

## CLINICO-EPIDEMIOLOGICAL AND HISTOPATHOLOGICAL CORELATIONOF POLYMORPHIC LIGHT ERUPTION

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**ABSTRACT: OBJECTIVES:** To study the incidence, clinical varieties, histopathological findings of PLE and to know its possible relationship to other cutaneous disorders and systemic diseases. **METHODS:** 70 patients of PMLE were considered and detailed general examination and biopsy of selected patients was done. 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.

**KEY WORDS:** PMLE, UV Rays, Photosensitivity.

**INTRODUCTION:** The term 'light' can be used to describe the visual sensation elicited when a portion of electromagnetic spectrum with wavelengths between 7000 and 3900A<sup>o</sup> reaches the photoreceptors of the eye, and is capable of stimulating in the normal human eye the sensation of vision and to denote the radiant energy itself. Radiant energy may be defined as electromagnetic energy of a fundamental spectral nature<sup>1</sup>.

Solar radiation is the main source of light to the world<sup>2</sup>. Very short wavelength UV light down to 100A<sup>o</sup> is known to be radiated from the sun as a result of internal thermonuclear reactions<sup>3</sup>. We are protected from very short more potential lethal wavelengths by the filtering action of oxygen and ozone layers in the outer atmosphere<sup>4</sup>. So less than 1% of the sun's radiation that reaches the earth's surface is of UV wavelengths (2900-4000A<sup>o</sup>)<sup>5</sup>. Only 0.2% of this UV light is of wavelength 2900-3200A<sup>o</sup> that causes sunburn in human skin<sup>6</sup>.

Human skin has neither a neural nor a humoral system that can immediately alert one as to when an overdose of harmful UV radiation is being received. The amount of harmful UV radiation that reaches the skin at any given time is dependent upon the latitude, season, time of the day, and the conditions of one's immediate environment. The lower the latitude, the greater will be the risk of sun damage to skin per hour, per day and per season. The time of greatest risk at all latitude occurs in the mid-summer between 10 AM to 2 PM<sup>7,8,9,10</sup>.

The factors in environment, increasing the total dose of radiation one receives are the white sand of the beach<sup>11</sup>, the snow<sup>12</sup>, the shiny metallic glazed and painted surface<sup>13</sup>. An overcast or light fog will scatter sufficient UV rays to cause reaction on the skin<sup>14</sup>.

There are two main factors that are related to attenuation of sunlight:

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- a) Wavelength in general the shorter the wavelength, the greater the attenuation;
- b) The thickness of the atmosphere – based on Bouguer- Lambert Law, there is loss in power of sunlight with atmospheric thickness.

Other factors, which play important roles are:

1. Atmospheric oxygen, which strongly absorbs all wavelengths shorter than 2000Å<sup>o</sup>;
2. Ozone which absorbs from near 2000Å<sup>o</sup> maximally at 2550Å<sup>o</sup> and much less about 3400Å<sup>o</sup>;
3. Water vapour and carbon dioxide which strongly absorbs certain infra-red bands;
4. Gas molecules which produce so called Rayleigh effect;
5. Suspended particles attenuated by more complex scattering process<sup>15,16</sup>.

Photosensitivity denotes either a quantitative or qualitative abnormality in skin's response to sunlight or artificial light exposure and these abnormal reactions to light in man have been classified into four major groups by Fitzpatrick et al.

1. Light Alone: Here the light alone is implicated in the causation of abnormal condition. The chromophores might well be the cutaneous DNA or component of the epidermal cells.
2. Light plus an exogenous agent: Here the exogenous agent is implicated in evoking the photosensitivity reactions when the skin is exposed to solar radiation. The chromophores are a chemical agent applied topically or administered orally. Phototoxic (non-immunologic) as well as photoallergic (immunologic) types of reactions are included here.
3. Light plus metabolite: In this group are several photosensitivity reactions in patients with various types of porphyrias. This is related to the overproduction of in-vivo of uroporphyrin, protoporphyrin, coproporphyrin and their precursors. The chromophores are the irreversible oxidized porphyrin molecules present in blood, plasma, skin, liver, stool or urine.
4. Light plus abnormal skin or disease: In this are several diseases where the exact chromophore is not known<sup>17</sup>.

