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# A rare case of meloxicam-induced pseudoporphyria

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## Abstract

Drug-induced pseudoporphyria is commonly linked to nonsteroidal anti-inflammatory drugs (NSAIDs) such as naproxen, oxaprozin, ketoprofen, and ibuprofen. The NSAID meloxicam is not a commonly reported inciting agent. We report a case of meloxicam-induced pseudoporphyria in a 55-year-old woman with a past medical history of hypertension, hyperlipidemia, gastroesophageal reflux disease, and osteoarthritis. She presented to the clinic with tense and denuded bullae on her dorsal feet, which was diagnosed as pseudoporphyria after further workup. Upon evaluating the patient's medication history, meloxicam was identified as the most likely inciting agent. The patient's condition resolved with the discontinuation of this medication. Our findings can help dermatologists effectively diagnose and treat meloxicam-induced pseudoporphyria in patients with similar cases.

*Keywords: meloxicam, NSAIDs, photosensitive rash, pseudoporphyria*

## Introduction

Pseudoporphyria is a rare disorder that usually presents as a photo-distributed bullous dermatosis closely resembling porphyria cutanea tarda (PCT), but without the classic derangement in porphyrin metabolism. The clinical features of pseudoporphyria consist of vesicles, bullae, milia, skin fragility, and scarring mostly on sun-exposed areas. This condition can be drug induced and when combined with sunlight exposure, can lead to a

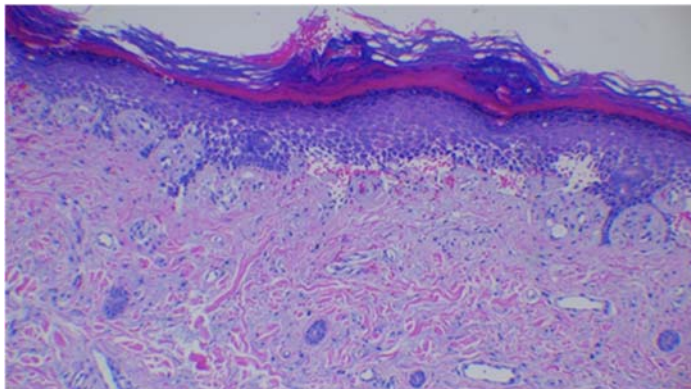
phototoxic cutaneous reaction that causes the formation of blisters.

## Case Synopsis

A 55-year-old woman presented with a three-week history of a painful, blistering rash on the dorsal toes. She reported working outside in her garden frequently while wearing open toed sandals. Her past medical history was significant for hypertension, hyperlipidemia, gastroesophageal reflux disease, and osteoarthritis. Medications included amlodipine, losartan, atorvastatin, omeprazole, and meloxicam. Physical examination revealed tense bullae on the dorsal aspect of the left second and third toes, and denuded bullae on the right second, third, and fifth toes and overlying the second to fourth metatarsals (**Figure 1**).



**Figure 1.** Tense bullae on the left dorsal foot.

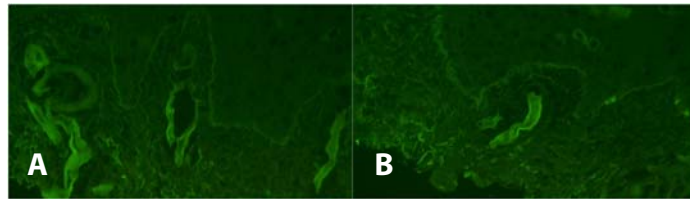


**Figure 2.** H&E showing subepidermal vesicular dermatitis with spongiosis and minimal associated inflammation, 100x.

Chlorazol black preparation was performed from a deroofed blister which was negative for hyphae. Punch biopsy was performed from an intact bulla which showed subepidermal vesicular dermatitis with spongiosis and minimal associated inflammation (**Figure 2**). Periodic acid-Schiff and gram stains were negative for fungi and bacteria, respectively. Direct immunofluorescence was performed from perilesional skin, which showed ribbon-like deposition of IgG, IgA, C3, and IgM in superficial dermal blood vessels consistent with porphyria or pseudoporphyria (**Figure 3**). Serum porphyrin levels were obtained which were within normal limits. A diagnosis of pseudoporphyria was made, with the most likely etiology being drug-induced from meloxicam. The patient discontinued meloxicam and was initiated on celecoxib as alternative treatment for her osteoarthritis. She was also counseled on strict sun protection. The bullae gradually resolved without formation of any new lesions.

### Case Discussion

The histologic and immunofluorescent findings of pseudoporphyria are often indistinguishable from PCT. Histologically, a subepidermal bullae is seen with scant to mild lymphocytic perivascular infiltrate, with or without festooning of dermal papillae [1,2]. Blood vessel thickening and sclerosis of collagen are present less frequently than in PCT. In both conditions, direct immunofluorescence usually reveals granular deposits of IgG and C3 at the



**Figure 3.** A) Direct immunofluorescence showing ribbon-like deposition of IgG in superficial dermal blood vessels, 100x. B) Direct immunofluorescence showing ribbon-like deposition of C3 in superficial dermal blood vessels, 100x.

dermoepidermal junction and in the upper dermal vasculature, whereas indirect immunofluorescence is negative [2].

Pseudoporphyria and PCT can be distinguished by porphyrin levels, which are usually less than 0.9 $\mu$ g/dl in pseudoporphyria. Patients with PCT, in contrast, have levels in the 5-40 $\mu$ g/dl range. The most frequent inciting agents for drug-induced pseudoporphyria include nonsteroidal anti-inflammatory drugs (NSAIDs), antibiotics, diuretics, retinoids, and oral contraceptive pills [1,3-9]. The most common NSAIDs to cause pseudoporphyria include naproxen, oxaprozin, ketoprofen, and ibuprofen [1,10-20]. Other NSAIDs which have been linked to pseudoporphyria include diflunisal, celcoxib, mefenamic acid, and nabumetone [1,10-14].

### Conclusion

Our case is unique as meloxicam is a lesser-known inciting agent for pseudoporphyria. Other causes of pseudoporphyria include hepatitis C infection, artificial tanning beds, and chronic renal failure, all of which were denied by the patient [2]. Treatment consists of strict sun protection and discontinuation of any responsible drug [3]. In drug-induced cases, patients can continue to be symptomatic for months to years after discontinuation of the offending agent [3]. Fortunately, our patient had almost complete resolution of lesions at the five-week follow-up visit.

### Potential conflicts of interest

The authors declare no conflicts of interest.

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