

How to Cite:

Manthale, N., Raikar, D. R., Raikar, S. R., Raikar, S. R., & Raikar, D. R. (2022). Polymorphic light eruption: Clinico epidemiological study and histopathological correlation. *International Journal of Health Sciences*, 6(S2), 5809–5817. <https://doi.org/10.53730/ijhs.v6nS2.6470>

Polymorphic light eruption: Clinico epidemiological study and histopathological correlation

Dr. Nagendra Manthale

Consultant Dermatologist Venereologist and Leprologist, Kalaburgi, Karnataka

Dr. Dayanand. R. Raikar

Associate Professor, Department of Dermatology Venereology and Leprology, Gulbarga Institute of Medical Science, Kalaburgi, Karnataka
Corresponding author email: drdraikar@gmail.com

Dr. Shrinivas. R. Raikar

Associate Professor Department of Pharmacology, BLDE (Deemed to be University), Shri.B.M.Patil Medical College, Hospital and Research Centre, Vijayapura, Karnataka

Dr. Shankar. R. Raikar

Consultant Cardiologist, Sirasi, Karnataka

Dr. Dattanand. R. Raikar

Consultant Pediatrician, Karwar, Karnataka

Abstract---Introduction: Polymorphic light eruption (PLE) is the most common photodermatoses encountered in clinical practice. It is an idiopathic acquired disorder in which a delayed response to Ultraviolet radiation occurs in the form of skin eruptions consisting of papules, vesicles or plaques over the sun exposed and rarely on partially covered areas, distribution is generally symmetrical. Diagnosis is clinical and histo-pathological features vary according to age of lesions sampled. Objective: To study clinical pattern of Polymorphic Light Eruption and Histo-Pathological Correlation Methods: The present study is a hospital based observational study, conducted over a period of one year. A total of 70 cases including male and female patients presenting with history of photosensitivity or with clinical manifestations related to photosensitivity were enrolled. All patients underwent clinical and systemic examination followed by routine and histo-pathological investigations. The findings were recorded in proforma for analysis and interpretation of data. Follow-up was done

to know the prognosis of the patients. Results: The study shows that females are more affected than males with a ratio of 1:2.33 and the maximum incidence in the age group of 11-20 years and minimum in the age group of 51-60 years. The most common form of PLE was papular variant and histopathologically hyperkeratosis and perivascular lymphocytic infiltration was observed in all the specimens. Conclusion: The study concludes that PMLE is photosensitive disorder, more common in childhood and adolescents with female preponderance and histopathologically shows non-specific dermatitis.

Keywords---PLE, sun allergy, clinico-epidemiology, histopathology.

