DERMATOLOGICAL CONDITIONS

Skin is comprised of an outer layer called the epidermis and an inner layer, the dermis. The epidermis contains primarily keratin-producing cells which perform the major barrier function of skin. Melanocytes are another cell type found in the epidermis and they produce the pigment melanin which determines skin color. The dermis is made up of a predominantly connective tissue layer with a deeper fat layer. It contains the blood and lymphatic vessels, nerve endings, sweat and sebaceous glands, and hair follicles. Hair and nails are non-living appendages of skin and are composed of the protein keratin.

ASSESMENT OF DERMATOLOGICAL CONDITIONS

HISTORY

During the course of a thorough medical history and physical examination will already have performed much of the dermatologic evaluation. However, when a patient presents with a dermatologic complaint, or when unexplained abnormal skin findings are noted during a general examination, should proceed with a systematic, focused approach to the problem, as would be done for any other organ system. History taking may need to be extensive with regard to the course of skin lesions and possible inciting factors when the etiology is unclear. A complete dermatologic history should elicit following information:

1. General medical history

Does the patient have any chronic medical problems or recent illnesses which may suggest a systemic cause of the skin findings? \

II. History of allergies Inquire about known allergic reactions to medications, foods and topical agents (i.e., cosmetics, soaps) as well as about hay fever and asthma.

III. History of medications It is essential to note both systemic and topical medications, including prescription and over-the-counter products. Some medications are more likely to cause rashes than others, but drug reactions are common and almost any agent may be implicated. How long has the patient been using each medication? If any were recently discontinued, when? Although new medications (taken for day or weeks) are the most likely to cause drug reactions, even those taken continuously for years may cause reactions.

IV. Family history Are there any family members with a similar skin condition? If so , this may reflect a contagious etiology or a common exposure among household members, or may suggest the possibility of a hereditary condition.

V. Course of skin lesions Different rashes may follow a characteristic pattern in their appearance over time, a pattern which may be crucial to identifying an etiology.

A. When did the lesions appear?

They may have appeared acutely within the past couple of days or, conversely, may have been present for many years. If the latter, why has the patient sought medical evaluation at this time.

B. How has the lesion or rash changed?

In the case of a persistent single lesion, has its appearance recently changed in any way? For example, recent rapid growth, color change or bleeding should make suspect malignant transformation, while cancer would be a less likely concern in a lesion unchanged for a few or more years. If a more generalized rash is present, where did it start, and what was the pattern and time course of spread? A rash such as that of measles classically begins on the face and then spreads to the trunk and limbs. Did the lesions look different initially, or were there any noted previously which are not visible now? For instance, vesicles may have been present at first, but resolved by the time of examination.

C. Does the patient have any associated symptoms? Pruritus (itching) is a complaint characteristically associated with a number of disorders, such as hives and contact dermatitis. Pain is a less frequently reported symptom but may be severe, as with herpes zoster (shingles).

D. Is there a correlation between the onset of skin lesions and any particular event or exposure? In some cases the patient will already have noted a clear association, i.e., a rash which appeared after the ingestion of a medication. Often, however, may need to ask several questions to elicit such associations, as patients are unaware of the importance of various factors. For example, if the patient denies taking any medications, should ask specifically about such over-the-counter agents as aspirin, cold remedies and birth control pills. Patients frequently will not report these items when being questioned more off-handedly. If an allergic contact dermatitis seems likely, the list of possible exposures to consider may be extensive, including cosmetics, detergents, topical medications, and occupational and recreational exposures. In other situations, exposure to sunlight may be an inciting or exacerbating factor (more common in summer months); or a recent flu-like illness, overlooked by the patient, may lead to suspect a viral exanthem.

E. Has the patient used any systemic or topical agents with subsequent improvement in appearance of skin lesions or symptoms?

Improvement with a particular agent may suggest one diagnosis more than another.

