)	Normal total and terminal follicles; increase in telogen hairs to > 20%, absence of inflammation and scarring, no vellus or miniaturized hairs	Normal number of total follicles, peribulbar mononuclear cell infiltrate, increased number of miniaturized follicles, increased telogen follicles at the active border

biopsies, mild follicular inflammation was seen in 45% of men and 38% of women and moderate to dense inflammation was seen in 40% of men and 16% of women.³² Some authors speculate that perifollicular inflammatory cells may play a role in the early pathogenesis of AGA in a genetically susceptible individual, or can be a consequence of androgen action on the pilosebaceous unit.²⁹ Separate but associated factors such as seborrhoeic dermatitis, production of porphyrins and ultraviolet radiation exposure have been implicated as possible causes of the inflammatory infiltrate.³³ Clinically, none of the patients with perifollicular inflammation developed scarring alopecia at follow-up 3–24 months after biopsy.

There is no approved therapy for AGA in the paediatric population. Topical minoxidil, a potassium channel opener and arterial vasodilator, which has proven safety and efficacy for AGA in adult men and women,^{34,35} has not been thoroughly studied in adolescents and data are limited to information from scientific meetings and case series. Topical minoxidil appears to be safe and effective in adolescents with AGA¹⁰ and was successful in our patients (aged 10–19 years). Nonetheless, considering several instances of cardiac side-effects in children using topical minoxidil,^{36,37} a more rigorous investigation of the pharmacokinetics, absorption and safety of topical minoxidil in the paediatric population is needed. Similarly, finasteride, a competitive inhibitor of 5- α -reductase that results in reduced levels of dihydrotestosterone, is approved by the U.S. Food and Drug Administration for AGA in men 18 years and older but is not well studied in

adolescents. Use of finasteride in men with AGA leads to increased target area hair counts,³⁸ longer and thicker hair³⁹ and sustained effect with continued use at 5 years.⁴⁰ Finasteride is generally safe and well tolerated but reversible sexual side-effects can be seen in 1.8% of men³³ and prostate-specific antigen levels are reduced.⁴¹ In our series, finasteride halted hair loss in six adolescent boys. In women, finasteride and other antiandrogens such as spironolactone and cyproterone acetate appear to be most useful if there is hyperandrogenism but data on their effect specifically on hair loss, and in adolescents, is limited.^{42–44} Spironolactone reversed hair loss in one 9-year-old girl with prepubescent AGA.⁴⁵ More prospective data evaluating the safety and efficacy of finasteride and other antiandrogens in adolescent hair loss are needed.

It is important to discuss expectations of treatments and natural progression of AGA with adolescents and their families. Both minoxidil and oral antiandrogens arrest progression of hair loss and this should be considered treatment success. Prolonged treatment is necessary to maintain results. In addition, camouflaging techniques, hair pieces⁴⁶ and hair restoration surgery should be discussed when appropriate.

AGA in adolescents is not uncommon. Children and adolescents with AGA are likely to report hair loss in at least one first- or second-degree family member. The clinical patterns of AGA in adolescents are more varied, and girls can have diffuse thinning while males often present with FPHL. Evaluation for hyperandrogenaemia is worthwhile in all girls and in boys with diffuse or FPHL and with aberrant pubertal development.

Accurately recognizing AGA in adolescents will help patients and their families understand the diagnosis and its natural progression, and allow for timely medical intervention for hair loss and any associated endocrine or psychosocial morbidity. The prevalence of AGA in adolescents reported here, and previously,^{3,4,10} draws attention to the need for further investigation into the pathogenesis, treatment and psychosocial impact of AGA in the paediatric population.

What's already known about this topic?

- Androgenetic alopecia (AGA) is the most common form of hair loss in adults and is clinically distinctive.
- Two studies have reported AGA in children and adolescents.
- In men, and some women, AGA is mediated by the action of androgens on a susceptible hair follicle.
- AGA is a hereditary condition, which is probably polygenic.
- Finasteride and topical minoxidil are safe and effective treatments for AGA in adults.

What does this study add?

- AGA is also common in adolescents.
- This is the largest series of paediatric patients with AGA to date, describing the clinical features, medical and family histories, histopathology, laboratory evaluation and treatment outcome in 57 patients.
- Evaluation for hyperandrogenaemia is worthwhile in adolescent girls with AGA.
- Topical minoxidil is well tolerated and can be effective at halting AGA progression in adolescents.
- Finasteride is effective for AGA in adolescent boys.

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