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# Chemotherapy-Induced Mucocutaneous Hyperpigmentation: A Case Series in Patients Receiving Etoposide-Based Regimens

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## ABSTRACT

*Background: Cytotoxic chemotherapy can produce a wide range of mucocutaneous and nail pigmentary changes that, although generally benign, are frequently distressing to patients and can be mistaken for disease progression or an unrelated dermatologic process. Etoposide-based regimens, commonly combined with bleomycin, platinum agents, or alkylating agents, represent a recurrent clinical context in which such changes are observed. Objective: To synthesize the published literature on chemotherapy-induced mucocutaneous and nail hyperpigmentation, with particular attention to reports arising in the setting of etoposide-containing regimens, and to place these findings within the broader spectrum of cytotoxic-agent-associated pigmentary toxicity. Methods: A narrative review was conducted of case reports, case series, and systematic reviews describing chemotherapy-induced hyperpigmentation, focusing on mechanistic explanations, clinical patterns associated with etoposide and its common regimen partners, agent-specific pigmentary syndromes across the wider cytotoxic drug class, and diagnostic and management approaches. Findings: Etoposide is rarely isolated as a sole causative agent; most reports implicate concomitant bleomycin, cyclophosphamide, or platinum compounds as the more likely proximate cause of the described pigmentation. Well-characterized entities across the broader cytotoxic drug class include bleomycin-induced flagellate hyperpigmentation, 5-fluorouracil-associated serpentine supravenous streaking (reported in 2-5% of treated patients), and hydroxyurea-related melanonychia after prolonged therapy. A large case series of 54 patients found cutaneous involvement in 83.3% and melanonychia in 18.5%, while a pooled meta-analysis estimated antineoplastic-agent-associated hyperpigmentation at approximately 35.5% of treated patients. Conclusions: Chemotherapy-induced mucocutaneous and nail hyperpigmentation is a common, pattern-recognizable, and generally self-limited toxicity best managed conservatively, with dermatologic referral reserved for diagnostic uncertainty or marked cosmetic distress; further case-level reporting specific to etoposide, distinct from its regimen partners, remains warranted.*

***Keywords:*** *chemotherapy-induced hyperpigmentation; etoposide; melanonychia; mucocutaneous pigmentation; serpentine supravenous hyperpigmentation*

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## Introduction

Pigmentary change is among the most visible and, for many patients, most distressing cutaneous consequences of cytotoxic chemotherapy. Unlike alopecia or mucositis, which typically resolve within weeks of treatment completion, drug-induced hyperpigmentation can persist for months to years after the causative agent has been withdrawn, and it frequently affects sites that are difficult to conceal, including the face, oral mucosa, nail bed, and palms and soles [1]. Systematic and pooled analyses of drug-induced pigmentary disorders indicate that antineoplastic agents are among the most frequently implicated drug classes, with hyperpigmentation reported in roughly a third of patients receiving pigmentation-associated cytotoxic regimens in aggregated cohorts, a burden comparable to or exceeding that attributed to antimalarials or minocycline [2]. Etoposide-based combination regimens—typically paired with platinum agents, bleomycin, ifosfamide, or cyclophosphamide for germ-cell tumors, small-cell lung cancer, and lymphoma—are a common context in which clinicians encounter these changes, yet etoposide itself is rarely isolated as the causative agent in the literature; more often it is one component of a multidrug regimen in which bleomycin, alkylating agents, or the combination as a whole is implicated in the resulting pigmentary pattern [3,4].

The clinical spectrum of chemotherapy-induced hyperpigmentation is broad, encompassing diffuse cutaneous darkening, localized or flagellate patterns, serpentine supravenous streaking overlying the infused vein, and mucosal and nail changes including longitudinal or transverse melanonychia, diffuse nail plate darkening, and blue or brown discoloration of the lunula and oral mucosa [5]. Because these findings can be alarming to patients and are sometimes mistaken for signs of disease progression, systemic toxicity, or an unrelated dermatologic disorder, accurate recognition of the characteristic patterns associated with specific cytotoxic agents is clinically important both for reassurance and for guiding any indicated evaluation. This narrative review synthesizes the published case report, case series, and systematic review literature on chemotherapy-induced mucocutaneous and nail hyperpigmentation, with particular attention to reports arising in the context of etoposide-containing regimens, across four domains: the proposed mechanisms of chemotherapy-induced melanogenesis, the clinical patterns most frequently described in association with etoposide and its common regimen partners, the wider spectrum of cytotoxic and targeted agents implicated in pigmentary change, and the diagnostic and management approach to the affected patient.

