Summary

Expression of CD\textsubscript{30} antigen is a distinct marker of lymphocyte activation that was originally described in the RS cells of Hodgkin's disease.

In addition to RS cells of Hodgkin disease, CD\textsubscript{30} is expressed on a subset of all cells of anaplastic large cell lymphomas, a variable proportion of bizarre large cell in lymphomatoid papulosis, angioimmunoblastic lymphadenopathy and peripheral pleomorphic T-cell lymphomas.

The CD\textsubscript{30} antigen is expressed in only a very small population of normal lymphoid cells (3%), whereas approximately 50\% of all malignant lymphomas are CD\textsubscript{30} positive, this occurs in almost all cases of Hodgkin's disease and the vast majority of anaplastic large cell lymphomas, so CD\textsubscript{30} +ve cell is considered a diagnostic feature of cutaneous CD\textsubscript{30} lymphoid proliferations.

Although, CD\textsubscript{30} is a marker of lymphoid activation that is characteristically expressed in lymphoproliferative disorders such as Anaplastic Large Cell Lymphoma (ALCL), Lymphomatoid Papulosis (LyP) and Hodgkin lymphoma but also, it has been reported in some cutaneous benign inflammatory infiltrates.

However, CD\textsubscript{30} is expressed by a subset of normal, activated T and B cells; little information is available regarding the mechanisms of induction of CD\textsubscript{30} on normal lymphoid cells (about 3\% of normal cells) or its normal cellular function.

It thought that CD\textsubscript{30} (Ki-1) positive cells found in normal human lymphoid tissue are rapidly proliferating cells and this could explain why
this population might undergo malignant transformation much more often than other cells in lymphoid tissue and this may explain some sort of CD30 function.

Previous studies demonstrated the expression of CD\textsubscript{30} in inflammatory infiltrate of atopic dermatitis and chronic scabies.

In this study, expression of CD\textsubscript{30} is examined in benign inflammatory diseases as psoriasis, lichen planus and scabies.

This study comprised 30 patients with various inflammatory skin diseases (psoriasis, lichen planus and scabies) and 10 normal volunteers as controls.

Skin biopsies were taken from all patients and normal controls following a written consent. All specimens were stained by hematoxylin and eosin for routine histopathological diagnosis. Each disease was classified pathologically into early inflammatory and late well developed stage. Immunodetection of CD\textsubscript{30} and its related antigens were performed.

The stained slides were then microscopically examined using the following parameters and the semi quantitative criteria for CD\textsubscript{30}(+ve) cells according to the distribution and the number of CD\textsubscript{30}(+ve) cells was evaluated as follows: isolated cells (+), small non-cohesive aggregates (3-5) of positive cells (++) and large aggregates (> 5 cells) of positive cells (+++).

Histopathologically, the skin lesions of the three diseases showed the classical pathological findings.

It was found that one out of 10 normal human specimens gave a weak positive epidermal staining for CD\textsubscript{30} while negative staining was found in 9 specimens.
Summary

All chronic cases of psoriasis (6 cases) stained positive for CD$_{30}$. In lichen planus the 4 late cases stained positive for CD$_{30}$. Also in scabies, the chronic lesions only stained positive for CD$_{30}$. 