Benha Journal of Applied Sciences (BJAS) **print: ISSN 2356–9751**
Vol. (9) Issue (8) (2024), (49- 52) **online: ISSN 2356–976x**
http://bjas.bu.edu.eg

Evaluation of serum Cold Inducing RNA Binding Protein level in Acne Vulgaris patients

Elaf A. Kamil[1], Amany I. Mustafa[1], Fatma M. El Esswey[1] and Rana A. Hussein[2]
[1]Dermatology, Venerology and Andrology department, Faculty Of medicine, Benha University
[2]Clinical and Chemical Pathology Faculty of Medicine, Benha University
E-mail: elaafaamer898@gmail.com

Abstract

Background: Acne vulgaris is a common cutaneous disorder of the pilosebaceous unit with a high prevalence in the general population. Objective: This article aims to review epidemiology, clinical characteristics, diagnosis and treatment of acne vulgaris. Acne affects 9.8% of women and 9.0% of men. However, men are more likely to have this syndrome in adolescence and early adulthood, although this reverses with age. Facial area is most typically afflicted in 99% of cases, followed by dorsal in 60% and thoracic in 15%. Studies that did not meet inclusion requirements were excluded. Ethical approval, eligibility criteria, controls, information, and evaluation measures were study quality assessment factors. A data collecting form was used to independently extract data from each qualifying research for our study results. Lack of a worldwide diagnostic or grading system makes acne prevalence difficult to estimate. Since acne is decreasing owing to therapeutic advances, estimates are revised often. Over 650 million people—9.4% of the global population have acne vulgaris. Medline databases (Pub Med and Medscape) were searched for acne aetiology, pathophysiology, clinical images, and decrease up to 2024.

Key words : Serum Cold ; RNA Binding Protein level ; Acne Vulgaris patients

Introduction

Acne vulgaris, a persistent pilosebaceous unit inflammation, affects both men and women. Acne affects 75–95% of teenagers. The aetiology includes hormonal factors, increased sebum production, follicular plugs, hyperkeratinization, colonisation of Cutibacterium acnes (previously Propionibacterium acnes), and inflammation **(1).**

Materials and methods

Data Sources: Medline databases (Pub Med and Medscape) were searched for acne aetiology, pathophysiology, clinical images, and decrease up to 2024.

Study Selection: Inclusion was independently evaluated for all trials. They were included if they met these criteria: 1. Published in English. 2. In peer-reviewed journals. 3. Review: melasma aetiology, pathophysiology, clinical images.

Data Extraction: Studies that did not meet inclusion requirements were excluded. Ethical approval, eligibility criteria, controls, information, and evaluation measures were study quality assessment factors. A data collecting form was used to independently extract data from each qualifying research for our study results **(2).**

Review of literature:

Common acne

The overall population is prone to acne vulgaris, a pilosebaceous unit skin ailment. The eighth most common skin disease globally is acne vulgaris, with 9.38% global prevalence **(3)**.

Epidemiology

Lack of a worldwide diagnostic or grading system makes acne prevalence difficult to estimate. Since acne is decreasing owing to therapeutic advances, estimates are revised often. Over 650 million people—9.4% of the global population—have acne vulgaris **(3)**.

All ages may have acne vulgaris, although teens are more likely. Girls get acne at 11 and guys around 12. Acne is becoming more common in youngsters as early as 8 or 9. A decline in puberty may explain the rise in acne at younger ages **(4).**

Acne affects 9.8% of women and 9.0% of men. However, men are more likely to have this syndrome in adolescence and early adulthood, although this reverses with age. Facial area is most typically afflicted in 99% of cases, followed by dorsal in 60% and thoracic in 15% **(5)**.

