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Half-and-Half Nails

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A 13-year-old boy was referred to the genetics clinic for evaluation of dysmorphic features associated with specific nail abnormalities, developmental delay, hypertension, and heart murmur.

History. The boy's nail abnormalities and speech delay had become prominent at age 15 months. Later, at the age of 5 years, he had received a diagnosis of benign essential hypertension, systolic heart murmur, atopic dermatitis, and chronic macrocytic anemia. A dermatologist had diagnosed half-and-half nails, possibly resulting from underlying kidney disease. Nevertheless, with the exception of a finding of macrocytic anemia on a complete blood cell count, results of renal ultrasonography and further laboartory investigations, including renal function tests, were unremarkable.

He was currently being cared for by a dermatologist, a nephrologist, a cardiologist, and a hematologist for his underlying conditions, and he had been receiving services for developmental delay.

His birth history was unremarkable. His family history showed a 2-year-old brother with similar findings of dysmorphic features and half-and-half nails that had become prominent at age 2 months and the later onset of developmental delay and heart murmur. He also had a healthy 17-year-old sister without nail abnormalities, and his father had the same nail abnormality but without systemic involvement.

Physical examination. Physical examination showed microcephaly, a high hair line, mild frontal bossing, up-slanting eyes with epicanthal folds, curved eyelashes, a grade 2/6 systolic murmur at the lower left sternal border, dry skin with multiple papules, and nails on the bilateral hands and feet with a proximal opaque white portion (4 mm) and distal dark brown portion (6 mm) (**Figure**).

