Large CD30- Positive Cells In Cutaneous Benign Lymphoid Infiltrate

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Citation

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Abstract

We are reporting a clinical presentation for a case that resamples CD30 positive lymphoproliferative disorder. The clinical challenge, the deferential diagnosis and review of literature have been discussed.

CLINICAL COURSE

This is the case of a 44 year old single smoker male with past medical history of duodenal ulcer, benign prostatic hyperplasia, diabetes mellitus, hyperlipidemia and hypertension. The patient presented to the hospital for evaluation of a 6 year history of multiple non pruritic red nodulo-plaque lesions on the head and neck regions without lymphodenpathy or any other systemic symptoms or signs (figure 1). One year before presentation patient had undergone “isotretinoin” therapy for 6 months for a clinical diagnosis of “ Granulomatous Rosacea” without any benefit.

Laboratory work up revealed normal cell blood count, normal electrolytes, normal urine analysis, negative anti-nuclear antibody (ANA), HBsAg and HCV none reactive. The patient was started on the following medications; glyburide, metformine, doxazosin and amlodipine about two years after the onset of the above complaint.

The first clinical impression was set for Jessner lymphocytic infiltrate with differential diagnosis of lupus erythematosus and cutaneous sarcoidosis.

The first skin lesion biopsy from the temporal area revealed normal epidermal histological features with dense perivascular and periadnexal lymphocytic infiltrate in the dermis. No epitheloid granuloma identified, and the findings most likely suggestive of Jessner lymphocytic infiltrate, and less likely to be lupus erythematosus.

Hence, the patient was treated with antimalarial drugs (hydroxychloroquine) 200 mg/day for 5 months but without improvement.

A second biopsy was performed later on from neck area and showed unremarkable epidermis with dense dermal perivascular and periadnexal lymphocytic infiltration that has a nodular appearance with occasional diffuse expansion within the dermis, it is composed mainly of small to medium sized lymphocytes admixed with histiocytes, smaller number of large lymphocytes, many eosinophils and rare plasma cells, the small lymphoid cells frequently show cleaved or irregular nuclear membrane. Mitotic figures are occasionally seen, and prominent vascular channels in many areas with plump endothelial cells identified (Figure 3 A, B).

Immunohistochemistry staining was performed and the large cells expressed CD3, CD30 and CD45 (Figure 3. C, D). And were negative for CD20, Alk, EBV (figure 3.E, F) and EMA. The proliferative index by Ki-67 labeling is 20%.

Therefore, the findings were in keeping with the diagnosis of CD30 positive T – cell lymphoproliferative disorder.

The patient was treated with prednisolone (40 mg/ day) for one week, reduced gradually to (5 mg/day). Methotrexate (25 mg /week) for 6 months and after treatment, lesions mildly have improved and decreased in size but fully recovery had not been accomplished and they relapsed in two months after ceasing methotrexate (figure 2).

Further molecular testing for T cell rearrangement was performed on paraffin embedded tissue and no monoclonality was detected.

Figure 1

Figure 1. Red nodulo-plaque lesions distributed over the head and neck area.

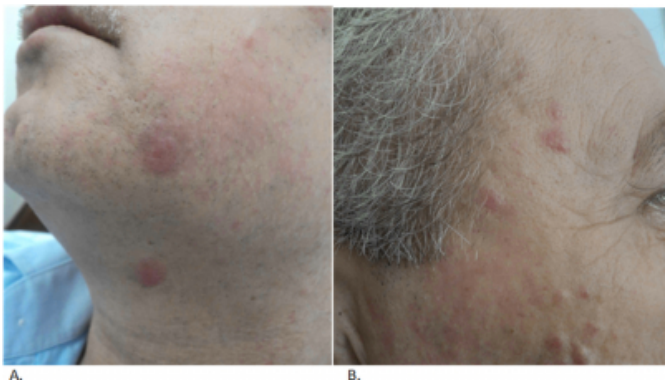


Figure 2

Figure 2.A. Head lesions after Methotrexate therapy. 2. B. lesions months after ceasing Methotrexate