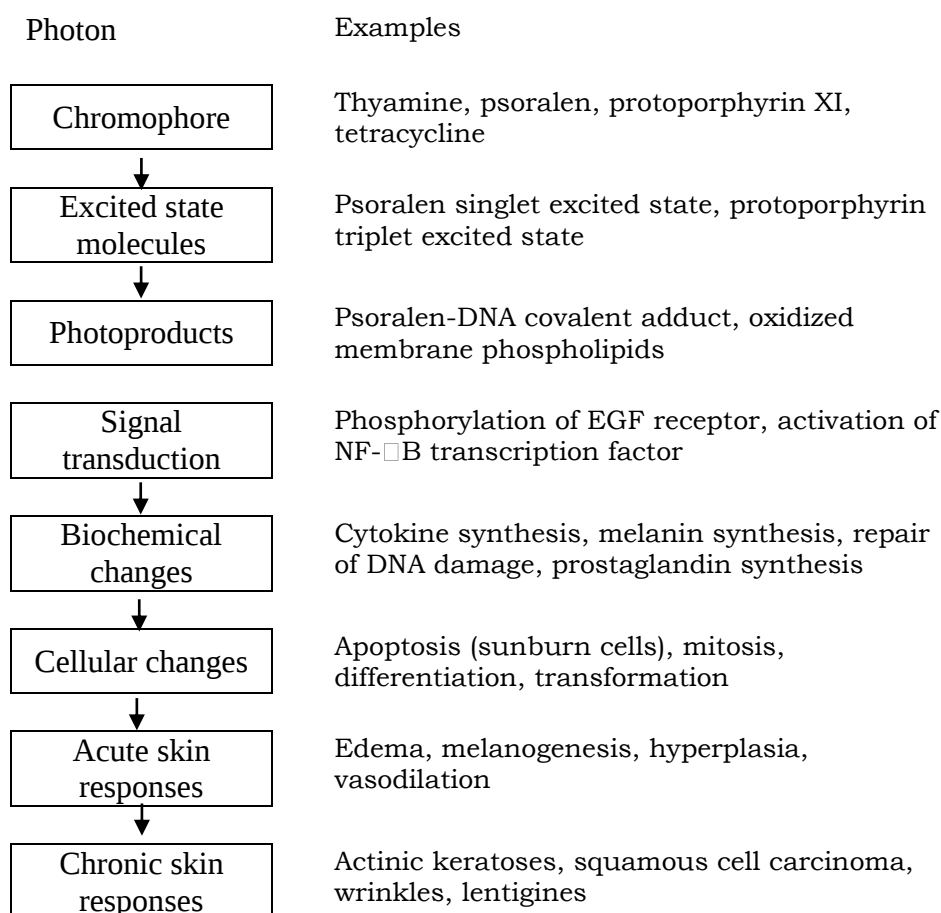
Introduction

Polymorphic light eruption (PLE) often incorrectly referred as “sun allergy”. It is the most common type of photodermatitis. It is recurrent, acquired sunlight-induced immune mediated and of delayed onset disorder. The prevalence of PMLE varies from 10 to 20% in various studies across the world. [1-5]. It is commonly seen in African, African Americans, Chinese and Indian. Its prevalence in India is 0.56%. Occurrence is more frequent at higher latitudes and altitudes.[6] It is common during spring or early summer. The severity increases as summer progresses and diminishes in winters. PMLE is generally observed in the first three decades of life. The mean age in females is 33 years whereas in males it is 35 years. Females are more often affected than males.[7] The etiology is unknown and is believed to be multi factorial. It has a polygenic mode of inheritance. It has many clinical variants, characterized by itchy papules, plaques and scales over exposed parts of body sometimes it can involve partially covered areas also. Diagnosis is clinical and histopathological findings are non-specific.[8] Histopathological findings vary as early lesions show mild spongiosis with focal lymphocytic exocytosis and sometimes mild to moderate superficial and deep perivascular, periadnexal lymphohistiocytic inflammatory infiltrate.[9] Condition is mostly self resolving. Treatment is symptomatic and further management depends on severity of disease.[10]

Table 1: Classification of photodermatosis

Immunologically mediated	Polymorphous light eruption Actinic prurigo Chronic actinic dermatitis Solar urticaria Hydroa vacciniforme
Drug- and chemical-induced Genodermatosis	Exogenous: phototoxicity and photoallergy Endogenous: cutaneous porphyrias Xeroderma pigmentosa Others
Photo aggravated conditions	Psoriasis Eczema- Atopic, seborrhoeic, allergic contact Lichen planus Rosacea and facial telangiectasia others

Relationship between photoproducts & biochemical and cellular changes¹⁸⁻³⁸. Skin is affected by UV and visible radiation that absorbed by molecules in skin. The pathway from light absorption to biological response can be broken into several steps. In the first step, molecules in skin such as pyrimidine nucleotides, DNA (e.g., thymidine, cytosine), and urocanic acid absorb radiation.[11] A light absorbing molecule is referred to as a chromophore. In some cases, the chromophore is an exogenous agent such as an ingested photosensitizing drug, for example, tetracycline or 8-methoxypsoralen. After absorbing the energy of the radiation, the chromophore is in an excited state.[12] Although this highly energetic species exists for only a fraction of a second, a chemical change may occur during this time to transform the molecule into a photoproduct such as an oxidized membrane lipid pyrimidine dimer, or psoralen-DNA adduct.[13] The presence of photoproducts in cells provokes biochemical responses, beginning with signal transduction processes that often involve adding and removing phosphate groups from proteins such as the growth factor receptors in the plasma membrane.[14] These changes may activate transcription factor such as AP-1 and NF- κ B, and thereby initiate cellular synthesis of new proteins, for example, melanin, cytokines, cyclooxygenase and matrix metalloproteinase-1.[15] Other important processes, such as repair of UV-induced DNA damage, also occur in response to photoproducts [16].



