Triflusal (2-acetyloxy-4-trifluoromethylbenzoic acid) is a fluorinated acetylsalicylic acid analog used as a platelet aggregation inhibitor in secondary prophylaxis after an ischemic coronary or cerebrovascular event.

We present a case of systemic photosensitivity to triflusal.

A 66-year-old man was referred for a 3-week history of lesions on the arms, hands, face, and neck. He had a past medical history of hypertension, dyslipidemia, psoriatic arthritis, hypothyroidism, benign prostatic hyperplasia, and ischemic heart disease. He was on multiple medications including lefunomide, adalimumab, levethyroxine, tamsulosin/silodosin, bisoprolol, triflusal, clopidogrel, and rosuvastatin. The rash was initially attributed to a second dose of subcutaneous adalimumab, which was administered as part of psoriatic arthritis treatment started 5 weeks earlier. Despite discontinuation of adalimumab and treatment with loratadine, the symptoms persisted.

Physical examination revealed intensely erythematous, scaly lesions, accompanied by areas of fissures with slight exudate, in regions exposed to UV radiation (Figure 1). He was treated with oral prednisone 0.75 mg/kg in a tapering fashion for 1 month and daily topical administration of betamethasone/gentamicin 0.5 mg/g + 1 mg/g. Photoprotection measures were also recommended, and rosuvastatin and enalapril were discontinued.

Improvement in his rash was evident after 3 weeks but recurred on decreasing the dose of prednisone. After consultation with cardiology, triflusal (initiated 5 months earlier) was discontinued, which resulted in a significant improvement in the lesions after 2 months and complete resolution after 4 months (Figure 2, A). After 1 year of follow up, he remains free of rash.

Arterial hypertension and dyslipidemia treatment were resumed with enalapril and atorvastatin without new lesions. Adalimumab was not reintroduced.

Phototesting (conducted once the lesions had been resolved) did not exhibit pathological UVB values for the patient’s phototype with a minimal erythema dose of 120 mJ. Photopatch testing was then performed using the Chemotechnique Extended European Photopatch Battery (Vellinge, Sweden) adding a patch with 1% triflusal in petrolatum. The results were read 48 hours after applying the patch, with no evidence of an eczematous reaction. We then applied UVA radiation at 5 J/cm² without observing any differences between the irradiated and nonirradiated sides. Nevertheless, after applying the same photopatch battery and assessing the results 48 hours after application, this time exposing one side to UVA (5 J/cm²) and the other to a suberythematogenic dose of UVB (100 mJ/cm²), only the area irradiated with UVB radiation produced a reaction (+++) (Figure 2, B).

Systemic photosensitivity associated with triflusal was first reported in 1987 by Serrano et al.² The hypothesis is that the trifluoromethyl group in the 4 position plays a crucial role in triflusal’s photosensitizing capacity. Triflusal’s maximum spectrum of action seems to lie within the UVB range, unlike most photosensitivity reactions that are normally associated with UVA wavelengths.

Given the scarcity of reported cases and their characteristics, it has been suggested that triflusal’s photosensitivity is caused by an immune mechanism. This phenomenon appears to involve a cell-mediated immune response, which would be initiated when a covalent bond is formed between active metabolite, 2-hydroxy-4-trifluoromethylbenzoic acid, and a UV radiation—activated protein.

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REFERENCES
**FIGURE 1.** A, Intensely erythematous, scaly lesions are observed in all regions exposed to UV radiation. B, The hands demonstrate fissures with slight exudate with the clock zone respected.

**FIGURE 2.** A, Complete resolution of the lesions after 4 months of the withdrawal of triflusal. B, Positive photopatch in the area irradiated with UVB.