Another classification by Harber et al is as follows <sup>17,18</sup>:

1. Genetic: Albinism, Xeroderma Pigmentosum, Bloom's, disease, Cockayne's disease, Rothmund-Thomson disease, phenyl ketonuria.
2. Metabolic: Pellagra, Hartnup disease, porphyrias.
3. Phototoxic: Systemic, contact
4. Photoallergic: Systemic, contact
5. Degenerative: Solar keratoses, Sailor's and Farmer's skin.
6. Neoplastic: Basal cell epithelioma, squamous cell carcinoma, malignant melanoma, keratoacanthoma.

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7. Photo aggravated: Herpes simplex, systemic lupus erythematosus, dermatomyositis, pemphigus erythematosus, Darier's disease, erythema multiforme, pityriasis rubra pilaris, psoriasis, lichen planus, lymphocytoma.
8. Idiopathic: Polymorphous light eruption, solar urticaria, hydroa vacciniforme, hydroa estivale, lymphogranuloma venereum.

Polymorphous Light Eruption<sup>17</sup> is a chronic, idiopathic, acquired or hereditary disorder of skin, characterized by a delayed abnormal response to electromagnetic radiation usually sunlight with a varied morphology of papules, plaques and vesicles on the exposed areas of the skin. The clinical types are plaque type, eczematous type, papular and vesicular type and erythematous type.

During the last three decades interest in the reaction of human skin to light has been renewed. This interest made us to take up the study of polymorphous light eruption – a type of reaction of the human skin to sunlight or artificial light.

**OBJECTIVES:** To study the incidence, clinical varieties and histopathological findings of PLE and to know its possible relationship to other cutaneous disorders and systemic diseases.

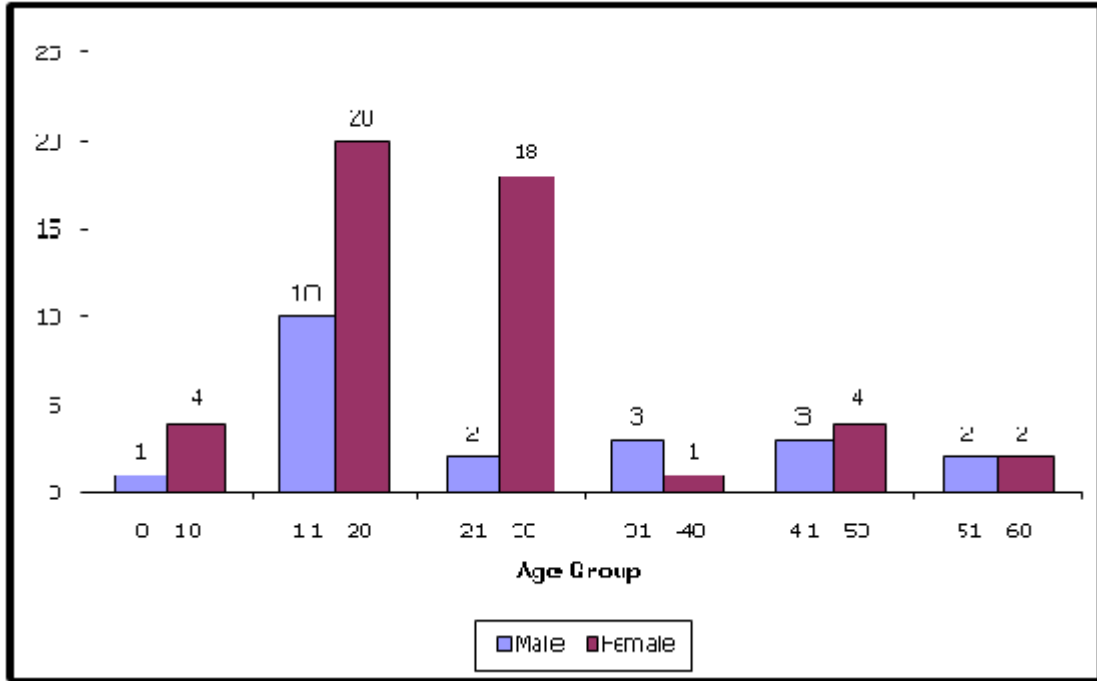
**MATERIALS AND METHODS:** Material for the present study consisted of 70 cases of clinically diagnosed untreated cases of polymorphous light eruptions, who were attending the skin and STD and Leprosy Department, Basaveshwara Teaching & General Hospital, attached to Mahadevappa Rampure Medical College, Gulbarga.

