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Evaluation of Serum UL16 Binding Protein 3 in Patients with Tinea capitis

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Abstract

Tinea capitis is a global public health concern, particularly in underdeveloped nations. Trichophyton and Microsporum are the only two genus of pathogens that cause the disease. NKG2D receptor ligands are found in the serum marker. A crucial regulator of both innate and adaptive immune responses, it plays a critical role. The marker is inactive in a healthy hair follicle. The dermal sheath and dermal papilla are markedly upregulated in the tissue marker expression, but not in control persons or those suffering from other inflammatory scalp conditions. Patients' scalp biopsy samples have also shown the presence of the tissue marker in hair follicles. The serum marker levels in individuals with TC were measured, and the research sought to determine the clinical importance of those levels. Methods: This research included 60 patients with TC of various kinds and severity levels, as well as 30 age and gender-matched controls. In this study, all participants were drawn from the Benha University Hospitals' outpatient clinic for the departments of dermatology, venereology, and andrology. Additionally, there was a substantial variance in the serum marker levels across the various species tested. Patients with a positive family history were found to have a median duration of 2 weeks and a range of 1-6 weeks. 38.3% of TC patients were infected through animal contact, while 61.77% were infected by human contact. 5 and 6 inches were the most common sizes of patches, respectively. Most patients (70%) had one patch on their scalp. Only 10 percent and 20 percent, respectively, had two or more spots on their scalp. There were 48.3 percent of cases in the parietal region, followed by 23.3 percent in the frontal region, and a further 3.3 percent in the vertex (21.7 percent). The occipital region was the least common location (10.0 percent). Black dot/scaly lesions were the least common kind of lesion, occurring in just 11.7% of cases, whereas scaly lesions accounted for 60.0% of all cases (5 percent). M.canis (41.7 percent) was the most common organism, followed by M.audouinii (25.0 percent). T. violaceum (18.3%) was the most often isolated dermatophyte that caused TC, followed by T. verrucosum (11.7%) and T. schoenleinii (10.1%). (3.3 percent). The serum marker may have a role in the pathogenesis of TC, according to our findings. the serum marker level may also be used as an independent risk factor for predicting TC vulnerability, activity, and severity.

Key words: Tinea capitis.

1. Introduction

TC is a severe public health problem in developing countries. Toxoplasmosis is caused by the Trichophyton and Microsporum genera (1).

When it comes to TC, M. canis was shown to account for most instances, followed by M. audouinii (0.4 percent) (52 percent and 36 percent respectively). According to several research, only T. violaceum was shown to be the single dermatophyte that was responsible for TC in Egypt (2).

Dermatophyte capacity to cause apparent inflammation and degree of host response are reflected in the pattern of clinical dermatophyte infection across distinct skin regions" (3).

As a member of the natural-killer group 2 family, the serum marker is a ligand for the NKG2D receptor. It is an essential modulator of both innate and adaptive immune responses. A healthy hair follicle has no activity from the marker (4).

Because the hair follicle is generally invisible to the immune system, the tissue marker is turned off in the typical hair follicle. It's conceivable that the marker has been upregulated.

in people who are genetically inclined to it has the same effect on setting off an immune response as an inflammatory cascade (5).

The markers in the dermal sheath and the dermal papilla are considerably elevated, but not in control people or those with other inflammatory scalp disorders. Hair follicles from those who are currently unwell are also found to carry the marker (6).

Individuals with TC had their blood marker levels tested to see whether they had any clinical significance.

2. Patients and Methods

Type of the study

This study was conducted as a case - control study.

Study population

The study included 60 patients suffering from TC (Group A). In addition to 30 apparently healthy individuals of matched age and sex was chosen as a control group (Group B).

All patients were selected from the outpatient clinic of Dermatology, Venereology and Andrology Department of Benha University Hospitals.

Administrative Design and Ethical Considerations

The study was approved by the local Ethic Committee of Benha Faculty of Medicine. Informed consents were taken from all participants or their parents before the start of the study.

Inclusion criteria

- Diagnosis of TC based on clinical and trichoscopic findings.
- Both sex were included.

Exclusion criteria

- Patients on topical therapy (2 weeks) or systemic therapy (1 month).
- Patients with other types of fungal infection.
- Patients with other autoimmune diseases or inflammatory disorders.

Methods

All patients were subjected to the following

A complete history taking:

- Personal history: Name, age, sex, occupation, residence, special habits of medical importance.
- Present history: Onset, course, duration of TC, Number, size and site of patches.
- Past History:
  Past history of prior episodes of TC. History of animal contact.
- History of medications (type, dose, duration).
- History of systemic diseases or other autoimmune diseases as rheumatoid arthritis, SLE and inflammatory bowel disease.
- Family History: History of other fungal infection in the family.

Clinical examination:

- Complete general examination: Complete clinical examination was done to exclude other autoimmune or systemic diseases.
- Clinical assessment of the skin (Local examination):
- Sites, number, size, morphology and configuration of TC lesions were recorded.
- Severity of TC according to CSS.

Clinical severity assessment score of Tinea capitis

Clinical severity score was assigned by the summation of the individual symptom scores (0-absent, 1-mild, 2-moderate and 3- severe) for eight clinical features of infection including; alopecia, scaling, crusting, erythema, inflammation, adenopathy, pain and itching.

