Correspondence

Analysis of androgenetic alopecia in Amerindian people (Mapuche) from southern Chile

Androgenetic alopecia (AGA) is a common cause of hair loss. International studies report racial differences in prevalence and clinical types of AGA in men and women.[1–6] Some authors predict that black and native Americans (Amerindian) have a lower prevalence and milder forms of AGA when compared to Caucasians.[7,8]

In Chile, the Amerindian population is made up of different ethnic groups; the largest one is the Mapuche community. Genetic studies evidence similar HLA profiles between Mapuche, other Amerindians, and Asians, although a small degree of admixture with Europeans is also admitted.[9] Our objective was to analyze frequency and clinical patterns of AGA in Chilean Mapuche.

The study group included adults with two or more surnames of Mapuche origin who attended clinics for general morbidity in Curarrehue (rural town in southern Chile), where most inhabitants are from this ethnic group. Participants were excluded if they had signs of cicatricial alopecia (scars, atrophy, and loss of follicular ostia) or other causes of non-cicatricial hair loss such as alopecia areata, trichotillomania, and isolated telogen effluvium. Subjects were diagnosed with AGA (clinical observation and dermoscopy) by a trained dermatologist, and clinical patterns were recorded according to the Norwood-Hamilton (NH) and Ludwig classifications.

Two hundred and thirty-one patients (88 males and 143 females) were included, men averaging 46.2 years (21–88) and women 40.7 years (20–81). The percentage of AGA in men was 32.9% and in women, 8.29%. Tables 1 and 2 show the age distribution and clinical types of AGA in Mapuche men and women, respectively.

International studies report differences in prevalence and clinical patterns of AGA between Caucasians and Asians. Twenty percent of Caucasian men have AGA by their mid-twenties, which increases by 10% for each decade.[1,2] Korean and Chinese studies show a lower prevalence in men, 14.1% and 19.4%, respectively.[4,5] In females, an American study found a global prevalence of 19% and in Britain, 6% under 50 years had AGA, which increased to 38% in subjects 70 years or older.[3,6] Asian publications in women show lower results, 5.6% in Korea and 3.1% in China.[4,5] We found a lower percentage of AGA in Chilean Mapuche compared to Caucasians, but it was higher than Asians. This may reflect the genetic admixture in the racial origins of our study group.

Table 1 Age (years) distribution and clinical pattern of AGA in Mapuche men

Clinical pattern	20–29	30–39	40–49	50–59	60–69	>70	Total
NH III vertex	1	0	0	2	2	0	5
NH IVa	0	0	0	0	0	1	1
NH V	0	0	1	0	0	1	2
NH VI	0	0	0	0	1	1	2
Female	0	2	1	2	7	11	23
Total AGA	1	2	2	4	10	14	33
Total subjects	16	19	10	14	15	14	88

AGA, androgenetic alopecia.

Table 2 Age (years) distribution and clinical pattern of AGA in Mapuche women

Clinical pattern	20–29	30–39	40–49	50–59	60–69	>70	Total
Ludwig I	0	2	2	1	4	3	12
Total AGA	0	2	2	1	4	3	12
Total subjects	38	38	27	18	15	7	143

AGA, androgenetic alopecia.

The percentage of AGA in our investigation increased with age as shown in previous publications. Regarding clinical presentation of AGA in men, Caucasian and Asian investigations report NH types III and III vertex as the most common.[1,2,4,5] Interestingly, we found that 27.6% of Mapuche men had a female pattern, a higher percentage than reported in China (0.5%) and Korea (11.1%). As in Korea and China, Ludwig type I was the most common pattern in Mapuche women.[4,5] This study of AGA is, to our knowledge, the first done in an Amerindian population. The similarities of AGA percentage and clinical patterns between Mapuche and Asians may support evidence of common anthropologic links.