The patients were selected at random irrespective of age, sex, socioeconomic status. A detailed history was taken with reference to the past history of similar episodes, family history of any similar illness, mode of onset, progression of the disease, seasonal variation and the extent of the involvement. An attempt was made to find out if any provocation factors like drugs, systemic illness, etc. were present. Local examination was carried out methodically in every patient to find out the morphological features of every skin lesion.

All the cases which were clinically diagnosed as cases of polymorphous light eruptions were investigated by taking a skin biopsy. Also urine albumin, sugar, microscopy and erythrocyte sedimentation rate were undertaken.

**RESULTS:** The results of the study are presented below.

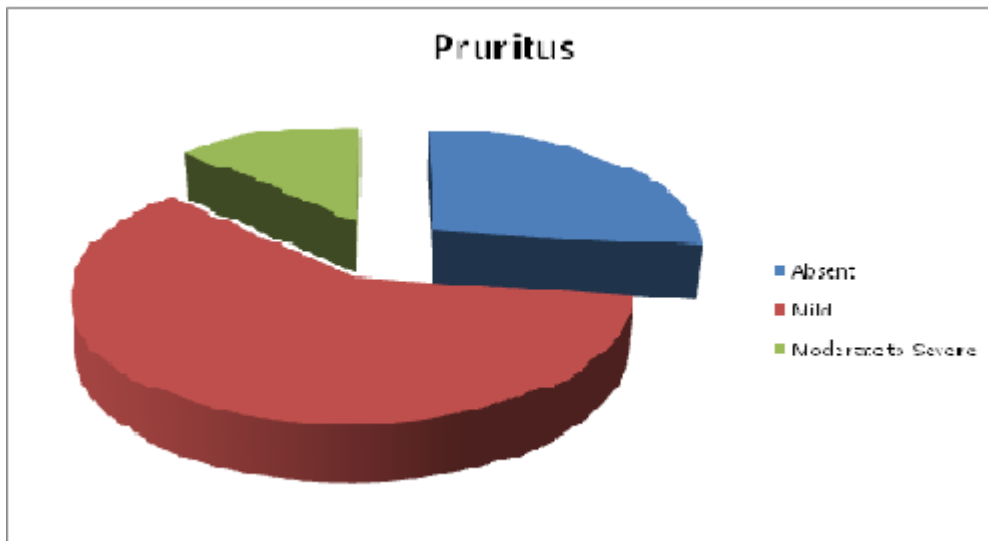
**Figure-1: Age and Sex Distribution**



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.

**Symptoms:**

**Figure 2: Incidence of Pruritus**



Pruritus was one of the symptoms observed in 51 of the 70 patients.

