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Relation between Acne Vulgaris and Metabolic Syndrome

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Abstract

Acne is a chronic inflammatory disease of the pilosebaceous unit resulting from androgen-induced increased sebum production, altered keratinisation, inflammation, and bacterial colonisation of hair follicles on the face, neck, chest, and back by Propionibacterium acnes. Acne vulgaris is an epidemic inflammatory disease of the human sebaceous follicle and represents the most common skin disease affecting about 85% of adolescents in Westernized populations. Acne vulgaris is primarily a disease of wealthy countries and exhibits higher prevalence rates in developed compared with developing countries. Prevention of acne relies on the successful management of modifiable risk factors, such as underlying systemic diseases and lifestyle factors. Several treatments are available, but guidelines suffer from a lack of data to make evidence-based recommendations. In addition, the complex combination treatment regimens required to target different aspects of acne pathophysiology lead to poor adherence, which undermines treatment success. Acne commonly causes scarring and reduces the quality of life of patients. New treatment options with a shift towards targeting the early processes involved in acne development instead of suppressing the effects of end products will enhance our ability to improve the outcomes for patients with acne. No acne has been found in non-Westernized populations still living under Paleolithic dietary conditions constraining hyperglycemic carbohydrates, milk, and dairy products. The high prevalence rates of adolescent acne cannot be explained by the predominance of genetic factors but by the influence of a Western diet that overstimulates the key conductor of metabolism, the nutrient- and growth factor–sensitive kinase mTORC1.

Keywords: Acne Vulgaris and Metabolic Syndrome, BMI.

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1. Introduction

Acne vulgaris is considered one of the most common skin disorders worldwide, affecting more than 85% of adolescents and young adults. Therefore, it is a vital issue during dermatological consultations because of its prevalence and impact on patients' social lives [1]. The first step in the pathophysiology of acne is increased sebum production, which causes follicular hyperkeratinization. This is followed by an infestation of Propionibacterium acnes (recently renamed Cutibacterium acnes), which causes the eventual release of inflammatory mediators. The usual inflammatory acne prevalent in the pubertal age group commonly occurs due to increased circulating androgen levels. Studies have also shown that an increased insulin level can aggravate acne [2]. The role of lipid metabolism and hormonal action in differentiation of sebocytes are causative factors for acne. Insulin-like growth factor-1 (IGF-1) has also been shown to cause excess sebum production and cause acne independently. A previous study reported elevated IGF-1 levels in cases of acne, potentially indicating a possible influence of insulin and growth hormone levels [3].

The metabolic syndrome (MetS), first described as Syndrome X by Reaven [4], comprises a set of laboratory and physical parameters predisposing the cases to the causation of cardiovascular diseases and diabetes mellitus (Type 2). Central obesity is considered one of the major constituents of MetS. The International Diabetes Federation (IDF) guideline regards it as a defining criterion. Subsequent consensus by the American Heart Association (AHA) and the National Heart, Lung, and Blood Institute (NHLBI) in 2009 recommended considering central obesity as just one of the criteria. Also, it recommended incorporating ethnicity-specific waist circumference (separately for males and females) [5]. Other parameters include elevated triglyceride levels, reduced high-density lipoproteins, elevated blood pressure, and increased fasting blood sugar. The presence of any three out of the five parameters in an individual is labeled as MetS. The pathophysiology of MetS is intricately associated with insulin resistance. The tissues like muscles, fat, and other cells, become insensitive to insulin levels in the bloodstream and fail to absorb blood glucose. Central obesity and adipose tissue accumulation play a significant role in developing insulin resistance [6].

2. MetS and the skin

Systemic metabolic derangements can often result in cutaneous manifestations and vice versa. The deposition of excess adipose tissue and insulin resistance in MetS

initiates a spectrum of hormonal disturbances. In the pathogenesis of MetS and acne, inflammatory markers like TNF-α, IL-17, IL-23, and oxidative stress have shown a possible correlation. With this study, we aim to analyze the changes in markers of MetS observed in patients with acne Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for Study of Obesity. Here, five parameters were considered, with the presence of any three confirming the diagnosis of MetS (Table I) [8]. A higher prevalence of MetS in acne patients of 32% also reported by Podder et al. [9]; however, difference in comparison to control group in their study was not significant (p=0.06). Nagpal et al., [10] also observed a higher proportion of acne cases having MetS. In their study, 17% of subjects fulfilled MetS criteria in acne group compared to 9% from control group (p=0.09). Podder et al. [9] Nagpal et al., [10] and Del Prete et al., [11] detected significantly increased fasting blood glucose values in case group.

