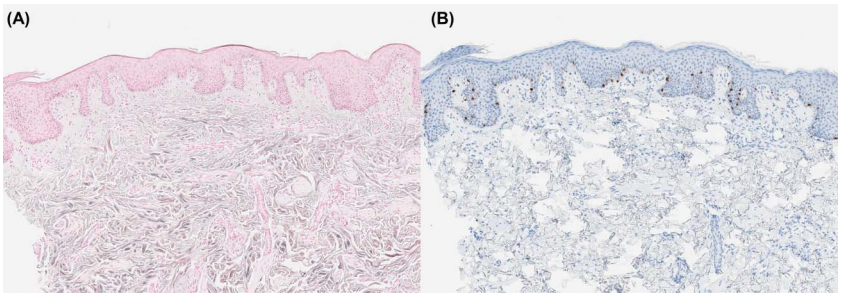

FIGURE 3. Staining for melanin pigment and melanocyte. A, The epidermal pigmentation has decreased (Fontana Masson stain, original magnification ×30). B, The number of melanocytes is adequate (immunohistochemical staining of MART-1, original magnification ×30).

diagnosis for diffuse hypopigmented patches present in infants since birth.

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Fibroblast Growth Factor Receptor 2 Overexpression in Multiple Familial Trichoepithelioma

To the Editor:

Multiple familial trichoepithelioma (MFT) is included within a spectrum of genodermatosis with development of different benign adnexal neoplasms and mutation described in the cylindromatosis tumor suppressor (*CYLD*) gene.

A 24-year-old woman, with no medical history, consulted for the appearance of asymptomatic facial lesions during the past 10 years. She had undergone CO_2 laser, 1% sirolimus cream, and 1% sunitinib cream with no response. Her father and paternal uncle had similar lesions on their faces. On examination, millimeter skin-colored papules were observed in the midfacial and malar region (Fig. 1). A biopsy of one of the papules showed histological features that led to the diagnosis of trichoepithelioma (Fig. 2). A genetic blood study identified a c.2686+1del mutation in the *CYLD* gene, and a next-generation sequencing study from tumoral tissue recognized an amplification of the genomic region associated with the fibroblast growth factor receptor 2 (*FGFR2*) gene. With all data, the diagnosis of MFT was then made.

MFT is a rare autosomal dominant disease, characterized by the progressive development of trichoepitheliomas, generally on the face and scalp.[1] MFT is considered within the spectrum of genodermatosis associated with a mutation in the *CYLD* gene, located in the chromosome 16q12. Mutations in the *CYLD* gene induce the appearance of different benign neoplasms of follicular origin, along with familial cylindromatosis (with development of cylindromas) and Brooke-Spiegler syndrome (with formation of cylindromas, spiradenomas, and trichoepitheliomas).[2,3] MFT is also linked to loss in the heterozygosity of the 9p21 gene.[4] The *PTCH* gene was also believed to be linked to the etiopathogenesis of MFT, but recent evidence suggests that it is rarely, if ever, mutated in this disease.[5]

The FGFR2 belongs to the FGFR family of tyrosine kinases receptors, involved in cell multiplication and maturation, angiogenesis, and the growth or development of bones. It has been linked to bone disorders and cancer. MFT is a benign entity, but in

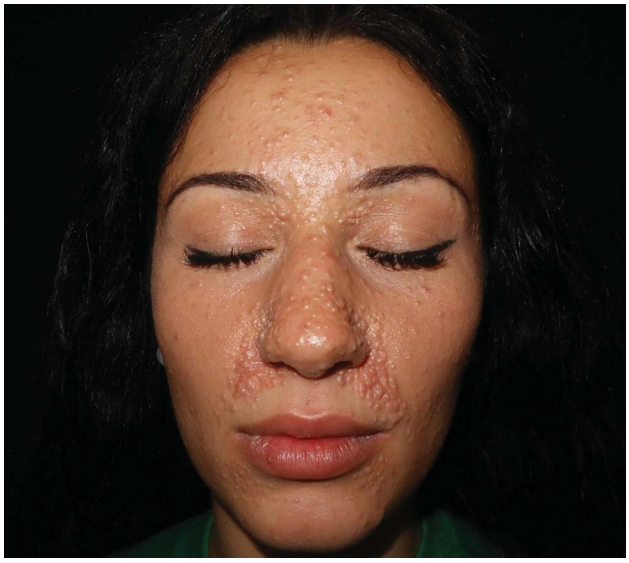

FIGURE 1. Clinical image of multiple familial trichoepithelioma. Millimeter skin-colored papules are observed in the midfacial.

The authors declare no conflicts of interest.

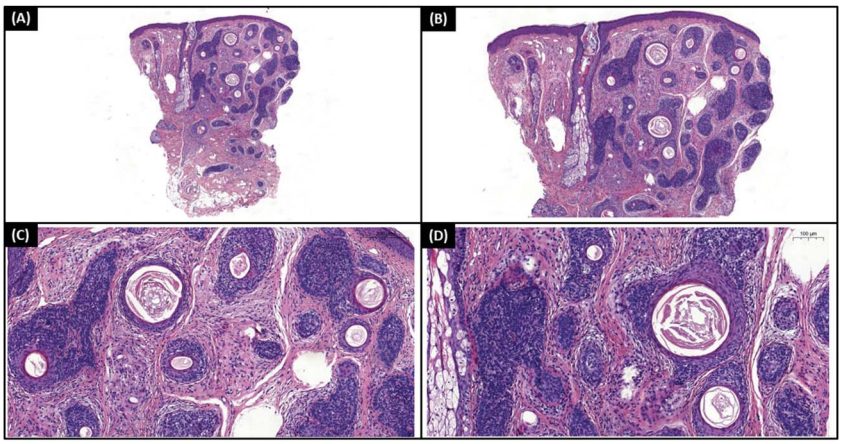

FIGURE 2. Histological images of trichoepithelioma. Nests of basaloid cells and a collagenous stroma can be seen. A, H&E, ×4. B, H&E, ×10. C and D, H&E, ×20.

some cases, the lesions can be disfiguring. No universally accepted therapy for MFT exists. Invasive procedures such as surgical excision, electrosurgery, or laser therapy have been used, but complications include pain or scarring and a high recurrence rate have been reported. Some topical agents such as 5% imiquimod or 1% sirolimus cream seem to decrease the size of the lesions, but they usually do not disappear completely.[6] Therefore, we present the only reported case of MFT with an amplification of the *FGFR2* gene, subsidiary of targeted treatment with a tyrosine kinase inhibitor of FGFR1–4, for example, erdafitinib.

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ACKNOWLEDGMENTS

The authors thank the patient for providing her informed consent for this publication.

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