ORIGINAL ARTICLE

Evaluation of Serum Testosterone Level in Male Patients with Tinea Corporis and Cruris versus Normal Subjects: A Comparative Study.

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ABSTRACT

Background: Several pathogenic fungi are known to interact and respond to various mammalian hormones. These interactions can influence the growth, and even the pathogenesis of the organism.

Aim of work: This study was designed to measure serum testosterone among patients with tinea corporis and cruris in comparison to normal subjects to evaluate its role in pathogenesis and progression of the disease

Patients and Methods: A total of 90 subjects, 30 male patients with tinea corporis, 30 male patients with tinea cruris and 30 age and gender matched healthy volunteers served as controls were enrolled in the study. After history taking and clinical examination, mycological study was done in the form of direct microscopy and fungal culture, then blood samples were withdrawn for serological evaluation of serum testosterone.

Results: Mean total testosterone serum level was significantly decreased in patients with tinea corporis and cruris than in controls but there was no statistically significant relation between species of dermatophytes and the serum levels of testosterone.

Conclusion: These results suggest a negative impact of dermatophytes infection on the total testosterone serum level.

INTRODUCTION

Fungal infections or mycoses are becoming common diseases. With the ease of worldwide travel, mycoses that were previously regarded as geographically limited can now be seen in any part of the world. Furthermore, in recent years the number of fungi recognized as human pathogens has risen, caused partly by an increasing population of immunocompromised patients and advances in molecular diagnosis.[1]

Fungi are unicellular or multicellular, eukaryotic, heterotrophic microbes. Each fungal cell contains a full array of organelles and is bound by a rigid cell wall containing chitin, glucan, and/or cellulose. Of the thousands of fungal species that are free-living in nature are pathogenic for plants, only a small group is known to be pathogenic for humans and animals.[2]

Superficial fungal infections are among the world's most common diseases and the prevalence of superficial mycotic infections has risen to such a level that skin mycoses affect more than 20-25% of the world's population. Dermatophyte infections are one of the earliest known fungal infections of mankind and are very common throughout the world.[3]

The prevalence of dermatomycoses or tinea infections has been studied in different parts of the world. The relative occurrence of the etiologic agents of these infections varies from

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country to country and from one climatic region to another.[4]

Dermatophytosis is common in tropical countries and may reach epidemic proportions in areas with high rate of humidity, over population and poor hygienic conditions.[5]

Dermatophytes have been divided into three groups, anthropophilic, zoophilic, and geophilic dermatophytes. The affinity of each of these groups to humans varies. For example, anthropophilic dermatophyte species are primarily associated with humans and rarely infect other animals. Zoophilic dermatophyte species commonly infect animals or are associated with animals, but occasionally infect humans. Geophilic dermatophyte species are primarily associated with keratinous materials, such as hair, feathers and horns, once they are dissociated from living animals and are involved in the process of decomposition. This type of dermatophyte may infect humans and animals through contact with soil.[6]

There are three closely related genera of dermatophytes: Trichophyton, Microsporum and Epidermophyton. The classic form of this mycosis is ringworm, a circular lesion with an active border, inflammation, pruritus and scaling.[7]

Though various species of dermatophytes produce clinically characteristic lesions; a single species may produce variety of lesions depending upon site of infection.[8]

Tinea corporis is a superficial dermatophyte infection characterized by either inflammatory or noninflammatory lesions on the glabrous skin (i.e., skin regions other than the scalp, groin, palms and soles).[9]

Tinea corporis is a common infection more often seen in typically hot, humid climates. *Trichophyton rubrum* is the most common infectious agent in the world and is the source of 47% of tinea corporis cases.[10]

Classically, tinea corporis manifests as well-bordered, erythematous, scaly, annular plaques widening from the center towards the periphery and having elevated borders. Itchiness is a frequently accompanying symptom. Sometimes, vesicles and pustules are observed. Rarely, even blister formation as a secondary change of severe inflammation might be observed.[11] As a result of the inflammation, scale, crust, papules, vesicles, and even bullae can develop, especially in the advancing border.[12]

Tinea cruris, or jock itch, occurs on the medial and upper area of the thighs and groin area and is more common in males than in females.[13] This disease is more often seen in men than in women, since the scrotum provides a warm and moist environment that encourages fungal growth and men are more likely to have tinea pedis and onychomycosis as a source of dermatophytes.[14] Signs of excessive moisture, pruritus and burning are often present.[15]

Dermatophyte infection, which might be observed at any age, spreads via direct contact with an infected person or animal, or indirectly via contaminated belongings. Also, spread via autoinoculation from a dermatophytical infection located in another body region is commonly observed.[11]