## Mechanisms of Chemotherapy-Induced Melanogenesis and Pigmentary Change

No single mechanism accounts for the diversity of pigmentary patterns produced by cytotoxic chemotherapy, and most reviews converge on a small set of overlapping pathophysiologic processes rather than a unified explanation [1,5]. Direct cytotoxic injury to basal keratinocytes and melanocytes is thought to trigger a reactive increase in melanin synthesis and transfer, analogous to post-inflammatory hyperpigmentation, and is invoked particularly for diffuse cutaneous and mucosal darkening following agents such as busulfan, cyclophosphamide, and hydroxyurea [6,7]. A second proposed mechanism is direct stimulation of melanocytes or upregulation of tyrosinase activity by the drug or a reactive metabolite, which has been suggested for hydroxyurea-associated nail and skin pigmentation on the basis of its antimetabolite mechanism and known effects on epidermal turnover [8,9]. Localized deposition of the drug or a pigmented metabolite in the dermis, sometimes with associated melanin incontinence and dermal macrophage uptake, has been described histologically in several case reports and is one explanation offered for the persistence of pigmentation long after drug discontinuation [10].

A distinct mechanism operates in serpentine supravenous hyperpigmentation, in which a linear or reticulate band of pigmentation develops directly over the superficial vein used for infusion; proposed explanations include a local irritant or toxic effect of the extravasating or diffusing drug on the vessel wall and adjacent epidermis, localized post-inflammatory change following a subclinical phlebitis, and a possible photosensitizing or direct pigmentary effect potentiated by sun exposure of the affected limb [11]. Trauma- or pressure-related potentiation of pigmentation has also been documented, most clearly for cisplatin, in which hyperpigmentation localized to areas of mechanical pressure or friction—such as beneath a tourniquet or adhesive tape—suggests that microvascular or mechanical injury can lower the threshold for a chemotherapy-induced pigmentary reaction at a given site [10]. Finally, flagellate pigmentation, most classically associated with bleomycin, has been attributed to linear excoriation from scratching following a pruritic or irritant reaction to the drug, with post-inflammatory melanin deposition along the resulting streaks, although the precise trigger for the initial pruritus remains incompletely defined [12,13]. Across these mechanistic threads, the recurring theme is that chemotherapeutic agents lower the threshold for melanocyte activation and epidermal or dermal melanin deposition through a combination of direct cytotoxicity, local irritant or vascular injury, and mechanical or inflammatory potentiation, with the resulting clinical pattern shaped by the route of drug delivery, the anatomic site affected, and patient-specific factors such as skin phototype.

## Etoposide- and Bleomycin-Associated Mucocutaneous and Nail Hyperpigmentation

Reports specifically describing pigmentary change in patients receiving etoposide are relatively uncommon and almost invariably occur in the setting of combination chemotherapy, making attribution to etoposide alone difficult. In one of the more directly relevant reports, a child treated with bleomycin, etoposide, and vinblastine for an endodermal sinus (yolk sac) tumor developed hyperpigmentation attributed principally to bleomycin alongside separate hypersensitivity reactions to etoposide and vinblastine, illustrating both the cutaneous pigmentary potential of bleomycin-containing etoposide regimens and the difficulty of isolating a single causative agent within a multidrug protocol [3]. Bleomycin, a frequent partner of etoposide in germ-cell tumor and Hodgkin lymphoma regimens, is the agent most classically associated with flagellate hyperpigmentation, a distinctive pattern of linear, whip-like streaks of brown-to-violaceous pigmentation typically affecting the trunk; case reports describe onset from days to a few weeks after drug exposure, frequently preceded by pruritus, with the eruption occurring even at cumulative bleomycin doses well below those associated with pulmonary toxicity [12,13].

