Clinical Course of Pediatric Androgenetic Alopecia

Lee, D [1] Eichenfield, D [2]

1. BA, BS University of California San Diego School of Medicine
2. Division of Pediatric and Adolescent Dermatology, Rady Children's Hospital, San Diego, California; Department of Dermatology, UC San Diego, La Jolla, California.

Background

Androgenetic alopecia (AGA) is a form of hair loss characterized by progressive thinning of scalp hair and loss of terminal scalp hair after puberty.[1] While AGA leans towards the adult population, the pediatric population may be affected. This has led to an under-recognized condition in which little is known about the natural history, risk factors, and treatment in pediatric patients.[2–5]

Objective

To better define the clinical characteristics and clinical course of pediatric androgenetic alopecia.

	Male	Female	Overall
Age of presentation	15.82 (11-18)	15.11 (9-18)	15.58
Age of onset of hair loss	14.52 (8-17)	13.18 (7-17)	14.02

Table 1. Total 52 patients stratified by gender. Age of onset for 5 male patients and 1 female patient were not reported or specified.

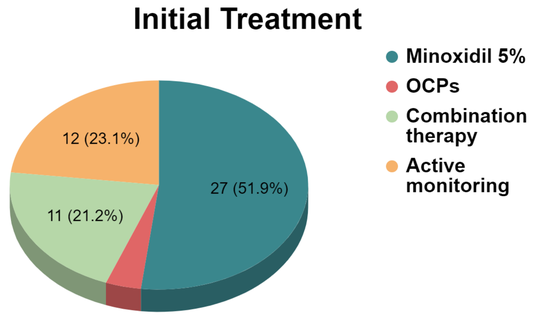

Figure 1. Initial Treatment. Minoxidil (n=27), OCPs (n=2), combination therapy (n=11) and active monitoring (n=12) was categorized. Combination therapy ranged from minoxidil 5%/ triamcinolone 0.1% ointment to minoxidil 5%/finasteride 1 mg once daily.

Methods

We conducted a retrospective study on AGA pediatric patients by evaluating the demographics, family history, treatment, adverse effects, and reported outcomes.

This study was approved by UC San Diego/Rady's Children Hospital Institutional Review Board. A retrospective chart review from 1/1/2009 - 1/1/2023 was performed to identify patients seen for AGA and the following was analyzed: biological sex, gender, race, age of presentation/onset, family history, treatment, side effects, and outcome.

Results

Fifty-two patients were seen for androgenetic alopecia, of which 17 were female and 35 were male based on biological sex. There was one transgender female patient. Average age of initial presentation and reported age of onset were 15.58 and 14.02, respectively (Table 1). Thirty-eight patients reported a family history of androgenetic alopecia, 13 had no family history, and 1 patient was adopted. Eleven patients were followed by endocrinologists. Dermatologists ordered various labs (e.g. DHEA-S, CBC) for 16 patients: 4 males to rule out secondary etiologies and 12 females for hyperandrogenism.

Initial treatment (Figure 1) included: minoxidil (n=27), OCPs (n=2), combination therapy (n=11) and active monitoring (n=12). Twenty-five had follow-up with dermatology, with an average of 2.44 follow-up visits. 12 patients reported an improved course, 3 with a worsened outcome, and 10 unchanged. Out of those with follow-up, 10 patients reported adverse effects (e.g. dizziness caused by OCPs).

Conclusion

Our study demonstrates AGA is prevalent among adolescents and children. A majority of patients had a family history of androgenetic hair loss. Females may present with signs of hyperandrogenism. A proportion was also seen by endocrinologists for related disorders. Prospective controlled studies will better define the relationship between AGA risk factors and recommended treatment.

References

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Rady Children's Hospital San Diego