Physiological mediators of human host that interfere with pathogenic fungi are of particular interest in clinical mycology. An example for such mediators is steroid hormones.[16]

The influence that hormones may have on mycotic infections in man has always been an intriguing question. Renewed attention was

drawn to this issue by the detection of fungal receptors for human hormones. It was also shown that the growth of yeasts as well as of dermatophytes can be influenced by steroid hormones *in vitro*. In human skin, the metabolism of such hormones within the pilosebaceous units is of particular interest to dermatomycoses. Among other steroids, androgenic hormones can be derived from metabolic pathways within follicular tissue.[17]

It has been shown that some fungi use "message molecules" including hormones to elicit certain responses, especially in the sexual cycle.[18] In some organisms, specific receptors for endogenous hormones have been demonstrated and various fungi interact with mammalian hormones.[19]

Fungi also utilize hormones as messenger molecules that regulate various activities of the organism. Primarily, these molecules are related to the control of sexual reproduction in various fungi, and take the form of steroids, peptides, and acid derivatives.[20]

Dermatophytic fungi have been demonstrated to be inhibited by the presence of various steroids including androgens and progesterone. The primary and most well-known androgen is testosterone.[21]

The aim of the present work was to measure serum testosterone among patients with tinea corporis and cruris in comparison to normal subjects to evaluate its role in pathogenesis and progression of the disease.

SUBJECTS AND METHODS

This comparative case-control study was conducted at Al-Hussein University Hospital, Department of Dermatology and Venereology, Faculty of Medicine, Al-Azhar University over a period of 8 months from April 2017 to November 2017. After approval of the Medical Research Ethics Committee of Al-Azhar University, informed written consents were obtained from all the participants in the study after explaining the nature of the study to them.

This study included ninety individuals:

Patients

Thirty male patients with tinea corporis and thirty male patients with tinea cruris were randomly chosen to participate in the study.

Controls

The control group comprised thirty age and gender matched healthy volunteers. The controls had no history of any skin abnormalities or any chronic-debilitating disease.

Patients were selected based on the following criteria:

Inclusion Criterion

Any patient with clinical diagnosis consistent with tinea corporis or tinea cruris.

Exclusion Criteria

1. Age below 16 and above 40 years.
2. Patients who had received any topical antifungals within the last two weeks and/or systemic antifungals during the last four weeks prior the study.
3. Patients on drugs that can affect the level of androgenic hormones such as: ketoconazole, hormonal replacement therapy, statins, opioids, anabolic steroids and chemotherapy.
4. Presence of other conditions causing or denoting change in the level of the androgenic hormones such as: hyperthyroidism, diabetes mellitus, metabolic syndrome, adrenal

gland tumors, congenital adrenal hyperplasia, hormone-dependent malignancy, precocious puberty and hypogonadism.

METHODS

The ninety subjects were divided into three groups:

Group I: Thirty male patients with tinea corporis.

Group II: Thirty male patients with tinea cruris.

Group III: Thirty age and gender matched healthy volunteers as a control group.

All subjects incorporated in the study were subjected to the following:

A. Careful History Taking: Demographic data such as age, course, duration, occupation, previous attacks, medical history, sexual history, family history, history of animal exposure and history of drugs and diseases that affect the testosterone level.

B. Thorough General Examination: To exclude signs of primary hypogonadism [such as gynecomastia, excessive growth of the arms and legs in relation to the trunk (span more than height), decrease in beard and body hair] and to exclude signs of other diseases that may affect testosterone level.

C. Dermatological Examination: To determine the extent and characteristics of the lesion.

D. Mycological Study:

For every patient, duplicate sets of skin scrapings were collected from the lesions. One set of skin scrapings was examined by direct microscopy and the other by culture. All cultures were done on Petri dishes.

DIRECT MICROSCOPY

- After cleaning the lesion with 70% alcohol, skin scales were collected by scraping the affected site. The specimen was placed on a clean glass slide, and a drop of 20% Potassium hydroxide (KOH) was added. A coverslip was applied with gentle pressure to drain away excess solution. The sample was then examined thoroughly for the presence of filamentous, septate, branched hyphae with or without arthrospores.

Fungal culture

- The second set of scrapings was inoculated onto two types of Sabouraud dextrose agar (SDA) culture media: one with cycloheximide (to suppress the growth of contaminant fungi) and the other without cycloheximide. Chloramphenicol was added to both culture media to prevent bacterial overgrowth. The media were then incubated in a warm, moist environment at 28°C and examined regularly to detect growth of any fungus. Observation for growth was done periodically up to 4 weeks, after which the cultures were reported as positive or negative.
- Regarding the identification of dermatophytes, we relied upon macromorphological and micromorphological characters of the isolates. The fungi were identified by noting their growth rate, colonial macroscopic morphology and microscopic structures. Macroscopic examination included color, size, texture and topography of the colony. The microscopic structures of fungi usually provide definitive identification. Using the tease mount technique, the following microscopic features were looked for: the type, size, shape and arrangement of spores and the size, color, septation and special shapes

of hyphae.