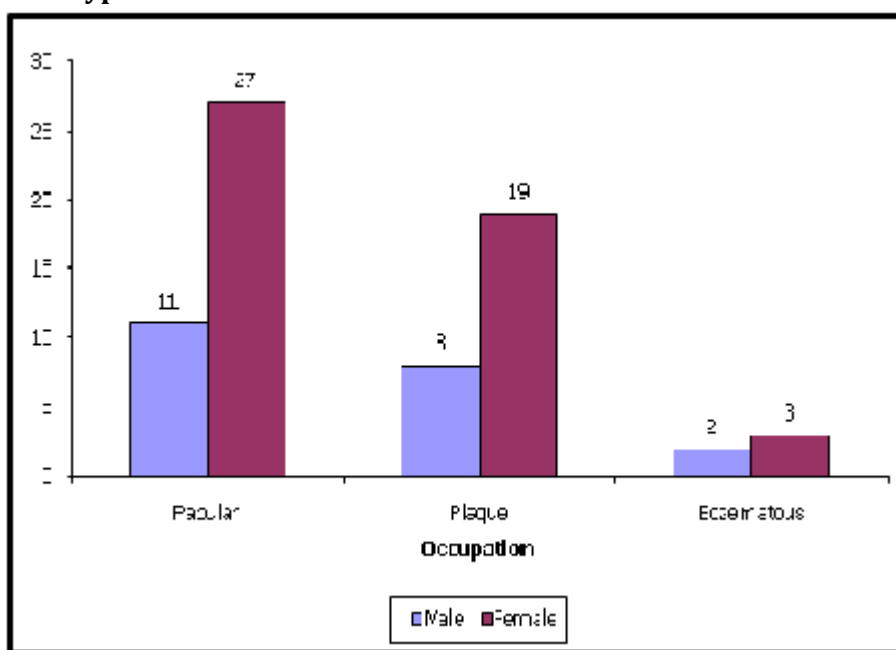
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**Table-1: Clinical Types**

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

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).

**Figure-3: Clinical Types**

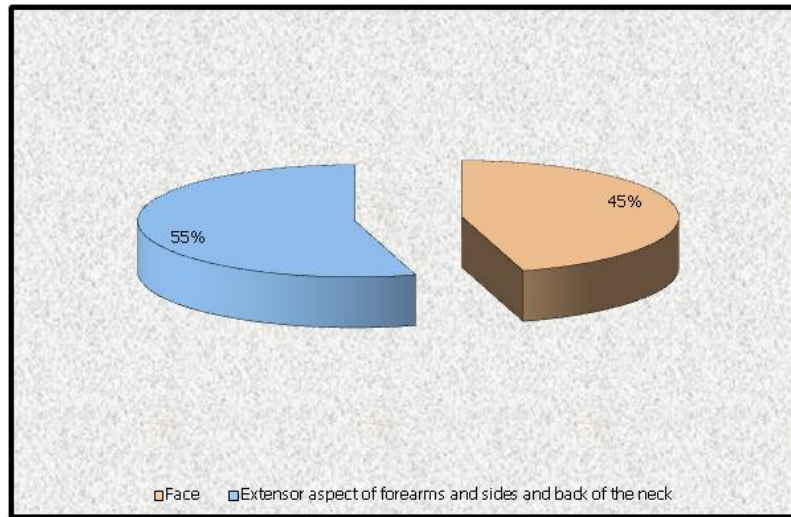


**Table-2: Distribution of Lesions**

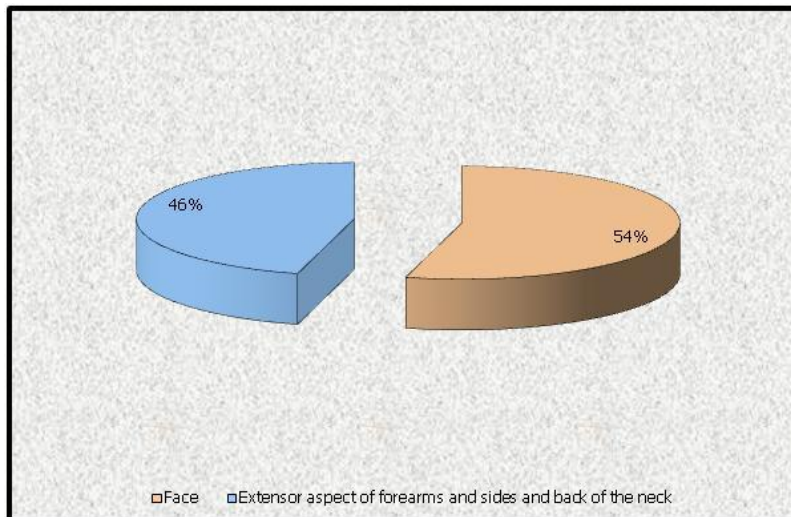
Site involved	Male	Female	Total	Percentage
Face	10	26	36	51.43
Extensor aspect of forearms and sides and back of the neck	12	22	34	48.57
<b>Total</b>	<b>22</b>	<b>48</b>	<b>70</b>	<b>100.00</b>

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.