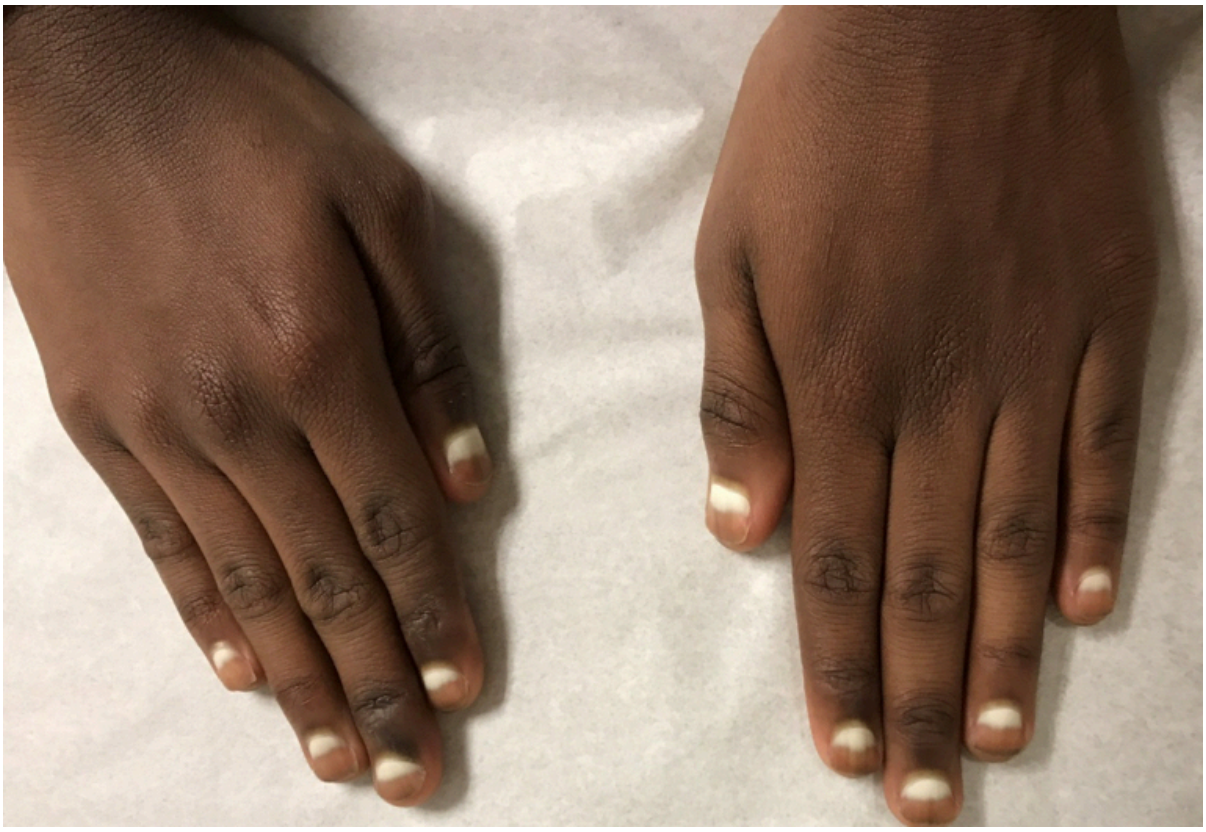

Figure. *Half and half nails with proximal white portion measuring 4 mm and the distal brown portion measuring 6 mm.*

Diagnostic tests. Due to the dysmorphic features and the constellation of systemic involvement, genetic studies including microarray, karyotype, and metabolic studies (plasma acylcarnitine profile, plasma amino acid analysis, and urine organic acid test) were performed. The results were unremarkable, and the boy was referred to a genetics specialists for further evaluation.

Discussion. Half-and-half nails, also known as Lindsay nails, are characterized by nail bed changes in which the proximal half of the nail appears white or pale pink, and the transverse distal portion appears to be pink or reddish brown, occupying 20% to 60% of the total nail portion.[1] There is a sharp demarcation between the 2 portions, which does not disappear with the application of pressure.

Half-and-half nails should be clinically differentiated from Terry nails, in which the proximal white area occupies 80% of the nail bed, while the distal pink-brown portion measures 0.5 to 3.0 mm.[1,2] In our patient, the proximal white portion occupied 40% of the nail, while the distal brown portion occupied 60%, thus fulfilling the criteria for half-and-half nails. This pattern was first

observed by Bean[3] in 1962 in patients with azotemia but was termed as half-and-half nails in 1967 by Lindsay.[1]

The prevalence of half-and-half nails in children is not known. The youngest patient reported with the condition was 10 years old.[4] However, studies in adults have shown that this phenomenon is most commonly associated with chronic kidney disease, with a prevalence of 20% to 50%,[1] with 14% of patients undergoing hemodialysis.[5] Other reported causes include yellow nail syndrome,[6] hyperthyroidism,[6] Crohn disease,[7] Kawasaki disease, Behçet disease,[8] cirrhosis, zinc deficiency, citrullinemia, HIV infection, and pellagra.[9,10] One pediatric case was reportedly acquired after chemotherapy.[11] It also can be present in healthy individuals without systemic involvement.[2,6] In all of the reported cases to date, the patients acquired the condition later in life, in contrast to the patient in our case.

The exact pathogenesis of half-and-half nails is unclear. However, Leyden and Wood proposed that, based on nail biopsy results, the brownish discoloration is secondary to melanin deposition by nail matrix melanocytes stimulated by a uremic environment resulting from renal failure.[12] Another theory postulates that increases in capillary density and wall thickening are responsible for half-and-half nails.[1] The most commonly accepted theory is that the condition could be a result of excessive development of connective tissue between the nail and bone that reduces the quantity of blood in the subcapillary plexus.[7,8] However, in our case, given that all possible etiologies for half-and-half nails were ruled out, that the condition appeared when the patient was a toddler, and that the family history was positive (occurring in his father and brother but not in his sister), the presence of half-and-half nails can only be explained by a pattern of inheritance.

To our knowledge, no studies or reports have documented the association of half-and-half nails with a syndrome, and further studies should be conducted to evaluate such an association. The case described here appears to be the first association of half-and-half nails with multisystem involvement and syndromic features. The patient's family members' similar findings should prompt further investigation into genetic causes.

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