E. Serological Evaluation of Serum Testosterone

Sample Collection

- Concerning the circadian hormone variation, all blood samples were taken between 8:00 A.M. to 10:00 A.M, when the hormone level is maximum.
- 3 ml venous blood was withdrawn from patients as well as control subjects under complete aseptic condition using a wide-bore syringe to avoid hemolysis of the red blood corpuscles.
- Samples were allowed to clot completely (within 30-60 minutes) at the room temperature.
- Centrifugation was done at 5,000 rpm for 5-10 minutes to separate the serum.
- The separated serum was stored at -20 °C in order to keep the serum stability until analyzed.

PROCEDURE

Testosterone assay by ELISA, Catalog Number: 10007 (Chemux Bioscience, Inc. South San Francisco, CA 94080, USA).

Principle of the test

The testosterone EIA is based on the principle of competitive binding between testosterone in the test specimen and testosterone-HRP (horseradish peroxidase) conjugate for a constant amount of rabbit anti-testosterone. In the incubation, goat anti-rabbit IgG-coated wells are incubated with 10 µl of testosterone standards, controls, patient samples, 100 µL testosterone-HRP conjugate reagent and 50 µL rabbit anti-testosterone reagent at 37°C for 90 minutes.

During the incubation, a fixed amount of HRP-labeled testosterone competes with the endogenous testosterone in the standard, sample, or quality control serum for a fixed number of binding sites of the specific testosterone antibody. Thus, the amount of testosterone peroxidase conjugate immunologically bound to the well progressively decreases as the concentration of testosterone in the specimen increases.

Unbound testosterone peroxidase conjugate is then removed, and the wells washed. Next, a solution of Tetramethylbenzidine (TMB) Reagent is then added and incubated at room temperature for 20 minutes, resulting in the development of blue color. The color development is stopped with the addition of 2 N HCl, and the absorbance is measured spectrophotometrically at 450 nm. The intensity of the color formed is proportional to the amount of enzyme present and is inversely related to the amount of unlabeled testosterone in the sample. A standard curve is obtained by plotting the concentration of the standard versus the absorbance. The testosterone concentration of the specimens and controls run concurrently with the standards can be calculated from the standard curve. Reference range for male testosterone was 3-10 ng/ml.

Data management and statistical analysis

The collected data was revised, coded, tabulated and introduced to a PC using Statistical package for Social Science (SPSS 15.0.1 for windows; SPSS Inc, Chicago, IL, 2001). Quantitative data were expressed as mean ± standard deviation (SD). Qualitative data were expressed as frequency and percentage.

The following tests were done

Chi-square test (X^2): was used for comparing non-parametric data.

Independent T-test: was used for comparing between two means.

A one-way analysis of variance (ANOVA): when comparing between more than two means.

Post Hoc test (Least significant difference LSD): was used for multiple comparisons between different variables.

Probability (P-value)

- P-value <0.05 was considered significant.
- P-value <0.001 was considered as highly significant.
- P-value >0.05 was considered insignificant.

RESULTS

This study included 30 male patients with T. corporis (group I), 30 male patients with T. cruris (group II) and 30 healthy male controls (group III).

The age ranged from 16 to 40 years (mean ± SD = 27.5 ± 7.2 years in group I; 27.4 ± 7.08 years in group II and 29.03 ± 6.8 years in group III). There was no statistically significant difference between studied groups as regard age (P-value > 0.05). (Table 1; Fig. 1).

The duration of the disease ranged from 3-60 days (mean ± SD = 19.4 ± 15.3 in group I, 19.1 ± 14.5 in group II). There was no statistically significant difference between groups I and II as regard duration of disease (P-value > 0.05). (Table 2; Fig. 2).

As regard mean serum total testosterone level, there was a statistically significant difference between each of group I and II in comparison to the control group (P < 0.001), while there was no significant difference between group I and II (Table 3; Fig. 3).

