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Hyperpigmentation in Chronic Myeloid Leukemia

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Abstract **Case Report**

Imatinib is widely used in the management of chronic myeloid leukemia and is associated with various cutaneous adverse events, most commonly hypopigmentary changes. Hyperpigmentation remains uncommon and may pose diagnostic difficulties, particularly when involving the face. We report the case of a 66-year-old woman treated with imatinib since 2018 who developed progressive, asymptomatic, bilateral blue-violaceous macules coalescing into patches over the temporal regions. Dermoscopy revealed a slate-blue pigmentation with sparse pigmented dots and globules. Skin biopsy showed dermal pigmentary incontinence with melanophages, without features supporting interface dermatitis or ochronosis. The clinical, dermoscopic and histological findings supported imatinib-induced cutaneous hyperpigmentation, and the causal relationship was confirmed by pharmacovigilance assessment. This observation highlights an uncommon pigmentary adverse effect of imatinib and the value of clinicopathological correlation in avoiding misdiagnosis and unnecessary modification of an essential therapy.

Keywords: Imatinib, Hyperpigmentation, Drug-induced pigmentation, Chronic myeloid leukemia, Dermoscopy, Tyrosine kinase inhibitor.

INTRODUCTION

Imatinib is an oral tyrosine kinase inhibitor targeting BCR-ABL, KIT and platelet-derived growth factor receptors, and it remains an established treatment for chronic myeloid leukemia [1]. Cutaneous adverse reactions to imatinib are frequent and include maculopapular eruptions, edema, pruritus, xerosis and pigmentary changes [2]. Among pigmentary adverse events, hypopigmentation is much better recognized than hyperpigmentation [3,4], and in vitro work supports an inhibitory effect of imatinib on melanogenesis [5]. Paradoxical hyperpigmentation has been reported only occasionally and may involve the skin, oral mucosa, nails, teeth, hair or gingiva [6–14]. The paradoxical occurrence of hyperpigmentation despite inhibition of the KIT pathway makes these cases diagnostically and mechanistically interesting. We report a case of bilateral, symmetric, slate-blue to blue-violaceous temporal facial hyperpigmentation in a woman receiving long-term imatinib for chronic myeloid leukemia.

CASE REPORT

A 66-year-old woman was evaluated for progressive facial hyperpigmentation. Her medical history included type 2 diabetes treated with oral antidiabetic agents, goiter and chronic myeloid leukemia treated with imatinib since 2018. The pigmented lesions had been present on the temples for approximately one year and had progressively increased in size. They were asymptomatic and no functional symptoms were reported.

Clinical examination showed bilateral and symmetric blue-violaceous to slate-blue macules coalescing into poorly demarcated patches over the temporal and frontotemporal regions (Fig. 1). The lesions formed sheet-like areas without visible erythema, scaling, ulceration, atrophy or inflammatory infiltration. Dermoscopy revealed a diffuse slate-blue to blue-gray structureless background with scattered gray-brown pigmented dots and a few pigmented globules; follicular openings and terminal hairs were preserved (Fig. 2). The

main clinical differential diagnoses were drug-induced pigmentation, melasma and lichen planus pigmentosus.

Figure 1: Clinical photographs showing bilateral, symmetric blue-violaceous to slate-blue macules coalescing into poorly demarcated patches over the temporal and frontotemporal regions, without overt erythema or scaling. Panels A and B show the two temporal regions

