A 49-year-old man presented with “bruising” of the fingernails and facial skin. He had recalcitrant rosacea and had undergone bilateral cataract extraction, but had no other significant medical history. He had not noticed a propensity to bruise, impairment of wound healing or any excessive gingival or nasal bleeding. He had been taking minocycline (100 mg orally twice daily) for his rosacea for the past 12 years.

On examination, the patient had marked blue-grey discoloration of the proximal nail beds with undisturbed nail formation (Figure 1A). Dark brown, patchy discoloration of the periorbital skin was subtle but present (Figure 1B). He was not cyanotic; capillary refill was normal bilaterally and the Allen test, in which ulnar artery patency is assessed by radial artery occlusion, showed sufficient collateral flow in both hands. Cardiovascular, respiratory and abdominal examinations were unremarkable. Laboratory investigations, including complete blood counts and testing of liver transaminase levels, kidney function, lipids, thyroid function and protein electrophoresis, did not show any abnormalities. The discoloration was thought to be a result of prolonged therapy with minocycline.

Nail bed discoloration is an uncommon side effect of minocycline therapy but often accompanies discoloration at other sites, including skin, sclera, palate, teeth, thyroid and bone.1 Incidence data are scant, but reports suggest that 3%—15% of those treated with cumulative doses greater than 100 g will develop discoloration in at least one site.1 Older age and a diagnosis of rosacea are more commonly associated with minocycline-induced discoloration, but this is likely attributable to higher cumulative doses in these patient groups.1

Three patterns of minocycline-induced pigmentation have been described.1 Type I results in blue-black macules localized to areas of active inflammation or scarring. This type appears to be dose-independent and results from local pigment deposition by macrophages.1 Type II pigmentation most commonly presents as blue-grey or brown patches on the shins, ankles and arms. Pigment complexes can be found in dermal macrophages or scattered freely within dermal collagen. Type III pigmentation is a symmetric and generalized pigmentation on sun-exposed areas. It is likely caused by increased melanin or melanin–minocycline complexes at the dermal–epidermal interface.1 The patient’s discoloration was thought to be most consistent with type II pigmentation of the nail beds and type III pigmentation of the face.

Six months after discontinuation of minocycline, the patient’s discoloration had not yet resolved. Pigmentation of the skin and nails may require months to years to fade after discontinuation of the drug, and other sites may remain permanently discoloured.1 Type III pigmentation is less likely to resolve despite discontinuation of minocycline.1 In light of the latter, it may be advisable for practitioners to warn patients about the risk of cutaneous discoloration when beginning minocycline therapy. For affected patients, timely recognition of this phenomenon may prevent unnecessary investigation or biopsy.

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REFERENCE