a monoclonal antibody that blocks interleukins 12 and 23, is rarely used to treat hidradenitis suppurativa, and conclusive evidence on its efficacy is lacking.

In the case described, after the patient's condition had failed to respond well to conventional therapy or 2 different TNF inhibitors, we requested authorization for off-label use of ustekinumab, a drug indicated for moderate to severe psoriasis. Treatment was initiated once the authorization was received, and the patient remains clinically stable at the time of writing after 1½ years' treatment.

A review of the literature shows that the experience with ustekinumab in hidradenitis suppurativa is anecdotal, with just one 3-case series in which response to treatment was uneven and 2 individual case reports of patients who had other associated inflammatory skin conditions (psoriasis and Behçet disease).[8–10]

The data presented suggest that ustekinumab could be a therapeutic option for treatment-refractory hidradenitis suppurativa.

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Probable Iatrogenic Xanthotrichia☆

Xantotriquia probablemente iatrogénica

We report the case of an 82-year-old man who came to our clinic because his hair had acquired a yellowish hue over the previous 10 months. Relevant medical history included chronic ischemic heart disease and dyslipidemia, which had been treated for over 10 years with acetylsalicylic acid (Adiro 1 × 100 mg tablet daily) and simvastatin (Pantok 1 × 20 mg tablet daily). One year before the consultation, he had been diagnosed with benign prostatic hyperplasia, which was being treated with once daily tamsulosin hydrochloride 0.4 mg (Omnic Ocas).

Physical examination revealed scalp hair with a yellow-orange hue, especially in the frontal and parietal regions (Fig. 1). The patient's natural hair color is gray and he denied using dyes and other hair treatments or making any change in his normal shampoo. His body hair retained its natural whitish color and the physical examination was otherwise normal. However, the patient also reported that for a few months his sweat had an orange hue while his tears and urine were normal.

Blood tests carried out included direct and total bilirubin, transaminases, alkaline phosphatase, albumin, prothrombin time, complete blood cell count, lactate dehydrogenase, haptoglobin, thyroid hormones, protein electrophoresis, glucose, lipids, and beta-carotene levels. The results of all blood tests and urinalysis were normal. In view of the bright yellow color of the tamsulosin hydrochloride tablet (Omnic Ocas, Astella Pharma), a color produced by yellow iron oxide (E172), and the fact that the onset of the symptom coincided with the introduction of this treatment, we decided to discontinue the drug after consultation with the urology and pharmacy departments. The yellow hair coloring gradually disappeared on follow-up and was undetectable at 10 months (Fig. 2). We have reported the reaction to the Valencian Regional Pharmacovigilance Centre by way of a yellow card. We have also reported it to the pharmaceutical company who manufacture the drug (Astellas Pharma). The company said it was unaware of any association between tamsulosin and this adverse effect and went on to say that the symptom could be caused by an adverse reaction to an excipient.

Hair color changes have been described in association with the consumption of certain drugs and other exogenous

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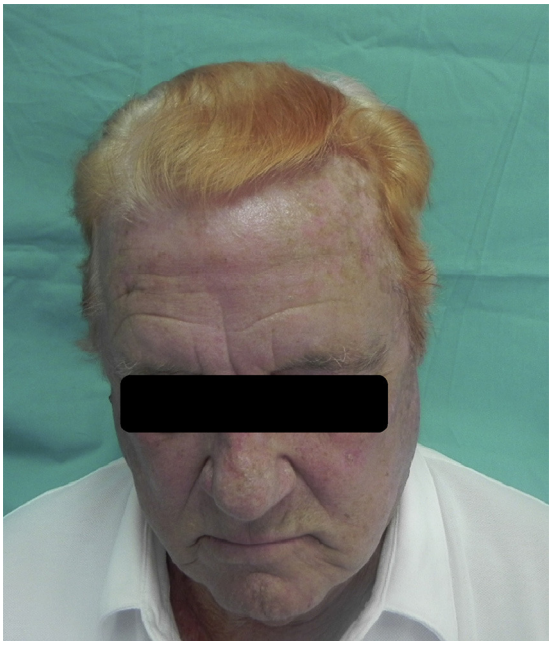

Figure 1 Yellow-orange discoloration of the patient's hair.

chemicals and can also be caused by some diseases. Hair color may darken in Addison disease and may become lighter in patients with hyperthyroidism or genetic disorders.[1–3] Professional or accidental exposure to arsenic, cobalt, lead, mercury, or silver can lead to hair discoloration ranging from blue to green tones and even black.[1] Greenish hair has also been reported in association with high levels of copper in the water supply.[4] A variety of drugs have also been implicated in hair color alterations, with different drugs producing lightening or darkening of the original color or even giving rise to a completely new color.[3] However, except in a few cases, the data are insufficient to establish a true causal relationship. The cases in which more evidence is available involve chloroquine and chemotherapeutic agents, but other products clearly related to xantotriquia are p-aminobenzoic acid, calcium pantothenate, anthralin, mephenesin, minoxidil, propofol, valproic acid, and verapamil.

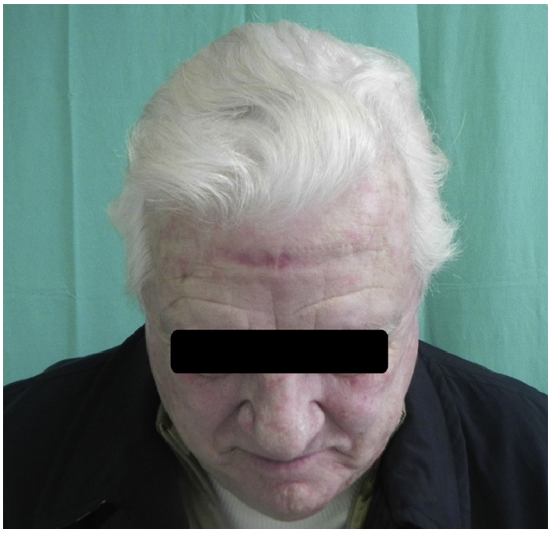

Figure 2 Return to normal gray hair 10 months later.

In the literature, xantotriquia or yellowing hair is considered to be caused mainly by exogenous chemical substances, such as those found in 2.5% selenium sulphide shampoo and dihydroxyacetone,[5] but the symptom has also been associated with certain drugs, including some chemotherapeutic agents (bleomycin, doxorubicin, and vincristine), p-aminobenzoic acid, topical anthralin, and minoxidil.[3,6,7]

Hair color in humans is due to the presence of melanin pigment in the keratinocytes of the hair cortex and medulla. Melanocytes present in the bulb can produce two types of melanin: eumelanin, which gives rise to brown and black hair, and pheomelanin, which gives rise to red or blond hair. Pheomelanin, which is found exclusively in the hair and not in the epidermis, is produced through a modification of eumelanin synthesis involving the interaction between dopaquinone and cysteine. Melanocytes are genetically conditioned to produce 1 type of melanin, but can produce both types under certain circumstances.[7] In the present case, xantotriquia may have been caused by the coloring agent used in the drug that was apparently responsible for the symptom or to the drug itself (or its metabolites) through the stimulation of pheomelanin production and/or the inhibition of eumelanin production. In conclusion, we present an interesting case of xantotriquia which was probably of iatrogenic origin according to the algorithm devised by Naranjo et al.[8] Unfortunately we were unable to confirm this origin with the reintroduction of the drug.

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