Evaluation of the Levels of Serum Testosterone and Some Lipid Profile in Patients with Nodulocystic Acne

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ABSTRACT

Background: Acne is a chronic inflammatory skin disease that occurs when dead skin cells and sebum close hair follicles. It is characterized by seborrhoea, the formation of open and closed comedones (primary lesion of acne), erythematous papules and pustules and possible scarring. Acne mainly affects skin areas with a large number of sebaceous glands, these areas include the face, back and upper chest. The outcome appearance may lead to anxiety, reduced self-esteem, in severe cases, depression and suicidal thoughts. Genetic factors play an important role (as a primary cause of acne) in about 80% of patients. The role of sunlight exposure, cigarette smoking and diet is still unclear. Overgrowth of Propionibacterium acnes (Gram-positive bacteria) which is normally found in the skin and hair follicles also play an important role in pathogenesis of acne. After puberty, in male and female, acne often appears to be an increase in hormones such as testosterone.

Objective: To assess levels of serum testosterone and some lipid profile in patients with nodulocystic acne.

Methods: This is an open, comparative, controlled investigative study. A total of sixty patients were enrolled in this study. Sixty patients were divided into 2 groups; each one consists of thirty patients: Group A: Nodulocystic acne patients. Group B: Acne vulgaris patients (pathological control).

Results: Analysis of the levels of cholesterol, triglycerides and androgen hormone (testosterone) in patients with nodulocystic acne patients and acne vulgaris patients. The results of the laboratory analysis between the two groups showed no significant difference.

Conclusion: The available results in our study showed no significant difference in the present hormonal level of testosterone and lipid profile between nodulocystic acne patients and acne vulgaris patients.

Keywords: Testosterone, Nodulocystic acne, Propionibacterium acnes.

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Acne is a chronic inflammatory skin disease that occurs when dead skin cells and sebum close hair follicles[1]. It is characterized by seborrhea, open and closed comedones (primary lesion of acne), erythematous papules and pustules and possible scarring[2-4]. Acne mainly affects skin areas with a large number of sebaceous glands, these areas include the face, back and upper chest[5]. The outcome appearance may lead to anxiety, reduced self-esteem and, in severe cases, depression and suicidal thoughts[6,7].

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Genetic factors play an important role (as a primary cause of acne) in about 80% of patient[3]. The role of sunlight exposure, cigarette smoking and diet is still unclear[8-9]. Over growth of Propionibacterium acnes (Gram-positive bacteria) which is normally found in the skin and hair follicles also play an important role in pathogenesis of acne. After puberty, in male and female, acne often appears to be an increase in hormones such as testosterone[10].

In 2015, acne was estimated to affect 633 million people all over the world, making acne the 8th most common disease worldwide[11,12]. In western countries, Acne affects 85% of teenagers, commonly in adolescence[13-15]. Low rates of acne are founded in rural areas[15,16]. Children (before puberty) and adults (after puberty) also can be affected by acne[17]. Nearly half of

patients, acne persists into their twenties and thirties and in some group continue into their forties[3].

Androgens hormones such as testosterone, dihydrotestosterone, de-hydroepiandrosterone sulfate (DHEAS) are genes regulator for sebaceous gland growth and sebum production[18]. The isozyme 5α-reductase type 1 is responsible for conversion of testosterone to 5α-dihydrotestosterone is expressed predominantly in skin[19]. Acne patients show higher activity of type 1, 5-α reductase, while higher levels of DHEAS is usually seen in pre-pubertal acne patients. DHEAS, especially in postmenopausal women, has been shown to regulate sebum production through indirect mechanisms[20]. Estrogen hormone has several mechanisms: Inhibition of androgen secretion, direct opposition effect on androgens, or modulation of genes responsible for sebaceous gland growth and function[21,22]. In patients with acne, decreased levels of estrogen have been found[23]. Low level of estrogens during the first few menstrual cycles at puberty suggested that exogenous estrogens have advantageous effect on acne[24].

