skin hypopigmentation despite similar mechanisms. One explanation for the discrepancy relates to the degree of effect on various tissues to the target-specificity of imatinib because different tissues may have diverse c-kit isotypes.\(^5\) Our patient showed graying of more than 50% of the scalp and pubic hair with generalized skin hypopigmentation, but the rest of the hairs were not involved. This may suggest that imatinib has target-specificity or a varying degree of c-kit inhibition not only between skin and hair but also between each hair. Besides hair graying, our patient also showed diffuse hair loss of the scalp, which is also a possible side-effect of imatinib. It is associated with platelet-derived growth factor receptor (PDGFR)-regulated maintenance of the anagen phase of the hair cycle.\(^3\) The hair loss is thought to be related to inhibition of PDGFR by imatinib with resulting telonization of the hair follicles that eventually led to telogen effluvium.

Both hair graying and hair loss are stressful for young female patients on antitumor therapy. Therefore, physicians should be aware of hair graying as an additional possible side-effect of imatinib and notify patients.

**CONFLICT OF INTEREST:** None.

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**Delayed-onset pachyonychia congenita caused by a novel mutation in the V2 domain of keratin 6b**

Dear Editor,

Pachyonychia congenita (PC) consists of a group of rare autosomal-dominant ectodermal disorders characterized predominantly by nail dystrophy. There are two main clinical subtypes of PC: PC-1 is caused by mutations in keratin 6a (*K6a*) gene or keratin 16 (*K16*) gene, accompanied by nail dystrophy, severe palmoplantar keratoderma and oral leukokeratosis; and PC-2 is linked to mutations in the keratin 6b (*K6b*) gene or keratin 17 (*K17*) gene, associated with nail dystrophy, focal palmoplantar keratoderma and multiple steatocysts.\(^1\)

A 9-year-old Chinese female was affected by thickened fingernails and toenails at 6 years of age. Physical examination showed hyperkeratotic fingernails and toenails (Fig. 1a,b). Steatocystoma multiplex, palmoplantar keratoderma and natal teeth were not found in this patient. Repeated fungal examination under a microscope and culture excluded onychomycosis. There were no other family members affected by this disease.

Following written informed consent, genomic DNA of the proband, her parents, and 100 unrelated and unaffected people was extracted from peripheral blood. Direct sequencing of DNA from the patient revealed a heterozygous 1495G→A mutation in the *K6b* gene. A heterozygous missense mutation 1495G→A (arrow), which predicts the amino acid change glycine to serine at codon 499 (G499S) was found. (d) Sequence in normal subjects.

**Figure 1.** (a,b) Photograph of the thickened fingernails. (c) Direct sequencing of the *k6b* gene. A heterozygous missense mutation 1495G→A (arrow), which predicts the amino acid change glycine to serine at codon 499 (G499S) was found. (d) Sequence in normal subjects.

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mutation in exon 9 of the K6b gene, in the tail domain, leading to the substitution of glycine 499 by serine (G499S; Fig. 1c). No mutations were found in K17, K16, K6a or K6c. No such mutation was found in her parents and the 100 unrelated controls (Fig. 1d).

Three mutations have been reported in K6b, but this is the first mutation to be reported in the tail domain of PC-2. Another known similar mutation was a insertion mutation first mutation to be reported in the tail domain of PC-2.4 Terrinoni et al.5 detected a case of late-onset PC association with a mutation K354N in the central 2B domain of K16. Our group has reported a mutation N109D in the second half of the 1A domain of K17 with delayed onset PC-2.6 Terrinoni et al.5 reported a postzygotic mutation in the V1 domain of K16 with unilateral palmoplantar nevus and delayed onset PC-1. In this report, we found this is the V2 domain of K6b associated with late onset and milder thickened nails. The patients listed above had delayed-onset PC, the milder phenotypes, and their mutation located at the less critical site of the keratins which is consistent with Connors et al. and Xiao et al.’s speculation.3,4

In conclusion, we reported a Chinese female affected with delayed-onset PC-2 caused by a novel mutation in the K6b V2 domain. The more PC patients that are reported, the more clinical data is accumulated. It will help expand the understanding of relationship between genotype and phenotype in PC and may give some clues to the cause of the phenotypic variability.

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REFERENCES

Generalized granuloma annulare after bacillus Calmette–Guérin vaccination, clinically resembling papular tuberculid

Dear Editor,

The bacillus Calmette–Guérin (BCG) vaccine contains a live, attenuated strain of bovine tuberculin bacteria Mycobacterium bovis. BCG vaccination has appeared to protect young children from more serious forms of tuberculosis. Real incidence of complications caused by BCG vaccination have been difficult to ascertain, but are extremely low in spite of the great number of vaccinations performed. Skin complications vary from local reactions to disseminated BCG infections.1 Herein, we report a 3-month-old boy with a complication of generalized granuloma annulare (GGA) mimicking papular tuberculid after BCG vaccination.

A 3-month-old boy was referred to our dermatology clinic because of generalized eruptions after BCG vaccination. The boy was a full-term baby and had no specific past and family history including tuberculosis. He received a BCG vaccination on his right arm 1 month after birth. He presented multiple small erythematous papules, plaques and crusts on the face, trunk and extremities symmetrically without symptoms occurring 7 weeks after BCG vaccination. The BCG injection site also showed papular lesions (Fig. 1a–c). On physical examination, no abnormal lymphadenopathy was seen and the overall clinical features were similar to popular tuberculid. Routine blood and biochemical tests as well as chest X-ray did not show any abnormal findings. QuantiFERON-TB Gold using serum and tuberculin skin test with purified protein derivative of tuberculin were negative. Histopathology showed a feature of chronic palisading granulomatous inflammation with central necrosis and suppuration(Fig. 1d,e). Multiple foci of inflammation with a central core of collagen degeneration surrounded

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