Laboratory investigations:

All the studied subjects were tested for serum kevel of the marker.

Sampling:

Five ml venous blood was collected from each participant under complete aseptic condition and put in a serum seperaty tube then was left for 30 mins till clotting then centrifuged (at 1500 rpm for 15 minutes). The separated serum was aliquoted and stored at -20°C for further testing.

Determination of serum marker level:

Enzyme linked immune sorbent assay (ELISA) technique was used to detect the serum marker level.

Principle of assay:

The serum marker's concentration was measured using a sandwich ELISA kit that used a twofold dose of antibody.

One well of the enzyme was precoated with the marker monoclonal antibody, and this was then added to.

An immunocomplex was formed with the addition of biotin-labeled serum marker antibodies and streptavidin-horseradish peroxidase.

Chromogen solutions A and B were then added after incubation and washing to eliminate any uncombined enzymes.

The liquid became blue as a result of the oxidation process. The sample's serum marker concentration was favourably associated with the chroma of the sample's colour.

3. Results

Most patients showed progressive course (90.0%). The median duration was two weeks and ranged from one to six weeks. Only 5% and 3.3% of the patients showed recurrence and positive family history, respectively. More than one-third (38.3%) reported animal contact. The median CSS was two and ranged from 1 to 6 (Table 1).

Table (1) Clinical characteristics in the studied groups

Clinical characteristics			
Course	Progressive	n (%)	54 (90.0)
	Stationary	n (%)	6 (10.0)
Duration (weeks)	Median (range)		2 (1 - 6)
Recurrence	n (%)		3 (5.0)
Family history	n (%)		2 (3.3)
Source of contact	Animal	n (%)	23 (38.3)
	Human	n (%)	37 (61.7)
CSS	Median (range)		2 (1 - 6)

CSS; Clinical severity score

Regarding the type of lesion, the most frequent was scaly lesions (60.0%), while the least frequent lesion was black dot/scaly lesions (5%) ***(Table 2)*.**

Table (2) Type of lesion in the studied patients

Type	n (%)
Black dot	14 (23.3)
Kerion	7 (11.7)
Scaly	36 (60.0)
Black dot/scaly	3 (5.0)

4. Discussion

Distantly linked to MHC class I molecules, the marker is a member of a family of human cell surface molecules (7).

Chromosome 6 encodes a cell-surface glycoprotein, which is the serum marker (8). A ligand for the NKG2 D receptor, as the name suggests. Innate and adaptive immune responses are both regulated by it (4).

For example, it may have the similar effect as in alopecia areata (AA) and/or trigger an inflammatory cascade that initiates the immunological response (6). In the hair follicles of healthy controls and AA patients, it was shown to be significantly upregulated, but not in the hair follicles of healthy controls or those with other inflammatory scalp illnesses (5).

NKG2 D is a protein identified by the killer receptor NKG2 D, which is targeted by viral micro ribonucleic acids (miRNAs) similar in sequence between JCV and BKV. Consequently, viral miRNA-mediated downregulation of NKG2 D-mediated NK cell killing of virus-infected cells resulted in decreased NKG2 D-mediated NK cell death. NK cells were more effective in killing infected cells when viral miRNA activity was reduced during infection (9).

A decrease in the marker's cell-surface expression is sufficient to shield cells from NK cell-mediated cytotoxicity when UL142 is co-precipitated from the marker-expressing cells (10).

NKG2D ligands induced the expression of serum markers characteristic to SLE serum. A greater amount of the serum marker was found on Tregs from individuals with severe SLE, compared to those with mild or moderate disease. SLE serum or IFN- may increase the expression of markers (11).

Among the cell lines investigated, the intensity of marker expressions was low. As a result, it is probable that NKG2 D ligands' protein and mRNA expression correlates positively (12).

LAM patients with sNKG2D ligands had a faster rate of loss in lung function. LAM-induced changes in NKG2D ligand and the NK environment are indicators of lung damage and progression, and NK cells are implicated in the pathogenesis of LAM (13).

Hair follicles are thought to be equipped with a variety of immunological abilities that help keep viruses from entering the body via the body's many hair follicles (14).

The tissue marker is switched off in a normal hair follicle because the hair follicle is normally undetectable to the immune system. In the dermal sheath and dermal papilla, the marker was significantly expressed (15).

TLRs and other surface-bound fungus-recognition components and nucleic acids have a role in fungus recognition. Fungal infection or cell stress may cause an increase in NKG2 D receptor expression through the TLRs pathway, which in turn affects NK cell activity (16).

Th1 cell counts and IFN- production are decreased in fungal infections. NK cell responses to healthy cells expressing NKG2 D ligand during inflammatory reactions may be limited by a natural negative feedback loop that is mediated by interferon gamma downregulation of the NKG2 D receptor (17).

Downregulation of NKG2 D ligands after IFN- has been hypothesised as a regulatory mechanism that allows for the transition from innate immune surveillance to adaptive immunological responses through MHC and T cells (18).

Intercellular communication between immune cells is facilitated by the NKG2 D ligands. Immune responses both innate and adaptive are influenced by these ligands, which were shown to be overexpressed on DCs and macrophages triggered by TLRs (19).

5. Conclusion

The serum marker may have a role in the development of TC. TC susceptibility, activity, and severity may also be predicted by the serum marker level.

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