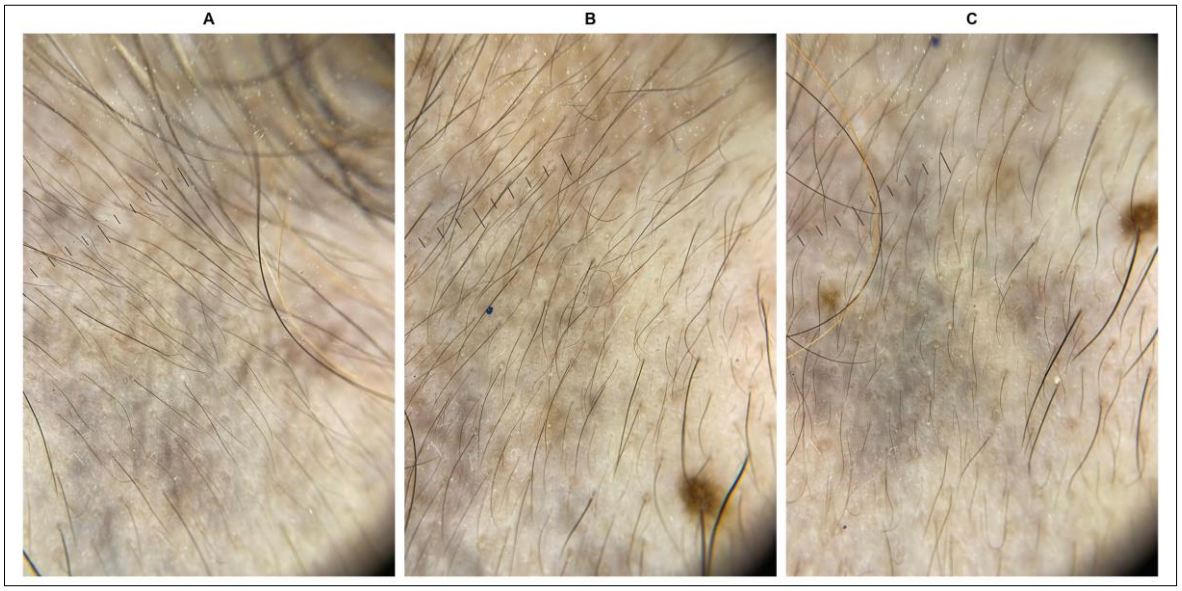

Figure 2: Dermoscopic views (A-C) showing a slate-blue to blue-gray structureless background with scattered gray-brown pigmented dots, a few pigmented globules and preserved follicular openings

A punch biopsy from a pigmented lesion was examined after hematoxylin and eosin staining. The epidermal thickness was substantially preserved, with a regular orthokeratotic stratum corneum and only slight effacement of epidermal ridges. The basal layer was preserved, without basal vacuolization or apoptotic keratinocytes. The dermoepidermal junction showed no lichenoid band-like infiltrate, and there was no notable basement membrane thickening. The superficial and deep dermis showed discrete-to-moderate pigmentary incontinence, consisting of free pigment and pigment phagocytosed by dermal melanophages. Mild-to-moderate hyalinization of superficial dermal connective tissue was noted, while the deeper dermal architecture was relatively regular. No significant dermal inflammatory infiltrate was observed. No characteristic ochronotic deposits, such as brownish homogenized banana-shaped collagen fibers, were identified. Adnexal

and visible vascular structures showed no notable abnormality (Fig. 3).

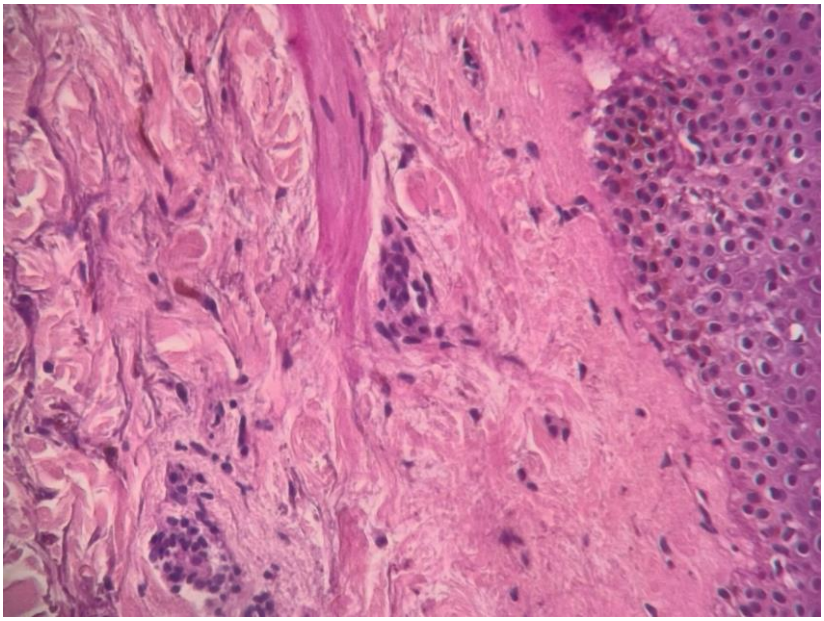

Figure 3: Histopathological photomicrograph showing no active interface dermatitis and the presence of dermal melanophages. The absence of brownish homogenized collagen fibers makes exogenous ochronosis unlikely (hematoxylin and eosin stain; original magnification ×200).