Balta et al. [12] had a similar observation of no significant difference with respect to fasting blood glucose in vulgaris in contrast to those with no skin manifestations. Identifying such a positive association between acne vulgaris and MetS at an earlier stage would enable us to take necessary preventive measures to minimize the brunt of the disease [7]. The diagnosis of MetS was based on the Joint case and control groups. Raised blood glucose values are associated with acne patients principally because an increase in blood glucose levels triggers insulin secretion, which decreases the binding protein for IGF-1, promoting cell proliferation by IGF-1. Higher fasting and postprandial insulin values can cause acne flare-ups by increasing basal keratinocyte proliferation. Insulin also causes stimulation of androgen secretion, ultimately leading to increased sebum production. Insulin sensitivity decreases during puberty and adolescence, along with an increase in IGF-1 and insulin serum levels. Sex hormone-binding globulin (SHBG) and insulin-like growth factor-binding protein-1 (IGFBP-1) serum levels show a reduction. Insulin and IGF-1 levels, for instance, peak in late adolescence and steadily fall until the third decade. Acne appears at about the same time as the preadolescent increase in plasma insulin, IGF-1, and BMI and usually clears up by end of puberty, even though circulating androgens stay unchanged [13].

Table I: Revised NCEP: ATP III Criteria for Metabolic Syndrome.

Parameters	Cut-off Values
Elevated Waist Circumference (Asian population)	Men: ≥90cm
	Women: ≥80cm
Elevated Blood Pressure	Systolic Blood Pressure: ≥130 mmHg and/or
	Diastolic Blood Pressure: ≥85mmHg
Elevated Triglycerides	≥150 mg/dL (1.7 mmol/dL)
Reduced High-Density Lipoprotein	Men: <40 mg/dL (1.0 mmol/L)
	Women: <50 mg/dL (1.3 mmol/L)
Elevated Fasting Glucose	≥100mg/dL

References

[1] P.C.T. Durai, D.G. Nair. (2015). Acne vulgaris and quality of life among young adults in South India. Indian journal of dermatology. 60(1): 33.

[2] W. Chen, B. Obermayer-Pietsch, J.B. Hong, B. Melnik, O. Yamasaki, C. Dessinioti, Q. Ju, A. Liakou, S. Al-Khuzaei, A. Katsambas. (2011). Acne-associated syndromes: models for better understanding of acne pathogenesis. Journal of the European Academy of Dermatology and Venereology. 25(6): 637-646.

[3] L. Tasli, S. Turgut, N. Kaçar, C. Ayada, M. Çoban, R. Akcilar, S. Ergin. (2013). Insulin-like growth factor-I gene polymorphism in acne vulgaris. Journal of the European Academy of Dermatology and Venereology. 27(2): 254-257.

[4] G.M. Reaven. (2005). Why Syndrome X? From Harold Himsworth to the insulin resistance syndrome. Cell metabolism. 1(1): 9-14.

[5] E. Oda. (2012). Metabolic syndrome: its history, mechanisms, and limitations. Acta diabetologica. 49: 89-95.

[6] D. Leroith. (2012). Pathophysiology of the metabolic syndrome: implications for the cardiometabolic risks associated with type 2 diabetes. The American journal of the medical sciences. 343(1): 13-16.

[7] E.C. Stefanadi, G. Dimitrakakis, C.-K. Antoniou, D. Challoumas, N. Punjabi, I.A. Dimitrakaki, S. Punjabi, C.I. Stefanadis. (2018). Metabolic syndrome and the skin: a more than superficial association. Reviewing the association between skin diseases and metabolic syndrome and a clinical decision algorithm for high risk patients. Diabetology & metabolic syndrome. 10: 9.

[8] K.G. Alberti, R.H. Eckel, S.M. Grundy, P.Z. Zimmet, J.I. Cleeman, K.A. Donato, J.-C. Fruchart, W.P.T. James, C.M. Loria, S.C. Smith Jr. (2009). Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation;

international atherosclerosis society; and international association for the study of obesity. Circulation. 120(16): 1640-1645.

[9] I. Podder, K. Agarwal, A. Anurag. (2021). Metabolic status, obesity, and quality of life in patients with acne vulgaris: a cross-sectional case-control study. Indian journal of dermatology. 66(2): 223.

[10] M. Nagpal, D. De, S. Handa, A. Pal, N. Sachdeva. (2016). Insulin resistance and metabolic syndrome in young men with acne. JAMA dermatology. 152(4): 399-404.

[11] M. Del Prete, M.C. Mauriello, A. Faggiano, C. Di Somma, G. Monfrecola, G. Fabbrocini, A. Colao. (2012). Insulin resistance and acne: a new risk factor for men? Endocrine. 42: 555-560.

[12] I. Balta, O. Ekiz, P. Ozuguz, I. Ustun, S. Karaca, S. Dogruk Kacar, M. Eksioglu. (2015). Insulin resistance in patients with post-adolescent acne. International journal of dermatology. 54(6): 662-666.

[13] R.N. Smith, N.J. Mann, A. Braue, H. Mäkeläinen, G.A. Varigos. (2007). The effect of a high-protein, low glycemic–load diet versus a conventional, high glycemic–load diet on biochemical parameters associated with acne vulgaris: A randomized, investigator-masked, controlled trial. Journal of the American Academy of Dermatology. 57(2): 247-256.