Materials and Methods

Material for the present study consisted of 70 cases of clinically diagnosed untreated cases of polymorphous light eruptions, who were attending the skin and STD and Leprosy Department, Basaveshwar Teaching & General Hospital, attached to Mahadevappa Rampure Medical College, Gulbarga. The patients were selected at random irrespective of age, sex, socioeconomic status. A detailed general examination was carried out in all cases with particular reference to find out the distribution of skin lesions, type of skin lesions, any secondary changes. Local examination was carried out methodically in every patient to find out the morphological features of every skin lesion. All systems were carefully examined to find out any associated abnormalities in other systems.

Skin biopsy of a classical skin lesion in 30 patients was taken. After thorough cleansing of the selected part with rectified spirit, the parts were infiltrated with two-percent xylocaine and bit of skin involving the whole thickness removed by using elliptical excision method from the edge of the involved skin and the specimens were fixed in aqueous solution of formalin 10.5% and submitted for histopathology studies at Pathology Department, Basaveshwar Teaching & General Hospital, Gulbarga. These biopsy specimens were processed in auto-technician processor wherein the specimens were passed first through increasing concentrations of ethanol for dehydration, then through xylene for lipid extraction and finally through several changes of hot, melted paraffin. After allowing the required time, these specimens were embedded with the epidermis upward in the still liquid paraffin, which is then allowed to harden. The specimens were cut on a rotatory microtome into section 5-7 micromillimeter thick and then they were stained with hematoxylin and eosin. With this statining method the nuclei stain blue whereas the collagen, muscles and nerves stain red. Later these stained histopathological slides were studied under light microscope using both low objective and high objective to find out the changes in the epidermis, dermis.

Results

Seventy clinically diagnosed and untreated cases of polymorphous light eruption were studied. These patients belong to both sex and were between the ages of 4 years and 60 years. They belong to various socioeconomic strata. They were drawn from those who were attending the Skin & STD Outpatient Department, Basaveshwar Teaching & General Hospital, Gulbarga. Out of 70 cases, 21 were male and 49 were female giving a male to female ratio of 1:2.33. The maximum incidence was noted in the age group of 11-20 years and the minimum was in the age group of 31-40 and 51-60 years. Youngest patient was four years old female patient, and the oldest was 60 years old female patient. Pruritus was one of the symptoms observed in 51 of the 70 patients. It was of moderate degree in 42 patients and was of moderate to severe degree in 9 patients. In all the cases, sunlight was provoking the pruritis. None of the patients gave history of neither drugs, neither application locally nor taking systemically. Out of 70 cases, 19 (8 males and 11 females) were agriculturist (27.14%), 23 were housewives (32.86%), 21 were students (30.00%) and 7 were doing miscellaneous occupations (10.00%).

Table-1: Age and Sex Distribution

<i>Age in Years</i>	Male	Female	Total	Percentage
0 – 10	1	4	5	7.14
11 – 20	10	20	30	42.86
21 – 30	2	18	20	28.57
31 – 40	3	1	4	5.71
41 – 50	3	4	7	10.00
51 – 60	2	2	4	5.71
Total	21	49	70	100.00

Table-2: Relation to Occupation

<i>Occupation</i>	Male	Female	Total	Percentage
Agriculturist	8	11	19	27.14
Housewife	--	23	23	32.86
Students	10	11	21	30.00
Other occupation	3	4	7	10.00
Total	21	49	70	100.00

The clinical types of the disease as observed in this series are presented in table-3. Out of 70 cases, 38 (11 male and 27 female) had papular type and 27 had plaque type (8 males and 19 females) and eczematous types were observed in 5 cases (2 male and 3 females). Polymorphous light eruption usually occurs in the sun-exposed areas of the body. The distribution of the lesions observed in this series is presented in table-4. Face was involved in 36 cases and extensor aspect of the forearm and sides of the neck, back of neck were involved in 34 cases.

