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CASE REPORT

Acquired Brachial Cutaneous Dyschromatosis with Coexisting Pigmentary Disorders: A Report of Two Cases with Clinicopathologic Findings

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ABSTRACT: Acquired brachial cutaneous dyschromatosis (ABCD) is an uncommon pigmentary disorder characterised by reticulated hyperpigmented and hypopigmented macules and patches on the dorsal forearms. It predominantly affects postmenopausal women and has been associated with chronic ultraviolet (UV) exposure and, in some reports, the use of angiotensin-converting enzyme inhibitors (ACEis). We report two cases of ABCD in the Philippines, highlighting its coexistence with facial iatrogenic dyschromia in one patient and idiopathic guttate hypomelanosis (IGH) in the other, raising the possibility of shared contributory factors despite these conditions representing distinct disease entities. Two 60-year-old Filipino women with Fitzpatrick skin phototype (FSP) IV presented with progressive reticulated hyperpigmented and hypopigmented macules and patches on the bilateral dorsal forearms. Histopathological evaluation of the forearm lesions in both patients demonstrated marked basal layer hyperpigmentation, sparse superficial perivascular lymphocytic infiltrates, and prominent solar elastosis, consistent with ABCD and chronic UV-induced photodamage. In Case 1, Melan-A immunohistochemistry of the forearm lesion demonstrated increased melanocyte density with an irregular distribution along the basal layer, supporting melanocytic hyperactivity. Concomitant facial pigmentary changes were consistent with iatrogenic dyschromia. In Case 2, concomitant discrete hypopigmented macules on the lower extremities were clinically and histopathologically consistent with IGH. These cases highlight the importance of correlating clinical, histopathological, and immunohistochemical findings in the evaluation of ABCD and provide further clinical observations supporting its proposed etiopathogenesis and possible association with concomitant pigmentary disorders. Management may focus on melanogenesis-modulating therapy and strict photoprotection to address pigmentary changes and minimise further pigmentary alterations.

Keywords: Acquired brachial cutaneous dyschromatosis, Dyschromia, Cutaneous pigmentary disorders, Idiopathic guttate hypomelanosis, Facial dyschromia

INTRODUCTION

Acquired brachial cutaneous dyschromatosis (ABCD) is an uncommon pigmentary disorder characterised by asymptomatic, reticulated hyperpigmented and hypopigmented macules on the dorsal forearms, most often observed in postmenopausal women [1,2]. ABCD has been associated with chronic sun exposure and, in several reports, angiotensin-converting enzyme inhibitor (ACEi) use [1]. Its distribution on the extensor upper arms, which are frequently exposed to ultraviolet (UV) radiation, may be consistent with a photoinduced mechanism. The proposed association between ABCD and hypertension or antihypertensive therapy may reflect the high prevalence of hypertension in middle-aged and postmenopausal individuals, along with the common use of these medications in this population [1]. Although ACEis are known to cause various cutaneous adverse reactions such as photosensitivity, they have not previously been implicated in causing pigmentary disorders [3].

Histopathologically, ABCD is characterised by an atrophic epidermis, basal layer hyperpigmentation, telangiectases, and prominent solar elastosis [1]. UV radiation induces keratinocyte and melanocyte injury, resulting in dysregulated melanin synthesis and transfer, which may be associated with histological findings of basal layer hyperpigmentation, epidermal thinning, superficial telangiectases, and solar elastosis. We report two cases of ABCD in the Philippines and document its

coexistence with facial iatrogenic dyschromia in one patient and with idiopathic guttate hypomelanosis (IGH) in another. Although ABCD, IGH, and depigmenting agents-induced dyschromia are pathogenetically distinct entities, their coexistence in our patients raises the possibility of shared contributory factors, including chronic photodamage. This case report aims to contribute to the limited literature on ABCD by highlighting its clinical and histopathological features that may aid in its recognition and differentiation from other pigmentary disorders with similar presentations, thereby supporting accurate diagnosis and appropriate patient management. Furthermore, given the challenges in treating ABCD and the lack of established optimal management strategies, this report aims to provide a basis for further investigation into its etiopathogenesis, potential contributing factors, and therapeutic approaches.