Table 1 Comparison between studied groups as regard mean age

Group Parameter	Group I N = (30)	Group II N = (30)	Group III N = (30)	ANOVA	
	Mean ± SD	Mean ± SD	Mean ± SD	F	P-value
Age (years)	27.5 ± 7.2	27.4 ± 7.08	29.03 ± 6.8	0.4	0.6
Least significance difference (LSD)					
	Group I vs Group II	Group I vs Group III	Group II vs Group III		
LSD	0.1	1.4	1.6		
P-value	0.9	0.4	0.3		

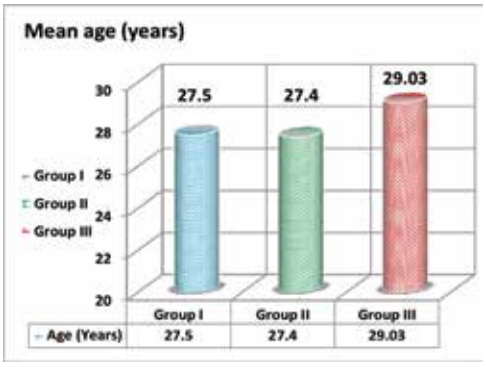

Fig. 1 Comparison between studied groups as regard mean age.

Table 2 Comparison between group I and II as regard mean duration of disease

Group parameter	Group I N = (30)	Group II N = (30)	T-test	
	Mean ± SD	Mean ± SD	T	P-value
Duration (days)	19.4 ± 15.3	19.1 ± 14.5	0.08	0.9

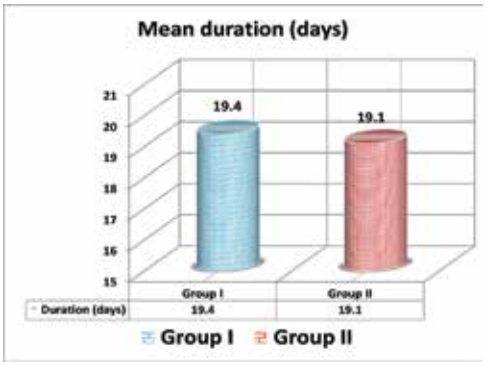

Fig. 2 Comparison between group I and II as regard mean duration of disease.

Table 3 Comparison between studied groups as regard mean total testosterone

Group parameter	Group I N = (30)	Group II N = (30)	Group III N = (30)	ANOVA	
	Mean ± SD	Mean ± SD	Mean ± SD	F	P-value
Testosterone (ng/dl)	4.7 ± 1.2	3.9 ± 1.5	7.4 ± 2.5	31.1	**< 0.001***
Least significance difference (LSD)					
	Group I vs Group II	**Group I vs Group III**	**Group II vs Group III**		
LSD	10.8	2.7	3.5		
P-value	0.09	**< 0.001***	**< 0.001***		

*: P-value < 0.001 is considered significant.

Table 3 shows:

- Statistically significant difference between studied groups as regard mean total testosterone (P-value < 0.001).
- Statistically significant difference between group I and III as regard mean total testosterone (P-value < 0.001).
- Statistically significant difference between group II and III as regard mean total testosterone (P-value < 0.001).
- No statistically significant difference between group I and II as regard mean total testosterone (P-value > 0.05).

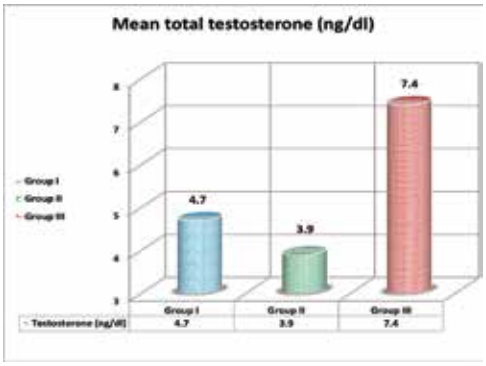

Fig. 3 Comparison between studied groups as regard mean total testosterone.

There was no statistically significant difference between mean serum testosterone and age and duration of disease in group I and II. Also in group III, there was no statistically significant difference between mean serum testosterone and age (P-value > 0.05) (Tables 4-6).

Table 4 Correlation between mean serum testosterone and age and duration of disease in group I

Parameters	(r)	P-value
Testosterone *(4.7 ± 1.2)* **vs age** *(27.5 ± 7.2)*	-0.03	0.8
Testosterone *(4.7 ± 1.2)* **vs duration of disease** *(19.4 ± 15.3)*	-0.006	0.9

(r): Pearson correlation coefficient

Table 5 Correlation between mean serum testosterone and age and duration of disease in group II

Parameters	(r)	P-value
Testosterone *(3.9 ± 1.5)* **vs age** *(27.4 ± 7.08)*	**0.3**	**0.08**
Testosterone *(3.9 ± 1.5)* **vs duration of disease** *(19.1 ± 14.5)*	**0.001**	**0.9**

Table 6 Correlation between mean serum testosterone and age in group III

Parameters	(r)	p-value
Testosterone *(7.4 ± 2.5)* **vs age** *(29.03 ± 6.8)*	0.1	0.4

Examination of the post-culture mounts in group I revealed *T. violaceum* in 12 patients (40%) followed by *T. rubrum* (10 patients; 33%), *M. audouinii* (3 patients; 10%), *M. canis* (2 patients; 7%), *T. verrucosum* (2 patients; 7%), and *T. tonsurans* (1 patient; 3%). While in group II mounts revealed *T. violaceum* in 15 patients (50%) followed by *T. rubrum* (11 patients; 37%), *T. verrucosum* (4 patients; 13%). There was no statistically significant difference between group I and II as regard isolated species (P-value > 0.05). (Table 7; Fig. 4-7).

