

Short Communication

Association of Impaired Fasting Glycaemia with Acne Vulgaris among Sudanese Population, 2017

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Abstract

Background: Acne Vulgaris is a chronic skin disease involving blockage and/or inflammation of pilosebaceous units. The disease has multifactorial etiology with variable prognosis and great psychological impact. The study aimed to evaluate the effect of impaired fasting glycaemia (IFG) on acne vulgaris.

Materials and Methods: A case control study was conducted with the recruitment of a total of 60 subjects. Participants were divided into two groups- 30 patients with acne vulgaris and 30 healthy individuals as matched controls. Blood samples were collected after overnight fasting and analyzed for blood glucose using chemical reagents. Informed consents were obtained from all participants in the study. Data were analyzed using appropriate statistical tests.

Results: Twelve patients with acne vulgaris (40%) had their fasting blood glucose levels above 110-126 mg/dl, specifying impaired fasting glycaemia; compared to only 10% of the controls. A p-value of 0.007 and an odd ratio of 6 indicated an association between impaired fasting glycaemia and acne vulgaris.

Conclusion and recommendations: The current study indicated a strong association between impaired fasting glycaemia (IFG) and acne vulgaris among Sudanese population.

INTRODUCTION

Acne vulgaris is a common chronic disease affecting pilosebaceous units (hair follicles and their accompanying sebaceous glands). Acne can present as non-inflammatory lesions, inflammatory lesions, or a mixture of both, affecting mostly the face but also the back and neck [1].

Acne develops from the following four factors: (1) follicular epidermal hyperproliferation with subsequent plugging of the follicle [2], (2) excess sebum production [3-6], (3) the presence and the activity of the commensal bacteria [7,8] *Cutibacterium acnes* (*Propionibacterium acnes*), and (4) inflammation [9].

Genetic predisposition plays a major role in the development of the disease [10]. Other aggravating factors like cosmetic agents and hair pomades [11], as well as certain medications [12] and diet [13-15] were also recognized.

Acne vulgaris is so common that it is referred to as a physiological condition. Almost 85% of young people between the ages of 12 and 24 years have acne, and while is most common in teenagers, acne affects 8% of adults aged 35 to 44 years. Acne in young adults may represent continuation of adolescent acne or development of late-onset disease [16].

Acne may cause long-lasting and detrimental psychological and physical effects. It is associated with depression and anxiety,

regardless of the disease severity. Furthermore, acne may cause permanent scarring which is difficult to correct [17,18].

The probable relationship between metabolic errors and certain cutaneous diseases has received the increasing attention of dermatologists within recent years.

As a result, numerous studies dealing principally with sugar metabolism and uric acid in such dermatoses as acne vulgaris have made their appearance in dermatologic literature. Association of acne with hyperglycemia has been surprisingly investigated very early. One study reported hyperglycemia in acne vulgaris [19]. Another study showed high blood glucose in over 50% of their 83 patients with the disease [20].

Similar studies concluded that insulin resistance seems to play the main role in the development of acne. Accordingly, insulin resistance could represent an effective target for therapy [21, 22]. These studies reported an association between high-glycemic diet and acne. The aim of this study was to compare impaired fasting glycaemia between acne vulgaris patients and healthy subjects.

MATERIALS AND METHODS**Study design**

This as a cross sectional study approach on patients with acne

vulgaris. It was conducted at the clinical chemistry laboratory, Faculty of Medicine, Alneelain University from November 2016 to June 2017. Patients were enrolled based on the following criteria: all acne vulgaris patients, both genders, aged 12-24 years. Patients with comorbidities or taking medications were excluded.

Study area and population

Thirty patients with acne vulgaris (23 females, 7 males) aged 12-24 years; and other 30 healthy subjects (matched for age and sex) were included in the study. Patients were selected from those residing in Khartoum State (Sudan) and attending Khartoum Dermatology and Venereology Hospital after fulfillment of inclusion criteria. All subjects signed informed consents and filled questionnaires. The study was ethically approved.

METHODOLOGY

All subjects were fasted over night and morning venous blood samples were taken. After plasma separation, two milliliters were put in a fluoride oxalate-containing tube for fasting blood sugar using the glucose oxidase reagent. Tests of independency (Chi-square and Fisher tests) were used to analyze categorical data. P value was considered significant when it is less than or equal to 0.005.

RESULTS

Out of thirty patients included in the study, 23 were females (76.7%) and 7 were males (23.3%). The main fasting blood glucose for patients with acne vulgaris was 103.37 mg/dl compared to 90.0 mg/dl for the controls. The majority of patients (18, 60%) had normal blood glucose levels while the remaining patients (12, 40%) had impaired fasting glycaemia. Only 3 patients (10%) of the control group had impaired fasting glycaemia (Table 1).

DISCUSSION & RECOMMENDATIONS

It is well established that drugs like steroids, certain cosmetics and diet all predispose to the development of acne vulgaris. The aim of the current study was to evaluate the role of impaired fasting glycaemia in acne vulgaris. In this study, the majority of patients were females with an approximate ratio of 3:1. This finding agreed with the assumption that females are more commonly affected than males during adulthood. Potential pathophysiological explanations; such as estrogen levels which based upon female gender, may apply to this group.

