Benha Journal of Applied Sciences (BJAS) **print: ISSN 2356–9751**
Vol. (9) Issue (2) (2024), (125-128) **online: ISSN 2356–976x**
http://bjas.bu.edu.eg

Pathogenesis of Acne Vulgaris in Adolescence

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Abstract

Objectives: Describe the pathophysiology of acne vulgaris and talk about how androgens contribute to the onset of the condition throughout puberty. Background: Acne is a skin ailment that often affects children and teenagers. It seems that the condition is triggered by an increase in the amounts of circulating androgens that originate from the adrenal glands and the testes. Research Methods: We scoured the Medline databases (Pub Med and Medscape) for research that elucidate the pathophysiology of acne vulgaris and address the part played by androgens in the onset of the condition in adolescents up to the year 2023. Research Prioritization: The inclusion of all research was determined by separate evaluations. Inclusion was contingent upon them meeting the following requirements: 1. Presented in an English language format. 2. Publicated in publications that undergo a rigorous peer review process. 3. Explain the process by which acne vulgaris develops and talk about how androgens play a part in this adolescent skin condition. Data Extraction: Studies were omitted from consideration if they failed to meet the inclusion criteria. The study's quality was evaluated based on a number of aspects, such as the following: the availability of sufficient information; the clarity of evaluation criteria; and the acquisition of ethical permission. For our concerned research outcomes, data were independently extracted from all qualifying studies utilizing a data collecting form. Conclusions: Acne is a problem that mostly affects children and teenagers, and it seems that the illness is triggered by elevated amounts of circulating androgens that originate from the adrenal glands and the testicles. Acne pathogenesis involves a series of events that occur in a relatively consecutive order: retained hyperkeratosis, hyperplasia of the sebaceous glands and increased sebum production, follicle colonization by Propionibacterium acnes, and inflammation around the follicles.

Key words: Acne vulgaris.

1.Introduction

Acne Psoriasis vulgaris is a common skin condition that mostly strikes the face, upper chest, and back because of the high concentration of sebaceous glands in these regions [1]. Its symptoms may appear as sudden attacks or gradually develop over time, and it has a history of recurrence or relapse [2].

A common occurrence in teenagers is the formation of visible, clinically identifiable blackheads or whiteheads (comedones) on the forehead. These comedones, which are initially imperceptible from a histological perspective, eventually mature into inflammatory red papules or pustules. Acne scarring may exacerbate such lesions [3].

Obese and non-obese AV patients had significantly greater cholesterol and low-density lipoprotein-C (LDL-C) levels, which significantly affected the severity of their condition. The production of adrenal and gonadal androgens both rely on cholesterol obtained from plasma [4], suggesting that total cholesterol levels may influence AV development.

2.Materials and methods

Data Sources: The A search of the Medline databases (Pub Med and Medscape) was conducted to source material on the pathophysiology of acne vulgaris in adolescents, with a focus on the years up to 2023.

Research Prioritization: Research studies were selected via an open, honest, and thorough selection procedure. Inclusion was contingent upon them meeting the following requirements:

1.Publicly released in English. 2. Featured in journals that strictly adhere to the principles of peer review.Thirdly, talk about how androgens play a role in the development of AV in teenagers and explain the pathophysiology of the disease.

Data Extraction: Studies were omitted from consideration if they failed to fulfill the requirements for inclusion. The study's quality was determined by its adherence to ethical approval, eligibility criteria, information, and well stated assessment methods. To ensure that our research results were accurately represented, data were independently extracted from all eligible and qualifying studies utilizing a data collecting form.

3.Review of literature:

Acne Vulgaris

Acne The inflammatory illness known as acne vulgaris affects the pilosebaceous unit and has a persistent but ultimately self-limiting course. In puberty, it is set off by the acne-causing bacteria Candida albicans (C. acne) in response to the typical levels of dehydroepiandrosterone (DHEA) in the blood. Inflammatory and non-inflammatory lesions may be seen on the face, upper arms, torso, and back of affected individuals; nonetheless, this skin ailment is rather frequent [5].

When there is a typical amount of androgens in the blood, the sebaceous glands become hypersensitive, resulting in acne [6]. Medications such as lithium, steroids, and anticonvulsants may aggravate acne, as can prolonged sun exposure and the use of occlusive clothing such as backpacks, headbands, and underwire brassieres. Polycystic ovarian syndrome and other endocrine diseases, as well as pregnancy; Part of sebum's composition is branched fatty acids [7].

