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Photodermatoses: Causes, Diagnosis and Treatment

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A B S T R A C T

A very significant and prevalent class of skin conditions known as photodermatoses is brought on by aberrant cutaneous responses when exposed to sunlight. Visible light (VL) and/or ultraviolet (UV) radiation is included in this exposure. Rashes, stinging, blistering, burning, or inflammation (eczematous reactions) on sun-exposed areas are some of the symptoms of these disorders. There may also be systemic symptoms such joint discomfort, exhaustion, or fever. Idiopathic photosensitive illnesses, medication or chemical-induced photosensitivity reactions, photoaggravated dermatoses (such Parthenium dermatitis), and hereditary disorders with impaired DNA repair are the four general categories into which photodermatoses fall. Since photodermatoses typically have no known treatment, photoprotection—either by avoiding light or by physically shielding oneself from the wavelengths that trigger them—is the cornerstone of management.

Keywords: Photodermatoses, Photoaggravated, Photoprotection, Photodermatitis, Photosensitive.

Introduction

Skin conditions known as photodermatoses are brought on by or exacerbated by exposure to sunshine, particularly ultraviolet (UV) radiation. Although the sun's light is necessary for many metabolic, endocrine, and physiological functions of life, its impact on the skin has drawn serious attention from the public and medical community. This is especially true in regions like India that receive a lot of sunlight. Immunologically caused photodermatoses, drug- and chemical-induced photosensitivity, photoaggravated dermatoses, and inherited conditions characterized by chromosomal instability or impaired DNA repair. Sun poisoning, sun allergy, photoallergy, and photosensitive dermatitis are common synonyms for photodermatitis. [1, 2, 3, 8]. Certain electromagnetic spectrum wavelengths can cause photodermatoses, with UVA (320–400 nm) and UVB (290–320 nm) being the main offenders. Reactions can also be triggered by visible light (400–760 nm), especially in cases of solar urticaria. [1, 2, 3, 22, 29, 33].

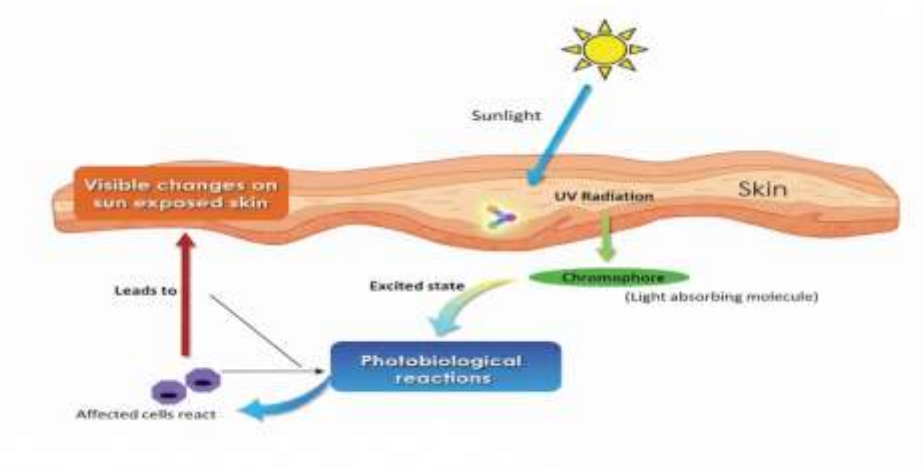

Fig.1- representation of photobiological response of skin

1. Idiopathic Photodermatoses

Idiopathic photodermatoses, also known as primary photosensitivity disorders or immunologically mediated photodermatoses, are a significant and prevalent group of skin diseases with an unknown or endogenous cause that are characterized by abnormal cutaneous reactions to solar radiation [12,5,33].

a. Polymorphous Light Eruption (PMLE):

