

Oral Fixed Drug Eruption Secondary to Isoniazid

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Oral Fixed Drug Eruption Secondary to Isoniazid

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Figure 1: Right upper mucosal lip, 3cm thickened, beefy red plaque with superficial fissures. Patient was on INH at the time.



Figure 2: Right upper mucosal lip, well healed lesion 3 weeks after INH was discontinued.

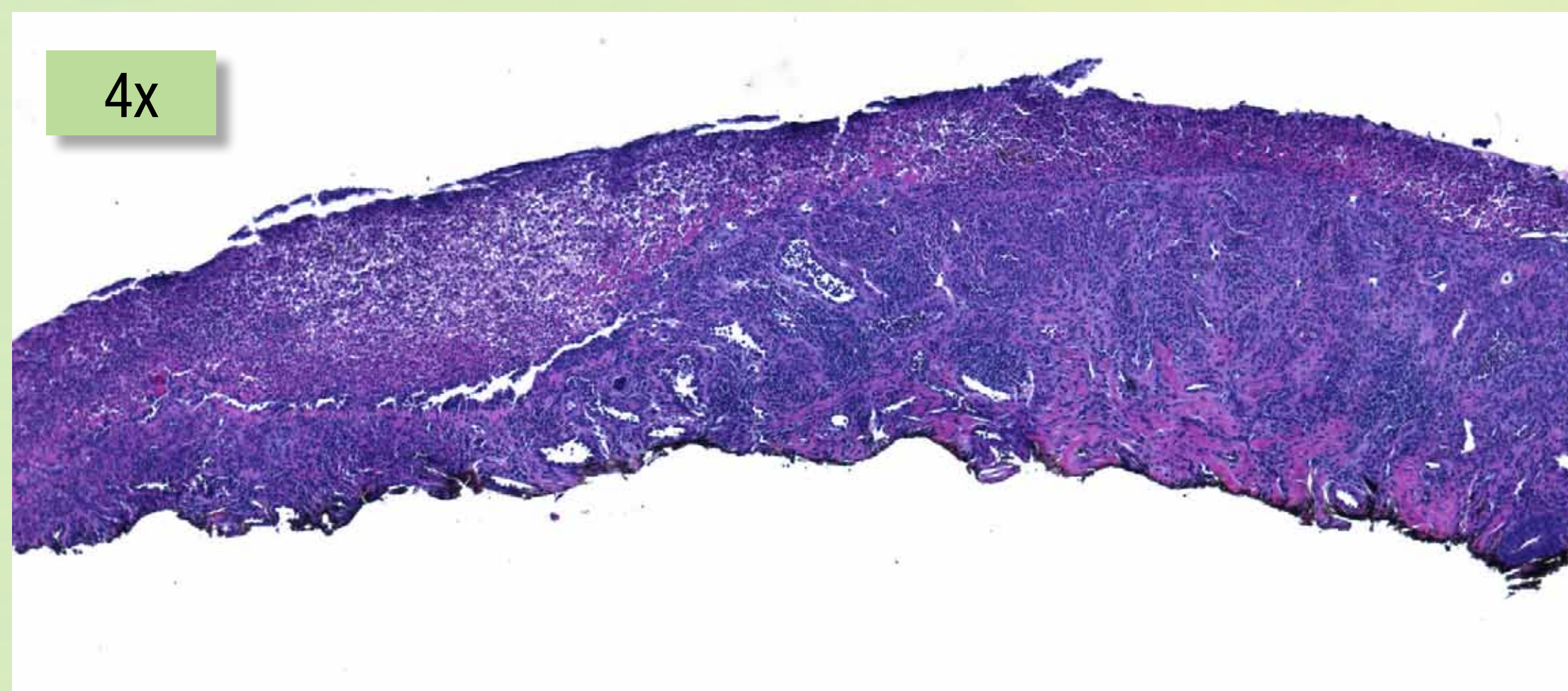


Figure 3: H&E (4x) Shave biopsy from the lesion in figure 3 showing acutely inflamed granulation tissue and overlying layer of fibrino-neutrophilic debris.