Table 7 Comparison between group I and II as regard isolated species

Groups parameter	Group I N = (30)		Group II N = (30)		Chi-square	
	N	%	N	%	X^2	P-value
T. violaceum	12	40 %	15	50 %	7.04	0.2
T. rubrum	10	33 %	11	37%		
M. audouinii	3	10 %	0	0 %		
M. canis	2	7 %	0	0 %		
T. verrucosum	2	7 %	4	13 %		
T. tonsurans	1	3 %	0	0 %		

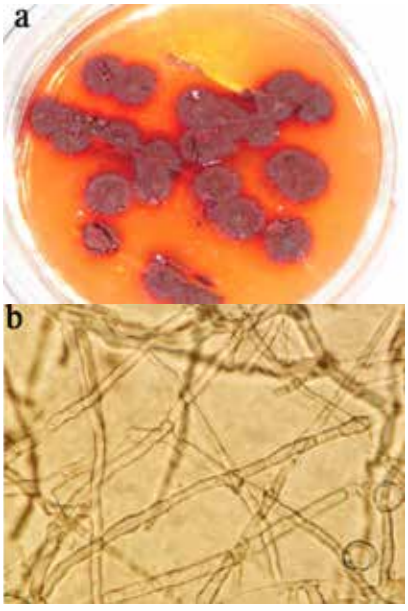

Fig. 4 Comparison between group I and II as regard isolated species.

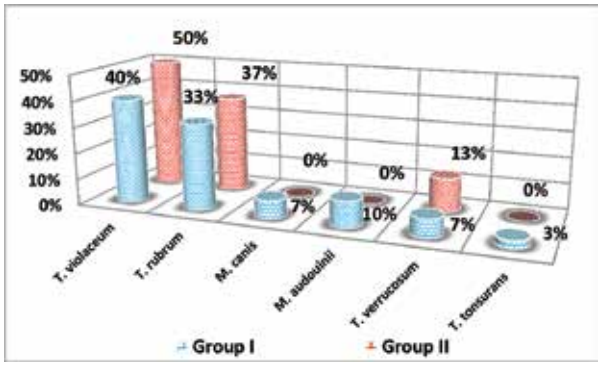

Fig. 5 (a) Macroscopic morphology of T. rubrum: colonies are flat to slightly raised, white to cream, suede-like to downy. (b) Microscopic morphology of T. rubrum: typical smooth thin-walled cigar-shaped septate macroconidia of T. rubrum (granular type) (water mount x100).

Table 8 Correlation between mean serum testosterone level and isolated species in group I and II

Groups parameter	Group I N = (30)	ANOVA		Group II N = (30)	ANOVA	
	Mean ± SD	F	P-value	Mean ± SD	F	P-value
T. violaceum	4.5 ± 1.6	0.8	0.5	3.7 ± 0.9	1.9	0.1
T. rubrum	3.7 ± 1.4			4.2 ± 1.9		
M. audouinii	4.1 ± 0.2			---		
M. canis	5.1 ± 1.4			---		
T. verrucosum	3.8 ± 1.1			4.7 ± 3.1		
T. tonsurans	4.5 ± 0.0			---		

Table 8 Shows no statistically significant difference between mean serum testosterone level as regard isolated species in both group I and II (P-value > 0.05).

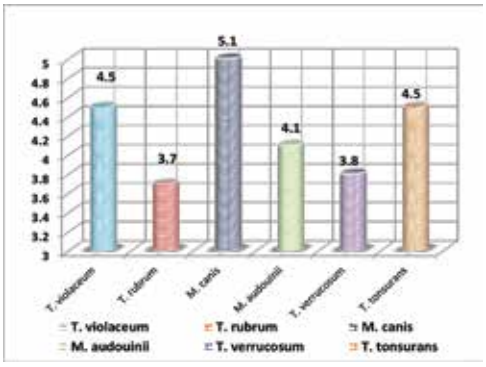

Fig. 6 Correlation between mean serum testosterone level and isolated species in group I.