On the basis of the clinical distribution, color, progressive course during long-term imatinib therapy and the histological findings, the diagnosis of imatinib-induced cutaneous hyperpigmentation was retained. The adverse event was reported through the pharmacovigilance system, and the subsequent assessment confirmed a causal relationship with imatinib.

DISCUSSION

Imatinib-associated pigmentary changes illustrate the complex role of tyrosine kinase signaling in melanocyte biology. Hypopigmentation is classically attributed to KIT pathway inhibition, with subsequent effects on melanocyte function and melanogenesis [3–5]. Conversely, imatinib-related hyperpigmentation remains rare and its mechanism is not fully understood [6–14]. Reported hypotheses include drug-melanin complex deposition, altered melanocyte signaling, a cytotoxic response to an epidermal neoantigen and activation or unmasking of dermal melanocytosis in predisposed patients [6,10,13].

Reported cases have described heterogeneous phenotypes, including melasma-like facial pigmentation, slate-gray pigmentation of the nose and hard palate, diffuse skin, mucosal and nail pigmentation, and acquired dermal melanocytosis with bilateral nevus of Ota-like macules [7–14]. Our case is closest clinically to reported blue-gray facial or temporal hyperpigmentation, yet the biopsy did not show dermal dendritic melanocytes; instead, the main finding was pigmentary incontinence with dermal melanophages. This pattern favors drug-induced dermal melanosis rather than acquired dermal melanocytosis.

The principal differential diagnoses were melasma, lichen planus pigmentosus and exogenous ochronosis. Melasma was less favored because of the slate-blue to blue-violaceous hue and the histological predominance of dermal pigment incontinence rather than simple epidermal hypermelanosis. Lichen planus pigmentosus and Riehl melanosis were not supported by the biopsy because there was no basal vacuolar degeneration, no apoptotic keratinocytes and no lichenoid interface infiltrate. Exogenous ochronosis was unlikely because no ochronotic banana-shaped collagen fibers were observed. The absence of visible inflammation and the symmetric temporal distribution further supported a non-inflammatory drug-related pigmentation.

Management of imatinib-associated pigmentation should be individualized. Because imatinib may be essential for hematologic disease control, discontinuation should not be considered without hematology input. In reported cases, treatment has ranged from reassurance and strict photoprotection to topical depigmenting agents when cosmetic concern is important, while imatinib was often continued [10,12].

CONCLUSION

Imatinib may rarely be associated with blue-gray or blue-violaceous cutaneous hyperpigmentation. In

a patient receiving imatinib, bilateral symmetric temporal facial pigmentation should prompt consideration of a drug-induced process. Correlation of clinical morphology, dermoscopic findings, histology and chronology is essential to distinguish this reaction from melasma, lichen planus pigmentosus and ochronosis. Recognition of this adverse event may prevent diagnostic errors and support coordinated dermatologic and hematologic management.

Declarations

Ethics statement: Ethical approval was not required for this single-patient case report.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical, dermoscopic and histopathological images.

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Conflict of interest: None declared

Author contributions: All authors contributed to the preparation and critical revision of the manuscript and approved the final version.

