

# Total melanonychia of 20 nails as a rare manifestation of vitamin B12 deficiency



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## CASE REPORT

A 47-year-old black male reported diffuse and progressive hyperpigmentation of his fingernails and toenails beginning 2 years earlier (Figs 1 and 2). There was no hyperpigmentation of the skin or mucous membranes, hair color change, or family history of melanonychia. He also complained of asthenia, hyporexia, postprandial epigastric pain, and a weight loss of 7 kg during the period. His medical history revealed use of fluoxetine at 20 mg/day for 6 months because of depressive disorder. There was no history of alcoholism, gastrointestinal bleeding, or abnormalities suggestive of autoimmune or endocrine diseases.

The laboratory evaluation revealed anemia (hemoglobin level 2.7 g/dL, hematocrit level 8.5%, red cell distribution width 39.1%, and mean corpuscular volume 99.5 femtoliters) and the presence of dacryocytes. Further investigation of the anemia revealed a vitamin B12 level of less than 30 pg/mL (normal 200-900 pg/mL), negative direct Coombs test result, and absence of significant changes in iron kinetics and levels of folic acid, indirect bilirubin, and lactate dehydrogenase. Upper digestive endoscopy showed gastric atrophy throughout the mucosa of the fundus, body, and antrum, with negative results for *Helicobacter pylori* testing.

Treatment was started with daily doses of 5000 µg cyanocobalamin, 100 mg pyridoxine, and 100 mg thiamine and 1 monthly maintenance dose of this compound, all intramuscularly for seven days. After 3 months of treatment, the patient reported improvement of the nail discoloration (Fig 3), as well as improvement in constitutional symptoms. Laboratory studies revealed levels of vitamin B12 at



**Fig 1.** Black pigmentation of the fingernails on the right hand.



**Fig 2.** Total melanonychia of the toenails.

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**Fig 3.** Marked improvement of the hyperpigmentation after 3 months of treatment.

940 pg/mL, hemoglobin at 14.4 g/dL, and hematocrit at 43.5%.

## DISCUSSION

Dark discoloration of the nails has a number of causes, including melanocytic activation, lentigo, melanocytic nevus, and melanoma.<sup>1</sup> Both melanin and hemosiderin are common pigments of the nail plate, but in rare cases the nail color is due to pigment deposition of endogenous or exogenous origin. The melanin deposits result from the activation or proliferation of nail matrix melanocytes, resulting in brown to black longitudinal, transverse, or total melanonychia. The latter in turn is a rare presentation.<sup>1,2</sup>

There are many dermatologic presentations related to cobalamin deficiency, including nail and mucocutaneous hyperpigmentation predominantly in pressure and flexural areas, angular stomatitis, glossitis, and hair changes such as poliosis.<sup>3</sup> The most common reported nail alteration is longitudinal melanonychia.<sup>3,4</sup> Hypocobalaminemia in vitro model experiments have demonstrated increased melanogenesis caused by tyrosinase activation, as well as increased reactive oxygen species and reduced glutathione in melanocytes.<sup>5</sup> It is hypothesized that the disinhibition of the tyrosinase caused by decreased glutathione could be involved in the production of melanin. In this regard, supplementation of vitamin B12 can reverse this process.<sup>5,6</sup>

Whereas frequent causes of melanocytic activation are associated with local disruption such as occupational trauma and onychotillomania or physiologic

events such as racial melanonychia and pregnancy, systemic causes are considered rare<sup>2,7</sup> and include endocrine disorders (Addison disease, Cushing syndrome, hyperthyroidism, and acromegaly), porphyria, AIDS, autoimmune diseases, hemosiderosis, malnutrition, and vitamin D deficiency.<sup>7-10</sup> Although there are case reports of longitudinal melanonychia in vitamin B12 deficiency, total melanonychia in the fingernails and toenails as reported here is a rare manifestation of this condition.<sup>10</sup>

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