immunoblastic morphology. The background can be 'inflammatory type', composed predominantly of polymorphs or of 'lymphohistiocytic' type. Necrosis, mitosis and considerable apoptosis can be seen in cases with high proliferation rate. Focal epidermotropismhas been reported and such cases must be carefully differentiated from primary cutaneous aggressive epidermotropic CD8+ cytotoxic T-cell lymphoma given the complete different prognostic implications and therapeutic approach for the two. Epidermal ulceration, granulomatous infiltrates and vascular infiltration are observed in some cases[1,2,5].

On immunohistochemistry, CD30 is expressed in more than 75% of cells and variable loss of CD2, CD3, and CD5 is noted. Classical cases are positive for CD4 and do not express CD8. Expression for cytotoxic proteins (granzyme B, TIA-1, perforin) is not uncommon. An occasional case may show positivity for CD56 with no prognostic implication. Most cases are negative for ALK and EMA. Very few cases show positivity for CD8 and such cases if present with a high Ki-67 index, follow a more aggressive clinical course. They need to be identified as CD8 positivity can commonly lead to misdiagnosis. The Ki-67 index not only correlates with the type and extent of skin involvement and clinical stage, but is also an independent adverse prognostic factor. DNA hybridization studies reveal proliferation of clonal T lymphocytes.

The differentials for CD8+ CD30+ immunophenotype include Lymphomatoid papulosis type D, mycosis fungoides, primary cutaneous aggressive epidermotropic CD8+ cytotoxic T-cell lymphoma, systemic lymphomas involving the skin and primary cutaneous peripheral T-cell lymphoma, unspecified. The distinction is not always possible on histopathologic grounds alone and Clinico-pathological co-relation forms the basis of making the correct diagnosis. CD8+ Lymphomatoid papulosis is clinically characterized by recurrent eruptions of small erythematous papulonodular lesions followed by spontaneous healing and scar formation. The CD30 positivity in these cases is found in 50% to 75% of cells[1,2,3].

In Mycosis Fungoides, lesions are usually generalized and show three phases of progression: Macular erythematous eruption → patch or plaque phase → Generalised erythroderma. Histomorphology reveals lichenoid infiltrate composed of mainly lymphocytes and histiocytes, but also a few atypical cells (small/medium-sized) with cerebriform nuclei and perinuclear halo usually confined to the epidermis (basal layer). Plaque stage shows more pronounced epidermotropism with occasional characteristic intraepidermal collections of atypical cells (Pautrier