ANDROGENETIC ALOPECIA

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Androgenetic Alopecia, also known as male or female pattern hair loss, is a disorder primarily determined by a genetic predisposition for hair follicles to have an excessive response to androgens. This excessive response leads to a progressive shrinking of hair leading to a transformation of terminal hairs to vellus hairs. In males, this hair loss presents primarily on the frontotemporal and vertex regions of the scalp while in women, it presents most commonly with loss in the central, frontal and parietal scalp, which may initially present with a widened frontal hairline. While there may be a psychosocial effect, the primary disorder of androgenetic alopecia is considered benign. However, in recent studies there is some evidence that suggests that those affected by androgenetic alopecia have an increased risk of cardiovascular disease, diabetes and prostate cancer.

Androgenetic alopecia is the most common form of alopecia in both males and females. The prevalence of the disorder increases with age, affecting nearly 50% of males and 25% of females by age 50. In less common instances, signs of androgenetic alopecia can begin as early as puberty. The cases are more prevalent and higher in severity in the Caucasian population followed by Asian and African populations. A son whose father is balding due to androgenetic alopecia has a five to six times increased relative risk of developing this condition.

It is believed that androgenetic alopecia has both a hormone (andro-) component and a hereditary (genetic) component to its development. Repeated activation of the androgen receptor on hair follicles leads to shortening of the anagen (growth) phase. This leads to progressive miniaturization of the follicle. Testosterone is able to bind and activate the androgen receptor on hair follicles, but its transformation from testosterone to dihydrotestosterone via the 5 alpha-reductase enzyme allows it to bind to and activate the receptor with a five times greater affinity. The hereditary component is considered polygenic (caused by several genes) in nature. Some of the involved genes include the androgen receptor gene on the X chromosome, the estrogen receptor-β gene and the aromatase gene that converts testosterone to estradiol. There are now genetic test kits on the market that look to predict the chances of developing androgenetic alopecia by analyzing some of these genes.

In most cases, the diagnosis of androgenetic alopecia is made clinically following a history of gradual hair loss beginning after puberty. The diagnosis can be aided by the use of dermoscopy. While the need for a biopsy is rare, in unusual or rapid onset cases, a biopsy may be indicated to rule out other causes of alopecia. In addition, patients may be evaluated with a CBC, iron studies and thyroid studies to further rule out other causes of alopecia.

Treatment varies for male and female patients. Male patients are generally treated with topical formulations of minoxidil (Rogaine) and/or oral finasteride. Minoxidil lengthens the anagen phase of hair growth by mechanisms that are poorly understood, while finasteride inhibits the 5α-reductase enzyme, reducing the amount of dihydrotestosterone which can bind to the follicle. In addition to these treatment options, surgical hair transplantation may also be viable. There are a wide array of other non-FDA approved treatment options including laser treatments, antiandrogen medications, prostaglandin analogues and more. However, some of these treatments have variable success rates and, in general, have not been studied as well as others.

Treatment of female patients usually includes topical minoxidil. In cases of hyperandrogenemia, oral antiandrogen medications can be considered, such as finasteride or spironolactone. It should be noted that women using either of these oral medications should also be instructed on proper methods of contraception as they can have deleterious effects on a developing fetus. Other treatment options, including surgical hair transplantation are similar to the treatment of male pattern baldness and, once again, have not been studied as extensively as others.