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EGFRI-Induced Skin Folliculitis Presenting on the Scalp: A Case Report

Faraz Yousefian^{1,2*}, Liliana Espinoza³, Sujitha Yadlapati⁴ and Terri J Nutt⁵

¹University of the Incarnate Word School of Osteopathic Medicine, San Antonio, Texas, USA

²Texas Institute for Graduate Medical Education and Research, USA

³Long School of Medicine, University of Texas Health San Antonio, San Antonio, Texas, USA

⁴HCA Corpus Christi Medical Center- Bay Area Dermatology Residency Program, McAllen, Texas, USA

⁵San Antonio Skin Care and Dermatology Clinic, San Antonio, Texas, USA

ABSTRACT

Skin toxicity, or folliculitis, is the most common adverse side effect resulting from the use of antineoplastic signal transduction inhibitors like the epidermal growth factor receptor inhibitors (EGFRI) panitumumab and poziotinib. This condition takes a toll on a patient's quality of life and can affect up to 90% of patients. Moreover, it is one of the primary contributing factors to the patient's decision to discontinue antineoplastic therapy, thus highlighting the need for better dermatologic interventions to help mitigate the side effects resulting from EGFRI treatment. We present the case of a 76-year-old female patient who presented with a sudden, severe folliculitis exclusively affecting her scalp following years of stable tumor suppressor therapy.

*Corresponding author

Faraz Yousefian, University of the Incarnate Word School of Osteopathic Medicine, Texas Institute for Graduate Medical Education and Research, USA. Tel No: 561-379-1136: E-mail: Yousefian.faraz@gmail.com

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Introduction

Epidermal growth factor receptor inhibitors (EGFRIs) are a group of anticancer agents approved for the therapeutic use on a wide array of solid tumors including, but not limited to, colorectal, non-small cell lung, and ovarian cancer [1-15]. EGFRs are commonly expressed in cancer cells, but can also be found in epidermal keratinocytes and sebocytes. Upon activation, EGFRs initiate a signaling cascade, promoting the uncontrolled cell division and proliferation of cancer cells. Consequently, EGFRIs prevent the signal transduction of such receptors, thereby impeding the growth of solid tumors [1, 2, 4-7, 11-15].

EGFRI therapy is known to cause mild to severe skin eruptions in upwards of 90% of patients [14]. The erythematous folliculitis that develops is often used by oncologists as an indicator of the agent's efficacy, with patients who respond best to therapy developing the most severe folliculitis [1, 2, 4, 6, 8-11, 13, 15]. The drug-induced folliculitis is most commonly present on a patient's upper body, face, and proximal arms [4, 6, 8, 15]. The case we present below is notable due to the severity of the folliculitis, which presented on the scalp of an otherwise stable patient

Management of the resulting skin toxicity is initiated once symptoms are apparent. Research has shown the efficacy of implementing a prophylactic skin regimen prior to the commencement of EGFRIs. Such consists of topical steroids, emollients, antibiotics on sub antimicrobial dosing like doxycycline, and a para-aminobenzoic acid (PABA)-free sunscreen [4, 8, 9, 11, 13, 14]. Depending on the

severity of the condition, some patients may decide to discontinue chemotherapy altogether. For this reason, better preemptive skin regimens can help reduce the severity of EGFRI-induced acneiform rash, all-the-while allowing patients to benefit from the agent's antineoplastic properties [6, 8, 9, 11-15].

Case Report

A 76-year-old female has been on the EGFR inhibitor poziotinib for 8 months since the year 2018 for the treatment of non-metastatic lung cancer. In addition to lung cancer, the patient's medical history is significant for essential thrombocytosis, atrial fibrillation, and basal cell carcinoma. The patient exhibited the expected acneiform and folliculocentric papules, which were satisfactorily controlled with topical steroids, and doxycycline 20 mg BID, alternating with doxycycline 100 mg BID in instances of flare-up. Despite no change in her chemotherapy treatment, the patient presented with a sudden, severe inflammatory flare-up affecting only the scalp. Her current medications include metoprolol, hydroxyurea, and poziotinib. Additionally, the patient has a family history of skin cancer.

Cutaneous examination of the scalp revealed alopecia accompanied with severe localized erythema and serocrusting lesions (Figure 1). Scalp skin cultures revealed heavy growth of group B streptococcus and light growth of H. parainfluenzae, with the remainder of the skin and nail examinations exhibiting her baseline acneiform and folliculocentric papule rash.

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Figure1: Clinical presentation of severe, serocrusting localized erythema and patchy, generalized alopecia of the scalp following chronic treatment with poziotinib before prednisone treatment.

Initially, she received doxycycline 20 mg BID, triamcinolone solution, clobetasol solution, betamethasone dipropionate ointment, 3% salicylic acid, topical clindamycin, and was switched to a gentle shampoo and a course of penicillin. Additionally, the patient was instructed to use doxycycline 100 mg BID and hydroxyzine 25 mg TID PRN in the occurrence of another flare-up.

Despite these regiments, in the three-month follow-up appointment the patient continued having severe inflammation of the scalp and alopecia, which caused her daily discomfort. Due to the patient's lung cancer, there was reluctance to prescribe the use of an oral steroid. However, after approval from her oncologist, she was placed on a three-week prednisone taper. The patient was followed up one month later and she reported satisfaction with the improvement of her scalp symptoms. She had residual erythema of the scalp, and she was placed on another three-week prednisone taper. The patient was reluctant to discontinue prednisone because of a potential reoccurrence of severe symptoms. With the consent of her oncologist, she continued on a daily dose of 5-10 mg prednisone and Fosamax, which ultimately helped resolve her skin findings (Figure 2)



Figure 2: Improvement of EGFRI-induced serocrusting localized erythema and patchy, generalized alopecia of the scalp following treatment with oral prednison

Comment

The use of targeted antineoplastic therapies for various types of cancers has been crucial to better disease prognosis. Still, continued use of these cancer drugs is associated with unwanted dermatologic side effects in a great proportion of patients. Although oncologists may see these manifestations as clinical markers of the drug's efficacy, severe skin eruptions often exert a negative influence on a patient's quality of life. With this in mind, diagnosis and treatment of mild to severe acneiform rashes resulting from the use of signal transduction inhibitors like EGFRs, can help discourage the discontinuation of cancer therapy agents.

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