EXAMINATION

A thorough dermatologic examination should include all visible body surfaces. Thus, should be evaluating not only the skin, hair and nails, but also the mucous membranes (mouth, eyes, nose, genitalia). An entire body examination is usually unnecessary for a readily identified localized process, but many findings may be missed if do not actively search beyond the most apparent pathology, or that which the patient points out. The examination is best performed in a room with bright overhead light. should also have a flashlight at hand; a ruler and magnifying glass are frequently necessary. The patient should disrobe completely except for a gown. The following outline provides a systematic approach to use each time perform a skin examination:

I. Note the overall appearance of the patient and skin. Is the patient healthy or ill-appearing? Is there an abnormal color to the skin? (i.e., paleness in anemia, yellow hue in jaundice, blue in cyanosis) Do see a generalized process involving a large part of the body surface?

II. Inspect each part of the body. Do so in the same order each time, as would in a general physical examination. A head-to-toe approach is suggested here.

A. Scalp and hair. If the patient has a full head of hair, may need to part it in several areas to see the scalp well. Hair bands should be removed so that the hair is loose. In evaluating hair, especially note any areas of alopecia (hair loss) or thinning.

B. Face, including eyes, nose, and mouth. In examining the eyes, have the patient look in all directions to reveal conjunctival lesions. In the nose, examine the outer nostrils and the nasal septum (using a flashlight). Mouth findings are often subtle and should always use a flashlight here. Have the patient move the tongue to either side so that the inner cheeks can be visualized; also inspect the palate and all surfaces of the tongue.

C. Ears and neck. Inspect the outer ear, the external ear canal and behind the ears. After examining the neck, feel for any enlarged lymph nodes locally, as well as elsewhere.

D. Chest and abdomen. Remember to inspect the axillae and under the breasts, and in skin folds of obese patients.

E. Back and buttocks. Also examine the intergluteal cleft and perianal region.

F. Arms, hands, fingernails. Inspect the sides of the fingers and web spaces, and distinguish lesions on the dorsum of the hand from those on the palms. The presence of palmar, usually along with plantar (sole) lesions is characteristic of certain diseases. When examining nails also look at the surrounding (periungual) area and the cuticles.

G. Legs, feet, toenails. The groin folds should be inspected at this time. Evaluate the feet in the same manner as the hands, including interdigital areas and the soles.

H. Genitalia. Inspect the pubic area and labia in women, and the pubic area, scrotum and penis in men. An uncircumcised male should retract the foreskin.

III. Description

Lesions found in each of the above areas should be characterized carefully. observations and descriptions are crucial here. A magnifying glass may be helpful, especially for smaller lesions. should note all the following characteristics of the lesions upon inspection:

A. Number and distribution. There may be one or more lesions in a localized

area, or numerous in several areas. The distribution often suggests an etiology. For example, a systemic process such as a viral illness or drug ingestion would be more likely to cause a generalized eruption than a localized one. An eruption confined to one dermatome (the cutaneous distribution of a single spinal nerve root) is classic for zoster. Lesions on sun exposed areas, such as the face, backs of the hands and upper chest suggest photosensitivity (sun-induced) reaction

Arrangement or configuration. Do the lesions form a pattern which can be described? For instance, an involved area with well-demarcated linear borders often suggest a contact dermatitis. Other configurations may be labeled annular (circular) or serpiginous (wavy). Often, the arrangement is random, forming no particular pattern.

C. Size of individual lesions. If several lesions are present, they may be similar in size, or there may be a range of sizes. If the latter, should indicate the range (i.e. 5-15 millimeters).

D. Color. The following terms are most commonly used in describing colors of skin:

1. Flesh-colored – no change from surrounding normal skin.
2. Hypopigmented – lighter in color than surrounding skin.
3. Hyperpigmented – darker in color than surrounding skin. Hyperpigmented lesions are usually various shades of brown from the pigment melanin.
4. Erythematous – red. Different lesions may vary from pale to very bright to deep red in color, and this should be indicated in description.
5. Violaceous – purple.

Skin lesions may also come in many other colors, including white, pink, blue, black, yellow and gray. Noting uniformity (or non-uniformity) of color is important. A lesion may have an erythematous ring around the periphery with a flesh colored center. Another lesion may be a uniform light brown or it may be unevenly colored shades of brown; both could be rightly labeled hyperpigmented, but a more precise description is necessary to distinguish the two.