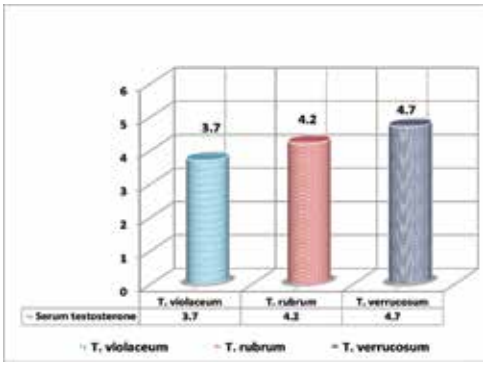

Fig. 7 Correlation between mean serum testosterone level and isolated species in group II.

In group I, 26 cases have irrelevant family history (87%) while only 4 cases (13%) showed positive family history of dermatophytes infection. In group II, 24 cases have irrelevant family history (80%) while only 6 cases (20%) showed positive family history. There was no statistically significant difference between group I and II as regard family history (P-value > 0.05) (Table 9; Fig. 8).

Table 9 Comparison between group I and II as regard family history

Group parameter	Group I N = (30)		Group II N = (30)		Chi-square	
	Positive	Negative	Positive	Negative	X^2	P-value
Family history	4 (13%)	26 (87%)	6 (20%)	24 (80%)	0.4	0.4

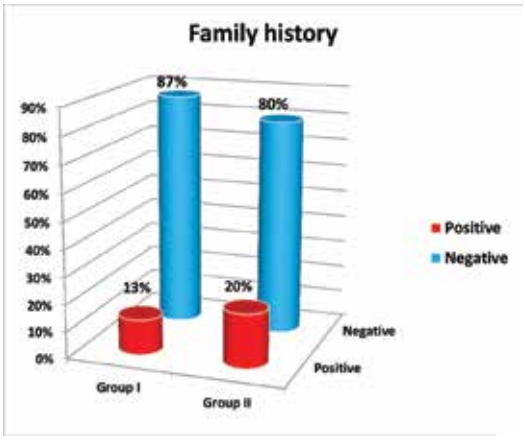

Fig. 8 Comparison between group I and II as regard family history.

In group I and II, 24 patients (80%) had no history of animal contact. In group I, 3 patients (10%) had a history of contact with dogs and 3 patients (10%) had a history of contact with cats, while in group II, 4 patients (13%) had a history of contact with dogs and 2 patients (7%) had a history of contact with cats (Table 10; Fig. 9).

Table 10 Description of animal contact in group I and II

Group parameter	Group I N = (30)			Group II N = (30)		
	Dogs	Cats	Nil	Dogs	Cats	Nil
Animal contact	3 (10%)	3 (10%)	24 (80%)	4 (13%)	2 (7%)	24 (80%)

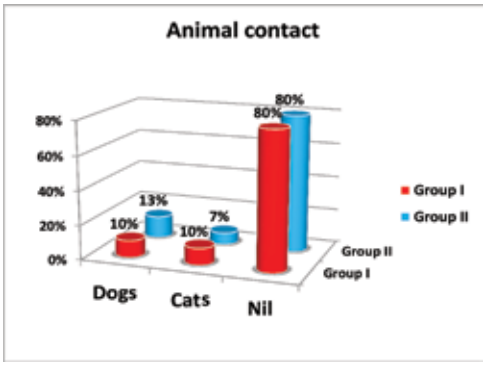

Fig. 9 Description of animal contact in group I and II.

Regarding the site distribution of the disease in group I, 12 cases (40%) showed affection of the extremities followed by face affection (10; 34%) while 4 cases (13%) showed neck affection and trunk being the least affected (2; 7%). Disseminated lesions were observed in only 2 cases (6%) (Table 11; Fig. 10-12).

Table 11: Distribution of lesions in group I.

Distribution of lesions	N	%
Extremities	12	40%
Face	10	34%
Neck	4	13%
Trunk	2	7%
Disseminated	2	6%

DISCUSSION

Dermatophytoses are fungal infections caused by three genera of fungi that have the unique ability to invade and multiply within keratinized tissue (hair, skin and nails). These fungi

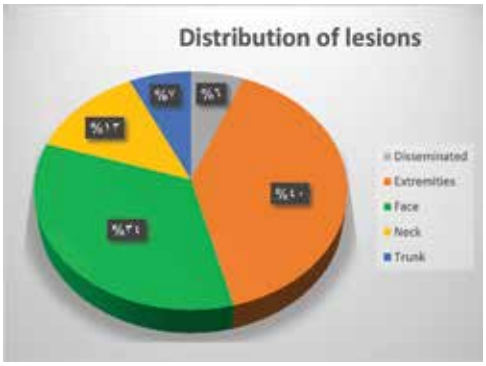

Fig. 10 Distribution of lesions in group I.