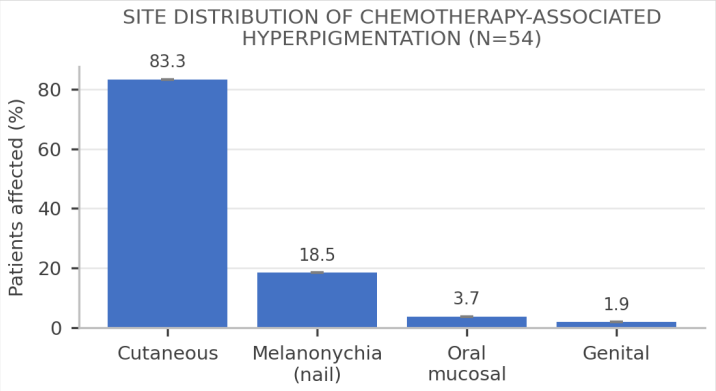
Nail and periungual changes are similarly prominent in regimens that pair etoposide with alkylating agents. Cyclophosphamide, commonly combined with etoposide in sarcoma, lymphoma, and pediatric solid tumor protocols, has been repeatedly reported to produce longitudinal, transverse, or diffuse melanonychia and less commonly diffuse skin darkening, with pigmentary bands sometimes tracking chemotherapy cycles in a manner that produces multiple parallel transverse bands across the nail plate [14,15]. Platinum agents, another frequent etoposide partner in germ-cell and lung cancer regimens, have been implicated in periungual and pressure-site hyperpigmentation with cisplatin, an unusual pattern in which pigmentation localizes specifically to sites of mechanical trauma or occlusion during treatment [10]. Oral and lingual mucosal pigmentation, including diffuse tongue darkening and blue discoloration of the nail lunula, has also been described in patients receiving multidrug regimens that included etoposide together with other cytotoxic and biologic agents, again generally attributed to the combination rather than to etoposide in isolation [16]. Taken together, the literature supports etoposide as a plausible contributing agent within combination regimens that produce mucocutaneous and nail hyperpigmentation, but it identifies bleomycin, cyclophosphamide, and platinum compounds as the components most consistently and specifically linked to the described pigmentary patterns, underscoring the importance of considering the full regimen, not etoposide alone, when counseling patients about expected pigmentary side effects.

## The Broader Spectrum of Cytotoxic Agents Implicated in Hyperpigmentation

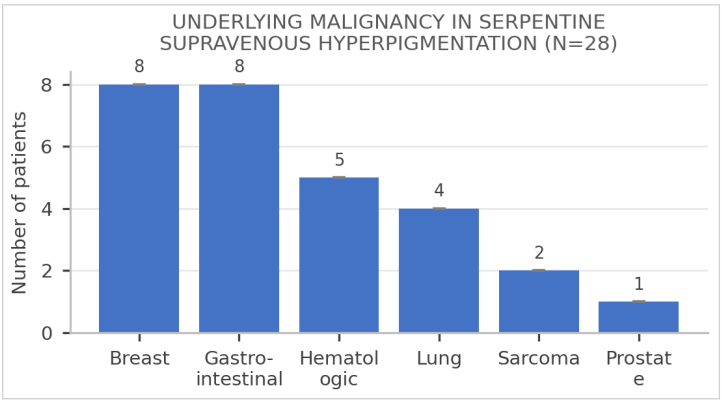
*Representative cytotoxic agents implicated in chemotherapy-associated mucocutaneous and nail hyperpigmentation, with characteristic clinical pattern and reported latency to onset.*

| Agent | Characteristic pigmentary pattern | Reported latency to onset | Representative report |
|---|---|---|---|
| Bleomycin | Flagellate (linear, whip-like) truncal hyperpigmentation, often pruritic | Days to a few weeks | Larson et al. 2017; Chen et al. 2025 |
| Cyclophosphamide | Longitudinal, transverse, or diffuse melanonychia; occasional diffuse skin darkening | Weeks to months; often tracks treatment cycles | Dave & Thappa 2003; Kumar et al. 2010 |
| Cisplatin | Periungual and pressure/occlusion-site hyperpigmentation | Weeks | Kim et al. 2002 |
| 5-Fluorouracil | Serpentine supravenous hyperpigmentation overlying infused vein | 30 minutes to 6 months | Shan et al. 2024 |
| Hydroxyurea | Melanonychia and diffuse skin hyperpigmentation, occasionally with dermal iron deposition | Months to years of continuous therapy | Gropper et al. 1993; Karanth et al. 2014; Letete & Vaz 2024 |
| Ifosfamide | Diffuse cutaneous hyperpigmentation | Weeks to months | Yule et al. 1994 |