Table 1: Impaired fasting glycaemia in acne patients versus controls.

| | | | Case | | Total |
|-------------------------------|---------------|---------------|---------|---------|-------|
| | | | Control | Patient | |
| Fasting blood glucose (mg/dl) | Less than 110 | Count | 27 | 18 | 45 |
| | | % within case | 90% | 60% | 75% |
| | More than 110 | Count | 3 | 12 | 15 |
| | | % within case | 10% | 40% | 25% |
| Total | | Count | 30 | 30 | 60 |
| | | % within case | 100% | 100% | 100% |

The mean fasting blood glucose levels for patients with acne vulgaris was 103.37 mg/dl compared to 90.0 mg/dl for the control group. Statistical analysis showed a significant difference at p-value of 0.001. Accordingly, patients with acne vulgaris tend to have higher levels of fasting blood sugar compared to the control subjects.

Levels of fasting blood glucose between patients and controls were significantly different at a p-value of 0.007 and the odd ratio of 6, meaning that the chance of a patient with impaired fasting glycaemia is 6 times subjects with normal blood glucose levels. The study agreed with both of Schwartz et al. and Levin and Khan. Further broader studies are required to address whether impaired fasting glycaemia maybe considered as a risk factor for acne vulgaris and whether patients with acne vulgaris may benefit from oral hypoglycemic agents.

REFERENCES

- Dawson AL, Dellavalle RP. Acne vulgaris. *BMJ*. 2013; 346: 2634.
- Noris JF, Cunliffe WJ. A Histological and immunocytochemical study of early acne lesions. *Br J Dermatol*. 1998; 118: 651-9.
- Pochi PE, Strauss JS. Sebaceous gland activity in black skin. *Dermatol Clin*. 1998; 6: 349-51.
- Lucky AW, Biro FM, Simbartl LA, Morrison JA, Sorg NW. Predictors of severity of acne vulgaris in young adolescent girls: result of a five-year longitudinal study. *J Pediatr*. 1997; 130: 30-9.
- Zouboulis CC, Böhm M. Neuroendocrine regulation of sebocytes – a pathogenetic link between stress and acne. *Exp Dermatol*. 2004; 13: 31-5.
- V Bataille, H Snieder, A J MacGregor, P Sasieni, T D Spector. The influence of genetics and environmental factors in the pathogenesis of acne: a twin study of acne in women. *J Invest Dermatol*. 2002; 119: 1317-1322.
- Jenny Kim, Maria-Teresa Ochoa, Stephan R Krutzik, Osamu Takeuchi, Satoshi Uematsu, Annaliza J Legaspi, et al. Activation of toll-like receptor 2 in acne triggers inflammatory cytokine responses. *J Immunol*. 2002; 169: 1535-41.
- Webster GF. Inflammatory acne represents hypersensitivity to propionibacterium acnes. *Dermatology*. 1998; 196: 80-1.
- Jeremy AH, Holland DB, Roberts SG, Thomson KF, Cunliffe WJ. Inflammatory events are involved in acne lesion initiation. *J Invest Dermatol*. 2003; 121: 20-7.
- Goulden V, McGeown CH, Cunliffe WJ. The familial risk of adult acne: a comparison between first-degree relatives of affected and unaffected individuals. *Br J Dermatol*. 1999; 141: 297-300.
- Cunliffe JW, Holland DB, Jeremy A. Comedone formation: etiology, clinical presentation and treatment. *Clin Dermatol*. 2004; 22: 367-74.
- Silverberg JI, Silverberg NB. Epidemiology and extracutaneous comorbidities of severe acne in adolescence: a US population-based study. *Br J Dermatol*. 2014; 170: 1136-1142.
- Spencer EH, Ferdowsian HR, Barnard ND. Diet and acne: a review of the evidence. *Int J Dermatol*. 2009; 48: 339-347.
- Adebamowo CA, Spiegelman D, Berkey CS, F William Danby, Helaine H Rockett, Graham A Colditz, et al. Milk consumption and acne in teenaged boys. *J Am Acad Dermatol*. 2008; 58: 787-793.
- Smith RN, Mann NJ, Braue A, Makelanian H, Varigos GA. The effect of

- a high protein diet, low glycemic load diet versus a conventional high glycemic load diet on biochemical parameters associated with acne vulgaris: a randomized, investigator-masked, controlled trial. *J Am Acad Dermatol.* 2007; 57: 247-56.
16. Collier CN, Harper JC, Cafardi JA, Cantrell WC, Wang W, Foster KW, et al. The prevalence of acne in adults 20 years and older. *J Am Acad Dermatol.* 2008; 58: 56-9.
17. Barnes LE, Levender MM, Fleischer AB Jr, Feldman SR. Quality of life measures for acne patients. *Dermatol Clin.* 2012; 30: 293-300.
18. Gollnick H, Cunliffe W, Berson D, Dreno B, Finlay A, Leyden JJ, et al. Management of acne: a report from Global Alliance to Improve Outcomes in Acne. *J Am Acad Dermatol.* 2003; 49: S1-37.
19. Schwartz HJ. sugar metabolism inn acne vulgaris. *J Cutan Dis.* 1916; 34: 159.
20. Albert S, Melvin AS. Sugar metabolism in acne vulgaris. *Arch Derm Syphilol.* 1929; 20: 705-711.
21. Michela Del, Maria Chiara Mauriello, Antongiulio Faggiano, Carolina Di Somma, Giuseppe Monfrecola, Gabriella Fabbrocini, et al. Insulin resistance and acne: a new risk factor for men? *Endocrine.* 2012; 42: 555-560.
22. Nazan Emiroğlu, Fatma Pelin Cengiz, Funda Kemeriz. Insulin resistance in severe acne vulgaris. *Postep Derm Alergol.* 2015; 32: 281-285.

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