exclude skin metastases and B-cell lymphoma. The diag-
nosis of these lesions must be confirmed histologically. Skin
metastases, most commonly from melanoma, are seen as
ova neoplastic structures with dense vascularization. 6 B-cell
lymphoma presents as a well-defined, hypoechoic nodular
lesion with a good vascular supply in the dermis or subcuta-
neous cellular tissue. 7

There is only 1 report of a case of cutaneous AHE that
gives a description of its sonographic pattern. 5 In that case,
a hypoechoic lesion was observed on the forearm and had
a peripheral hypoechoic ring and increased blood flow on
Doppler study. The absence of other reports is probably
because the condition was considered part of the spectrum
of Kimura disease until a few years ago. The 2 diseases,
though clinically similar, are now considered to be differ-
et entities because of the differences in extracutaneous
involvement, laboratory tests, and histological findings,
leading to a completely different prognosis. A larger number
of articles have been published on Kimura disease. The char-
teristic image is of a heterogeneous hypoechoic mass with
poorly defined borders, situated in the dermis and subcuta-
neous cellular tissue, with intermingled, hypoechoic and
hypoechoic curvilinear structures, which has been called a
"woolly" pattern. 9 The vascularization of the lesions is
variable. 10 Our case shows the sonographic characteristics
of both diseases as the wooly pattern was observed, associated
with hypervascularization and a hypoechoic halo.

Skin ultrasound is a useful tool for the diagnosis of
subcutaneous lesions. Although the woolly pattern is char-
acteristic, it does not enable us to distinguish between AHE
and Kimura disease. These findings testify to the difficulty
of differentiating between the 2 diseases and to the need
for histological confirmation.

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Ustekinumab for Hidradenitis Suppurativa: A Case Report
crossMark

Ustekinumab en hidradenitis suppurativa: a propósito de un caso

Hidradenitis suppurativa is a chronic and recurrent inflam-
matory disease that causes disfiguring lesions in areas rich
in apocrine sweat glands. 1

Prevalence is estimated at 1% to 4%, women are affected
more often than men (at a ratio of 3:1), and onset is typically
in the second or third decade of life. 1,2

While its etiology and pathogenesis are largely unknown,
hidradenitis suppurativa is considered a multifactorial dis-
ease in which the immune system plays a prominent role.
Management of hidradenitis suppurativa should be tailored
to lesion severity and distribution assessed according to
the Hurley staging system. 3 As no specific treatments are
available, a wide range of therapeutic options are used
with highly variable results. Antibiotics, corticosteroids,
and retinoids may be helpful in the early stages and during
exacerbations; in advanced or extensive forms of the dis-
ease, surgical resection of the affected tissue is imperative. 4
Biologic agents, particularly tumor necrosis factor (TNF)
inhibitors, have been proposed in recent years for recalc-
itrant forms of hidradenitis suppurativa. Experience with
other biologic agents having different mechanisms of action,
such as p40 inhibition, is anecdotal.

We report the case of a 50-year-old female smoker with
moderate-to-severe hidradenitis suppurativa (Hurley stage

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In April 2009, as the condition had proved refractory to treatment, the patient started a new treatment with an 80-mg loading dose of adalimumab, followed by 40 mg every 14 days beginning one week later. Her condition remained stable for 1 year, after which the dosing interval was increased to 21 days. However, 6 months after this change the lesions had worsened and the 14-day regimen was reinstated, with the addition of prednisone for 1 month. Treatment was discontinued 6 months later owing to loss of efficacy (i.e. lesion recurrence) and adverse effects.

After 2 years of adalimumab, the patient was switched to infliximab 5 mg/kg at weeks 0, 2, and 6; however, this regimen was discontinued after 3 doses because the lesions worsened.

In October 2011, because of the severity of her lesions and the lack of other options, off-label treatment was proposed with 45 mg of subcutaneous ustekinumab at weeks 0 and 4, and at 12-week intervals thereafter, and hospital authorization was granted. Disease activity ceased to progress from the start of treatment, although 3 months passed before a clear improvement of the lesions was observed. After 8 months the disease was no longer active (Figs. 1 and 2). At the time of writing, 1½ years into treatment with ustekinumab, the patient has no active lesions and is tolerating therapy well. During the treatment period there were 2 exacerbations. Both were resolved with a 3-week course of antibiotics (amoxicillin-clavulanic acid at 500 mg/8 h in one case, and rifampicin at 300 mg/12 h in the other), plus prednisone.

Hidradenitis suppurativa is an orphan disease, not because its prevalence (1%-4%) is low, but because there are no therapies that produce sustained clinical remission, let alone any curative effect.

Recent studies in hidradenitis suppurativa have highlighted the role of the immune system and its proinflammatory action, including overexpression of interleukins 12 and 23 and of TNF.5,6

TNF inhibitors are a therapeutic option for advanced-stage patients who fail to respond to conventional treatment, but response, if achieved, appears to last only as long as treatment is continued. Because of this drawback, as well as their side effects and high cost, TNF inhibitors are relegated to second- or third-line treatment.7 Ustekinumab,
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, Astellas 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