E. Shape. Individual lesions are most commonly round or oval, but may also be linear or other shapes. Next inspect the borders. Are they well-demarcated or indistinct, even or jagged? Also note the three-dimensional shape. A lesion may be raised above the level of the skin, even with it, or depressed below the surface; or it may be a combination, such as with raised edges and a

central ulceration. Some common shapes of raised lesions are dome-shaped, flat-topped and filiform (finger-like). At the skin surface a lesion may also be broad-based or pedunculated (on a stalk).

F. Surface characteristics. Examples are smooth, rough, shiny, dull, waxy and verrucous (warty).

PALPATION

The final step is palpation. Carefully run index finger over the lesion and note the texture of the surface (i.e. rough or smooth). At times may only be able to distinguish a slightly raised lesion from a flat one by careful palpation with eyes closed. Next determine the consistency by pressing on the lesion and then palpating it between fingers. Terms used to describe consistency include rock-hard, firm, rubbery, fluctuant and soft. Also note how far the lesion extends below the skin surface by feel. If a lesion is completely below the surface, is it fixed in place (thus attached to the epidermis and/or dermis) or freely moveable (thus completely underlying the skin)? Lastly, subtle changes in the feel of the skin surrounding a lesion (i.e. an apparently small malignancy to the eye) may indicate more extensive involvement of the area, and may have important therapeutic implications.

Terminology

I. Primary lesions = lesions which have not been altered by external forces or time

A. Flat, non-palpable lesions (demarcated only by color change from the surrounding skin; cannot be distinguished by touch)

1. Macule – smaller than 2 cm. In diameter

2. Patch – larger than 2 cm.

B. Solid, palpable lesions

1. Papule – smaller than 1 cm., elevated above the skin surface.

2. Plaque – larger than 1 cm., flat-topped and elevated above the skin surface. Plaques may be very large, covering extensive areas. Smaller plaques may be composed of grouped, confluent papules.

3. Nodule – Usually spherical, a nodule may be palpated deeper than a papule or plaque and may be below the skin surface.

4. Wheal – A papule or plaque which is formed by edema in the skin, and which typically disappears after a short time.

II. Fluid-filled lesions \

1. Vesicle – smaller than 1 cm and raised above the skin surface; a vesicle usually contains a clear serous fluid, but may also contain blood. Vesicles are commonly called blisters.

2. Bulla – Larger than 1 cm with the same contents as a vesicle.

3. Cyst – A firm-walled lesion usually containing a semisolid material. A cyst may be distinguished from a nodule by its softer, more rubbery feel.

4. Pustule – Small lesion raised above the skin which contains purulent (opaque) material (pus). Lesions which contain pus and that are larger and extend deeper are called in order of the increasing size, furuncles, carbuncles (made up of multiple furuncles) and abscesses.

III. Depressed lesions

1. Erosion – An area of skin loss, usually with a moist, erythematous

base. An erosion is fairly superficial and does not extend below the epidermis.

2. Ulcer – An area of skin loss extending into the dermis or deeper.

3. Fissure – A linear erosion or ulcer.

IV. Secondary lesions – These occur as the result of change in primary lesions over time, or from exogenous manipulation of the skin.

1. Scale – White flakes from the top of the epidermal layer (stratum
corneum) which are retained on the skin surface.

2. Crust (scab or eschar) – A solid, brownish covering over a lesion which is composed of old dried serum, blood or exudate.

3. Erosion – As described above. An erosion may be secondary if it results from the rupture of a vesicle or bulla. An excoriation is erosion created by scratching and is usually linear or angular.

4. Lichenification – Thickening of the skin with accentuation of skin lines. Lichenification results from repeated rubbing and scratching

PSOARIASIS

Definition

Psoriasis is a noncontagious, chronic inflammatory disease of the skin characterized by clearly defined dry, rounded red patches with silvery white scales on the surface.