The most prevalent immuno-mediated photodermatosis is PMLE, often known as sun allergy. Although the exact etiology of PMLE is uncertain, it is thought to be a delayed type hypersensitivity reaction. The V-area of the chest, arms, forearms, thighs, upper back, and less frequently the face are affected by lesions that develop on sun-exposed areas [9,11,12,21]. Patients may experience macular, papular, vesico-papular, or plaque-like skin lesions that are extremely irritating or burning. A few hours to many days following sun exposure, the rash develops [9,10,12]. Tests for diagnosis: Photoprovocation: Through experimental provocation, the diagnosis can be verified. Patients with PMLE typically have a normal Minimal Erythema Dose (MED). Differential Diagnosis: It's important to differentiate PMLE from other illnesses such as photoallergic eczema and lupus erythematosus (LE) [9,12,29]. Treatment: Glucocorticoids used topically can hasten the healing process. Phototherapy ("Hardening"): The skin's tolerance can be raised by carefully exposing it to artificial UV light sources prior to the sunny season. Antimalarials: Hydroxychloroquine is a potentially useful and affordable substitute for phototherapy. Immunosuppressants: Azathioprine or cyclosporine may be taken into consideration for severe and refractory cases. Strict Photoprotection: it is crucial to use full-coverage, tinted sunscreens and light-blocking clothes for effective sun protection [5,9,10,11,22].

b. Chronic ActinicDdermatitis

A sun-exposed skin and can spread to covered areas is called chronic actinic dermatitis (CAD) [7,12,10]. Since the cause is unknown, it is categorized as an idiopathic photodermatosis [12]. A DTH reaction to an endogenous, cutaneous, photo-induced allergen that clinically resembles allergic contact dermatitis is the most likely etiology [10,21]. Clinical Features: Although it can spread to covered areas, CAD is characterized by persistent, lichenified dermatitis on sun-exposed areas such as the hands, face, scalp, and neck [9,10,11]. Diagnostic tests include patch tests and phototests. Airborne contact dermatitis can mimic or worsen these conditions, so it's critical to rule them out. Differential Diagnosis: The illness needs to be distinguished from systemically caused photoallergic reactions, mycosis fungoides, and atopic eczema [9,10,11,20]. Photochemotherapy and Topical Therapies: Flares can be controlled with brief treatments of topical steroids and emollients. Systemic Therapies: Glucocorticosteroids, azathioprine, cyclosporine, or mycophenolate mofetil are examples of systemic drugs used for more severe or resistant cases [9,10,11,28].

c. Solar Urticaria

The unusual IgE-mediated photodermatosis known as solar urticaria (SU) is typified by the quick, fleeting emergence of wheals after exposure to UVR or visible light. Within 5–10 minutes of sun exposure, symptoms start as a tingling feeling over exposed areas, which is quickly followed by erythema and wheals. After the sun stops shining, the eruption goes away entirely in one to two hours [9,10,11,18]. Clinical Characteristics: Within minutes of being exposed to sunlight or artificial UV sources, urticarial skin lesions, also known as wheals, develop. Initially, patients may feel a tingling feeling. After stopping exposure, the rash usually goes away in one to two hours [9,10,11]. Diagnostic Tests: Differential Diagnosis: Erythropoietic protoporphyria and urticarial variants of PMLE are conditions to take into account [9]. Therapy: Primary Care: Avoiding light and shielding oneself from the triggering wavelengths are the cornerstones. Phototherapy, photochemotherapy, and antihistamines [9,10,11,29].

d. Actinic Prurigo (AP):

AP is a persistent, itchy skin condition that often begins in childhood and lasts for a long time due to an aberrant reaction to sunlight [11,7,9]. Excoriated papules and nodules, which are primarily present on the face and limbs, are a characteristic of AP. [7,11]. Clinical Characteristics: On sun-exposed areas, especially the face and limbs, the condition manifests as itchy, excoriated papules and nodules. [7,9,11]. Tests for diagnosis: Histopathology: The diagnosis is made by a skin biopsy. Tests in the lab, differential diagnosis: It must be differentiated from photo-aggravated atopic eczema, PMLE, and chronic actinic dermatitis [7,9,29]. Therapy: Photoprotection: Using sunscreens with a high SPF and limiting UV exposure are essential. exposure to sunlight [9,10,11,22].

e. Hydroa Vacciniforme (HV):

HV is an extremely uncommon condition. [7,9,]. After a few days of exposure to the sun, HV produces crops of distinct erythematous macules that develop into blisters (vesicles) [9,11]. Clinical Features: Its unique clinical appearance serves as the basis for diagnosis. It involves hemorrhagic vesicles on the hands and face that appear suddenly. The fact that these lesions heal and leave behind varioliform scars, which resemble smallpox, is a crucial diagnostic characteristic [9,11]. Treatment: Although treatment is challenging, sunscreens that filter UV rays and limiting UVR exposure may be beneficial [11].