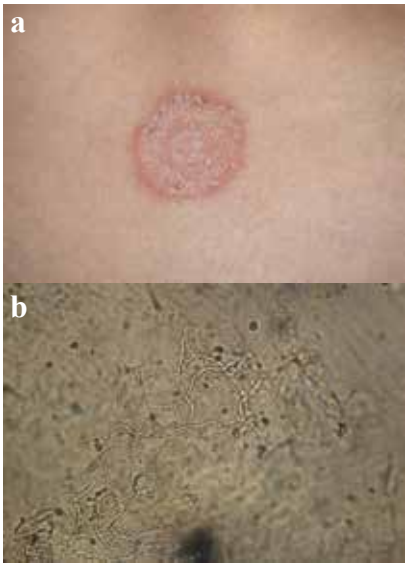

Fig. 11 (A) Typical lesions of tinea cruris involving the groin and sparing the scrotum. (B) Lateral view showing the active raised erythematous edge of the lesion.

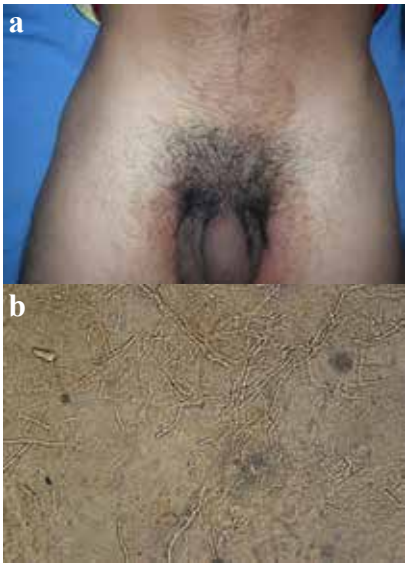

Fig. 12 Tinea cruris showing extension of infection to the lower abdomen. (b) Septated and branched hyphae observed in KOH preparation (x100).

"dermatophytes" are alike in their physiology, morphology and pathogenicity. The three genera are Microsporum, Trichophyton and Epidermophyton.[22]

Tinea corporis usually involves the trunk, limbs, and occasionally the face.[23] The infection commonly appears as annular, scaly patches or plaque with raised, scaling border and central clearing.[24] Tinea cruris is a dermatophyte infection of the inguinal region, in particular the inner aspects of the upper thighs and crural folds, with occasional extension onto the abdomen and buttocks.[22]

The finding of interactions of mammalian hormones with fungi and subsequent functional responses by the fungi, suggest that hormonal interactions with fungal systems has been conserved throughout evolution and have an important role in fungal pathogenesis, as well as in the overall biology of the organisms.[19]

Mammalian hormones, acting through fungal receptors, may affect pathogenicity. Fungal ligands, which may achieve high concentrations at the interface between individual fungi and individual host cells, may affect this interaction and thus affect pathogenicity. The ligands themselves are also of interest for their biological effects on the fungus and their possible clinical use.[25]

In this study, we were trying to investigate the possible relation between serum testosterone level and pathogenesis of tinea corporis (group I) and cruris (group II) versus healthy controls (group III).

As regard the age, all the subjects were at age between 16 and 40 years to avoid marked hormonal variation in relation to age between the selected individuals. The mean age of control

group was matching the patient groups age.

There was no statistically significant relation between age and serum levels of total testosterone. Also, there was no statistically significant relation between duration of the disease and serum levels of total testosterone. To the best of our knowledge, no available reports about the relation between serum testosterone level and age or duration among patients with dermatophytosis.

In our study, total testosterone serum level was significantly decreased in both groups (I and II) than in controls, but there was no statistically significant relation between species of dermatophytes and serum levels of total testosterone. This comes in agreement with our results. Also compatible with our results, Brasch and Gottkehaskamp, (1992) who concluded that progesterone, testosterone and estradiol proved to reduce fungal growth.[26]

El-Sherif and Refai, (1976),[27] found that many hormones including testosterone caused inhibition of growth of dermatophytes. Dermatophytic fungi have been demonstrated to be inhibited by the presence of various steroids including androgens and progesterone[17,25,28] An *in vitro* study was made by Chattaway and Townsley, (1962)[29] who reported inhibitory activity of testosterone on T. rubrum. Additionally, a dematiaceous fungus (*Phialophora verrucosa*) has also been demonstrated to be inhibited by the mammalian hormones progesterone and testosterone.[30]

In the present study, decreased testosterone in both groups of patients may be explained through the following:

- Androgenic hormones present within the pilosebaceous units of human skin have different inhibitory effects on the growth of some dermatophytes. These hormones are metabolized within human follicular tissue; therefore, it can influence the colonization of hair follicles by dermatophytes.[16]
- Receptor-mediated effects and an unspecific interference with fungal sterol metabolism are probable mechanisms of the fungal growth inhibition by steroidal hormones. However, it had been shown that some fungi could escape from this inhibitory effect by metabolizing these hormones to low potent derivatives.[16]