Table-3: Clinical Types

<i>Clinical Types</i>	Male	Female	Total	Percentage
Papular	11	27	38	54.28
Plaque	8	19	27	38.57
Eczematous	2	3	5	7.15
Total	21	49	70	100.00

Table-4: Distribution of Lesions

<i>Site involved</i>	Male	Female	Total	Percentage
Face	10	26	36	51.43
Extensor aspect of forearms and sides and back of the neck	12	22	34	48.57
Total	22	48	70	100.00

Perivascular lymphocytic infiltration was observed in all of 30 specimens studied. The infiltration was mild in 10 cases, moderate in 13 cases and was dense in 6 cases. Other investigations performed in these cases included routine urine examination for sugar, albumin, microscopy and Blood examination for

hemoglobin percentage, leukocyte count, both total and differential, erythrocyte sedimentation rate were within normal limits in all the patients.

Discussion

Polymorphous light eruption is an idiopathic disorder characterized by a delayed, abnormal response to electromagnetic radiation, usually sunlight, with a varied morphology of papules, plaques and vesicles on exposed areas of the skin. In each patient a single morphology predominates and remains constant [17]. The pathogenesis of PLE is uncommon, but because of delay in onset of lesions and lymphocytic infiltration in involved skin, many studies have suggested an immunological basis for its pathogenesis. The present study was undertaken mainly to study the various clinical manifestations that may occur in polymorphous light eruption, and the histopathological changes in skin in patients with polymorphous light eruption. Totally 70 cases of clinically diagnosed and untreated polymorphous light eruption were included. These patients belong to both sex and were between the ages of 4 years and 60 years. They belong to various socioeconomic strata. Results of the study are critically analyzed in this section¹⁰¹.

The maximum incidence was noted in the age group of 11 to 20 years and the minimum was in the age group of 30-40 and 51-60 years. Thus, this observation confirms to the observations mentioned in textbooks [18]. It was noted the early age of onset in 3 years, whereas in the present series, the age of onset was 4 years. [18] Author reviewed 100 patients in London and found that in 69 of them the age of onset was 30 years or less. From these different studies, it is clear that the age of onset varies considerably, although there seems to be a definite tendency for this disorder to begin in early adulthood in majority of patients.[19] In our series, females are predominant. There were 21 male patients and 49 female patients giving male to female ratio of 1:2.33. Similar observations were seen in the study of Tutrone WD et al (2003), in which females are affected two to three time more than males. According to Dermatologic Disease Database (2006), female to male ratio was 2:1. [20]

Pruritus was one of the symptoms observed in 51 patients out of 70 patients. It was moderate degree in 42 patients and was of moderate to severe degree in 9 patients. According to Vande Pas et al (2004) transient, non-scarring, pruritic papules and vesicles, typically developing hours or days after sun exposure and resolving over several days without sequelae. [21] According to Harber and Bickers (1981), pruritic burning or stinging sensation preceded or accompanied the development of visible lesions. They also noted that most patients are symptomatic in spring and early summer and are comparatively asymptomatic in the fall and winter months. [22]

They are of papular, papulo-vesicular, plaque, vesiculobullous eczematous, insect bite like and erythema-multiforme like variants have been described. In our series, papular type occurred in 38 patients, plaque type in 27 patients and eczematous type in 5 patients. The commonest form was the papular type, second most in plaque type. A similar observation was seen by Dermatologic Disease Database (2006). [23] According to Reinhard et al (2003), papular and papular-

vesicular eruptions were the most common. [24] Lamb et al (1957) observed plaque type to be most common [24] but according to Kontus AP et al (2002), papular type was common. [25] In our series, sunlight is a provoking factor for the development of the disease.