**Figure-4: Distribution of Lesions in Males**



**Figure-5: Distribution of Lesions in Females**



**Table-3: Histopathological changes**

Epidermal/ Dermal changes	Male	Female	Total	Percentage
Epidermis				
Hyperkeratosis	9	21	30	100.00
Parakeratosis	3	6	9	30.00
Spongiosis	3	9	12	40.00
Acanthosis	5	12	17	56.67
Hydropic degeneration of basal cells	3	10	13	43.33
Increase in melanocytes	8	10	18	60.00
Thinning of epidermis	1	3	4	13.33
Subcorneal vesicles with exocytosis	0	1	1	3.33
Dermis				
Perivascular lymphocytic infiltration	15	15	30	100.00

### **Epidermal Changes:**

Hyperkeratosis was observed in the biopsy specimens of all 30 cases, whereas parakeratosis was observed in 9 cases. Spongiosis was observed in 12 cases only. Acanthosis was observed in 17 biopsy specimens. The hydropic degeneration of the basal cell layer was observed in 13 biopsy specimens. The melanocytes were increased in 18 biopsy specimens studied. Thinning of the epidermis was observed in 4 biopsy specimens. There were subcorneal vesicles with exocytosis in 1 biopsy specimen studied.

**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<sup>19,20</sup>. 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 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<sup>21</sup>.

MastelieuKerl H and Wolf P (1998) noted the early age of onset in 3 years, whereas in the present series, the age of onset was 4 years<sup>22</sup>.

According to Morison, the age of onset varies between 20-40 years<sup>23</sup>. According to MastelieuKerl H and Wolf P, the median age of onset of PLE was 26 years<sup>22</sup>.

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, in which females are affected two to three time more than males<sup>24</sup>.

According to Dermatologic Disease Database, female to male ratio was 2:1<sup>25</sup>. According to Morrison study, female preponderance was more than male with a ratio of 3:1<sup>23</sup>. According to Sophie

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Shirin, females are affected 2-3 times more than males<sup>26</sup>. These studies almost correlate with the present study.

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 transient, non-scarring, pruritic papules and vesicles, typically developing hours or days after sun exposure and resolving over several days without sequelae<sup>27</sup>.

John LM et al, classified polymorphous light eruption into many clinical types. They are of papular, papulo-vesicular, plaque, Vesiculobullous eczematous, insect bite like and erythema-multiforme like variants<sup>28</sup>.

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<sup>25</sup>.

According to Reinhard et al (2003), papular and papular-vesicular eruptions were the most common<sup>29</sup>.

According to Kontus AP et al, papular type was common<sup>30</sup>. As observed by Dermatologic Disease Database papular was most common and plaque as the second most common type of eruption<sup>25</sup>. 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. This observation correlates with the observation by Fitzpatrick<sup>17</sup>, Epstein JH<sup>31</sup> and also by Holzte et al<sup>32</sup>. The histological picture in the papular type, the infiltrate was distributed in a family, tight pattern around sub pupillary plexus of extending to deeper vessels predominantly mononuclear cells mainly by T-lymphocytes.

No consistent increase in mast cell, neutrophil or eosinophil numbers. This observation compared with studies of PG Norris et al<sup>33</sup>. The absence of hydropic degenerations of the cells of the basal layer was observed by Wright and Winer<sup>34</sup>.

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<sup>35</sup>. 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.

Millard TP et al<sup>36</sup> observed prevalence of polymorphic light eruption was 215 and 18% in monozygotic and dizygotic twins respectively.

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.



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The papular form of polymorphous light eruption was seen in 38 patients and was the most common variant. 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.

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.

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