The pathogenesis of acne vulgaris

Hormones cause sebaceous glands to overproduce sebum. The whole body has these glands, although they are most plentiful in the upper trunk, chest, and back. They are absent from the palm, sole, and dorsum of the foot, where there are no hair follicles. Sebum overproduction impairs follicular keratinisation, blocking the sebaceous gland pore and causing acne. The following stages occur during pathogenesis. 1) Overproduction of sebum by sebaceous glands, 2) excessive keratin formation resulting in small comedones that grow into larger ones, and 3) anaerobic bacteria colonising the hair follicle and causing inflammatory reactions.

1) The study of genes and heredity.

Genetics have a major role in acne production, according to family and twin studies. Multiple genetic variants impacting gene expression/function have been studied. Acne was linked to the insulin-like growth factor (IGF1) (CA) 19 repeat polymorphism, the PPARG124 Pro12Ala polymorphism, the IL6 572 G/C polymorphism, and the IL1A 889 C/T polymorphism **(6).**

2) Hormonal influences:

Androgens enhance sebum production throughout puberty. 5-alpha reductase turns testosterone into DHT, which binds to sebaceous gland receptors. This increases sebum production. Thus, the outer layer of skin cells grows excessively, accumulating sebum. Distended follicles rupture and release pro-inflammatory chemicals into the dermis, causing inflammation. The user typed "C." Acnes, Staphylococcus epidermis, and Malassezia furfur inflame and proliferate hair follicle epidermal cells **(7)**.

Menstrual cycles and pregnancy affect oestrogen, oestradiol, and oestrone production. This influences acne vulgaris development. Higher DHEA levels are typical, according to **(8)**. There is no significant difference in 17α hydroxyprogesterone levels between acne-afflicted and acne-free women. Women's high oestradiol levels protect them **(9)**.

3) Nutrition:

Insulin-like growth factor signalling, the major endocrine route for sexual maturation, is activated by hyperglycaemic diets and contributes to acne. High quantities of glycaemic carbs, saturated fats, and dairy products in the Western diet produce sebaceous lipids and sebum **(10)**. Dairy products cause acne vulgaris. Acne was linked to full-fat, skimmed, and low-fat milk consumption. Regular omega-3 fatty acid and low glycaemic index diets significantly reduce acne **(10).**

The Western diet is high in BCAAs, glutamine, and palmitic acid. Insulin and IGF-1 suppress FoxO1's metabolic transcription factor activity. Insulin, IGF-1, BCAAs, glutamine, and palmitate activate mTORC1, which promotes anabolism and lipogenesis. FoxO1 represses transcription factors implicated in sebum lipogenesis, including as androgen receptor, PPARγ, liver X receptor-α, and SREBP-1c. According to **(11)**, mTORC1 increases the transcription of PPARγ and SREBP-1c, leading to increased sebum production.

4) Function of linoleic acid:

Linoleic acid is an essential fatty acid found in sebum. Lack of linoleic acid weakens the hair follicle barrier, enabling bacterial lipases to release more free fatty acids. Exogenous fatty acids worsen linoleic acid deficiency, creating unusually thick sebum that obstructs the sebaceous gland duct **(12)**.

5) Inflammation's Function:

The onset, development, and cure of acne vulgaris depend on inflammation. Most acne inflammation is caused by IGF-1 and pathogenic P. acnes. In primary human sebocytes, IGF-1 alone induces pro-inflammatory cytokines. **(13)** found that IGF-1 stimulation increased NF-κB, IL-1β, IL-6, IL-8, and TNF-α in sebocytes cultured in vitro.

In healthy guys, testosterone raises IGF-1 levels in the circulation, which may have similar effects. Sebocytes release cytokines and MMPs after IGF-1 activation, attracting inflammatory cells to the pilosebaceous unit. MMPs tear the follicular membrane, releasing fatty acids into the dermis and breaking down the extracellular matrix **(14)**.

Toll-like receptors 2 (TLR-2) on monocytes and macrophages detect P. acnes and release IL-12 and IL-8. P. acnes also upregulates caspase-1 and NLPR3, which rupture lysosomes and release cathepsin B. This mechanism produces ROS, potassium efflux, and IL-1β **(14)**.