The mental toll that acne takes is well-established. Low self-esteem, social isolation, sadness, and anxiety are some of the negative outcomes that may stem from acne-related body image difficulties [8].

Acne Vulgaris Risk Factors

The stress hormone cortisol is elevated because it triggers the secretion of inflammatory cytokines and the corticotrophin-releasing hormone (CRH). Acne may be made worse by the lack of sleep that comes with contemporary women's hectic lives, which in turn affects the hypothalamic-pituitary-adrenal axis and causes an increase in the release of hormones that are connected to stress [9]. The HPA axis and the sympathetic nervous system are activated by stress signals, which can trigger the release of various neurotransmitters, cytokines, and hormones. These substances have skin receptors and may exacerbate many skin conditions, such as acne. It seems that the CRH plays a significant role in how acne lesions form [10]. Many adults and teenagers with acne (ranging from 50 to 80 percent) have validated what many patients have said: that emotional stress exacerbates their acne [11].

Variables at the genetic level: An individual's vulnerability to comedogenesis and the severity of the illness may be impacted by genetic factors. There has been research into the possible hereditary links between acne and a number of genes, but chromosomal abnormalities and HLA phenotypes also have a role in the development of acne [12]. The HSD3B gene family and the cytochrome P450 (CYP) gene family are two examples of gene families that have been investigated extensively and may have a role [13].

Sugary sweets, refined grains, a lot of protein, fatty dairy products, and a lot of sugar are all hallmarks of the Western diet. Obesity, insulin resistance, and acne are so prevalent in younger generations at alarming rates [16]. Anabolic androgen precursors of dihydrotestosterone (DHT), such as 5-pregnanedoine and other exogenous steroid hormones, are known to be present in cow's milk [15].

Micro and macro comedones, with minimal inflammatory lesions, describe the comedonal form, which is more common in smokers [9]. The sebaceous gland responds to nicotine by releasing acetylcholine, which is a neurotransmitter. Inducing hyper-keratinization and affecting sebum production and composition, acetylcholine leads to cellular regulation and differentiation [12].

The Outward Signs of Acne vulgaris in Clinical Practice

Nodules, pustules, comedones, and inflammatory papules are the typical lesions seen in acne. Cysts were the old name for some of the bigger nodules, and nodulocystic was a word for more severe inflammatory acne [16]. Comedones, both open and closed, are acne lesions that do not cause inflammation. Because they look like white or flesh-colored papules with an apparently closed surface, closed comedones are sometimes called whiteheads. The follicular openings are widened by brown to black plugs, which are either flat or slightly elevated, and are known as open comedones (black heads). Because the melanin pigment has oxidized, it has taken on a dark hue [17].

Clinical Variants of Acne Vulgaris

Neonatal acne: This occurs often and is characterized by open and closed comedones, papules, and pustules. There will be no visible scarring when the condition, which seems to have its roots in fetal hormone activity, goes away on its own within one to three months after giving birth [18].

Acne vulgaris that starts early: Lesions may begin to appear more often in families prone to acne at the age of six or seven and continue well into adulthood. Puberty is the most common time for inflammation to worsen, however it may happen years before [12].

An Acne Follicularis Systemic symptoms such as fever, arthralgias, anorexia, leukocytosis, and sometimes evidence for localized lytic bone lesions accompany the abrupt development of profoundly inflamed lesions [19].

Females are more likely than men to have Tarda acne (5:1).Acne tarda in women, which manifests with inflammatory lesions of the lower face [20], may be caused by dehydroepiandrosterone sulfate.

Acne Venenata: The development of large comedones is a result of contact with comedogenic substances. The most typical workplace environments include chlorinated hydrocarbons (chloracne), cutting oils, petroleum oil, coal tar, and pitches [3].

One kind of cosmetic acne, known as pomade acne, is exclusive to black people and happens when they apply oils and greases to their scalps. Typically, the areas affected include closed comedones on the chin, cheeks, forehead, and temples [3].

How Acne Vulgaris Develops

What Does C. acnes Do?

