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Erythema Dyschromicum Perstans with Islands of Sparing: A Case Report

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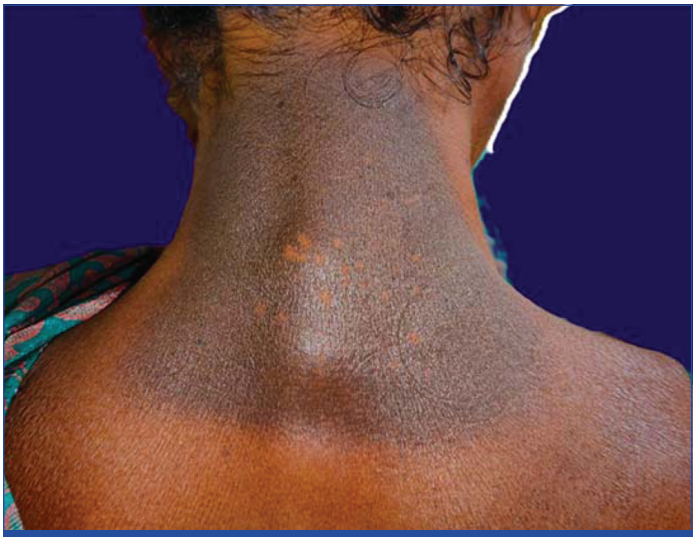
A 46-year-old female presented with a six-month history of gradually progressive asymptomatic dark coloured skin lesion over back of the neck. She did not reveal any history of topical application prior to onset of lesions, usage of hair cosmetics or chronic mechanical irritation at the site. The patient did not report any preceding redness, induration, history of trauma or any other previous skin lesions at that particular site. She denied history of similar lesions anywhere else in the body. There was no history of fever, abdominal pain, altered bowel habits or significant weight loss and antecedent drug intake. Past history and family history was insignificant. She was a paddy field worker and used to have chronic exposure to sunlight and pesticides. On general examination, the patient was conscious and oriented. Her vital signs showed a blood pressure of 120/80 mmHg, a pulse rate of 78/min, and she was afebrile.

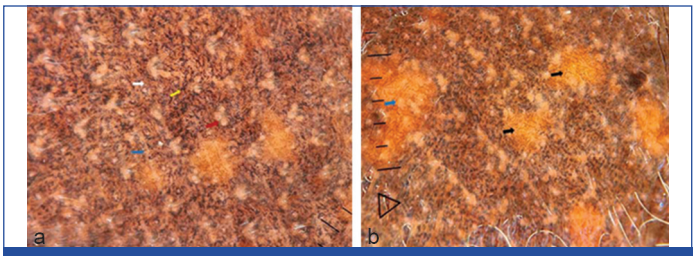
Dermatological examination revealed a single, symmetrical, well-defined greyish-brown patch measuring 15 × 8 cm over the posterior aspect of the neck, extending to the upper back. Within the patch, multiple discrete circular areas of normal skin measuring <0.5 cm were noted, predominantly at the centre, representing islands of sparing. [Table/Fig-1]. Palpation revealed no oedema, induration, thickening or sclerosis. There was no evidence of similar lesions elsewhere. Examination of hair, nail and mucosa was normal. Systemic examination was unremarkable.



[Table/Fig-1]: A single well defined hyperpigmented gray brown patch with islands of normal skin distributed more at the centre.

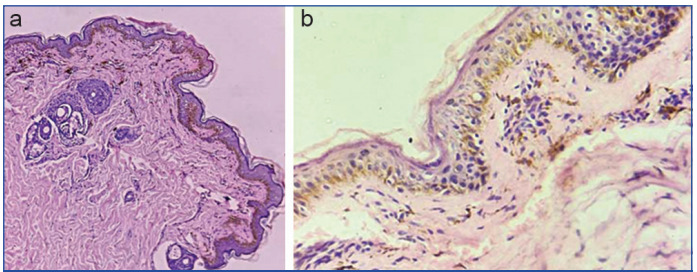
Differential diagnosis of Erythema Dyschromicum Perstans (EDP), Fixed Drug Eruption (FDE), Lichen Planus Pigmentosus (LPP), Post Inflammatory Hyperpigmentation (PIH) and macular amyloidosis were considered. Her blood investigations revealed a haemoglobin level of 12 g/dL and a total leukocyte count of 7500 cells/mm^3. The random blood sugar level was 129 mg/dL. Liver function tests were within normal limits, with total bilirubin 0.9 mg/dL, SGOT 23 U/L, and SGPT 37 U/L. Blood urea was 34 mg/dL, and serum creatinine was 0.8 mg/dL. Thyroid function test showed TSH levels 2 mIU/L and was euthyroid. Antinuclear antibody was negative. Viral markers including HIV and HBsAg was non-reactive.