The maximum incidence was observed in housewife, comprising 32.86%. The incidence was 30% in students and 27% in agriculturists. As observed by Dermatologic Disease Database (Jan. 2006) papular was most common and plaque as the second most common type of eruption. [26] According to Main RA (1966) observation was similar to our study, but in the study plaque type was most common type.[27]

30 biopsy specimens were studied in this series. Hyperkeratosis was observed in all the 30 biopsy specimens, parakeratosis was observed in 9 out of 30 biopsy specimens and acanthosis in 17 specimens. There was a focal basal cell layer, hydropic degeneration in 13 biopsy specimens, melanocytes were increased in 18 specimens. There was thinning of the epidermis in 4 specimens. Perivascular lymphocytic infiltration at the mid and upper dermis was observed in 30 biopsy specimens. The histological picture in the papular type, the infiltrate was distributed in a family, tight pattern around subpapillary plexus of extending to deeper vessels predominantly mononuclear cells mainly by T-lymphocytes. No consistent increase in mast cell, neutrophil or eosinophil numbers. In our study, the age of onset of lesions was late in male as compared to females. Similar observations were seen by Boonstra HE et al (2000). [28] The age at onset differed significantly between men and women (average age 46 and 28 years respectively). In our study, 13 patients have positive family history. Orr PH O Birt AR (1984) observed family history of photosensitivity, suggesting an autosomal dominant trait with incomplete penetrance. [29] Millard TP et al (2000) observed prevalence of polymorphic light eruption was 215 and 18% in monozygotic and dizygotic twins respectively. [30] Family history of polymorphic light eruption in first degree relative was present in 12% of affected twin pairs compared with 4% of unaffected twin pairs.

Conclusion

Females were more affected than males giving a male to female ratio of 1:2.33. The maximum incidence was noted in the age group of 11-20 years and the minimum was in the age group of 31 to 40 years and 51-60 years. Pruritus was one of the symptoms observed in 51 out of 70 patients. It was moderate in 42 patients and moderate to severe in 9 patients. 19 patients were agriculturists, 25 were housewife, 22 were students and 7 were doing miscellaneous occupations. The popular form of polymorphous light eruption was seen in 38 patients. The plaque form was observed in 27 patients and eczematous form was observed in 5 patients. Face was the more commonly involved site as it was involved in 36 (51.43%) cases. Outer and extensor aspects of forearm and arms and sides and back of neck were involved in 34 (48.57%) cases.

Histopathological study was done in 30 patients. In these 30 biopsy specimens studied, hyperkeratosis was observed in all the 30 specimens and parakeratosis in 9 specimens. Hyperkeratosis was mild in 13 cases, moderate in 15 cases and

intense in 2 cases. Parakeratosis was only focal in all 9 cases. Spongiosis was observed in 6 cases only and in all specimens were focal. Acanthosis was observed in 17 biopsy specimens. There was focal hydropic degeneration of the basal cell layer in 13 biopsy specimens. The melanocytes were increased in 18 biopsy specimens studied. Thinning of the epidermis was observed in 4 biopsy specimens. Perivascular lymphocytic infiltration was observed in all 30 specimens. It was mild in 10 cases, moderate in 9 cases and dense in 11 cases. Routine blood and urine examination did not reveal any abnormality.

References

1. Verma K, Rokde R, Singh U. A clinic epidemiological and histo-pathological study of polymorphic light eruptions in malwa region. *Indian J Clin Exp Dermatol* 2019;5:24-9.
2. Wadhvani AR, Sharma VK, Ramam M, Khaitan BK. A clinical study of the spectrum of photodermatoses in dark-skinned populations. *ClinExp Dermatol* 2013;38:823-9.
3. Chacko E, Vellaisamy SG, Gopalan K, Nanjappachetty G. A clinic-epidemiological study of polymorphic light eruption in a tertiary care centre in Salem: a region of South India. *Int J Res Dermatol* 2017;3:113-119.
4. Dogra S, Narang T. Emerging atypical and unusual presentations of dermatophytosis in India. *Clin Dermatol Rev* 2017;1:S12-8.
5. Agarwal SB, Godse K, Patil S, Nadkarni N. Knowledge and attitude of general population toward effects of sun exposure and use of sunscreens. *Indian J Dermatol* 2018;63:285-91.
6. U. Mastalier and H. Keri, "Clinical, Laboratory, PhotoTest and Phototherapy Findings in Polymorphic Light Eruption: A
7. AlJasser MI, Lui H, Ball NJ, Kalia S. Persistent polymorphous light eruption after ultraviolet A1 phototherapy. *Photodermatol Photoimmunol Photomed* 2013;29:52-4.
8. Baliah K, Maduravasagam K, Radhakrishnan L, Jambulingam J. A prospective observational study on clinico - pathological correlation in polymorphous light eruption. *IOSR J Dent Med Sci (IOSR-JDMS)* 2016;15(10):01-7.
9. Elmetts CA, Cala CM, Xu H. Photoimmunology. *Dermatol Clin* 2014;32:277-90.
10. Singh M, Sharma E. Novel and secure trend for photo protection-A Hallmark of plant metabolites as UV filters. *Int J Rec Sci Res* 2014;5:322-5.
11. Gruber-Wackernagel A, Byrne SN, Wolf P. Polymorphous light eruption. Clinic aspects and pathogenesis. *Dermatol Clin* 2014;32:315-34.
12. Singh S, Singh A, Mallick S, Arava S, Ramam M. Lichenoid pseudovesicular papular eruption on nose: A papular facial dermatosis probably related to actinic lichen nitidus or micropapular polymorphous light eruption. *Indian J Dermatol Venereol Leprol* 2019; 85:597-604.
13. Ibbotson S, Dawe R. Cutaneous Photosensitivity Diseases. In: Griffiths CEM, Barker J, Bleiker T, Chalmers R, Creamer D, editors. *In Rook's Textbook of Dermatology*. 9th ed. John Wiley & Sons; 2016.p. 127.1-127.35.
14. Pathania V, Shankar P, Shelley D, Baveja S, Sinha A. A case of Hansen's disease masquerading as polymorphous light eruption. *J Mar Med Soc* 2019;21:196-8.

