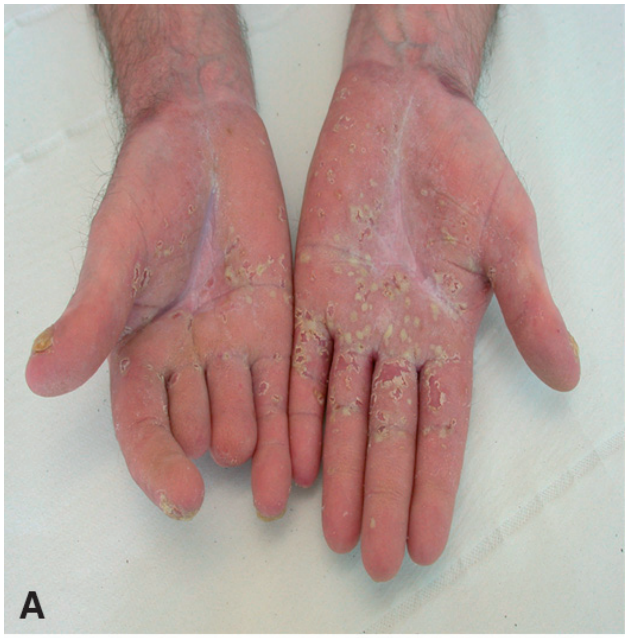

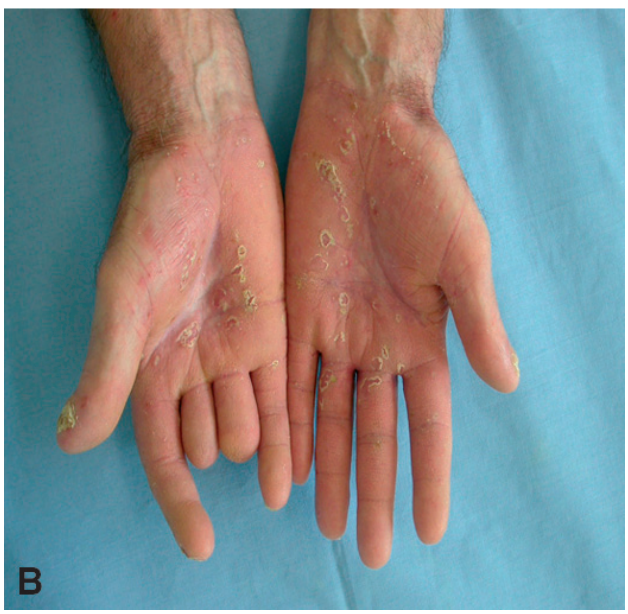

Figure 1. A) Many pustules sometimes joining in big lakes of pus on palmar region observed at the first visit. **B)** An important decrease of pustules with just a remaining light desquamation observed after 3 months of therapy with efalizumab.

compensatory increase in the number of CD11. In this way the domains cannot be saturated by the biological drug at conventional doses. A second hypothesis is a possible development of anti-efalizumab antibodies, even if they are not considered relevant in the efficacy of the biological drug [6]. Another explanation is that CD11, once blocked, promotes the starting of alternative mechanisms which are able to go over the block and hence support the dermatosis. The last hypothesis for the good and rapid initial response could be related to a placebo effect of the new drug, but it is improbable that such an improvement of clinical features can be easily induced by a "mind effect", since the important results of other cases are reported in the literature[2]. ■

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Immunohistochemical analysis of the immune cells in the epithelioid cell granuloma of tuberculoid leprosy

Leprosy, a disease caused by Mycobacterium leprae (*ML*), is characterized by several immunological alterations. Tuberculoid leprosy (TL) is characterized by the formation of epithelioid cell granulomas, which reflects delayed-type cellular immunity toward antigens of *ML*. Understanding the cellular composition of these granulomas would have some clinical ramifications [1-4]. To achieve this, we examined the immune profile of epithelioid granulomas in TL.

Materials and methods

Formalin-fixed, paraffin-embedded skin biopsy specimens representing thirty untreated cases of TL were obtained from the Pathology Department of Sohag University, Egypt. Immunoperoxidase staining methods and specific polyclonal antibodies (PGL-1 antibodies) against phenolyic glycolipids of mycobacterium lepra were used to confirm bacteriological diagnosis (Fite stain). Immunostaining was carried out as previously described [5]. Sections were incubated with mouse monoclonal antibodies for 30 min at room temperature (Clone L26, PC3/188A, PG-M1, and GrB-7 for CD20, CD3, CD68 and Granzyme B, respectively, DAKO corporation, Denmark) [5]. Immune cells were analyzed in the adjacent clinically normal skin. Positive control specimens consisted of lymph nodes with reactive lymphoid hyperplasia (CD3, CD20, CD68 and Granzyme-B). Reactivity was membranous (CD3 or CD20) and cytoplasmic (CD68 and Granzyme-B). Additional sections, running in parallel but with omission of the primary antibody served as negative controls. The cells

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were counted in ten different fields. The results were expressed as mean values (mean ± SEM). A student t-test was done and a significance level of $p < 0.05$ was used (SPSS software version).