Aetiology

Age: Common age of first occurrence is 15-30 years. It can occur as ng as 2 years. Also it can start as late as 80 years.

Sex: Both sexes are equally affected.

Climate: The condition is worse in damp, cold climates. It has been known to clear if a patient who suffers quite badly in the UK goes to a sunny climate.

Predisposing / precipitating factors

A number of factors appear to predispose or precipitate an exacerbation of the psoriasis. These are:

Heredity: There is an inherited defect in the skin which results in psoriasis developing in certain circumstances; 30 percent of patients have blood relative with the condition.

Infection: Psoriasis has been known to develop after, for example, an upper respiratory tract infection.

Trauma: Lesions tend to develop at sites of potential or actual trauma, e.g. mechanical friction, cuts, stings etc.

Anxiety: Psoriasis often appears in relation to mental stress, e.g. bereavement, examinations etc.

Drugs: Some drugs, e.g. chloroquine, may precipitate the condition.

Diabetes: Some patients with diabetes develop the condition. Arthropathy: Sero-negative arthritis develops in some patients.

Causes

Lesions of psoriasis are caused by an increase in the turnover rate of dermal cells from the normal 23 days to 3-5 days in affected areas. Silver scale on the surface of lesions is a layer of dead skin cells and may be scraped away from most lesions even if the scale is not apparent on visual inspection. Patients with psoriasis have a genetic predisposition for the disease. Perceived stress can cause exacerbation of psoriasis. Autoimmune function - significant evidence is accumulating that psoriasis is an autoimmune disease. Lesions of psoriasis are associated with increased activity of T cells in underlying skin. Guttate psoriasis has been recognized to appear following certain immunologically active events, such as streptococcal pharyngitis, cessation of steroid therapy, and use of antimalarial drugs.

Pathological changes :

Epidermis:

There is increased reproduction in the stratum germinativum. The stratum spinosum is thicker due to an increased number of cells plus edema. The stratum granulosum is absent.

The strata lucidum and corneum are replaced by several layers of nucleated, incompletely keratinized, soft cells (para-keratotic cells).

There is no time for the normal changes to take place through the skin layers. The cells at the surface are sticky and do not fall off like normal keratin.

Accumulation of these cells forms scales, which over 2-3 weeks dry out and fall off in big flakes.

Dermis

The capillaries are dilated with increased blood flow. The papillae are elongated and there are chnges of inflammation.

Healing

The center of the patch heals first causing circular lesions. Normal skin recovery takes place without scarring.

Clinical features:

Sharply defined red and pink areas are termed as plaques. \

Scales look silvery due to light reflecting from the swollen stratum spinosum.

Distribution: Elbows, knees, scalp and sacrum are covered in thickly scaled patches. Plaques of varying sizes appear any where on the body.

Nail become pitted, ridged or separated from the nail bed. This can be the only evidence of the disorder in some people.

Skin contact areas can be badly affected- between fingers, axillae, groin, between toes, under breast and behind ears. The face is rarely affected.

The lesions are usually symmetrically distributed and are characteristically located on the ears, elbows, knees, umbilicus, gluteal cleft and genitalia. The joints (psoriatic arthritis), nails and scalp may also be affected. :

- The size of plaques and distribution varies so that different types are described. They are:

Plaque psoriasis is characterized by raised inflamed lesions covered with a silvery white scale. The scale may be scraped away to reveal inflamed skin beneath. This is most common on the extensor surfaces of the knees, elbows, scalp, and trunk.

Guttate psoriasis presents as small red dots of psoriasis that usually appear on the trunk, arms, and legs; the lesions may have some scale. It frequently appears suddenly after an upper respiratory infection (URI). This type responds well to UVR. Inverse psoriasis occurs on the flexural surfaces, armpit, groin, under the breast, and in the skin folds and is characterized by smooth, inflamed lesions without scaling.

Pustular psoriasis presents as sterile pustules appearing on the hands and feet or, at times, diffusely, and may cycle through erythema, pustules, and scaling. UVR has limited success in this type.