REFERENCES

1. O'Brien, S. G., Guilhot, F., Larson, R. A., Gathmann, I., Baccarani, M., Cervantes, F., Cornelissen, J. J., Fischer, T., Hochhaus, A., Hughes, T., Lechner, K., Nielsen, J. L., Rousselot, P., Reiffers, J., Saglio, G., Shepherd, J., Simonsson, B., Gratwohl, A., Goldman, J. M., … Druker, B. J. (2003). Imatinib compared with interferon and low-dose cytarabine for newly diagnosed chronic-phase chronic myeloid leukemia. *New England Journal of Medicine, 348*(11), 994–1004. https://doi.org/10.1056/NEJMoa022457
2. Valeyrie, L., Bastuji-Garin, S., Revuz, J., Bachot, N., Wechsler, J., Berthaud, P., Tulliez, M., & Giraudier, S. (2003). Adverse cutaneous reactions to imatinib (STI571) in Philadelphia chromosome-positive leukemias: A prospective study of 54 patients. *Journal of the American Academy of Dermatology, 48*(2), 201–206. https://doi.org/10.1067/mjd.2003.44
3. Tsao, A. S., Kantarjian, H., Cortes, J., O'Brien, S., & Talpaz, M. (2003). Imatinib mesylate causes hypopigmentation in the skin. *Cancer, 98*(11), 2483–2487. https://doi.org/10.1002/cncr.11812
4. Arora, B., Kumar, L., Sharma, A., Wadhwa, J., & Kochupillai, V. (2004). Pigmentary changes in chronic myeloid leukemia patients treated with imatinib mesylate. *Annals of Oncology, 15*(2), 358–359. https://doi.org/10.1093/annonc/mdh068
5. Cario-André, M., Ardilouze, L., Pain, C., Gauthier, Y., Mahon, F.-X., & Taïeb, A. (2006). Imatinib mesilate inhibits melanogenesis in vitro. *British Journal of Dermatology, 155*(2), 493–494. https://doi.org/10.1111/j.1365-2133.2006.07359.x
6. Balagula, Y., Pulitzer, M. P., Maki, R. G., & Myskowski, P. L. (2011). Pigmentary changes in a patient treated with imatinib. *Journal of Drugs in Dermatology, 10*(9), 1062–1066.
7. Li, C.-C., Malik, S. M., Blaeser, B. F., Dehni, W. J., Kabani, S. P., Boyle, N., Toner, M., & Woo, S.-B. (2012). Mucosal pigmentation caused by imatinib: Report of three cases. *Head and Neck Pathology, 6*(2), 290–295. https://doi.org/10.1007/s12105-011-0325-4
8. Song, H. S., & Kang, H. Y. (2014). Imatinib mesylate-induced hyperpigmentation of the nose and palate. *Annals of Dermatology, 26*(4), 532–533. https://doi.org/10.5021/ad.2014.26.4.532
9. Balasubramanian, P., Jagadeesan, S., & Thomas, J. (2015). Imatinib-induced extensive hyperpigmentation in a case of chronic myeloid leukemia. *Indian Journal of Dermatology, 60*(5), 523. https://doi.org/10.4103/0019-5154.164447
10. Ghunawat, S., Sarkar, R., & Garg, V. K. (2016). Imatinib induced melasma-like pigmentation: Report of five cases and review of literature. *Indian Journal of Dermatology, Venereology and Leprology, 82*(4), 409–412. https://doi.org/10.4103/0378-6323.182387
11. Di Tullio, F., Mandel, V. D., Scotti, R., Padalino, C., & Pellacani, G. (2018). Imatinib-induced diffuse hyperpigmentation of the oral mucosa, the skin, and the nails in a patient affected by chronic myeloid leukemia: Report of a case and review of the literature. *International Journal of Dermatology, 57*(7), 784–790. https://doi.org/10.1111/ijd.13931
12. Rehman, H., Hakim, N., Sugarman, R., Seetharamu, N., & Saif, M. W. (2020). Hyperpigmentation due to imatinib: A rare case of cutaneous involvement. *Journal of Oncology Pharmacy Practice, 26*(6), 1511–1515. https://doi.org/10.1177/1078155220903364
13. Jang, J. W., Song, C. H., Jung, Y. J., Cheon, D. U., Koh, W. S., Ro, Y. S., & Ko, J. Y. (2021). Imatinib mesylate-induced acquired dermal melanocytosis and acquired bilateral nevus of Ota-like macules. *Annals of Dermatology, 33*(3), 293–294. https://doi.org/10.5021/ad.2021.33.3.293
14. Garraway, C., Yang, K., Kantamneni, K., Kissel, R., & Pavlidakey, P. G. (2024). Imatinib-induced hyperpigmentation. *JAAD Case Reports, 52*, 28–30. https://doi.org/10.1016/j.jdcr.2024.06.038