

Skin diseases affect on people's health



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In modern society, more and more people are attacked by a variety of diseases. In medicine, relevant skin diseases seriously affect people's health. As one of the common diseases, skin diseases such as leprosy, scabies, fungal disease, bacterial skin infections appear frequently. With the form, structure and functions changing, skin (including hair and armor) is influenced by external and internal factors, which produce the pathological process, and the corresponding produce all sorts of clinical successively performance. This is the cause of skin diseases. They have a high incidence of dermatitis, but relatively the symptoms are not serious, they often do not affect health, but a few heavier are even life-threatening.

As a typical kind of skin diseases, photodermatoses are among the most common skin disorders in the world. Some of them acquired a particular importance in some regions because of their high frequency, severity, and also because of their different diagnostic and therapeutic approaches.

Photo medicine is a rapidly developing subspecialty of dermatology concerned with skin diseases caused by radiation in the UV and visible spectra. Initiation or exacerbation of a rash after sun exposure that occurs in typical light-exposed areas is features that point toward a sunlight-induced condition. The diagnosis of photosensitive conditions may be difficult, and the use of investigations such as light, patch, and photopatch testing may be necessary to confirm the diagnosis. [1]

Background and history

With the development of modern medicine, every Teaching Hospital Department is in treatment development dilemmas. For becoming involved

in a new therapy, they still need to promise to be at the speculative stage. In the early 1990s, the problem about whether to actively become involved in the development of PDT for skin cancers was discussed by the Photobiology Unit within the Department of Dermatology in Dundee. As a new investment significant resources, it's so difficult for PDT to have a fairly certain outcome. By 1998, with the position changed and enough good quality data existed, treatment outcomes justified become involved in the development of both PDT and photodiagnosis (PD) for pre-malignant and malignant skin lesions. From a clinical research and therapeutic point of view, the skin has two huge advantages. Firstly, it can be easily examined with the naked eye, and secondly, it is the most accessible organ for investigation, biopsy and treatment. Although PDT firmly has its roots at the beginning of the last century, it is only over the last 15 years that it has gained considerable popularity as a topical treatment of great promise for the treatment of skin cancers.[2]

In 1900 a German medical student Oscar Raab famously reported the concept of cell-induced death subsequent to light interacting with chemicals. In subsequent experiments he demonstrated that this effect was greater than with alcidine red alone, light alone or alcidine red exposed to light and then added to the paramecium. He postulated that in vitro toxicity occurred as a result of fluorescence caused by the transfer of energy from the light to the chemical. Professor von Tappeiner soon after predicted the future of fluorescent substances in medicine.

In 1904 von Tappeiner and Jodlbauer identified that oxygen was integral component in photosensitisation reactions and termed the phrase "

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photodynamic action” in 1907. Since its incidental discovery in 1900 photodynamic therapy (PDT) and all aspects relating to it from mechanism of action, differing photosensitisers through to clinically based applications have been studied. Three components are required for PDT to occur; a photosensitiser, oxygen and a light source. [3]

The Photobiology Unit (photobiology = the study of light on living systems) has the purpose in Scotland of diagnosing light sensitive skin disease (the photodermatoses) and the development of new forms of light therapy (phototherapy). This Centre, which has been in existence since 1973, has always combined clinical skills (photodermatology) with a strong scientific base (photophysics) and laboratory biology (photobiology). This combination of applied science and clinical service in the same unit has provided exciting research opportunities. Applied photo physics, through the Medical Physics Department, has dedicated members of staff whose only role is optical physics. The necessary expertise in light delivery and measurement is essential for predictable PDT and PL.

Basic knowledge of sun and the skin

Nm 254 290 320 360

X-rays

UVC

UVB

UVA

Visible light

Figure 1 the place of ultraviolet radiation in the electromagnetic spectrum

Figure 1 illustrates the relationship between ultraviolet radiation and the other types of non-ionizing radiation, such as natural light and infra-red radiation. It will be seen that ultraviolet radiation from the sun is divided into three different wavelengths—UVA, UVB, and UVC. The UVA waves are the longest and the UVC the shortest.

