observed reaction is also doubtfully drug related, since there were no liver test abnormalities found. The dermatophytid (id) reaction was excluded by the following observations: First, clinical lesions of id reactions are typically intensely pruritic and develop quite distantly from the site of infection. In the present case, the patient denied pruritus, and the exacerbation involved the primary lesions without production of any new ones. Second, in id reactions, no fungal forms are recovered from the lesions; in the present case, the patient’s lesions yielded fungal growth. Third, over the course of id reactions, no generalized symptoms (eg, fever), as seen in this patient, are normally observed.

Altogether, this report presents an unusual case of a paradoxical reaction resembling, to some extent, the JHR, after treatment of dermatophyte infection with terbinafine.

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**Complete Remission of Squamous Cell Carcinoma After Treatment With Panitumumab in a Patient With Cetuximab-Induced Anaphylaxis**

A patient with locally advanced cutaneous squamous cell carcinoma (cSCC) who had had an anaphylactic reaction to an epidermal growth factor receptor (EGFR) inhibitor, cetuximab, responded to panitumumab.

**Report of a Case** | An 89-year-old man presented with a locally advanced cSCC of the nose that was 35 mm in diameter. The exophytic, ulcerated, necrotic tumor invaded local cartilage, but there was no metastasis (T4NOM0) (Figure, A). The large size of the lesion and difficulty in repairing the defect after excision made surgery inappropriate. In addition, the patient’s other comorbidities precluded general anesthesia. He received radiation therapy at a dose of 40 Gy in 10 fractions. The tumor progressed immediately after irradiation with a voluminous...
infiltrated peripheral bulge. Skin biopsy confirmed the persistence of invasive cSCC. Owing to the patient’s age and medical history, the multidisciplinary team recommended systemic palliative treatment with an EGFR inhibitor (cetuximab, 400 mg/m²) beginning 3 months after cessation of the radiation therapy. The first infusion immediately led to an anaphylactic reaction. Anti-galactose-α-1,3-galactose (α-gal) antibodies were detected. The cetuximab treatment was stopped.

Treatment with another EGFR inhibitor, panitumumab, 6 mg/kg every 14 days, was administered, followed by rapid regression of the peripheral bulge around the ulceration (Figure, B). After 15 treatment cycles, complete remission was obtained and biopsies of the remaining ulceration showed only epidermal hyperplasia. Thereafter, panitumumab injections were given monthly. After 20 cycles, treatment was stopped, and complete remission persisted for 6 months without any further treatment (Figure, C).

Discussion | Managing locally advanced unresectable or metastatic cSCC in elderly patients is a challenge.1,2 Radiotherapy is sometimes inappropriate or ineffective, and platinum-based chemotherapy may be given alone or in combination with an EGFR inhibitor.1,2 In elderly patients, an EGFR inhibitor as a single-agent therapy has been considered palliative.1,2 The EGFR gene codes for a transmembranous tyrosine kinase receptor responsible for cellular proliferation and epithelial growth. Cetuximab, a chimeric monoclonal antibody IgG1 EGFR inhibitor, was approved for colorectal cancer treatment and head and neck squamous cell carcinoma and has shown efficacy in cSCC in a phase 2 trial3 with a limited number of patients. Panitumumab is a fully human monoclonal antibody IgG2 EGFR inhibitor indicated for colorectal cancer treatment. It has shown noninferiority to cetuximab4 and is currently in development for head and neck SCC. Panitumumab treatment consists of 1 infusion every other week vs cetuximab, which is administered weekly. There is also a difference in infusion-related reactions, which occur in less than 0.5% of patients treated with panitumumab vs 2% in those treated with cetuximab.4 This difference in infusion reactions is due to α-gal, a ubiquitous glycan epitope, which is not expressed in humans but is present on the Fab portion of cetuximab. Panitumab does not exhibit the α-gal epitope, which explains the lower rate of grades 3 and 4 infusion reactions.5 Specific IgE α-gal antibodies are associated with red meat delayed anaphylaxis and with cetuximab immediate anaphylaxis.6 A phase 2 study of panitumumab monotherapy in patients with incurable cSCC has shown its safety and efficacy.6 These data, as well as the outcome of the present case, suggest that panitumumab is a good alternative to cetuximab when anaphylaxis occurs. The overall response rate with panitumumab of 31% (2 of 16 complete responses) in a previously extensively pretreated cohort seems to be promising in cSCC6 and must be confirmed in randomized clinical studies.

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Rapid Clearance of Necrolytic Migratory Erythema Following Intravenous Administration of Amino Acids

Necrolytic migratory erythema (NME) is a rare paraneoplastic skin disorder, considered the hallmark clinical sign of glucagonoma syndrome. Its recognition allows for early diagnosis of the tumor, which might lead to a better prognosis. Additional mucocutaneous features can include angular stomatitis and glossitis. We present a case of NME that cleared rapidly following intravenous administration of amino acids.

Report of a Case | A woman in her 50s was evaluated for a persistent pruritic eruption lasting for 3 months. The eruption started on the lower extremities and perianal area and rapidly expanded to involve the genital area. Her medical history included sicca syndrome for the last 20 years. In the last 2 years, the patient had developed angular cheilitis and glossitis. She also reported weakness, anorexia, weight loss (approximately 20 kg within a year), numbness and tingling sensation in the extremities, and instability during walking.

Physical examination revealed erythematous migratory plaques with irregular, erosive, scaly borders and hypopigmented centers on the perianal and genital areas. Lichenified erythematous and hyperpigmented plaques were evident on the distal shins. The patient had an erythematous tongue with angular fissures (Figure 1). Cardiovascular, abdominal, and respiratory examination findings were normal.

Laboratory tests revealed slight anemia (hemoglobin, 11.4 g/dL; reference range, 12-16 g/dL) and borderline increased hemoglobin A1c (5.8%; reference range, 4.0%-5.7%). Further evaluation revealed increased levels of gastrin (129 ng/mL; reference range, 13-115 ng/mL), chromogranin A (223 ng/mL; reference range, 19-98 ng/mL), and neuron-specific enolase (21.11 ng/mL; reference range, 0-12 ng/mL). Somatostatin receptor scintigraphy revealed high concentrations of somatostatin receptors in the pancreatic head and adjacent lymph nodes. Computed tomography demonstrated an abdominal central tumor in the pancreatic head, medial to the duodenum, causing superior mesenteric vein blockage. Endoscopic ultrasonographic examination and needle biopsy showed a neuroendocrine tumor, grade 2, and based on findings of immunohistochemical staining (MIB-1 labeling index, 3%) and markedly increased glucagon levels (>500 pmol/L; reference range, <50 to 150 pmol/L) the patient was diagnosed with glucagonoma.

Histopathologic examination of a skin biopsy specimen from the perianal area showed nonspecific subacute dermatitis with mild perivascular lymphocytic infiltrate accompanied by plasma cells and a few eosinophils. There was no evidence of necrolysis in the upper epidermis.

Two weeks after establishing the diagnosis of glucagonoma, treatment was initiated with intravenous administration of a commercial mixture of 500 mL of amino acid solution (Vamin 18 electrolyte free; Fresenius Kabi) for 12 hours, once a day, for 2 consecutive days. Marked improvement was observed after 24 hours, with reduced erythema and scaling, reepithelialization of the erosions in the genital area, and closing of the fissures on the tongue. The skin lesions continued to resolve rapidly over the subsequent weeks.