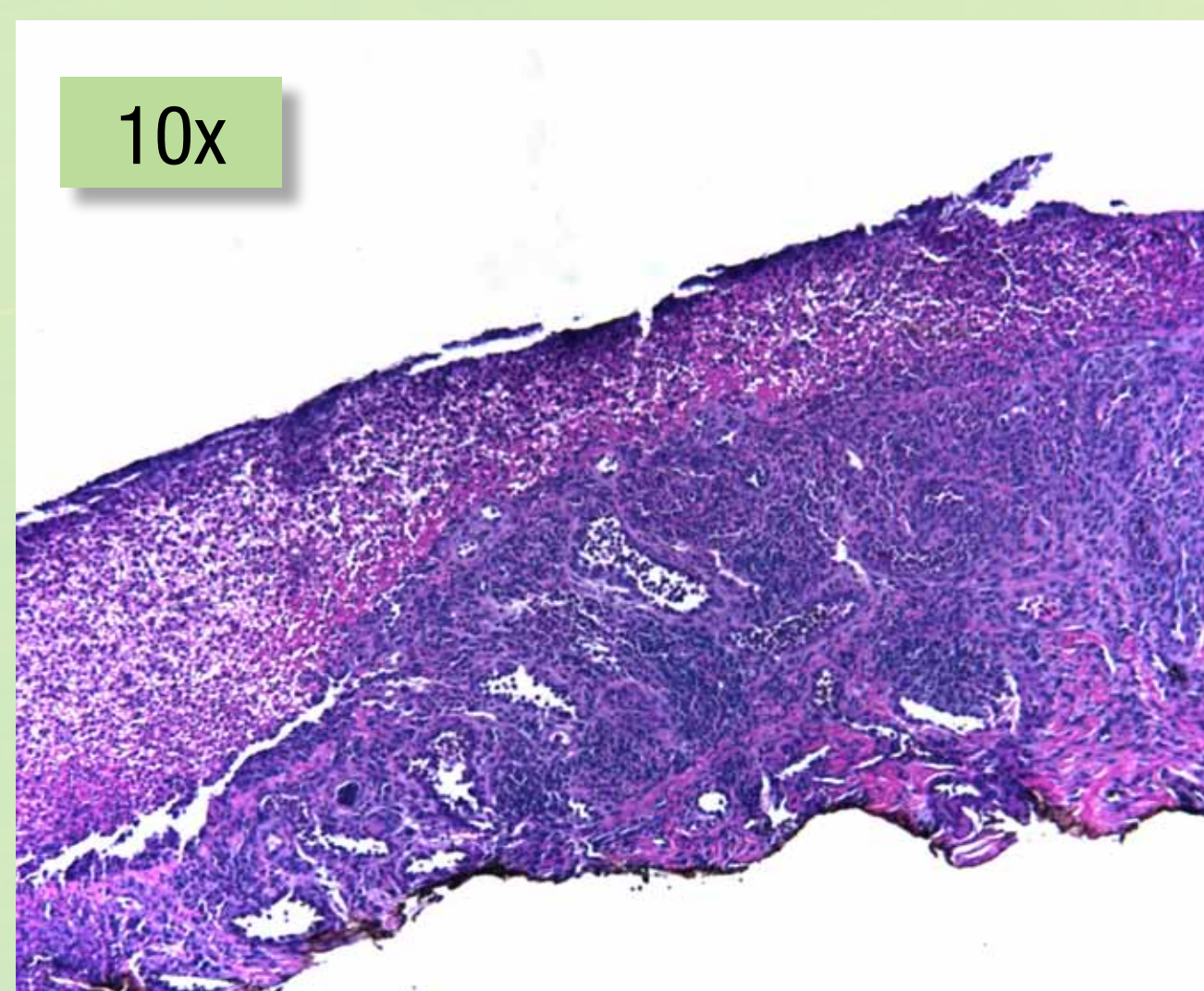


Figure 4: H&E (10x) Higher power of fibrino-neutrophilic debris and granulation tissue.

