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47.1 Introduction

Polymorphous light eruption (PMLE) is the most common of the immunologically mediated (formerly categorized as idiopathic) photodermatoses. The prevalence of PMLE ranges from 10 to 20 %, depending on the geographic location [1]. Onset is typically within the first three decades of life [2–5]. Females are two to three times more affected than males [2–5].

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47.2 Clinical Features

PMLE lesions can present as non-scarring, erythematous, pruritic papules, vesicles, papulovesicles, plaques, or nodules [1]. Pinpoint variant of PMLE is the most common morphology seen in individuals with skin phototypes IV–VI (Figs. 47.1 and 47.2) [3]. Pinpoint PMLE is characterized by the development of pinpoint papules, 1–2 mm, on sun-exposed areas minutes or hours after ultraviolet radiation [6]. PMLE has a predilection for the arms, forearms, hands, head, and neck region.



Fig. 47.1 Pinpoint papules due to PMLE located on the cheek and perioral skin in an African American male (Photograph reprinted with written permission from the *Journal of Drugs in Dermatology*)



Fig. 47.2 Pinpoint papules due to PMLE on the cheek of an African American male (Photograph reprinted with written permission from the *Journal of Drugs in Dermatology*)

47.3 Natural History and Prognosis

PMLE lesions present hours to days after ultraviolet (UV) light exposure. The lesions usually last over 1–7 days and completely resolve without scarring [3]. PMLE typically begins in the spring, improving by late summer with “hardening” due to increased tolerance of the skin [4]. These patients have decreased ability to be locally suppressed upon exposure to UV, which explains the “hardening” response seen clinically [5].

47.4 Histopathological Features

Histology of the skin lesion is considered nonspecific, revealing superficial and deep dermal inflammatory cell infiltrates [1]. Variable epidermal changes can occur that range from mild spongiosis to acanthosis [1].

47.5 Diagnosis and Differential Diagnosis

Diagnosis of PMLE is typically made through history, morphology of lesions, and clinical course; phototesting and photopatch testing are not routinely performed [6]. Selected laboratory examinations such as antinuclear antibody (ANA), anti-Ro (SSA), anti-La (SSB), plasma porphyrin levels, and in some cases biopsy of persistent lesions may assist in making the diagnosis [2]. Differential diagnoses include, but are not limited to, systemic lupus erythematosus, eczema, erythropoietic protoporphyria, solar urticaria, and actinic prurigo.

47.6 Treatment

Prevention is essential with sun avoidance, utilization of broad spectrum sunscreen, and photoprotective clothing [7]. Additionally, light tolerance or “hardening” can be accelerated using narrowband UVB phototherapy or, less commonly, psoralen plus UVA (PUVA) before the sunny period of the year [7]. Other treatment modalities include topical corticosteroids and antimalarials. Systemic corticosteroids may rarely be required in the setting of acute exacerbation of the disease or during a brief winter vacation to a sunny locale [8].

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