CASE PRESENTATION

Case 1: A 60-year-old Filipino woman with Fitzpatrick skin phototype (FSP) IV presented with a 20-year history of progressive hyperpigmentation over both malar areas. She reported intermittent use of various over-the-counter depigmenting creams containing tretinoin (0.25%) and hydroquinone (2%). Within 2–3 days of application, she developed mild erythema without associated stinging, burning, pruritus, or scaling. The reaction was observed shortly after application and was self-limited but was not consistently observed with each depigmenting cream used by the patient. Despite continued use over several years, the malar hyperpigmentation showed minimal improvement, and she subsequently developed multiple irregularly shaped, non-scaly hypopigmented macules and patches on both malar areas (**Figure 1**). One year prior to consultation, she noted the development of multiple irregular, geometric tan-to-brown patches interspersed with hypopigmented macules and scattered telangiectatic vessels on the bilateral dorsal forearms (**Figure 2**). The lesions gradually progressed with chronic sun exposure. She had no history of hypertension, denied use of ACEis, and reported no recent intake of phototoxic medications. No similar lesions were reported in her family.

Histopathological examination of a 4-mm punch biopsy from the facial lesion demonstrated epidermal thinning, basal layer hyperpigmentation, and sparse superficial perivascular lymphocytic infiltrates (**Figure 3A**), findings consistent with iatrogenic dyschromia. In contrast, the forearm biopsy revealed marked basal layer hyperpigmentation, an ill-defined grenz zone, prominent solar elastosis, pigment-laden macrophages, hemosiderin deposition, and fibrillar collagen changes (**Figure 3B**), consistent with ABCD. Immunohistochemical staining with Melan-A of the facial lesion demonstrated irregularly-shaped and apoptotic melanocytes (**Figure 3C**), whereas the forearm specimen showed epidermal melanocytosis consisting of irregularly-shaped melanocytes along the basal layer and increased melanin in the basal cell layer (**Figure 3D**).

Figure 1. Facial iatrogenic dyschromia showing multiple hypopigmented macules and telangiectasias against a background of hyperpigmented patches over the malar areas.

Figure 2. Reticulated hyperpigmented and hypopigmented macules and patches over the bilateral dorsal forearms, consistent with ABCD.