Genetic factors, environmental factors, Cytokines have an important role in pathogenesis of acne (multifactorial). Tumor necrosis factor (TNF) -α-308 gene in acne susceptible patients might be a predisposing factor with no evident relation to its severity[25].

Acne develops as a result of an interplay between the following factors[26,27].

1. Follicular hyperkeratinization and follicular plugging.

2. Propionibacterium acne colonization of the follicle.

3. Excessive sebum production.

4. Release of inflammatory mediators.

Research for diet and nutrition, oxidative stress and genetics have been produced some interesting insights into acne development[28]. The key component in the pathogenesis of acne is the inflammation[29].

This study aims to assess levels of serum testosterone and some lipid profile in patients with nodulocystic acne.

Methods

This is an open, comparative, controlled, investigative study. A total of sixty patients were enrolled in this study. Sixty patients were divided into two groups; each of thirty patients: Group A; including nodulocystic acne patients. Group B; including Acne vulgaris patients (pathological control).

The study was conducted in the department of Dermatology and Venereology in Salah Alden General Hospital, during the period between January and September 2017. The diagnosis was done clinically by the dermatologist. The sixty patients were included in this study from both genders, their ages were ranged from (14-35 years) mean ±S.D (23.8 ±5.093). Acne vulgaris patients that were receiving or treated with hormones, anti-lipid drugs, steroids are excluded from the study. Also, we excluded obese patients, hirsute, pregnant and lactating women and patients with known history of cardiovascular diseases or lipid disorder.

The evaluation of the sixty patients with acne was including gender, age, onset, duration and grading of acne. Serum testosterone and lipid profile were analyzed according to the results of laboratory tests ordered in outpatient department of dermatology of Salah Al den General Hospital. Lipid profile was including only cholesterol and triglycerides according to availability of investigations in the hospital.

Estimation of Cholesterol: (The test was conducted on fasting patients). Reagent 1 (Buffer solution) phosphate buffer. Reagent 2 (Enzymes) Cholesterol oxidase, Cholesterol esterase. Preparation of working solution by dissolving reagent 2 (powder) in bottle of reagent 1 (Buffer solution) then keep at 2-8 °C. This working solution, at this temperature is stable for 8

months. Mix and incubate for 10 min at 37°C, then read the absorbance in spectrometer at 505 nm. The color is stable 30 minutes.

Estimation of Triglycerides: (The test were conducted on fasting patients). Reagent 1 (Buffer solution) p-chlorophenol. Reagent 2 (Enzymes) 4-aminoantipyrine. Preparation of working solution is the same as cholesterol.

Estimation of testosterone: Into labeled tube, collect 4-5 mL of blood and allow it to clot. Centrifuge and remove the serum layer carefully. Store at -10°C or lower if the analyses are to be done at a later date, or at 4°C for up to 24 hours. No need for sample pretreatment (direct system assay).

Results

During the course of study, thirteen patients defaulted (for unknown reason). While the remaining sixty completed the study. Thirty patients (50%) were male and thirty patients (50%) were female. Sixty patients were included in this study from both genders, their ages were ranged from (14_35 years) mean ± S.D (23.8± 5.093).

According to grading:

Cholesterol: From analysis of cholesterol level, the level of cholesterol in nodulocystic acne patients (G1) mean ±SD (255.6 ±44.0) gave higher level than acne vulgaris patients (G2) mean ±SD (231.9 ±36.7), (Table 1). But the difference between nodulocystic acne patients and acne vulgaris patients was not significant.

Triglycerides: The level of triglycerides in nodulocystic acne patients (G1) mean ±SD (172.4 ±23.4) gave lower level than acne vulgaris patients (G2) mean ±SD (185 ±37.9), (Table 2). But the difference between nodulocystic acne patients and acne vulgaris patients was not significant.

Table 1: Results of cholesterol, triglyceride and testosterone according to the severity.

