Journal of Clinical & Experimental Dermatology Research

OPEN ACCESS Freely available online

Perspective

A Brief Description of Erythema Dyschromicum Perstans

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DESCRIPTION

Erythema Dyschromicum Perstans (EDP) is a type of acquired hyperpigmentation of the dermis, characterized by round to oval or irregular grey spots at the face, neck and trunk. This is a form of acquired dermal pigmentation. Erythema Dyschromicum Perstans (EDP) can be difficult to diagnose and treat. Therefore, distinguish between Lichen Plano Pilaris (LPP) and EDP and determine the most effective treatment for EDP. Erythema Dyschromicum Perstans was treated successfully with Narrow Band Ultraviolet B (NBUVB). Erythema Dyschromia, also known as grey dermatosis or dermatosis senicienta, is an acquired dermatitis characterized by slowly progressive greyish-grey spots/spots that are symmetrically distributed to the trunk and proximal end. It is a chronic pigmented disorder. Ramirez called with this condition los cenicienta which in Spanish means the ashcolored ones because of the characteristic ashy color of the lesions. In 1961 the word "Erythema Dyschromicum Perstans" was coined by Convit. one researcher reported that numerous macules of greyish color with slightly raised, firm erythematous border. Cause of erythema dyschromia remains unknown. Due to its histopathological characteristics, it is often classified as a variant of lichen planus.

Causes

- Genetic susceptibility.
- Contact allergies to cosmetics and hair dyes.
- Toxic effects of chemicals such as ammonium nitrate and barium sulphate.
- Infestation of whipworm.
- Virus infection.
- Drugs and side effects of drugs.

Damage to melanocytes and basal cell keratinocytes seen in EDP is due to an abnormal immune response to antigens predominantly $CD8^+$ T lymphocytes and $HLADR^+$ in the dermis, intercellular adhesion molecule keratinocytes in the genetic sensitivity of the epidermis. Genes in the major histocompatibility complex (mainly HLADR4) were also present. Local calcineurin inhibitors (tacrolimus, pimecrolimus) bind to the cytoplasmic immunophilins FKBP12 and form a complex that inhibits the activity of the enzyme calcineurin, which is required for T cell activation. Inhibition of calcineurin prevents dephosphorylation of the cytoplasmic component of activated T cell nuclear factor that regulates mRNA transcription of inflammatory cytokines in the Th1 (T-helper cell) and Th2 pathways (IL2 (Interleukin), IFNg (Interferon Gamma) and IL4, IL10). An abnormal cell mediated immune response has been postulated to play a role in the pathogenesis of EDP possibly as a result of involvement of cell adhesion and activation molecules (ICAM1, LFA1α (Lymphocyte Function-Associated Antigen-1)). This is evident from the predominant presence of $CD8^+$ T cells in the dermis, HLADR+ (Human Leukocyte Antigen–DR isotype) and $ICAM1^+$ (Intercellular Adhesion Molecule 1) expression in epidermal keratinocytes and exocytosis of Cutaneous Lymphocyte Antigen (CLA) cells in areas with damaged basal cells. Topical tacrolimus show its therapeutic effect in EDP by virtue of its immunomodulatory effects. Tarolimus provides an effective and safe alternative therapeutic action in EDP.

Erythema dyschromia should be distinguished from idiopathic eruptive macular pigmentation and lichen planus. In idiopathic eruptive macular pigmentation, the lesions are taupe, not confluent, small, and tend to regress over time. In lichen planus, the lesions are brown to tan spots/spots, itching, and there is no border of active erythema. Lesions often occur in the interstitial area exposed to sunlight and may be accompanied by mucous membranes. Further differential diagnosis includes macular papilla mass, post-inflammatory hyperpigmentation, morphia, rosacea, azison's disease, hemochromatosis, arsenic, contact dermatitis, multiple fixed drug rashes, confluent and reticular papillomatosis. Erythema dyschromia can be cosmetically awkward and socially embarrassing, especially when lesions occur in visible areas such as the face. The rash usually resolves after a few years in prepubertal children, but persists in adults. In some people the issue lesions will be reassured with the beign nature the issue will be resolved with the time.

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Received: 05-Jan -2022, Manuscript No. JCEDR-22-597; **Editor assigned:** 07-Jan -2022, PreQC No. JCEDR-22-597 (PQ); **Reviewed:** 14-Jan-2022 QC No. JCEDR-22-597; **Revised:** 21-Jan -2022, Manuscript No. JCEDR-22-597(R); **Published:** 28-Jan -2022, DOI: 10.35248/2155-9554.22.13.597

Citation: Jones K (2022) A Brief Description of Erythema Dyschromicum Perstans. J Clin Exp Dermatol Res.13:597.

Treatment

- EDP can be treated with fractional lasers it has been unsuccessful and in several occasions were associated with post inflammatory pigmentation changes.
- Q-switched lasers have been used.
- Topical steroids.
- Exposure to UV light.
- Pigment laser (e.g., B.Q switch ruby laser).
- Chemical peels.

The conclusion for EDP, NBUVB and tacrolimus are promising and effective treatments with significantly fewer side effects compared with clofazimine, and opposed to LPP, for which sun avoidance is recommended. In several cases after discontinuation of these drugs like isotretinoin, dapsone, and griseofulvin lead to reoccurance of the EDP. Lasers were consistently ineffective treatments. macrolide antibiotic medication like Tacrolimus having the properties of immunosuppressive, exerting its effects through direct the inhibition of calcium dependent events such as the Inter Leukin (IL), gene transcription, nitric oxide synthase activation, cell degranulation, and apoptosis. Tacrolimus was described with success in the treatment of EDP either as a monotherapy or in combination with laser and with NBUVB. NBUVB has successfully treated in EDP all with minimal or no side effects.

EDP is a rare skin disorder, it is important to consider other skin disorders that may resemble erythema dyschromia, such as: IT cannot be cured. Several different topical and systemic therapies have been tried, but none have been consistently successful. In children, spontaneous appearance of EDV is possible within months to years.