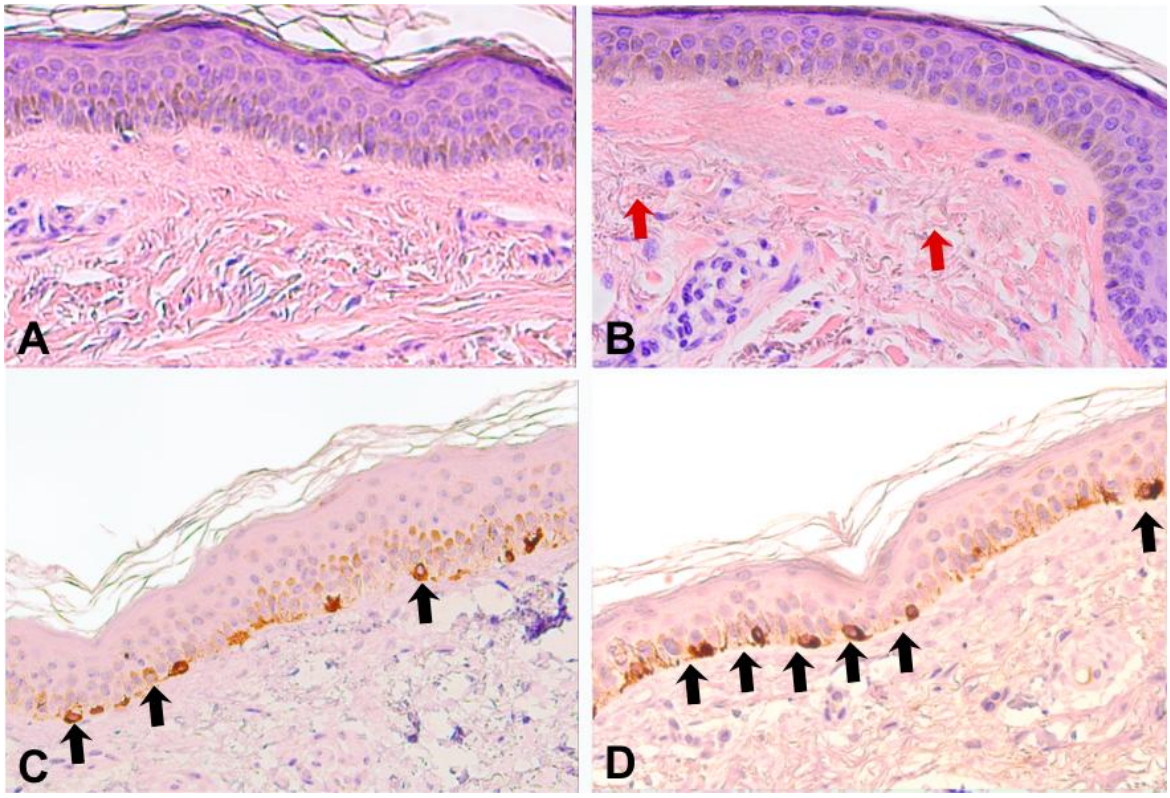

Figure 3. (A) Facial iatrogenic dyschromia showing epidermal atrophy and basal layer hyperpigmentation and sparse superficial perivascular lymphocytic infiltrates (H&E, ×400). **(B)** ABCD of the dorsal forearm showing epidermal atrophy, basal layer hyperpigmentation, and prominent solar elastosis (red arrow) (H&E, ×400). **(C)** Melan-A immunostaining of the facial lesion demonstrating irregularly-shaped and apoptotic melanocytes (black arrow) (×400). **(D)** Melan-A immunostaining of the forearm lesion highlighting epidermal melanocytosis, with irregularly shaped melanocytes and increased melanin in the basal layer (black arrow) (×400).

Case 2: A 60-year-old Filipino woman with FSP IV, presented with 2-year history of gradual development of hypopigmented and depigmented macules involving the lower extremities (**Figure 4**). Over time, multiple, reticulated, brown patches interspersed with hypopigmented macules also became apparent on the bilateral dorsal forearms (**Figure 5**). The lesions were asymptomatic and progressively more noticeable. The patient denied any history of topical hypopigmenting agent use, including hydroquinone, retinoids, or other depigmenting creams, and reported no prior cosmetic procedures. There was no history of hypertension or intake of ACEis, and no recent use of phototoxic medications. The patient reported no significant history of excessive sun exposure and denied any family history of similar pigmentary disorders.

Histopathological examination using a 4mm punch biopsy of the hypopigmented macule on the thigh revealed epidermal atrophy with basal layer hyperpigmentation with skipped areas of retained melanin in the basal layer alternating with large areas of melanin loss, and a sparse perivascular lymphocytic infiltrate, findings consistent with IGH (**Figure 6A**). In contrast, the forearm lesion demonstrated epidermal acanthosis with basal layer hyperpigmentation, prominent solar elastosis, and a sparse perivascular lymphocytic infiltrate, consistent with ABCD (**Figure 6B**). Although Melan-A immunostaining was not performed for Case 2 due to financial constraints, the H&E findings supported the diagnosis of ABCD in the appropriate clinical context.

MANAGEMENT AND OUTCOME

Both patients were initiated on a topical serum (Neotone®) containing niacinamide, trimethoxybenzyl acetylsinapate, licorice root extract, and diacetyl boldine, applied twice daily, with the aim of addressing pigmentary changes. They were also counselled on strict daily broad-spectrum photoprotection and advised to avoid potential irritant topical agents and bleaching agents, particularly hydroquinone. Neither patient showed clinical improvement after six months of Neotone serum use, resulting in limited patient satisfaction due to the lack of visible response.

Subsequent management would emphasize continued photoprotection and individual treatment strategies. Potential options include melanogenesis-modulating agents, such as topical tacrolimus or bimatoprost, and targeted phototherapy for focal hypopigmented areas, although their efficacy and safety in ABCD have yet to be established. Procedures targeting dermal photodamage, including microneedling, chemical peels, and mild ablative resurfacing, may also be cautiously considered.

Figure 4. Multiple hypopigmented to depigmented macules on the lower extremities, consistent with IGH.

Figure 5. Reticulated hyperpigmented and hypopigmented macules and patches over the bilateral dorsal forearms, consistent with ABCD.

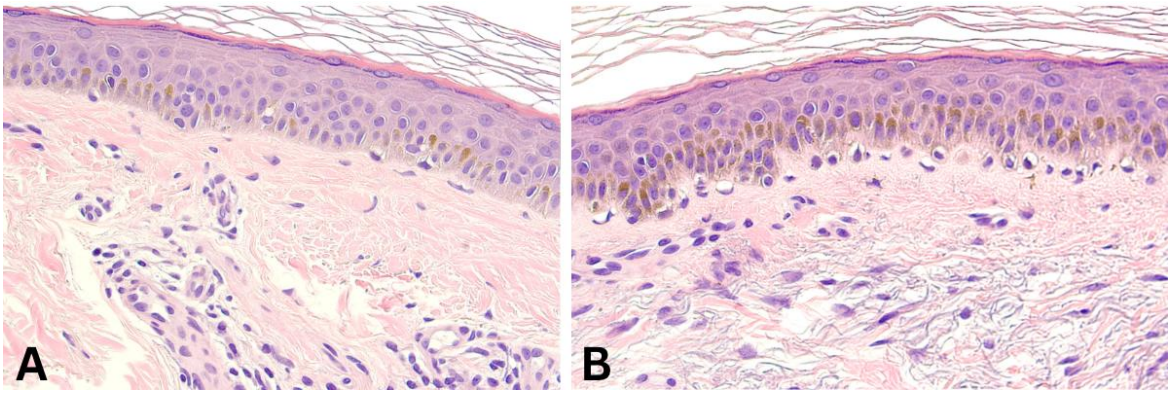

Figure 6. (A) IGH showing epidermal atrophy and basal layer hyperpigmentation with skipped areas of retained melanin (H&E, ×400). **(B)** ABCD of the dorsal forearm showing epidermal atrophy, basal layer hyperpigmentation, and prominent solar elastosis in the dermis (H&E, ×400).

