leukocidin, the toxin responsible for the virulence of some strains of *Staphylococcus aureus*.

In contrast to other SCoN, *S. lugdunensis* presents an excellent sensitivity profile to a number of families of antimicrobials, including the penicillins, which makes control easier. Despite this, cases of methicillin resistance have already been reported.[10]

It should be noted that the rate of error of identification in automated instruments is not insignificant, meaning that the frequency of infection may be underestimated.

The growing interest in this bacterium in dermatology is based not only on its elevated capacity to cause skin and soft tissue infections, but also on the correct interpretation of the culture, in order to recognize it as a pathogenic organism and not rule it out as a skin comensal.

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Prenatal Screening for Bathing-suit Ichthyosis After Diagnosis in an Older Sibling☆

Ictiosis en bañador y diagnóstico prenatal en subsiguiente embarazo

Bathing suit ichthyosis (BSI) is a rare form of ichthyosis in the group of autosomal recessive congenital ichthyoses (ARCI), together with lamellar ichthyosis (LI), congenital ichthyosiform erythroderma (CIE), harlequin ichthyosis, and self-healing collodion baby (SHCB). These in turn form part of the nonsyndromic ichthyoses,[1,2] and their estimated prevalence in Spain is of 7.2 cases per million population, with a total of 144 cases reported. Among these, only 2 cases of BSI have been documented, 1.4% of all cases of ARCI.[3] BSI was first described as a distinct disease in 2005[4] and it is characterized by presentation at birth as collodion baby; the characteristic distribution typically develops within a few months, with the appearance of thick brownish scales on the trunk, with sparing of the limbs and face, giving the appearance of a woman's bathing suit.[2,5] This condition is due to mutations in the *TGM-1* gene,[1,2,5–8] which provoke a phenotype similar to LI but milder; the distribution depends on body temperature, as the function of the enzyme transglutaminase-1 is only altered in the warmest areas of the body, typically the central regions.[8]

We presented the case of a 6-year-old girl of Spanish origin, with no family history of interest and no known consanguinity, product of a well-controlled second gestation. At birth the condition presented as collodion baby. This resolved without complications, but when the infant was 6 months old, thick brownish scales started to develop in a specific distribution, only affecting the inguinal and axillary regions (Figs. 1 and 2) and the central area of the trunk, with sparing of the face, limbs, hands, and feet. Diffuse desquamation and erythema of the scalp were also observed. Notably, there was no involvement of the nails or hair, no alteration of sweating, and no other signs of systemic involvement, nor was any deficit detected in the child's psychomotor development. On suspicion of BSI, we decided to perform genetic analysis, which confirmed the suspected diagnosis after finding 2 known pathogenic mutations in heterozygosis, in both alleles of the *TGM-1* gene: c.424C > T; p.Arg142Cys described in BSI[8] and c.919C > G; p.Arg307Gly described in LI.[9]

The patient's clinical course has at all times been characteristic of the disease (chronic and recurrent), and the manifestations have been managed using moisturizers and

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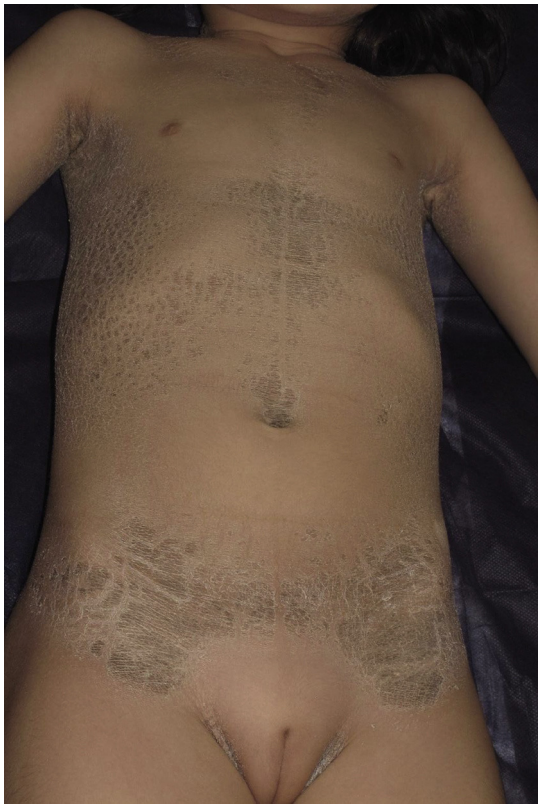

Figure 1 Body distribution of the lamellar scales outlining the form of a woman's bathing suit, with sparing of the limbs and of the central area of the abdomen.

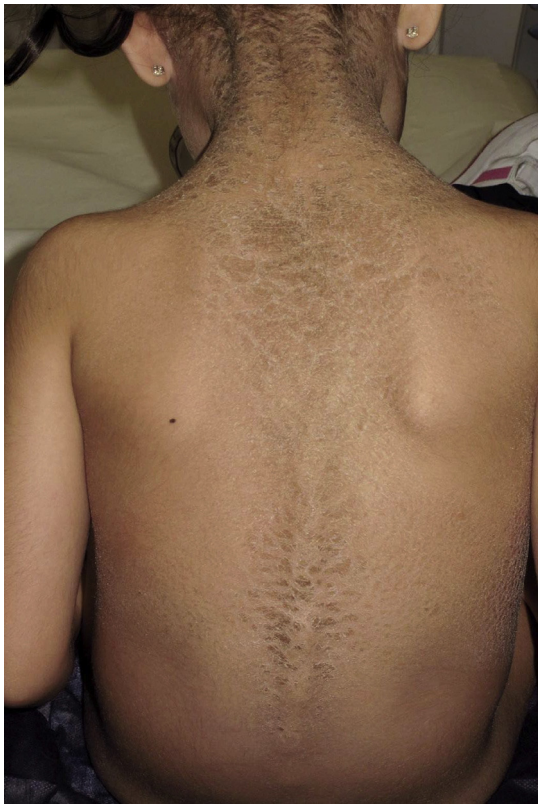

Figure 2 Thick dark scales down the midline of the back.