Patient: P.S. is a 60 year-old Caucasian female.

History of Present Illness: The patient presented to our dermatology practice complaining of 2 months duration of painful sores inside her mouth. Two weeks prior to the eruption she was started on isoniazid (INH) and vitamin B-6 for treatment of a positive PPD found on routine screening. The patient was seen by her PCP before presenting to our office and was put on valacyclovir for a presumed HSV outbreak, without improvement. Viral and bacterial cultures were negative; however the patient was on valacyclovir at the time. She offered no complaints other than the burning and occasional bleeding inside the mouth.

Medical History/Surgical History: HTN, osteoporosis, psoriasis, psoriatic arthritis, seasonal allergies, tuberculosis, joint replacement, appendectomy, hysterectomy (uterine tumor)

Medications: Celebrex, hydroxychloroquine, methotrexate, teriparatide, simvastatin, folic acid, vitamin D, rifampin

Previous Treatments: Valacyclovir, prednisone taper, desoximetasone topical gel

Physical Examination: The right upper mucosal lip has a 3 cm thickened, beefy red plaque with superficial fissures. Her right lower mucosal lip has a healing erosion. On the left side of the dorsal tongue a 6 mm x 3 mm ulceration is noted. No lesions were seen on the genital mucosa and no other cutaneous findings were present.

Studies: Viral and bacterial cultures: negative

Biopsy: CBLPath (D10NY1-0379575, 11/5/10) Lower lip: "Mostly acutely inflamed granulation tissue and overlying layer of fibrino-neutrophilic debris. The findings appear nonspecific."

Direct Immunofluorescence: Negative

Reason for Presentation: Interest

Discussion: Fixed drug eruption (FDE) is a distinctive variant of a drug eruption with characteristic recurrence at the same site of skin or mucous membranes and spontaneous resolution upon discontinuation of the causative agent. The initial eruption is often solitary and frequently located on the lip or genitalia. Involvement of the mucosa only without any skin involvement, as in the case of our patient, is extremely rare.

Clinically, the lesions on mucosal surfaces may be edematous plaques or pigmented patches with an erythematous halo. The lesions may often become vesicular or bullous and rupture leaving erosions. With the initial attack, the period required for sensitization is highly variable ranging from a few weeks to several years. Subsequent re-exposure to the medication results in

reactivation of the old lesion and occasionally development of new ones, within approximately 2 hours.

The exact mechanism for the development of the FDE lesions is unknown. It is believed to be triggered by activation of intraepidermal CD8+ T cells and additional recruitment of CD4+ and CD8+ T cells from the circulation causing extensive tissue damage observed in fully evolved lesions. It is hypothesized that intraepidermal CD8+ T cells are strategically seeded to the epidermis upon contact with infection or trauma as they are similar in phenotype and function to virus-specific effector-memory CD8+ T cells.

The major categories of causative agents of FDE include antibiotics, antiepileptics, nonsteroidal anti-inflammatory agents (NSAIDs), and phenothiazines. The drugs frequently associated with mucosal FDE are sulfonamides (especially TMP-SMX), NSAIDs (in particular, phenazone derivatives) and tetracyclines. Recent literature describes case reports of oral FDE secondary to fluconazole and gabapentin.

Different adverse dermatological reactions have been reported with INH. Many of these are hypersensitivity reactions such as urticaria, angioedema and morbiliform eruptions. To the best of our knowledge, this is a unique case of oral FDE caused by INH.

The main goal of treatment is to identify the causative agent and avoid it. Rechallenging the patient, either by patch testing the lesional skin or oral provocation (most reliable test) are the only known tests to possibly identify the suspected agent. Our patient refused a rechallenge because of great discomfort associated with the eruption. However, resolution of her oral lesions within 3 weeks and no recurrence at 12 weeks follow-up since discontinuation of INH, strongly supports it being the causative agent.

FDE lesions can be treated for symptomatic relief. In this case, the patient did well with a potent topical corticosteroid and a short, low-dose prednisone taper.

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