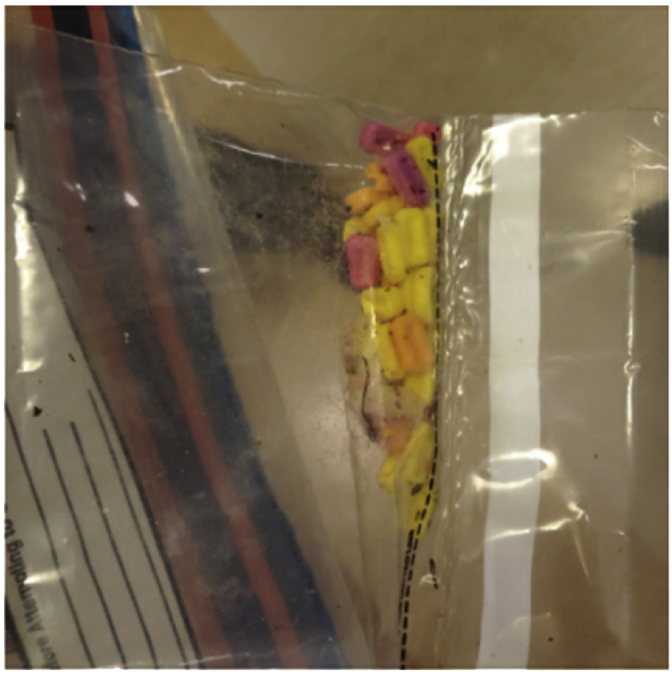

Figure 2. Additional etizolam pills obtained from patients.

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A Neonate with Feathery Scales

A female neonate was referred to the dermatology clinic for evaluation of skin lesions. Streaks of large feathery scales that were tightly adherent to the skin with distribution along the Blaschko lines were apparent (**Figure, A**) along with an atrophic erythematous track on the dorsomedial aspect of the left foot (**Figure, B**). The proximal portion of the left arm was shorter and the fourth and fifth fingers of the left hand were fused (**Figure, C**).

The constellation of findings suggested chondrodysplasia punctata, also known as Conradi-Hunermann-Happle syndrome. Genetic testing revealed a heterozygous mutation on the *Emopamel binding protein* (*EBP*) gene consistent with the clinical diagnosis.

Chondrodysplasia punctata is an X-linked dominant disorder characterized by the presence of stippled calcification of the epiphyses accompanied by other skeletal, cutaneous, and ocular abnormalities.[1] Skin lesions include transient linear ichthyosiform erythroderma that resolve within the