Parameter	Nodulocystic Acne mean ±S.D	Vulgaris Acne mean ±S.D	t-test
Cholesterol	255.6 ±44.0	231.9 ±36.7	0.60
Triglyceride	172.4 ±23.4	185 ±37.9	1.55
Testosterone	2.55 ±3.12	4.12 ±3.160	1.93

Table 2: Age and sex parameter for nodulocystic acne and acne vulgaris.

Parameter	Nodulocystic Acne	Vulgaris Acne
Age G1 = 15 -21 years G2 = 22 -28 years G3 = 29 -35 years **Mean 23.8** **SD ± 5.093** **No. 60**	30	30
Sex male 30 Female 30	21 18	9 12

The level of testosterone in nodulocystic acne patients (G1) mean ±S.D (2.55 ±3.12) gave lower level than acne vulgaris patients (G2) mean ±SD (4.12 ±3.160). According to table 1, the difference between nodulocystic acne patients and acne vulgaris patients is not significant.

Discussion

The results of the present study showed there was no significant difference of lipid profile and testosterone levels between nodulocystic acne patients and acne vulgaris patients.

Statistically using t test and P-value not indicating any difference in lipid profile and testosterone levels between nodulocystic acne patients and acne vulgaris patients.

Minimal reports are available on the relationship between blood lipids such as cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) and acne. Most of available literature was about

the effect of drugs used in acne treatment on these parameters[31]. Androgens represent the most important of all hormones regulating sebum production. This androgen-dependent secretion of sebum is mediated by potent androgens such as testosterone and DHT and weaker androgens[31].

From analysis of cholesterol level, the level in nodulocystic acne patients (G1) (mean 255.6) gave higher level than acne vulgaris patients (G2) (mean 231.9), according to Table 1. This result may be due to use of systemic retinoids which is used for treatment of severe acne, this is agree with a study about systemic retinoid in Oman[32]. Or may be due to the effect of a fatty meals, high-protein and high glycemic diet, which is agree with a study which compare the effect of an experimental low glycemic–load diet with a conventional high glycemic–load diet on clinical and endocrine aspects of acne vulgaris. This study suggests nutrition-related lifestyle factors play a role in acne pathogenesis[33]. According to Tables 1 and 2 difference between nodulocystic acne patients and acne vulgaris patients (triglycerides and cholesterol) is not significant.

This is agree with a study of one hundred and sixty-six acne patients and 105 age and sex matched healthy control. The age of both, acne patients and controls was ranged from 13 to 42 years. Plasma cholesterol analysis showed that there were no significant differences in plasma total cholesterol levels between acne patients and controls[30]. Also, triglycerides plasma levels also showed no significant difference in both sexes between acne patients and controls[30].

According to Table 1, the level of Testosterone in nodulocystic acne patients (G1) (mean 2.55) gave lower level than acne vulgaris patients (G2).

This disagreed with a study that was conducted in Hilla city, 2009.

The control group includes forty-one apparently healthy individuals, after having been asked about their health.

Serum testosterone was significantly higher in moderate acne and in severe acne patients compared with control group in both genders[34].

Also disagreed with study in Erbil city, included two groups, the control and the study groups. Each group consisted of 60 individuals. The mean age was 20.82 years for study group and 20.75 years for control group. The study showed presence of a significant association between serum testosterone level and acne vulgaris in female patients[35].

In conclusion: The available results in our study showed there was no significant difference in hormonal level (testosterone) and lipid profile (cholesterol and triglycerides) between nodulocystic acne patients and acne vulgaris patients.

Patients with nodulocystic acne need to test for triglycerides when there's arrangement to use systemic retinoid, to follow the hypertriglyceridemia that proved to occur during treatment with systemic retinoid.

Previous beliefs of severe acne is due to high hormonal level of testosterone, in this current study, no significant differences in testosterone levels between nodulocystic acne patients and acne vulgaris patients.

Hormonal therapy against hyper-androgenemia will give no benefit for nodulocystic acne patients than systemic retinoid due to normal level of testosterone in these patients.

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