2. Drug Induced Or Chemical Induced Photodermatoses

When a medication or its metabolites absorb UVR or visible light, it causes "photosensitization," which is the first step toward photosensitivity and results in biochemical alterations in the tissue [6,32]. According to their fundamental mechanism, the resulting reactions are divided into two primary categories: phototoxic reactions and photoallergic reactions [6,24,26,27].

a. Drug-Induced Phototoxic Reactions:

The most prevalent kind of drug-induced photosensitivity is phototoxic reactions [9]. Nonallergic inflammatory skin reactions brought on by photochemical processes are known as phototoxic reactions [6,9]. Direct cellular damage is one of their characteristics [6]. When photons are absorbed by the photosensitizing drug molecules, they are stimulated into an unstable, reactive triplet state [6]. Usually, phototoxic reactions are dose-dependent, which means that the drug and light doses both affect how severe the reaction is [6]. Numerous pharmacological types, such as diuretics, antimicrobial

medications, and nonsteroidal anti-inflammatory medicines (NSAIDs), are currently linked to high levels of severe photosensitivity [4,5,30]. Clinical Characteristics: It usually presents with burning or severe erythema (redness), oedema (swelling), and occasionally vesicles or blisters, much like an excessive sunburn. Severe hyperpigmentation may follow [6,9,10,24,25].

Drug-Induced Photoallergic Reactions

Compared to phototoxic reactions, photoallergic reactions are less frequent [9]. Induced by Drugs Immune-mediated (T-cell-mediated) reactions, photoallergic reactions necessitate prior exposure to the photosensitizing agent (a chemical or medication) in order to develop a particular sensitization [6,9,10,24,25]. The photosensitizing medication undergoes a structural alteration as a result of the interaction of UV light, most frequently UVA [6,10,30]. A photoallergen (hapten) is produced when this altered drug structure attaches to skin proteins [6,10]. Epidermal Langerhans cells present this photoallergen to T lymphocytes, which causes a Type IV hypersensitivity reaction, which is a delayed hypersensitivity reaction [6,7,10,24,25]. Usually, these don't depend on dosage [5,6,32]. Clinical Characteristics: It begins as an eczematous dermatitis that is irritating and characterized by papules, vesicles, crusting, and leaking. Later on, it may become scaly and lichenified [6,7,10]. Diagnostic Tests: A number of tests can be carried out to confirm the diagnosis and rule out other illnesses when the diagnosis is unclear. Histopathology (Skin Biopsy): By ruling out other dermatoses, a biopsy can assist confirm the diagnosis. Systemic photoprovocation, photo-onycholysis, hyperpigmentation and dyschromia, etc. [6,10,29].

Treatment: Identifying and stopping the causal substance, controlling acute symptoms, and putting strict photoprotection in place to stop recurrence are the main strategies for treating both phototoxic and photoallergic drug- or chemical-induced photosensitivity (DIP). [29, 30].

3. Photoaggrivated Photodermatoses

disorders known as photoaggravated dermatoses occur when exposure to sunlight, especially UV light, exacerbates or flares up pre-existing skin disorders [7,10,11]. To put it simply, these conditions are common dermatoses that are extremely sensitive to light exposure rather than primary photosensitivity illnesses [9,11,31]. Secondary heliotropic illnesses are another name for them [9]. Primary Cause: In addition to light exposure, the underlying disease has "another genesis altogether" (a separate origin) [9,11]. The disease is triggered or made worse by sunlight [7, 10]. In addition to sun protection, the management strategy calls for proper treatment of the underlying main condition [11]. UVR is reported to aggravate the following conditions [10,11,16].

a. Lupus Erythematosus (LE):

Photosensitivity is a characteristic of both systemic lupus erythematosus (SLE) and cutaneous lupus erythematosus (CLE), especially discoid lupus erythematosus (DLE), which might worsen in a tropical environment [10,11].

b. Eczema and Rashes:

Sunlight exposure can exacerbate conditions like rosacea, seborrheic dermatitis, and atopic eczema [10].

