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The SAHA Syndrome

Constantin E. Orfanos Yael D. Adler Christos C. Zouboulis

Department of Dermatology, University Medical Center Benjamin Franklin, The Free University of Berlin, Berlin, Germany

Key Words

SAHA · Seborrhoea · Acne · Hirsutism · Androgenetic alopecia

Abstract

The presence of **seborrhoea**, **acne**, **hirsutism** and **alopecia** in women has first been summarized as SAHA syndrome in 1982 and can be associated with polycystic ovary syndrome, cystic mastitis, obesity and infertility. In 1994, the association of these androgen-dependent cutaneous signs, was classified according to their etiology into four types: (1) idiopathic, (2) ovarian, (3) adrenal, and (4) hyperprolactinemic SAHA. The HAIRAN syndrome has been currently described as a fifth variant with polyendocrinopathy. The SAHA syndrome generally occurs in young to middle-aged women and involves either the presence of elevated blood levels of androgens or increased androgen-driven peripheral response with normal circulating androgen levels. Peripheral metabolism of androgens takes place in various areas within the pilosebaceous unit, as indicated by local differences in the activities of aromatase, 5 α -reductase as well as of the presence of the androgen receptors. In cases of SAHA syndrome, careful diagnostic and clinical evaluation has to be performed in order to identify the cause for peripheral hyperandrogenism and to exclude androgen-producing tumors. Treatment will target the etiology, whereas the management in idiopathic cases will aim to improve the clinical features of SAHA.

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Introduction

The SAHA syndrome summarizes the major cutaneous features indicating peripheral hyperandrogenism in young females. This term, introduced by us in 1982 [1], stands for **seborrhea**, **acne**, **hirsutism** on face, trunk and extremities and androgenetic **alopecia** of the scalp. Although all four signs of SAHA syndrome are only present in approx. 20% of the patients [2], its knowledge is important for recognizing hormonal disorders involving androgen metabolism.

In skin, the formation of excessive active androgen metabolites in the pilosebaceous unit is responsible for the appearance of cutaneous hyperandrogenism [3, 4], however, the characteristic clinical phenotype does not always correlate with elevated blood levels of androgens [5]. In cases of systemic virilization, additional signs are present, such as deepening of the voice, increased muscle bulk, clitoris hypertrophy, loss of smooth skin contours or obesity, irregularities of the menstrual cycle and infertility. Many clinical characteristics of the SAHA syndrome are common with those seen in cases with polycystic ovaries (PCO) and similar disorders [6].

Pathogenetic Background

Women produce androgens in the ovaries, the adrenal glands and also in peripheral organs, especially the skin, the skeletal muscles and the liver [7]. The ovaries secrete