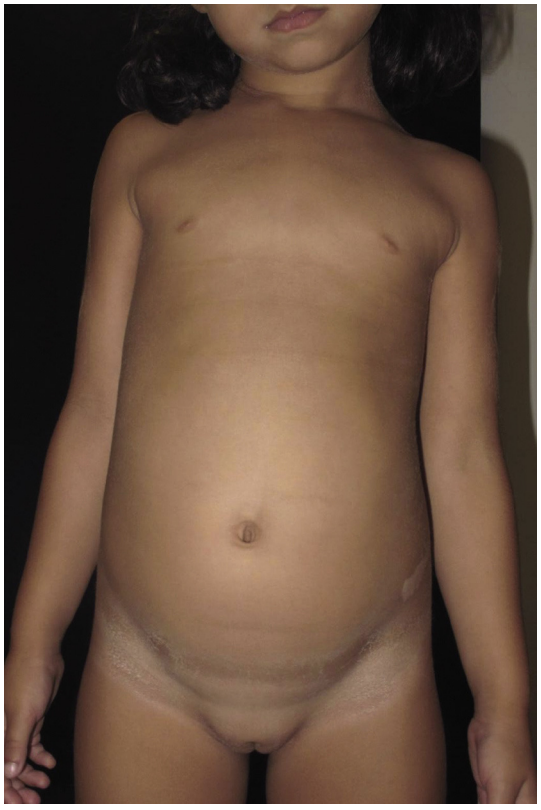

Figure 3 Good control of the disease with the preparation of 10% N-acetyl-cysteine plus 5% urea in a moisturizing body milk.

the application of preparations of 10% N-acetyl-cysteine plus 5% urea[10] (Fig. 3). However, the bad smell of the N-acetyl-cysteine preparation has occasionally interfered with adherence to treatment.

After 3 years of good control, the mother consulted for a new pregnancy and the possibility of giving birth to another child affected by BSI. There are now 7 genes known to be implicated in different types of ARCI. Of these, the gene most frequently implicated is, without doubt, *TGM-1*.[6,7] However, this is the only gene known to be implicated in BSI.[6] Twenty different mutations of this gene have been detected, 9 of which are exclusive to BSI, whereas the other 11 are mutations shared with other types of ARCI.[5] Thus 2 individuals with the same mutations can present different phenotypes.[5,6] In fact, families have been described in which siblings with the same genetic changes have presented different types of ARCI.[5] There are also isolated reports of cases in which the phenotype has changed with the age of the child, passing from BSI to generalized forms of LI/CIE or even to self-healing collodion baby.[5,6] It would appear likely that environmental factors may influence the phenotypic expression of these genetically identical conditions. In our case, one of the mutations found had already been reported in LI.[9] Given the possibility described in the literature of LI in siblings of patients with BSI with the same mutations,[5,6] we agreed with the mother to perform prenatal screening: mutations of the *TGM-1* gene in the fetus were excluded. The gestation was uneventful and the infant was healthy.

In conclusion, BSI is a rare genodermatosis belonging to the group of ARCI. It has a series of clinical and diagnostic peculiarities that we should be aware of. Although the diagnosis is usually clinical, confirmation can only be made by genetic analysis of the *TGM-1* gene. This is the only gene implicated in this condition, but its mutations are also the most prevalent in other forms of ARCI, and many of the mutations are common to the different forms; thus, individuals with the same genetic load can develop different phenotypes, and these can even be dynamic, with changes occurring during life. All these features are important with respect to the prognosis in our patients and to correct genetic counselling.

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Histologic diagnosis of ossifying fibromyxoid tumor: 2 cases in the past 20 Years☆

Diagnóstico histológico del tumor fibromixoide osificante: 2 casos en los últimos 20 años

To the Editor:

The ossifying fibromyxoid tumor (OFMT) was originally described as a small benign tumor of the subcutaneous tissue. It is formed of small cells arranged in cords and nests in a fibromyxoid stroma, covered by a bony capsule.[1] However, recent publications have reported histologic findings of malignancy associated with metastatic disease.[2] There is controversy regarding the histologic origin of the tumor. Despite initially being considered to be distinct from schwannian or cartilaginous tumors,[1] based on ultrastructural and immunohistochemical characteristics (positivity for protein S-100), more recent proteomic and genetic analyses support a neuronal or myoepithelial origin.[3,4] In the last decade, the idea that malignant OFMTs do not exist has been proposed, as they do not satisfy the traditional histological description, and could correspond to other malignant soft tissue tumors.[5]

In the last 20 years, 2 cases of OFMT have been identified in our hospital, one on the scalp of a 55-year-old man the other on the hand of a 46-year-old man (Fig. 1). The tumors were painless. Histology of the excisional biopsies were consistent with the classic description of OFMT: well-defined capsule; areas of fibrosis formed of laminae of uniform, ovoid cells with round nuclei in a hyaline stroma; other areas of myxoid appearance with lower cellularity; and moderate diffuse positivity for protein S-100 (Fig. 2). The surgical margins were not extended in either case. No signs of local recurrence or metastases have been detected after follow-up of 18 years and 21 months, respectively.

This tumor typically affects men, and the mean age at presentation is 50 years. It usually arises in the proximal regions of the limbs, most commonly the lower limbs.[1,3–6]

Immunohistochemistry closely reflects the controversy regarding the histogenesis of the tumor. The origins that have been postulated with greatest emphasis are Schwann

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