MORE THAN SKIN DEEP – SEVERE SEBORRHEIC DERMATITIS IN THE SETTING OF UNDIAGNOSED HODGKIN LYMPHOMA

Fatima Arif M.Sc, Luis J Borda MD, Julia Burden MD
EVMS Department of Dermatology, Eastern Virginia Medical School, Norfolk, VA

EVMS Eastern Virginia Medical School

Introduction

Seborrheic Dermatitis (SD) is an inflammatory skin condition that affects areas rich in sebaceous glands such as the face, scalp, neck, upper chest, and back. Onset of severe SD could represent a red flag for underlying immunodeficiency, such as HIV/AIDs, malignancy, or Parkinson's disease. Up to date, there have been three case reports linking lymphoma's immunosuppression with the development of SD. We present a 16-year-old female who presented with severe SD in the setting of undiagnosed Hodgkin Lymphoma (HL).

Case

16-year-old African American female with nonsignificant past medical history, presented for bilateral scaly thick yellow plaques on the nasolabial areas and eyebrows, and scaly scalp for about 9 days after admission for a one-week history of 103 F fever, fatigue, loose bloody stools, abdominal pain, vomiting, body aches, tender cervical, axillary and inguinal lymph nodes and mild URI symptoms.

Patient had erythematous, well-marginated plaques with white scales on the frontal, vertex, postauricular scalp, preauricular cheeks, eyebrows, paranasal cheeks, and perioral skin **(Figure 1)**.

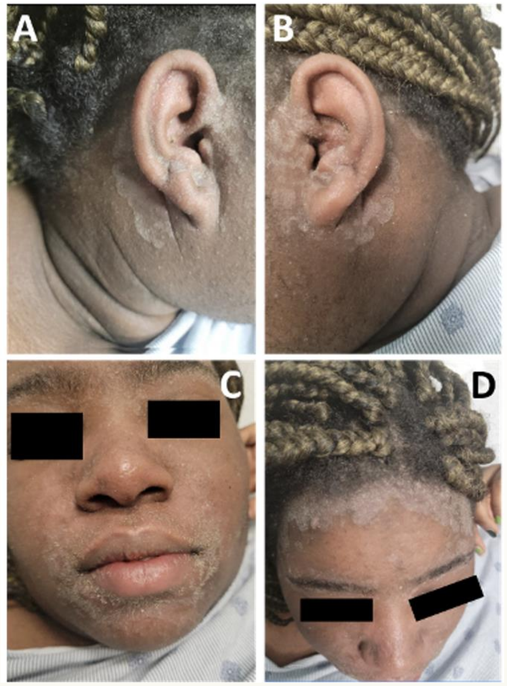

Figure 1. Clinical Findings. Well-marginated plaques with overlying whitish scale appreciated on pre- and post-auricular skin (**A, B**), nasolabial and perioral areas (**C**) and frontal scalp/superior forehead (**D**).

Initial lab work was notable for leukocytosis 34K, elevated absolute lymphocytes 20K, mild hyponatremia 133mEq/L, AST 80 U/L, ALT 88 U/L, BUN 33 mmol/L, Cr 2.2 mg/dL, lipase 121 U/L, CRP 3.4 mg/L, uric acid 10.5 mg/dL and LDH 1700 IU/L. Rapid strep, CMV/EBV, HIV, and COVID testing, and urinalysis were negative.

Abdominal CT revealed multifocal enlarged intraperitoneal lymph nodes. Chest CTA showed axillary lymphadenopathy and splenomegaly. Abdominal US revealed adjacent reactive lymph nodes at the pancreatic neck.

Due to the sudden onset and severity of the patient's SD flares, thorough workup for underlying immunosuppression found decreased C4 and normal C3, ANA, dsDNA, and Sjogren labs. However, a right axillary lymph node biopsy revealed mixed cellularity HL with CT staging establishing Stage IV HL.

The patient was treated with ketoconazole shampoo 2% 2-3 times per week and triamcinolone 0.1% ointment twice daily with noticeable improvement of her SD flare.

For Stage IV HL, the patient was started on doxorubicin, vinblastine, dacarbazine, brentuximab, and dexamethasone.

Discussion

Patients with underlying malignancies or HIV/ AIDs, have a lower level of skin free fatty acids (FFA) and a higher level of skin triglycerides (TG) compared to healthy controls, indicating that a difference in skin surface lipid composition may play a part in SD development in immunodeficient patients. *Malassezia* lipases degrade TGs and use the skin FFA for proliferation, which can cause severe SD flares seen in patients with immunodeficiencies. Additionally, skin surface FFA are converted to proinflammatory eicosanoids (i.e leukotriene B4) which further increases severe SD flares.

Conclusion

- Sudden onset of severe SD could be a manifestation of an underlying immunosuppression, such as HIV/AIDs, and hematologic malignancies, such as lymphomas as seen in our case.
- With raising further awareness of the link between underlying immunosuppression/malignancy, our hope is that a more comprehensive discussion and malignancy workup be performed for any patient presenting with severe acute onset of SD flares.

References