At present, UVC is prevented from reaching the earth's surface by the ozone layer, and is not therefore a natural hazard. There is, however, concern that the loss of the protective layer of ozone above the earth's atmosphere will continue, and that in future more UVB might reach the earth. The main ultraviolet component of natural light that reaches the earth's surface is UVB. This penetrates the epidermis and reaches the more superficial layer of the dermis—the papillary dermis. UVA is also present in sunlight and, in the early spring, a high proportion of natural sunlight in countries at latitudes 50 degrees or more north or south of the equator is composed of UVA. As the summer develops the proportion of UVA falls. UVA is the main, but not the only, wavelength found in the long tubes in UVA sunbeds. The effects of UVA go deeper into the skin than those of UVB. A very simple rule of thumb is that chronic over-exposure to UVB causes wrinkles, chronic over-exposure to UVA causes sagging, and chronic over-exposure to both increases the risk of developing skin cancer. One of the important points of difference between UVB and UVA exposure is that acute over-exposure to UVB causes the redness and soreness recognized as sunburn. This is maximal 12-24 hours

after the exposure has taken place, and is a useful warning that the skin should be protected for a few days until the redness has disappeared.

The chemicals in sun-screens that protect against UVA and UVB can be divided into those that absorb ultraviolet radiation and those that reflect it away. The absorbing chemicals include para-aminobenzoic acid-PAMA-cinnamates, and salicylates, which protect against UVB alone.

Benzophenones protect against both UVB and UVA and are also chemical sun-screensers.[4]

Photosensitivity

The skin is our main defense against light, and in particular against ultraviolet (UV) radiation. Sometimes the skin reacts abnormally to light by becoming inflamed. This is called photosensitivity.

There are many causes of photosensitivity. Some of the most important are below:

Acute parts like Sunburn Xeroderma pigmentosum, Porphyria, Solar urticaria, Pellagra, and Photosensitivity disorders like Polymorphic light eruption, Juvenile spring eruption, Hydroa vacciniforme, actinic prurigo. Disorders exacerbated by light include Drug reactions, Lupus erythematosus, Rosacea Darier's disease Eczema (including actinic dermatitis and photo contact dermatitis Psoriasis Lichen planus.

These reactions are either a direct toxic effect of light, or have an immunological component, either provoked by light alone or in conjunction with something else such as a drug.

Diagnosis and treatment of common causes of photosensitivity

The acute effects of sun on the skin are all too familiar. They are caused largely by medium wavelength UV radiation (UVB), but the “ dose” required producing sunburn depends on:

(1)An individual’s skin type

(2)The intensity of the radiation (greatest near the equator and around midday)

(3)The length of exposure to UVB

Mild sunburn causes erythema: more severe damage leads to extensive blistering and epidermal loss. Treatment makes little difference to the acute changes, but symptomatic relief can be obtained with soothing lotions, such as calamine. These include avoiding the midday sun, seeking shade, wearing appropriate clothing and eyewear, and using sunscreens, this is more important for those with skin type I and II than for those with a more radiation skin.

There are several special examples which are listed and explained:

(1) Porphyria:

Some forms of porphyria are associated with photosensitivity. In a European child the most common is erythropoietic protoporphyria, whereas an adult presenting for the first time probably has porphyria cutanea tarda. The latter is often associated with alcoholic liver disease. Screening tests involve

blood , urine and stool samples and are best undertaken in a specialist setting.

(2)Solar Urticaria: Rarely, exposure to light leads to urticarial weals.

(3)Pellagra:

In western societies, nicotinic acid deficiency is seen most commonly in alcoholics. It presents a triad of changes: Diarrhoea Dementia Dermatitis, which is light sensitive.

(4)Polymorphic light eruption

This is perhaps the most important, and certainly the commonest of the primary photosensitivity disorders. Patients often refer to their skin changes as” prickly heat”, but true prickly heat (or miliaria rubra)is quite different.

Polymorphic light eruption presents a day or two after sun exposure, with changes on light exposed areas, for example the forearms, legs the “ V” of the neck and the face. The lesions are itchy and morphologically variable (hence “ polymorphic”). There may be papules, plaques, and blisters of areas resembling eczema. They increase in intensity over a week or so before subsiding.

Treatment with topical steroids provides some relief, but some patients require systemic steroids to control an acute attack. Prevention is a better approach. Unfortunately, sunscreens are often not effective, but pre-season PUVA works well and can last for a whole summer. An alternative is the use of antimalarial medication (notably hydroxychloroquine) taken during sunny

periods, or while abroad. A variety of polymorphic light eruption occurs almost exclusively in boys. Clusters of small blisters appear on the tops of the ears, especially in early spring. The condition settles spontaneously with age.

Clinics in Photodermatosis

Actinic Prurigo

Actinic prurigo (AP) is a dermatosis that belongs to the group of idiopathic photodermatoses. Many names have been given to it, such as: solar dermatitis, Guatemalan cutaneous syndrome, solar prurigo, light-sensitive eruption in American Indians, familial actinic prurigo, polymorphous light eruption, (prurigo type) solar prurigo of high plateaus, and hereditary polymorphic light eruption of American Indians; however, it is the term “ actinic prurigo,” coined by Londono¹¹ in 1968, which is preferred and used by most authors today.