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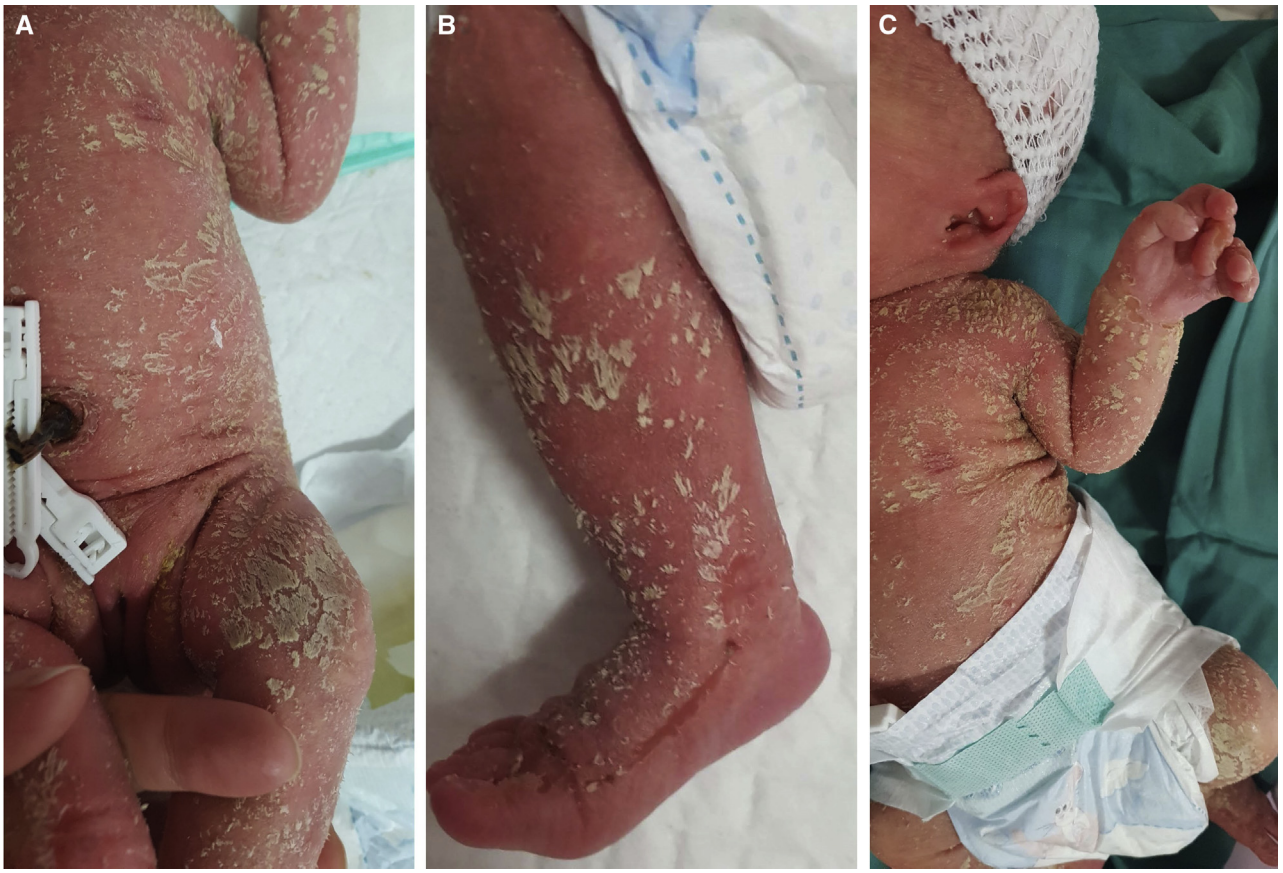

Figure. **A,** feathery scales. **B,** Atrophic track. **C,** Syndactyly.

first few months of infancy leaving behind follicular depressions (follicular atrophoderma) and areas of hypopigmentation.[2] Affected individuals often develop swirly cicatricial alopecia with coarse lusterless hair.[3] Skeletal defects are often asymmetric and include rhizomelic shortening of the limbs, polydactyly, syndactyly, scoliosis, and joint contracture. Craniofacial defects such as frontal bossing, saddle nose, and malar hypoplasia are common. Cataracts are reported in 67% of patients and are usually apparent at birth. Microphthalmia, microcornea, glaucoma, nystagmus, and optic nerve atrophy have been reported.[3] Additional anomalies of the trachea, heart, and kidneys have been described, but the central nervous system seems to be spared in affected females.[3] The presence of the condition in male infants is attributed to postzygotic mutations or gametic half-chromatid mutations that allow the survival of the fetus by alleviating the severity of the anomalies.

The *EBP* gene responsible for the condition is assigned to Xp11.23-22 and encodes an enzyme (3β-hydroxysteroid-Δ8, Δ7-isomerase) involved in the biosynthesis of cholesterol. The mutations result in enzyme deficiency and subsequent accumulation of 8(9)-cholesterol and 8-dehydrocholesterol that can be used as screening biomarkers.[4]

A multidisciplinary approach is needed for the treatment of chondrodysplasia punctata that often includes surgical correction of skeletal and ocular abnormalities. ■

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Data Statement

Data sharing statement available at www.jpeds.com.

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