C. Parthenium Dermatitis:

Photoaggravation is a characteristic feature of this illness [7, 10]. Airborne allergens, including oleoresins from the Parthenium hysterophorus weed, can more readily enter the damaged skin of those with chronic eczematous dermatitis (such as those with Chronic Actinic Dermatitis). Instead of the allergen being the initial photosensitizing cause of the disease, this leads to a secondary contact dermatitis that worsens the pre-existing condition [10,16]. Identifying the underlying skin condition and establishing a documented history of exacerbation or triggering by sun exposure are the two main aspects in diagnosing photoaggravated dermatosis [11,31].

Drug- or chemical-induced photosensitivity (DIP) is treated with a multifaceted strategy that includes prompt reaction management, withdrawal of the causing agent, symptomatic relief, and—above all—strict photoprotection and patient education to avoid recurrence of the condition [6,10,11,28,31].

4. Genetic or Inherited Photodermatoses

The term "genetic photodermatitis" describes photosensitivity linked to pre-existing, usually uncommon, hereditary abnormalities. These disorders belong to a particular class of photodermatoses, which are characterized by aberrant skin responses to visible light or ultraviolet (UV) radiation. DNA repair-deficiency photodermatoses, hereditary illnesses with chromosomal instability, or faulty DNA repair are other names for these syndromes [7, 10]. Porphyrias: Since porphyrias are metabolic illnesses caused by either acquired or hereditary abnormalities in specific enzymes along the heme biosynthesis pathway, they are categorized as secondary photodermatoses [9,10,23]. Patients may not always be aware of the link between sun exposure and these skin alterations since they often occur in subtle ways on traumatized or sun-exposed areas (such as the face, neck, dorsal sides of the fingers, and hands) [10,15,19,23].

A deficiency in DNA repair causes cellular hypersensitivity to UV light in Xeroderma Pigmentosum (XP), a rare autosomal recessive condition. Photosensitivity, pigmentary alterations, early skin aging, and the emergence of malignant tumors are its clinical hallmarks. According to the sources, its occurrence in India is negligible [10,13,14]. Porphyrias are a class of hereditary or acquired diseases that impact the enzymes involved in the heme-producing pathway. Porphyria cutanea tarda and erythropoietic protoporphyria are two examples of cutaneous porphyrias, which are those that result in symptoms related to the skin [9,10,14,15]. Diagnosis: Making a diagnosis depends on identifying the distinctive clinical characteristics of each individual

illness.Treatment: Since there is no known cure for genetic photodermatoses, careful photoprotection is the mainstay of treatment because there aren't many reliable pharmaceutical treatments. [5,22].

Conclusion

Due to overlapping clinical characteristics and the difficulties in pinpointing specific triggers, particularly in patients using several drugs, photodermatoses are still difficult to diagnose. Therefore, education of physicians and patients is essential for primary and secondary prevention. For example, there have been few research on the prevalence of Polymorphous Light Eruption (PMLE) in the general population, despite the fact that it is frequent. Due to patients' frequent ignorance that sun exposure is the cause of a skin eruption, the overall prevalence of acute and chronic photosensitivity is unknown and probably underdiagnosed. The frequency and prevalence of photosensitive dermatitis are also unknown at this time because diagnoses are uncommon, most likely as a result of possible misdiagnosis. To more accurately evaluate the effectiveness of preventative and treatment approaches, additional randomized, controlled trials are required. There is still much to learn about the precise mechanisms, especially with regard to idiopathic illnesses like PMLE and the photoexacerbation of autoimmune diseases. Severe photosensitivity's psychological and social effects, which include loneliness and a lower quality of life, emphasize the necessity of efficient management. There is continuous worry about the possible connection between phototoxic medicines and an elevated risk of skin cancer. In severe circumstances, persistent use of certain phototoxic medicines may result in permanent scarring, chronic disorders like erythroderma, or an increased risk of skin cancer. However, advancements have been achieved, especially in the creation of sunscreens that are more affordable and effective. In the end, timely clinical diagnosis, avoiding triggers, and thorough patient education regarding sun protection are now the mainstays of photodermatosis management.

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