DISCUSSION

ABCD is characterised by asymptomatic, irregular grey-brown patches with geographic borders, sometimes accompanied by hypopigmented, slightly atrophic macules, most commonly on the bilateral forearms. We present two cases in which ABCD coexisted with facial iatrogenic dyschromia in Case 1 and IGH on the lower extremities in Case 2. In Case 1, facial iatrogenic dyschromia was associated with the use of topical skin-lightening agents. Histopathological findings may include basal layer hyperpigmentation, dermal pigment incontinence with melanophages, and mild perivascular lymphocytic infiltrates; epidermal atrophy or vacuolar degeneration may also be present, depending on the causative agent [4]. ABCD, on the other hand, shows focal increased pigmentation of the epidermal keratinocytes against a background of solar elastosis in the dermis [5]. Melan-A staining in facial iatrogenic dyschromia revealed an irregular but patchy pattern, which may correlate with melanocytopenia [4], compared with melanocytosis or increased melanocyte density [5] in ABCD.

In case 2, IGH is a distinct hypopigmentary disorder characterised clinically by multiple, small, sharply demarcated, round to oval, hypopigmented to depigmented, porcelain-white macules on sun-exposed extremities [6]. This condition may be associated with senile degeneration, UV exposure, genetic factors, local trauma, autoimmune factors, and local inhibition of melanogenesis [7]. Histopathological findings include an atrophic epidermis with effacement of the rete ridges, a thick basket-weave orthokeratotic stratum corneum, and focal areas of retained melanin, which are relatively specific histopathological features of IGH [8]. These findings contrast with the increased melanin density in the basal layer and prominent solar elastosis in the papillary dermis of ABCD [5].

There is currently no established standard treatment for ABCD. Management primarily involves a multimodal therapeutic approach in conjunction with strict photoprotection [9]. Broad-spectrum sunscreen use, photoprotective clothing, and behavioral sun avoidance are recommended as first-line measures. In these cases, we used a topical serum formulation

(Neotone®), which has been investigated for other pigmentary disorders such as melasma, with good results [10,11]. However, no visible improvement was observed in our patients for these cases at the six-month follow-up. Alternative therapeutic approaches need to be explored.

Therapies aimed at melanocyte modulation, including topical tacrolimus, bimatoprost, or targeted phototherapy such as excimer laser treatment, may be considered for focal hypopigmented areas. These therapies have been used to treat hypopigmentation in other pigmentary disorders, such as IGH [12], and could potentially be explored for focal hypopigmented areas of ABCD. Topical retinoids [1], including tretinoin and other vitamin A derivatives, may improve epidermal turnover and contribute to dermal remodelling, particularly in the setting of photodamage. Procedural interventions targeting dermal photodamage, such as microneedling, chemical peels, or mild ablative resurfacing [1], may be considered cautiously in selected cases, particularly where solar elastosis is prominent. Adjunctive topical therapies, such as antioxidants including vitamin C [10], may theoretically help mitigate oxidative stress.

CONCLUSION

These cases aim to highlight the clinical and histopathological features of ABCD to facilitate its recognition and differentiation from other pigmentary disorders with similar clinical presentations. They also contribute to the limited literature on ABCD and may provide a basis for further research into its underlying pathogenesis, potential risk factors, and therapeutic approaches. The concomitant presence of iatrogenic dyschromia and IGH illustrates the diverse pigmentary changes that may occur in chronically sun-exposed skin. These coexisting disorders may reflect different melanocyte responses associated with chronic UV exposure and other contributing factors. Clinicians should maintain a broad differential diagnosis when evaluating ABCD in chronically sun-exposed skin and avoid attributing all pigmentary changes to a single process. Accurate recognition of these distinct entities is important to prevent misdiagnosis and inappropriate pigment-targeted therapy. Management should prioritise sustained photoprotection, with treatment strategies tailored to the underlying pigmentary disorder.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest related to the publication of this case report. Informed consent was obtained from the patients for publication of the case report, including the clinical photographs.

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