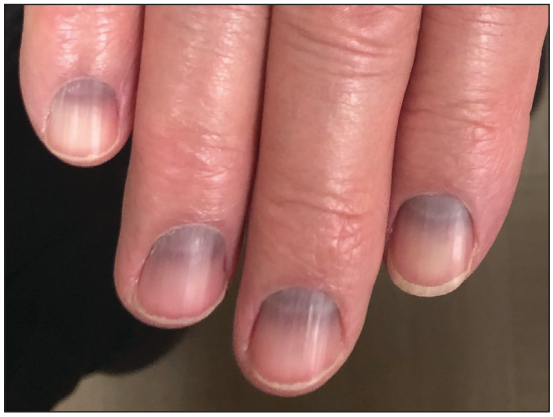
Blue to Slate Gray Discoloration of the Proximal Fingernails

Marlee Hill, BS; Chance Morris, MD; Allison Hood, MD



An 88-year-old man presented with asymptomatic and unchanging discoloration of the proximal fingernails of both hands of 50 years' duration. Physical examination revealed blue to slate gray, subungual pigmentary changes of the fingernails of both hands sparing the nail bed distal to the lunulae. There was no overlying plate dystrophy, toenail involvement, or additional mucocutaneous abnormalities. His medical history was notable for heart failure, obstructive sleep apnea, and type 2 diabetes mellitus. He had no history of hepatic, ophthalmologic, or neurologic dysfunction.

WHAT'S YOUR **DIAGNOSIS?**

a. argyria-induced azure lunulae
b. chloronychia
c. hereditary acrolabial telangiectasia
d. nail bed cyanosis
e. Wilson disease

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From the University of Oklahoma, Oklahoma City. Marlee Hill is from the College of Medicine, and Drs. Morris and Hood are from the Department of Dermatology, Health Sciences Center.
The authors report no conflict of interest.
Correspondence: Marlee Hill, BS, University of Oklahoma College of Medicine, 940 Stanton L. Young Blvd #357, Oklahoma City, OK 73104 (Marlee-hill@ouhsc.edu).
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THE **DIAGNOSIS:** Argyria-Induced Azure Lunulae

Argyria is an acquired condition resulting from excessive exogenous exposure to silver with subsequent gastrointestinal absorption and pigmentary tissue deposition. Upon further questioning, our patient disclosed a lifetime history of colloidal silver use, both as a topical antiseptic agent and intraorally for aphthous ulcers. Silver has a predilection for granular deposition in stromal tissues and basement membranes with sparing of the epidermis, manifesting as progressive, permanent, blue to slate gray discoloration of sun-exposed skin, mucous membranes, and nail beds.[1] The patient was advised to discontinue use of colloidal silver to avoid development of further pigmentary changes. The appearance of his nails remained unchanged in the months following initial presentation, as expected, since argyria pigmentation is not anticipated to reverse upon colloidal silver cessation.

Nail involvement may be an early presentation of generalized argyria or may be found in isolation, as seen in our patient. Early recognition and patient education are essential to minimize cumulative silver deposition. Although dyspigmentation may impact psychosocial well-being secondary to aesthetic concerns, there is limited research supporting adverse systemic effects of argyria confined to the nail beds. Similarly, the majority of generalized cases are not associated with systemic complications; however, potential toxicities, as described in isolated case reports without conclusive causal relationships, include nyctalopia, renal or hepatic toxicity, pulmonary fibrosis, and neuropsychiatric events.[1-6] Successful treatment of cutaneous argyria has been reported with the 1064-nm Q-switched Nd:YAG laser; however, there have been no reported treatments for nail bed involvement.[7] Due to the absence of systemic symptoms, additional mucocutaneous dyspigmentation, or cosmetic concerns regarding nail bed lunulae discoloration in our patient, no further intervention was pursued, except for continued colloidal silver cessation.

The differential diagnosis of blue-gray nail bed dyspigmentation is broad and includes cyanosis secondary to cardiopulmonary disease, drug-induced dyspigmentation, Wilson disease, argyria, chrysiasis, hereditary acrolabial telangiectasia, and pseudomonal infection or chloronychia.[1,8,9] Etiologic insight may be provided from a thorough review of prescription and over-the-counter medications as well as careful attention to the distribution of dyspigmentation. Medications commonly associated with bluish nail bed dyspigmentation include antimalarials, amiodarone, minocycline, clofazimine, chlorpromazine/phenothiazines, and various chemotherapeutic drugs; our patient was not taking any of these.[1,9]

Cyanotic nail bed dyspigmentation secondary to cardiopulmonary disease likely manifests with more diffuse nail bed dyspigmentation and is not confined solely to the lunulae. Only drug-induced dyspigmentation, classically due to phenolphthalein-containing laxatives; Wilson disease; and argyria have a tendency to spare the distal nail bed, which is a presentation termed *azure lunulae*.[8] The toenails typically are spared in argyria, while toenail involvement is variable in Wilson disease, and additional systemic symptoms—including hepatic, ophthalmologic, and neuropsychiatric—as well as potential family history would be expected.[8] Phenolphthalein is no longer available in over-the-counter laxatives, as it was formally banned by the US Food and Drug Administration in 1999 due to concerns of carcinogenicity.[10]

Hereditary acrolabial telangiectasia is a familial condition with autosomal-dominant inheritance that can manifest similarly to argyria with blue-gray discoloration of the proximal nail bed; however, this condition also would demonstrate involvement of the vermilion border and nipple areolae, often with associated telangiectasia and migraine headaches.[11]

Chloronychia (also known as green nail syndrome) is an infection of the nail bed with *Pseudomonas aeruginosa* that more commonly presents with green-black discoloration with variable involvement of the fingernails and toenails. Chloronychia, often with associated onycholysis, typically is found in individuals with repeated exposure to water, soaps, and detergents.[12] Our patient's long-standing and unwavering nail bed appearance, involvement of all fingernail lunulae, lack of additional symptoms, and disclosed use of over-the-counter colloidal silver supported a clinical diagnosis of argyria-induced azure lunulae.

Argyria-induced azure lunulae secondary to colloidal silver exposure is an uncommon yet clinically significant cause of nail bed dyspigmentation. Prompt identification and cessation of the offending agent can prevent progression of mucocutaneous dyspigmentation and avoid potential long-term sequelae from systemic deposition.

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