*Site distribution of chemotherapy-associated hyperpigmentation among 54 patients treated with anti-tumor chemotherapy regimens, illustrating the predominance of cutaneous involvement over nail (melanonychia), oral mucosal, and genital patterns (Hanafi et al., 2020).*




*Underlying malignancy distribution among patients with serpentine supravenous hyperpigmentation identified in a systematic review of 28 patients across 26 reports, showing the predominance of breast and gastrointestinal malignancies among affected patients (Shan et al., 2024).*




Beyond etoposide-containing regimens, a wide range of cytotoxic and antimetabolite agents has been associated with characteristic pigmentary patterns, providing important comparative context for interpreting findings in etoposide-treated patients. The antimetabolite 5-fluorouracil is the agent most consistently identified as the leading cause of serpentine supravenous hyperpigmentation, a linear or reticulate streak of pigmentation overlying the vein used for infusion that has been reported in an estimated 2 to 5 percent of treated patients and can persist for months to more than a year after chemotherapy is discontinued [11]. A systematic review of this entity identified breast, gastrointestinal, hematologic, lung, sarcomatous, and prostate malignancies as the underlying cancers most frequently represented among affected patients, with onset ranging from thirty minutes to six months after the initial infusion [11]. Ifosfamide, another alkylating agent occasionally combined with etoposide in sarcoma and relapsed lymphoma regimens, has been reported to cause diffuse cutaneous hyperpigmentation in a pattern distinct from its structural analogue cyclophosphamide [17]. Hydroxyurea, used chronically in myeloproliferative neoplasms rather than as part of etoposide-based protocols, nonetheless offers one of the best-documented models of chemotherapy-associated nail and skin pigmentation, with case reports describing melanonychia, diffuse cutaneous darkening, and, in some instances, associated dermal iron deposition after months to years of continuous therapy [6,7,8,9].

A large single-center case series of 54 patients with anti-tumor chemotherapy-associated hyperpigmentation, encompassing a range of cytotoxic regimens, found cutaneous involvement in 83.3 percent of cases, melanonychia in 18.5 percent, and oral mucosal or genital involvement in a small minority, with a mean latency to onset of approximately six weeks and a predominance among patients with darker constitutive skin phototypes (III and IV), suggesting that baseline pigmentary reserve is an important determinant of clinical expression [4]. Broader reviews of cutaneous reactions to cytotoxic and targeted anticancer therapy corroborate this pattern, cataloguing hyperpigmentation alongside other pigmentary and structural nail changes as a recurring toxicity across taxanes, alkylating agents, antimetabolites, and select targeted agents, and noting that certain patterns, such as reticulate hyperpigmentation described with paclitaxel, add further phenotypic diversity to the broader category of chemotherapy-associated pigmentary change [18,19,20]. A recent clinico-epidemiological study of cutaneous adverse reactions to targeted chemotherapeutic agents further

situates classical cytotoxic-agent hyperpigmentation within an expanding landscape that now includes pigmentary changes associated with kinase inhibitors and other molecularly targeted drugs, and a pooled meta-analysis across drug classes estimated antineoplastic-agent-associated hyperpigmentation at approximately 35.5 percent of treated patients in the studies analyzed, an incidence broadly consistent with the case series data above [21,2].

## Diagnostic Evaluation and Clinical Management

The evaluation of a patient who develops new pigmentary change during or after chemotherapy begins with a careful clinical history establishing the temporal relationship between drug exposure and the onset of pigmentation, together with a detailed description of the pattern, distribution, and any preceding pruritus, trauma, or venous irritation at the affected site, since pattern recognition alone often permits a confident clinical diagnosis without biopsy [1,5]. Distinguishing chemotherapy-induced pigmentation from other causes of new skin darkening—including post-inflammatory hyperpigmentation from an unrelated dermatosis, endocrinologic causes such as adrenal insufficiency, and, most importantly, cutaneous metastasis or a second primary pigmented lesion—remains an essential part of the initial assessment, particularly when pigmentation is localized, asymmetric, or associated with other concerning features, in which case dermoscopy or skin biopsy may be warranted to exclude a melanocytic or metastatic process [18]. Reviews of drug-induced pigmentation emphasize that a comprehensive medication history, including recently discontinued agents, is important because the latency between chemotherapy exposure and clinically apparent pigmentation can range from days, as with bleomycin-associated flagellate change, to several months, as with serpentine supravenous hyperpigmentation and some nail findings [12,11].