Actinic prurigo is a chronic photodermatosis that has frequently been confused with polymorphic light eruption (PLE). However, there are now enough clinical, histologic, epidemiological, and immunogenetic data to suggest that they are two different diseases.

AP begins in the first decade of life usually around ages 4 to 5, affects females more than males (ratio 2: 1). The lesions are symmetrical in the sun-exposed areas of the face (eyebrows, dorsum of the nose, malar regions, upper and lower lips), V-area of the neck, dor-sum of hands and forearms. The primary lesions are erythematous papules although excoriations, crusts, and lichenified plaques are commonly seen. Pruritus is a rule and usually

very intense. One of the clinical features which distinguish this disease from PLE is the absence of vesicles as primary lesions in AP. Of course, whenever a secondary dermatosis such as eczema, impetigo, or contact dermatitis ensues, vesicles may be seen. Two other differences between AP and PLE are lip and conjunctival affection in AP. Cheilitis of actinic prurigo affects 84% of patients. Although lesions are usually seen in both lips, the lower one being more exposed to sun rays is affected first and more intensely; severe cases show edema, crusts, fissures, shallow ulcerations, and hyperpigmentation, while in mild cases only dry lips with scaling may be found.

Atopic dermatitis with photosensitivity where the key findings are a familial incidence, an early infancy onset, the presence of xerosis, the sparing of the tip of the nose, and a good response to topical cortico-steroids and emollients.

Chronic actinic dermatitis which is quite infrequent, starts much later in life has a reduced UVB minimal erythema dose induction, and/or positive photopatch testing. Persistent light reactors and actinic reticuloid show histologically dense lymphocytic infiltrates, which especially in the latter may resemble true lymphomas. [5]

Phytophotodermatitis

Phytophotodermatitis is a very common skin disease in some countries. It is an acute phototoxic reaction where a substance containing psoralens comes in contact with the skin which is then exposed to UVA light. The clinical picture is that of sunburn ranging from mild erythema to severe blistering; it is usually accompanied by stinging or burning sensations. A residual

hyperpigmented macule is the final stage of the disease and usually persists for weeks to months; some dark-skinned individuals (skin types IV to VI) may only present with this hyperpigmentation without any previous signs or symptoms of sunburn.

The most common phototoxic compounds are the furocoumarins which contained in a wide variety of plants, especially of the Umbelliferae, Rutaceae, and Moracea families. Commonly occurring photosensitizing plants include citrus fruits such as limes and oranges, figs, and many vegetables such as celery, parsnip, parsley, carrots, and dill³²; furocoumarins in lime pulp are 13 to 182 times less concentrated than those in the peel.

Treatment is only symptomatic. Topical corticoste-roids help alleviate the burning sensations but do not seem to improve skin healing. Sunscreen use helps the resolution of the hyperpigmentation, and is the most important prophylactic measure for high-risk people.

Melasma

Melasma (chloasma) is characterized by light or dark brown hyperchromic macules with undefined borders, affecting mainly and symmetrically, the sunlight-exposed areas of the face. Melasma predominates in women, although it also affects men and has been related to sunlight exposure as well as hormonal, racial, and hereditary factors. Melanocytes are well known to be stimulated by estrogen and other sex-related hormones. Pregnancy and the ingestion of oral contraceptives can produce or exacerbate melasma, with an increase of melanogenesis and the presence of large

melanocytes. Facial melano-cytes appear to be especially sensitive to hormonal influences but sunlight is always necessary for melasma to occur.

Melasma has been reported to have a greater incidence in tan or dark-skinned persons, especially in skin types III, IV, and V, but also depends, as stated before, on sunlight exposure (including UVA and visible light).

Piquero-Martin has observed that pigment disposition (as seen with wood's light), tends to occur more superficially in lighter skins and deeper in darker skins, the latter being more difficult to treat and eradicate.

In some dark-skinned patients with clinically evident melasma, under woods light, the hyperpigmentation disappears. Apparently, this can be related to melanic pigment located deep in the dermis.

Differential diagnoses include Rhiel melanosis, Ashy dermatosis, Addison disease, pigmented lichen planus, and postinflammatory hyperpigmentation.

Many treatment modalities have been used, such as mercury compounds, vitamin C, tretinoin, topical steroids, glycolic acid, azelaic acid, and hydroquinone.

Pytirisias Alba

Pytirisias alba is a chronic asymptomatic dermatosis of unknown etiology, characterized by hypochromic macules, 1 to 5 cm in diameter, covered by a very fine scale, with an ill-defined and occasionally hyperpigmented border. The macules are mainly located on the sun-exposed areas of the face (malar areas, forehead, around the nasal and moth orifices) and extremities.