15. Prasad PV, Kaviarasan K, Sidhu U.A clinic-pathological study of polymorphous light eruption. *J Cosmet Dermatol Sci Appl* 2012;2:219-23.
16. Retrospective Study of 133 Patients," *Eur J Dermatol* 8; 8;1998:554-559.
17. Sharma D, Dhillon K S. Polymorphic light eruption: A clinicoepidemiological study. *Paripex- Indian J Res* 2017;6(4): 661-663.
18. Kerr HA, Lim HW. Photodermatoses in African Americans: a retrospective analysis of 135 patients over a 7-year period. *J Am Acad Dermatol* 2007;57:638-643.
19. Lal BM, Devi AS, Suhasini K. The study of clinical variations and histopathological findings in polymorphous light eruption. *J Evid Based Med Healthc* 2016;3:2899-904.
20. Rodríguez-Pazos L, Gómez-Bernal S, Rodríguez-Granados MT, Toribio J. Photodistributed erythema multiforme. *Actas Dermo-Sifiliográficas* 2013;104:645-53.
21. Van de Pas LB, Kelly DA, Seed PT et al. Ultraviolet radiation induced erythema and suppression of contact hypersensitivity responses in patients with polymorphic light eruption. *J. Invest Dermatol.* 2004; 122: 295-9.
22. Harber and Bickers. Photosensitivity disease. Philadelphia, Saunders. 1981; 109: 170-182.
23. Dermatologic Disease Database: Polymorphous light eruption: <http://www.aocd.org/skin/dermatologic-disease/polymorphous-light-eruption.htm>. on 21st Jan. 06.
24. Reinhard et al. Clinical and therapeutic aspects of polymorphous light eruption. Dept of Dermatology, University Hospital, Zurich, Switzerland. *Dermatology* 2003; 207: 93-95.
25. Kontus AP et al. Polymorphous light eruption in African Americans: Pinpoint papular variant. *Photodermatol Photoimmunol Photomed.* 2002 Dec; 18(6): 303-6.
26. Abeyama K et al. A role for NF kappa B-independent gene transactivation in sunburn. *J Clin Invest.* 2000; 105: 1751.
27. Main RA. Polymorphous light eruption. *Practitioner.* 1966; 196: 654-60.
28. Bounstra HE et al. Polymorphous light eruption: A clinical, photobiologic and follow-up study of 110 patients. *J Am Acad Dermatol.* 2000 Feb; 42(2 pt 1): 199-207.
29. Orr PH, Birt AR. Hereditary polymorphic light eruption in Canadian unit. *Int J Dermatol.* 1984; 23(7): 472-5.
30. Millard TP et al. The heritability of polymorphic light eruption. *J Invest. Dermatol.* 2000 Sept. 115 (3): 467-70.