6) Bacterial factor:

Rod-shaped, gram-positive, facultative, anaerobic P. acnes bacterium. It comprises 87% of human skin clones, together with Staphylococcus, Corynebacterium, Streptococcus, and Pseudomonas. P. acnes is inactive before puberty. However, puberty increases androgenic hormones, which increase sebum production. This sebum rise stimulates bacteria, promoting fast growth. Sebaceous glands generate sebum, which contains fatty acids that support the body. Follicle bacteria get energy and nutrition from sebum, cellular debris, and skin tissue metabolic byproducts. Therefore, follicular blockage may foster bacteria growth **(15)**.

P. acnes' cell wall peptidoglycan activates toll-like receptors, triggering acne inflammation. P. acnes activates follicular keratinocytes, releasing proinflammatory cytokines such IL-1 β, IL-8, GM-CSF, and TNF-α. This stimulation causes keratinocyte proliferation and preclinical microcomedo **(16)**.

7) Function of sebum:

The sebaceous glands and hair follicles form the pilosebaceous unit, which produces sebum. Disruptions in lipid metabolism modify sebum amount and composition, causing acne vulgaris and AD. Pathogens like P. acnes strains and TLR2 and TLR4 ligands can also activate sebocytes to release pro-inflammatory cytokines, chemokines, and antimicrobial peptides **(17)**.

Fat cells emit adipokines in reaction to stimuli, which may cause acne. IL-6,

adiponectin, resistin, leptin, serpin E1 (plasminogen activator inhibitor 1), visfatin (nicotinamide phosphoribosyltransferase), apelin, chemerin, RBP4, and MCP1 are included in this group **(18)**.

Overview and classification of acne vulgaris:

Open or closed comedones are the major feature of acne. Whitehead and blackhead acne comedones are closed and open, respectively. Pustules and nodules may accompany acne. Normal distribution includes sebaceous gland-rich face, upper back, chest, and shoulders. Scarring, post-inflammatory hyperpigmentation, and erythema are secondary lesions **(19).**

The classification of acne:

Acne vulgaris severity can be assessed using clinical examination, lesion counting, and more advanced methods like photography, fluorescent photography, polarised light photography, video microscopy, and sebum production measurement. Grading and lesion counting are common. Pillsbury, Shelley, and Kligman published the first grading system in 1956. Since then, several grading systems have been developed, including the Global Acne Grading System (GAGS) **(20)**.

The Global Acne Grading System (GAGS) measures acne severity numerically. This was developed by Doshi and colleagues in 1997. The severity score is the sum of six regional evaluations. The variables allocated to face and body regions are multiplied to calculate each location. These contain 2 forehead, 2 cheek, 1 nose, 1 chin, and 3 chest and back elements. The highest weighted lesion in each region comedon, papule, pustule, and nodule is multiplied **(21).**

References

[1] Adityan, B.; Kumari, R. and Thappa, D. M. (2009): Scoring systems in acne vulgaris. *Indian Journal of Dermatology, Venereology, and Leprology*, *75*(3), 323.

[2] Baldwin, H. and Tan, J. (2021): Effects of diet on acne and its response to treatment. *American journal of clinical dermatology*, 2(5), 1-11.

[3] Cong, T. X.; Hao, D.; Wen, X. and Jiang, X. (2019): From pathogenesis of acne vulgaris to anti-acne agents. Archives of dermatological research, 311(5), 337-349.

[4] Coyner, K. S. (2018): Dermatology lesions and differential diagnoses. *Clinical Atlas of Canine and Feline Dermatology*, 2(2), 23-46.

[5] Doshi, A.; Zaheer, A. and Stiller, M. J. (1997): A comparison of current acne grading systems and proposal of a novel system. *International journal of dermatology*, *36*(6), 416-418.

[6] Dréno, B. (2017): What is new in the pathophysiology of acne, an overview. *Journal of the European Academy of Dermatology and Venereology*, *31*, 8-12.