Results and discussion

Our data demonstrated three observations: i) TL granuloma is composed of a mixture of CD3+ T cells, CD68+ histiocytes, CD20+ B cells and plasma cells; ii) CD3 +T cells and CD68+histioicytes are predominant cell populations; iii) a relatively large number of T lymphocytes (CD3+ cells) had cytotoxic activity (GRB+); and iv) compared to normal skin, the counts of immune cells were statistically significantly higher in the lesional tissue ($p < 0.05$). A summary of these results is shown in *table 1*.

The presence of a mixture of immune cells in granulomas of TL concurs with previous reports [6] , and suggests that the evolution of these lesions entails participation of several immunocytes. As compared to clinically adjacent normal skin, there was an increased density of immune cells (CD3+ T cells, CD68+ histiocytes, GRB+ Cytotoxic T cells, CD20+ B cells, eosinophils, and plasma cells) in TL granulomas. This may be due to an increase in the antigenic load in the damaged tissues. These antigens include both *Ml* specific antigens as well as cross-reactive mycobacterial antigens [1-6]. The numeric dominance of CD3+ T cells, CD68+ histiocytes, suggests their critical roles in the development of these granulomatous lesions. The concomitant increase in CD3+ T cell and CD68+ histiocytes in TL granuloma suggests that activation of T lymphocytes occurs in close collaboration with CD68+ antigen presenting cells [7].

The numerical dominance of CD3+ T lymphocytes in TL granulomas concurs with previous investigations and raises the notion that T lymphocytes have a central role in both the evolution and modulation of these lesions [1]. Several experiments support the central role of CD3 T cells in TL: i) T-cell clones generated from the lesional skin of TL patients recognize a large number of different antigenic proteins, and ii) non-protein antigens, such as PGL-I show T-cell reactivity [1-6].

The cytotoxic T-lymphocytes are able to recognize and destroy target cells through release of cytoplasmic granules such as GRB. The significantly higher density of GRB +cells in TL as compared to normal skin suggests possible pathogenetic roles for GRB in TL [1-6]. Here we report immunophenotypic characterization of epithelioid cell granulomas in TL. Possible diagnostic, prognostic and therapeutic ramifications of our findings are open for further investigations. It would be interesting for further studies to compare immune cells along the entire spectrum of leprosy and to examine the expression of HLA-DR on macrophages. ■

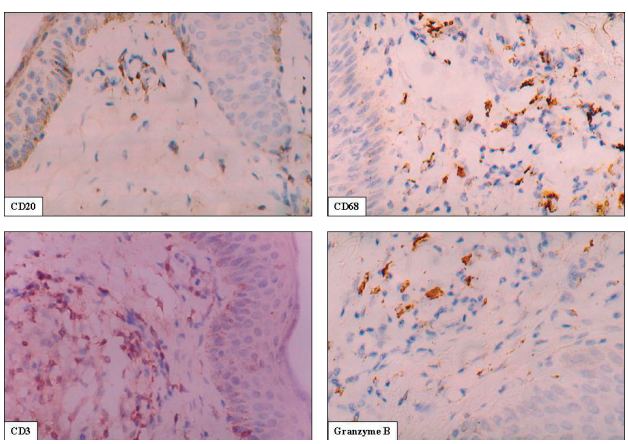

Figure 1.

Table 1. The mean counts of the inflammatory cells in the epithelioid cell granuloma of tuberculoid leprosy. Immune cells were analyzed in the adjacent clinically normal skin Expression p < 0.05 was used for all of the true values which are lower than 0.05

Cell type	Normal skin	Tuberculoid leprosy	*p* value
T cells (CD3+)	2.8 ± 1.0	54.0 ± 2.2	< 0.05
Histiocytes (CD68+)	3.6 ± 1.0	41.1 ± 2.5	< 0.05
Cytotoxic T cells (GRB+)	0.6 ± 0.2	10.9 ± 3.3	< 0.05
B cells (CD20+)	0.0 ± 0.0	4.3 ± 0.40	< 0.05
Eosinophils	0.0 ± 0.0	0.8 ± 0.01	< 0.05
Plasma cells	0.0 ± 0.0	0.6 ± 0.01	< 0.05

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Waterproof camouflage for vitiligo of the face using Cavilon™ 3M as a spray

Vitiligo vulgaris has profound psychological and social effects. Patients with vitiligo may suffer from low self-esteem and poor body image, experience discrimination from others and feel stigmatized [1]. Various medical therapies for vitiligo vulgaris are available, though they are often not effective, especially for segmental vitiligo [2].

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