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Extensive acne in Apert syndrome

A 15-year-old female patient presented with acrocephaly, hypertelorism, proptosis, strabismus, interrupted eyebrows, and hooked nose. Maxillary hypoplasia, mandibular overjet, malocclusion, mouth breathing, highly arched palates, and misalignment and crowding of the teeth were also present (Fig. 1a). Symmetric syndactyly of the hands and feet was associated with nail dystrophy (Fig.1b and 1c), palmoplantar hyperhidrosis, and plantar hyperkeratosis. She also presented severe scoliosis due to hemivertebrae and ankylosis. The diagnosis of Apert syndrome (AS) was established at birth, and she was submitted to multiple consecutive craniectomies (Fig. 1d) and hand microsurgery to enable brain mass growth and pincer grasp, respectively. She has five nonsyndromic older siblings, and her parents age at conception was 44 years.

She was examined in our dermatological outpatient clinic for extensive acne, which began 3 years ago. Multiple comedones were observed, which besides the classical localization of acne (Fig. 2a,b) were found in the forearms down to the wrists and thighs (Fig. 2c). Papules, nodules, and residual acne scars were seen with the same distribution. Scattered lesions were found on the abdomen and back. She was started on a course of isotretinoin (0.84 mg/kg daily; with a cumulative dose of 150 mg/kg after 6 months), obtaining excellent response, with no significant side effects and no recurrence in a 1-year follow-up.

Apert syndrome (MIM ID no. 101200) is a severe congenital disorder characterized by premature fusion of the skull sutures and epiphyseal closure, resulting in intrauterine craniosynostosis and symmetric syndactyly in addition to a variety of abnormalities of the skeleton, skin, and visceral organs.[1,2] The estimated frequency of the condition ranges from 1 : 80,000 to 1 : 160,000 live births.[3,4] AS is an autosomal dominant disease that usually arises by new mutation in the gene encoding the fibroblast growth factor receptor 2 (FGFR2) with a paternal age effect.[3,4]

The dermatologic hallmark of AS is the development of inflammatory acne that begins in adolescence and extends to areas usually spared by acne vulgaris, such as the forearms, buttocks, and thighs.[3] A mosaic cutaneous manifestation of AS, called Munros acne nevus (MN), is characterized by sharply bordered acneiform lesions along the lines of Blaschko.[2,5]

The early and irregular ossification of the bones in AS causes asymmetric growth and alters the shape and function of the cranium, limbs, and spine. Characteristic craniofacial abnormalities of AS include acrocephaly, hypertelorism, and parrot-beak nose along with proptosis and strabismus due to shallow orbits.[1,2] As a result of underdevelopment of the upper jaw, prognathism, narrow palate, and crowding of the teeth are found. Symmetric severe syndactyly of the hands and feet added to shortening of the arms and forearms results in limited movements.[1,6] Ankylosis of the tarsal and metatarsal bones implies adaptations of the march and transfer of weight bearing, leading to lateral plantar hyperkeratosis.[2]

The relationship between seborrhea, acne, and skeletal abnormalities remains uncertain. Sebaceous gland activity, along with closure of the epiphysis, is under the influence of androgens.[7] In AS, plasma androgens and androgen receptor distribution and density are within normal ranges.[3,8] Increased FGFR2 signaling has been proposed to be involved in the pathogenesis of acne vulgaris and explains acne lesions in AS and MN associated with gain of function point mutations of FGFR2.[9] Androgen-mediated upregulation of FGFR2 signaling could be the initiating signal in the pathogenesis of acne and premature epiphyseal closures in AS.[2,3,8]

Apert syndrome-associated acne is typically recalcitrant to standard therapy.[8] Oral isotretinoin has been reported to lead to healing in these patients.[8,9] These good results are explained by evidence that suggests that the retinoids have an inhibitory effect at the FGFR2-signaling cascade.[9,10]

A splice variant of FGFR2 is FGFR2b, which is expressed exclusively in the suprabasal spinous layer of