The sebaceous glands produce more oil because C. acnes raises diacylglycerol acyltransferase activity, which worsens androgen-related seborrhea that already exists [21]. The triglycerides released by the sebaceous glands are broken down by the C. acnes, which then releases free fatty acids. One of

the primary components of sebum is squalene, and the porphyrins released by C. acnes serve as catalysts for this process. Comedogenesis is promoted by oxidized squalene and free fatty acids [22]. In addition to increasing vascular permeability and chemotactic leukocyte participation in inflammatory reactions, C. acnes activates the classical and alternative complement pathways to produce C3a and C5a. In addition, C. acnes secretes transforming growth factor-β (TNF-β), interleukin (IL)-1β, and IL-6, which activate sebocytes and encourage the transformation of naïve T cells into T helper (Th) 17 cells. According to reference 23, C. acnes has the ability to trigger the production of IL-1β, IL-8, and TNF-α from sebocytes by activating the NLRP3 inflammasome. The presence of activated Th1 cells in early inflammed acne lesions is another way that C. acnes triggers the adaptive immune response. Inflammatory acne is characterized by a humoral immune response to C. acnes. It is true that C. acnes causes AV patients to produce high serum antibody titers that target many putative surface proteins [24].

Interaction Between Hormones and the Development of Acne Vulgaris

Androgens' Function: Enhanced androgenic hormone sensitivity in the sebaceous gland: In adult female acne, similar to AV, the quantity and sensitivity of the androgenic hormone receptors found in sebocytes and keratinocytes are increased. An increase in sebum production, driven by androgens, occurs in both sexes as puberty begins [24]. Increased pre-hormone peripheral conversion of DHEA-S, androstenedione, and testosterone into more potent androgenic hormones (testosterone and DHT) due to hyperactivity and abnormal activity of enzymes involved in the metabolism of androgenic hormones, such as 5-alpha reductase, 3-beta-hydroxysteroid dehydrogenase, and 17-hydroxysteroid dehydrogenase. There is less metabolic breakdown of DHT, and its potency is five to ten times that of testosterone [25].

Estrogen Function: Many believe that progesterone is to account for the fact that a woman's sebum production varies during her menstrual cycle. Progesterone may exacerbate acne lesions by increasing sebum production, which in turn increases free fatty acids in the surface lipids, stimulates keratinocyte growth, and is regulated by the menstrual cycle. It has been shown that IL-6 concentrations rise during the luteal phase of the menstrual cycle, which may be the reason for the increase in acne lesions at this period [3]. Progestins have the ability to boost monocyte production of proinflammatory cytokines.

Impact of Estrogen: There are a number of ways in which estrogen might exert its effects. One is by blocking androgen production in the gonadal tissue, which in turn inhibits pituitary release and regulation genes that have a negative impact on sebaceous gland growth and lipid production [26]. Another way is by acting directly against androgen effects within the sebaceous gland.

How Being Obese Affects Acne Vulgaris The development of disease

Increased sebum production and the onset of severe acne may be associated with obesity, peripheral hyperandrogenism, insulin resistance, and adipose tissue, the location of androgen biosynthesis. When calculating the prevalence of overweight and obesity, body mass index is a popular statistical tool [27].

An increase in androgen production, which stimulates sebaceous glands, and an increase in endogenous androgen production, which causes acne to develop or worsen, are both possible outcomes of hyperinsulinaemia. Therefore, AV sufferers may benefit from a weight reduction regimen. Metaformin use and weight loss are associated with decreased levels of insulin, androgens, and insulin-like growth factor-1, all of which are thought to have a role in skin breakouts [9].

Acute Acne Vulgaris Severity

A sensible and efficient categorization system was established, and many dermatologists throughout the globe progressively started using it. The following AV grading system is proposed by this consensus: mild acne, moderate acne with comedones and few to several papules-pustules; severe acne with many nodules and either numerous or extensive papules-pustules; and very severe acne, including the most destructive forms of the disease like follicular occlusion triad, acne conglobata, and acne fulminans [3].

4. Conclusion

It seems that elevated amounts of circulating androgens originating from the adrenal glands and the gonadal glands are the initiating factors in acne, a malady that mostly affects children and adolescents. Acne pathogenesis involves a series of events that occur in a relatively consecutive order: retained hyperkeratosis, hyperplasia of the sebaceous glands and increased sebum production, follicle colonization by Propionibacterium acnes, and inflammation around the follicles.

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