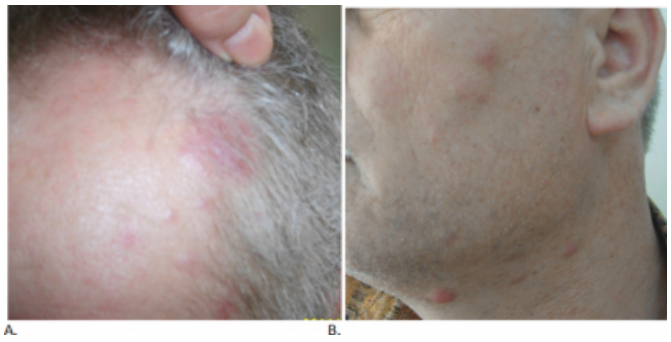
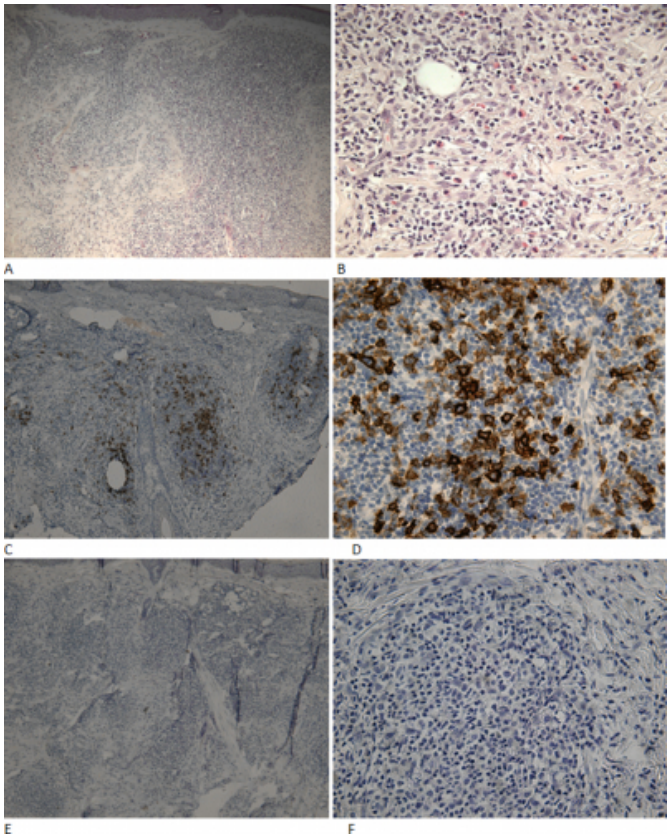


Figure 3

Figure 3.hematoxyline&eosin stain showing A. low power X10, intensive subcutaneous lymphocytic infiltration. B. same section in high power X 40 revealing small and large atypical lymphocytes with eosinophils. C and D, low and high power imaging, revealing positive immunohistochemistry staining of CD30. E & F. low and high power imaging, revealing negative immunohistochemistry staining of EBV.



DISCUSSION

The clinical presentation of this case was confined to the head and neck regions along with normal laboratory findings and no waxing or waning of recurrent papule-nodular lesions.

In addition, verities of medications have been given to the patient with mild or no benefits.

However, the clinical course of this disease appears very indolent or benign with consideration to the discomfort that that patient has over years of this chronic lesion.

The final clinical impression is in favor of non-malignant condition of cutaneous CD30-positive T-cell Lymphoproliferative disorder (CD30+TLPD) which was supported by the clinical, histological, phenotypic, and molecular genetic findings as well as Immunohistochemistry

staining (1).

In one study, the presence of CD30-positive atypical lymphoid cells was 71.4% of the common non-neoplastic cases studied, even in the presence of clonal B-cell populations, and this warrants caution in the interpretation of these cells as malignant, particularly when dealing with the differential diagnosis of lymphomatoid papulosis or neutrophil-rich anaplastic large cell lymphoma (2).

Non-malignant CD30+TLPD conditions include but not limited to reactive lymphoid hyperplasia, viral infections such as (herpes virus or EBV), drug reactions, arthropod assaults, pityriasislichenoides, pseudolymphoma and others. And some of the malignant conditions that are listed under (CD30+TLPD) are primary cutaneous anaplastic large cell lymphoma, lymphomatoid papulosis(LyP) and transformed mycosis fungoides.

Occasional transformation from benign cutaneous (CD30+TLPD) to cutaneous lymphoma has occurred.

Similar cases have been reported in literature (3) and in order to single out the correct diagnosis; further testing such as immunohistochemistry, PCR for viral DNA and T cell rearrangement is needed. The management and the clinical follow up are depending on the clinical pathological correlation and the responding to chosen therapy.

The term CD30+ pseudolymphoma is proposed to designate inflammatory processes with CD30+ T cells.

The disorders often show broad patches and plaques and often mimic cutaneous T-cell lymphoma. It is not specific disease but rather an inflammatory response to known or unknown stimuli that results in a lymphomatous-appearing but benign accumulation of inflammatory cells. Examples include actinic reticuloid, lymphomatoid contact dermatitis, and lymphomatoid drug eruptions. The pseudolymphoma should be reserved for idiopathic cases if no other cause is identified (1).

Viruses and drugs are the most common cause for occurrence of large CD30-positive cells in cutaneous pseudolymphomatous infiltrates. Arrangement of these large, CD30-positive cells in small clusters is not unique to cutaneous CD30-positive lymphomas, and in many cases a precise diagnosis can be made only upon accurate clinic-pathological correlation or using ancillary methods such as polymerase chain reaction analysis for viral DNA (3).
Even though, this case showing benign course and the patient still have healthy life and there is no evidence so far about transforming to worse course; we believe this case and other similar cases should be studied more since they may recognized as a separate identity.

References

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