The cause of this dermatosis is unknown, but many factors have been attributed to it. The main cause is sunlight exposure on a tan or dark skin, and it has been described as a reactive dermatosis to a distant staphylococcal or streptococcal upper respiratory tract infection.

The lesions have been attributed to postinflammatory hypopigmentation, with a decrease in melanosome number and size, low keratinocyte melanin uptake, and light refraction from a hyperkeratotic and parakeratotic stratum corneum. The histopathology shows an epidermis with hyperkeratosis and parakeratosis, acanthosis, and mild spongiosis. In the papillary dermis, vasodilation and mild perivascular inflammatory infiltrate and occasional pigment incontinence can be seen.

Sunlight radiation (UVA, UVB, and visible light) in atopic patients with tan or dark skin are the most commonly affected.

Treatment of pityriasis alba must include sunlight protection, sun filters, low potency corticosteroids with a topical antibiotic (mupirocin, vioform). If an upper respiratory tract infection is detected, specific treatment should be given.

Pellagra

Also named Gaspar Casal's disease, pellagra is a nutritional disorder due to nicotinic acid deficiency, a B complex vitamin. Niacin is a nonessential vitamin and can be synthesized from the amino acid tryptophan via the kynurenine pathway. It is mainly expressed on the skin, gastrointestinal and central nervous system. Pellagra is a bilateral and symmetrical dermatosis affecting sun-exposed areas.

The avitaminosis can be caused by drugs, such as isoniazid, 6-mercaptopurine, 5-fluouracil, and chloram-fenicol. Phenytoin and sodium valproate⁵¹ have also been related to nicotinamide deficiency.

Clinical findings in pellagra include dermatitis, diarrhea, and dementia (the “3Ds”).

Pellagra is characterized by a intense red, scaly and hyperpigmented plaques on areas exposed to sun, heat, friction, or pressure. The lesions can be edematous with a burning sensation and occasional vesicles and des-quamation.

In chronic lesions, the skin is thickened, hyperpig-mented with a dark brown hue.

A typical finding is Casal’s “ necklace,” a scaling collarette around the neck which extends down toward the sternum. Flexural fold may be macerated, and on seborrheic areas, follicular hyperkeratotic plugs are frequently present. Frequently, angular cheilitis, glossitis with papillary atrophy with a tender “ beefy” red tongue, and esophagitis are seen. Manifestations in ad-vanced disease cases are vomiting, diarrhea, and weight loss with secondary anemia or amenorrhea. Central nervous system symptoms include irritability, headaches, insomnia, amnesia, and anxiety. Later on, patients develop tremor, movement disorders, numbness, encephalopathy, paralysis, and psychosis.[5]

PDT in dermatology

Photodynamic therapy (PDT) harnesses the power of light and oxygen to enact biologic change. In its infancy, the use of PDT in the treatment of dermatologic disease was limited due to the prolonged and pronounced photosensitivity resulting from systemic photosensitizing agents. However, in the early 1990s Kennedy and Pottier described the use of topical 5-aminolevulinic acid (ALA) to create endogenous protoporphyrin IX (PpIX) from which came a limited, localized, photodynamic response. With this development, many of the early limitations of PDT were alleviated, and the treatment became much more convenient. Early application focused primarily on the treatment of dysplastic and neoplastic disease; however, during the past few years, the versatility of PDT has been more fully realized, and it is now also being used to treat a wide variety of inflammatory and infectious processes.

The effectiveness of PDT depends on the photosensitizer used, its ability to selectively penetrate diseased tissue, and the duration of application; the activating light source, its ability to penetrate to the desired target, and its duration of exposure; and the type of target cells and their oxygenation status. To be effective, the damage resulting from PDT must surpass cellular repair mechanisms, a feature referred to as the minimum photodynamic dose. [6]

Summary

According to the lecture the Professor Moseley gave us and the materials I get from relative books and papers. We can know that the photodermatology is a huge branch of skin disease. First I give the introduction and a review of <https://assignbuster.com/skin-diseases-affect-on-peoples-health/>

background and history, it can clear explain what is photodermatology especially photodermatosis in clinic use. Next I focus on Photosensitivity, PDT in dermatology and Clinics in Photodermatosis, which explain different parts of the whole photodermatology. In Clinics of photodermatosis, there are many kinds of photodermatosis listed and illustrated, including the principles, treatment and prevention. As new to dermatology, PDT is an effective therapy to cure dermatosis by penetrating diseased tissue.