Management is largely conservative once the diagnosis is established, since most reported cases of chemotherapy-induced hyperpigmentation are self-limited after treatment discontinuation, although resolution can be slow and, in some agents such as hydroxyurea and bleomycin-associated changes, pigmentation may persist for years [6,13]. Sun protection of affected areas is commonly recommended given evidence that ultraviolet exposure can potentiate or prolong drug-induced pigmentation, and in the case series of 54 patients, dermatologic intervention including photoprotection counseling was associated with clear or moderate cosmetic improvement in the large majority of patients followed over time [4]. Because chemotherapy-induced hyperpigmentation, including patterns arising in etoposide-containing regimens, is rarely dose-limiting or a marker of an otherwise dangerous drug reaction—flagellate pigmentation and pressure-site pigmentation being usually free of systemic implications, in contrast to a true hypersensitivity reaction as separately described for etoposide and vinblastine in the same case—most authors advise continuing the causative regimen when clinically indicated, with dermatologic referral reserved for diagnostic uncertainty, marked cosmetic distress, or coexisting features suggestive of an alternative diagnosis [3,22]. Patient counseling before treatment initiation, particularly for regimens containing bleomycin, cyclophosphamide, or 5-fluorouracil, is a low-cost intervention that may reduce anxiety when expected pigmentary changes subsequently appear.

***Reported incidence of chemotherapy- and antineoplastic-agent-associated hyperpigmentation across key case series and pooled analyses.***

| Study | Design / population | Reported incidence or key finding |
|---|---|---|
| Hanafi et al. 2020 | Single-center case series, 54 patients on anti-tumor chemotherapy | Cutaneous involvement 83.3%; melanonychia 18.5%; mean onset ~6 weeks |
| Alharithy et al. 2026 | Systematic review and meta-analysis, drug-induced hyperpigmentation | Antineoplastic-agent-associated hyperpigmentation ~35.5%; overall pooled incidence 36.7% (95% CI 29.1-44.4%) |
| Shan et al. 2024 | Systematic review, 26 reports / 28 patients with serpentine supravenous hyperpigmentation | Incidence 2-5% of treated patients; 5-fluorouracil most commonly implicated agent |

## Conclusion

Chemotherapy-induced mucocutaneous and nail hyperpigmentation is a common, clinically distinctive, and generally benign toxicity that spans diffuse cutaneous darkening, flagellate and serpentine supravenous patterns, and a range of nail and oral mucosal findings [1,2]. Reports arising specifically in the context of etoposide-based regimens are limited and consistently implicate concomitant agents—principally bleomycin, cyclophosphamide, and platinum compounds—as the more likely proximate cause of the observed pigmentary pattern, even as etoposide itself remains a component of many of the multidrug protocols in which these changes have been documented [3,4]. The broader literature on cytotoxic-agent hyperpigmentation, anchored by well-characterized entities such as bleomycin-induced flagellate pigmentation, 5-fluorouracil-associated serpentine supravenous streaking, and hydroxyurea-related melanonychia, provides a useful comparative framework for clinicians evaluating new pigmentary change in patients on etoposide-containing therapy, supporting a conservative, pattern-based diagnostic approach that reserves biopsy and further workup for atypical or diagnostically ambiguous presentations [11,6,10]. As combination cytotoxic regimens incorporating etoposide continue to be used widely across germ-cell, small-cell lung, and hematologic malignancies, continued case-level documentation of pigmentary findings attributable specifically to etoposide, as distinct from its regimen partners, remains a worthwhile area for future clinical reporting.

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## Conflict of Interest

The authors declare no conflict of interest.

## Author Contributions

Balodis L conceived the review, conducted the literature search,

and drafted the manuscript. Kovac I contributed oncologic and treatment-regimen context, assisted with literature screening, and critically revised the manuscript for important intellectual content. Balazova J contributed expertise on oral and mucosal pigmentary findings, provided additional context on diagnostic evaluation, and approved the final version for submission.

## Ethics Statement

Not applicable. This is a narrative review of previously published literature and did not involve new experimental data, human participants, or animal subjects.

## Data Availability

No new data were generated or analyzed for this review. All sources synthesized are cited in the reference list and are available through their original publishers.

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