Oral Communications

SUNITINIB-INDUCED FOLLICULAR OCCLUSION DISORDER OF THE FACE: A CASE REPORT

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Background. Sunitinib is a multi-targeted receptor tyrosine kinase (RTK) inhibitor widely used for the treatment of metastatic renal cell carcinoma (RCC). While cutaneous adverse events such as hand-foot skin reaction and hair depigmentation are common, the paradoxical induction of follicular occlusion disorders is rarely reported. We present a case of follicular occlusion disorder of the face developing in a patient undergoing Sunitinib therapy.
Case Presentation. A 70-year-old male with no prior history of acne or follicular occlusion disorders presented with a 2-month history of painful facial and cervical lesions. The patient had been receiving Sunitinib for metastatic RCC for seven months. Dermatological examination revealed lesions resembling ectopic Hidradenitis Suppurativa (HS) and dissecting cellulitis (DC), with deep inflammatory nodules, fibrous pedunculated papules at the mandibular angle and lateral neck, follicular cicatricial lesions on the nape, and multiple ice-pick scars on the cheeks. Notably, comedones were entirely absent. Lesions were intermittently suppurative. Based on the clinical morphology, the lack of primary comedones, and the temporal correlation with the targeted therapy, a diagnosis of Sunitinib-induced follicular occlusion disorder of the face was established. A conservative topical approach was chosen to minimize systemic drug interactions. The patient was started on 1% clindamycin gel twice daily.
Results. At the 3-week follow-up, the patient showed a discrete clinical improvement. There was a reduction in both local pain and active suppuration of the inflammatory nodules, although the fibrotic and cicatricial outcomes naturally persisted. The topical treatment was well-tolerated, without interfering with the oncological therapy's safety profile.
Conclusions. Multi-kinase inhibitors targeting VEGFR and PDGFR, such as Sunitinib, can disrupt follicular homeostasis and local angiogenesis, potentially triggering deep infundibular hyperkeratosis and the subsequent inflammatory cascade typical of HS and DC, even in atypical extra-intertriginous locations. The strict absence of comedones crucially differentiates this adverse event from standard acneiform eruptions. This case highlights the importance of recognizing paradoxical inflammatory cutaneous toxicities from targeted therapies.