Dermoscopy of the hyperpigmented area showed black and brown dots with perifollicular hypopigmentation and sparing of creases suggestive of EDP [Table/Fig-2a]. These dermoscopic characteristics resembled the appearance of Wagyu meat as reported by Tommi K et al., in a case report of EDP [1]. Two separate dermoscopic characteristics, namely pigmentation in the crista cutis and brown dots combines to produce this appearance. Pigmentation in crista cutis corresponds to epidermal papillomatosis and small brown dots correspond to pigmentary incontinence [1]. The absence of brown dots in reticuloglobular pattern or hem like pattern differentiates EDP from FDE and LPP [2,3]. Islands of sparing showed reticular network and normal eccrine duct openings [Table/Fig-2b]. Dermoscopy of postinflammatory hyperpigmentation exhibits non-specific brown pigment pattern and may sometime show features of original parent lesion [4]. Macular amyloidosis shows incomplete hub and spoke wheel pattern with cluster of pigmented dots and globules [5] which was not observed in the present case report.



[Table/Fig-2]: a) Dermoscopy of the hyperpigmented area showed black dots (yellow arrow), brown dots (white arrow) with perifollicular hypopigmentation (red arrow) and sparing of creases (blue arrow), (Heine Delta 30:10x Magnification: Polarised mode); b) Dermoscopy of Islands of sparing showed reticular network (Black arrow) and normal eccrine duct openings (Blue arrow), (Heine Delta 30:10x Magnification:Polarised mode).

A punch biopsy was done from hyperpigmented skin over the lesion and showed epidermal papillomatosis, basal cell vacuolisation, melanin incontinence and dermal lymphocytic infiltrates suggestive of EDP [Table/Fig-3a,b].



[Table/Fig-3]: a) Histopathology (Haematoxylin & Eosin) of the lesional skin under low power (10x) showing epidermal papillomatosis, basal cell vacuolisation, increased melanin in basal layers, melanin incontinence and dermal lymphocytic infiltrates; b) Histopathology (Haematoxylin & Eosin) of the lesional skin under high power (40x) showing melanophages and dermal infiltrates.

The FDE can be differentiated histopathologically from EDP by the presence of eosinophilic infiltrate and severe interface dermatitis [6]. There is a significant overlap between histologic features of EDP and LPP but characteristic dermoscopic features differentiates both the conditions clinically. Brown dots in hemlike or speckled pattern, as seen in LPP was not observed in present case report and the characteristic dermoscopic findings were brown dots resembling wagyu meat with perifollicular hypopigmentation [7]. PIH shows melanin in basal and suprabasal layers in epidermal type and dermal melanophages in dermal type of PIH without any interface changes [8]. The histopathology of present case report did not reveal any eosinophilic amyloid deposits which are characteristic of macular amyloidosis hence excluding the same [9]. Based on the history, clinical examination, a suggestive histopathology and further supported by dermoscopic findings, she was diagnosed as a case of EDP. The patient was started on topical tacrolimus 0.1% ointment once daily, along with broad-spectrum sunscreen. Two months following regular application she appreciated mild improvement in pigmentation and is continuing the same treatment along with Tablet ascorbic acid 500 mg twice daily.

EDP is a form of acquired dermal macular hyperpigmentation characterised by multiple discrete macules and patches with an erythematous red border in early phase typically distributed symmetrically over the body. These lesions are usually uniformly pigmented and homogenous, with the trunk being the most common site involved. Aetiopathogenesis of EDP is attributed to CD8 + T lymphocyte induced damaged to melanocytes and basal keratinocytes leading to dermal pigment incontinence. Possible triggers for EDP include ingestion of substances such as ammonium nitrate, oral contrast agents and certain medications like benzodiazepines and penicillin. Additionally, exposure to various pesticides, fungicides, or toxins may play a role. Endocrine disorders, including thyroid disease, and infections like whipworm and HIV have also been implicated as potential factors [10]. The phenomenon of sparing has been documented in various dermatological disorders including pityriasis rubra pilaris, vitiligo, and alopecia areata and may be associated with localised immune dysregulation. The immune response within a defined cutaneous region is orchestrated by a complex interplay of cellular interactions, resulting in the migration of immunocompetent cells through lymphatic channels and the modulation of signalling pathways mediated by neuromediators released from peptidergic nerve fibers, which engage immune cell membrane receptors. Any disruption in lymphatic drainage or impairment in the communication between peripheral neuromediators and immune cells can significantly alter the local immune milieu and modify the cutaneous immune response [11]. EDP is an immune-mediated condition, and the observed islands of sparing reported in this case may be attributed to the preferential infiltration of CD8+ T lymphocytes in the affected skin, reflecting localised immune dysregulation potentially triggered by an unidentified inciting factor. This case is reported for its unique presentation of EDP as an isolated large hyperpigmented patch with islands of sparing, at an uncommon site which has not been documented previously in the literature.

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