In this study, total testosterone serum level was lower in group II than group I. Although this was not a statistically significant decrease, it could be explained by the following:

- The most important metabolic pathway of androgenic hormones in skin is through activity of 5 α-reductase which metabolize testosterone to dihydrotestosterone.[31] Dihydrotestosterone has a low inhibitory effect on dermatophytes. The groin region contains a much higher activity of 5 α-reductase, compared with hair follicles and other parts of the skin.[32]
- Therefore, the high activity of 5 α-reductase in the skin of the groin can be a hypothesis for the high affinity for colonization of dermatophytes in the groin.

Of the 60 isolates in both group I and II, *T. violaceum* was recovered in 27 cases followed by *T. rubrum* in 21 cases. This result is similar to that of Ellabib et al., (2002) who isolated *T. violaceum* and *T. rubrum* as important causes of tinea corporis in Libya.[33]

Epidemiological studies carried out in Egypt reported high isolation rate of *T. violaceum*.[34] re-

ported that the most frequently isolated dermatophyte species was *T. violaceum* (71.1% of all recovered dermatophytes). Amer et al., (1981) demonstrated that *T. violaceum* was the most commonly isolated pathogen (44%) in cases of dermatophytosis in Al-Sharkia Governorate.[35] Also agreeing with this study, Ansari and Siddiqui (2006)[36] in their epidemiological study reported that *T. violaceum* was the most prevalent organism, isolated in 41% of cases.

The increasing incidence of *T. violaceum* as a major cause of tinea corporis in Egypt is probably because the fungus is indigenous to North Africa.[23] *T. violaceum* has been reported as the most common etiologic agent causing dermatophytosis in Egypt reported is *T. violaceum* was the chief isolate from scalp infection (eight out of 35 and 21 out of 33, respectively).[37-39]

In Egypt, large family sizes, close familial contact, and sharing of personal items such as hairbrushes, hats and towels are common. This, in addition to overpopulation, hot humid atmosphere and increased environmental exposure to fungi, may be responsible for the predominance of certain dermatophytes causing tinea infections. Dispersal of fungi from the patient's scalp or foot lesions to the trunk and groin or from sharing of nonpersonal objects can also occur.

In contrast, Bhavsar Hitendra et al., (2012)[40] isolated *T. rubrum* as the main dermatophyte (55.26%) from tinea corporis and tinea cruris cases, followed by *T. mentagrophytes* (27.63%) They failed to isolate any *T. violaceum* from skin lesions. Also, Abd Elmegeed et al.,(2015)[41] reported that *T. tonsurans* and *M. canis* were the main causative agents in cases of tinea corporis. Other studies also revealed that *T. rubrum* was the main dermatophyte isolated from ringworm lesions, particularly tinea corporis.[5,42,43] This difference in dermatophyte isolation could be explained by different samples of the study population and again emphasizes the role of different environmental conditions and degree of exposure to the pathogens. The predominance of certain dermatophytes varies geographically depending on ethnicity and different environmental factors such as climate, humidity, occupation and different lifestyles.

As *M. audouinii* is usually not identified or is rare to be isolated from dermatophytosis in Egypt, especially from tinea corporis, the isolation of *M. audouinii* in 10% of cases in group I is very interesting. The isolation of *M. audouinii* in Egypt could be explained by the fact that, because of its anthropophilic nature, the fungus is accessible to the general population who was exposed to it during their routine living conditions. This is particularly enhanced by the increased national and international immigration.

In group I of the present study, 40% of patients had skin lesions on extremities, 34% on the face, 13% on the neck and 7% on the trunk, while 6% had disseminated disease. This result is in accordance with the results obtained by Nejad et al., (2007)[44] who detected that the patients had tinea corporis lesions on upper limbs (33%), lower limbs (27%), face (13%) and trunk (9%) in descending manner. The variability of the sites affected by tinea corporis is meaningful as the most involved sites are related to the site of maximum exposure to sources of infection as extremities and face while the least involved sites are neck and the trunk which are more hidden and somewhat protected.

From this study, we can conclude that the se-

rum level of total testosterone was significantly lower in patients with tinea corporis and cruris than in healthy controls. This suggests that there might be a relation between the low serum testosterone levels and dermatophytoses.

Further studies on larger population to confirm correlation between cutaneous fungal infection and serum testosterone. Follow up of patients after treatment with re-evaluation of serum testosterone level. Trials for hormonal treatment with testosterone either topical or systemic in cases of infection with dermatophytoses.

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