[7] Dréno, B.; Pécastaings, S.; Corvec, S.; Veraldi, S.; Khammari, A. and Roques, C. (2018): Cutibacterium acnes (Propionibacterium acnes) and acne vulgaris: a brief look at the latest updates. *Journal of the European Academy of Dermatology and Venereology*, *32*, 5-14.

[8] Heng, A. H. S., Say, Y. H., Sio, Y. Y., Ng, Y. T., & Chew, F. T. (2022). Epidemiological risk factors associated with acne vulgaris presentation, severity, and scarring in a Singapore Chinese population: a cross-sectional study. *Dermatology*, *238*(2), 226-235.

[9] Kim, K.; Ha, I.; Kim, E. and Kim, K. (2017): A comparative study of biological and metabolic biomarkers between healthy individuals and patients with acne vulgaris: A cross-sectional study protocol. *Medicine*, *96*(45), 125-130.

[10] Kovács, D.; Fazekas, F.; Oláh, A. and Törőcsik, D. (2020): Adipokines in the Skin and in Dermatological Diseases. *International Journal of Molecular Sciences*, *21*(23), 9048.

[11] Kovács, D.; Lovászi, M.; Póliska, S.; Veres, I. and Törőcsik, D. (2016): Sebocytes differentially express and secrete adipokines. *Experimental dermatology*, *25*(3), 194-199.

[12] Kozłowski, M., Niedzielska, M., Lorenz, A., Brodowska, A., Malanowska, E., Przepiera, A., ... & Sowińska-Przepiera, E. (2023). Metabolic and Dietary Factors in Acne Vulgaris and Evaluation of the Acne Vulgaris Treatment with Oral Contraceptive-Based Therapies in Young Adult Women. *Nutrients*, *15*(6), 1488.

[13] Kutlu, Ö., Karadağ, A. S., & Wollina, U. (2023). Adult acne versus adolescent acne: a narrative review with a focus on epidemiology to treatment. *Anais brasileiros de dermatologia*, *98*, 75-83.

[14] Liu, P. F.; Hsieh, Y. D.; Lin, Y. C. and Huang, C. M. (2015): Propionibacterium acnes in the pathogenesis and immunotherapy of acne vulgaris. *Current Drug Metabolism*, *16*(4), 245-254.

[15] Melnik, B. C. (2018): Acne vulgaris: The metabolic syndrome of the pilosebaceous follicle. *Clinics in dermatology*, *36*(1), 29-40.

[16] Qin, Z.; Li, X.; Han, P.; Liu, H.; Tang, J. and Zhang, W. (2017): Association between polymorphic CAG repeat lengths in the androgen receptor gene and susceptibility to prostate cancer: A

systematic review and meta-analysis. Medicine, 96(25) 12-16.

[17] Rodriguez Baisi, K. E., Weaver, A. L., Wi, C. I., Shakshouk, H., & Tollefson, M. M. (2023). Socioeconomic status, race, and preadolescent acne: A population- based retrospective cohort analysis in a mixed rural–urban community of the United States (Olmsted County, Minnesota). Pediatric Dermatology, 40(3); 428-433.

[18] Tahir, C. M. (2016): Pathogenesis of acne vulgaris: simplified. *Journal of Pakistan Association of Dermatology*, *20*(2), 93-97.

[22] .

[19] Teder-Laving, M., Kals, M., Reigo, A., Ehin, R., Objärtel, T., Vaht, M., ... & Kingo, K. (2023). Genome-wide meta-analysis identifies novel loci conferring risk of acne vulgaris. *European Journal of Human Genetics*, 1-8.

[20] Woo, Y. R., & Kim, H. S. (2022). Truncal Acne: An Overview. *Journal of Clinical Medicine*, *11*(13), 3660.

[21] Zaenglein, A. L. (2018): Acne vulgaris. *New England Journal of Medicine*, *379*(14), 1343-1352