Erythrodermic psoriasis presents as generalized erythema, pain, itching, and fine scaling. This type does not usually respond to UVR. Scalp psoriasis affects approximately 50% of patients, presenting as erythematous raised plaques with silvery white scales on the scalp. Nail psoriasis may cause pits on the nails, which may develop yellowish color and become thickened. Nails may separate from the nail bed.

Prognosis

Psoriasis clears completely with no marks but unfortunately can recur. There can be no sign in the evening and next morning it has started. It tends to be better in summer, worse in winter and recurs if the patient is worried. It has lifelong involvement, with waxing and waning, with progression to arthritis in about 10% of cases. It is usually benign. It may be may be refractory to treatment.

Treatment

This may be considered in four headings: General Management Topical Applications Systemic Applications

Physiotherapy Management

General management A sympathetic, considerate approach is required together with reassurance. Any anxiety or worry should be identified and the patient encouraged to relax or seek appropriate help. Reassurance that it is not infectious or disfiguring must be given to both patient and family. Also open door system should operate so that the patient can get to a dermatologist or physiotherapist immediately there is an eruption. Dieting may be tried if there appears to be any allergy factor.

Topical Applications Many patients do well on topical treatment. Treatment may be: Simple bland aqueous cream. Coal tar applications with salicylic acid and zinc oxide in soft paraffin may be used alone or with UVR. The patient is usually admitted to hospital. The ointment is applied every day to the whole body except face and scalp. Every 24 hours it is washed off in a bath containing coal tar solution. If UVR is given, it must be after a bath because suberythema general treatment is given daily using the Theraktin. This is the Goeckerman regimen. Diathranol in Lassar paste is used for resistant psoriasis. It is highly effective but can burn the normal skin. The patient may be admitted to hospital or treated as an outpatient. If the patient is applying the physiotherapist should look out for blisters or reddish purple stains on the skin and warn the patient of the danger. UVR with the Theraktin may be given in conjunction with diathranol as a daily suberythema dose. The paste is removed in coal tar bath before the UVR and is then reapplied afterwards.

Corticosteroids cream produces good results at first but when treatment stops the diseases can return worse than before. It is useful in an acute eruption and on the face and hands because there is greater absorption in moist areas. The dangers of side effects make long-term use inadvisable.

Systemic Applications Retinoids- a variant of vitamin A- taken in tablets form produces marked improvement. Retinoic acid or etritinate is marketed as Tigason. Unfortunately, this produces unpleasant side effects such as dryness and cracking of the mouth, alopecia and pruritus. It is teratogenic (produces malfunction in a fetus), therefore must be avoided in pregnancy. Cytotoxic drugs such as methotrexate are sometimes used in severe cases. These have dangers such as

damage to bone marrow, intestinal and liver tissues. Cyclosporine also may be useful in severe cases.

Physiotherapy Management

Psoriasis can be treated very successfully with UVR. Two sources are used: the Theraktin and PUVA. The Theraktin This is usually in the form of a tunnel with four fluorescent tubes. The patient lies flat for the treatment, therefore in order to treat the whole body the patient is generally naked and lies supine for half the treatment session and prone for other half. The spectrum of UVR emitted is 390-280nm and peak emission is around 313nm,therefore this constitutes UVB treatment. It may be used alone or in conjunction with coal tar or diathranol.

Treatment A suberythema dose is given daily or three times a week. The prominent parts of the body have a mild erythema, which fades before the next treatment is due. The time is maintained to maintain the reaction (e.g. 12.5% every 1-2 treatments.). When the lesions start to flatten and heal the same time is repeated and frequency of treatment reduced to twice weekly, once weekly and then once a fortnight. The course of treatment may be spread over 8-12 weeks. These patients tend to deteriorate during the autumn and need treatment in the winter or spring. About 75% of patients with guttate psoriasis respond to UVB.

PUVA This is psoralen plus UVA and is used for resistant psoriasis. Psoralen is photosensitizing substance, which occurs in plants such as parsley, parsnips and celery. The one used for psoriasis is 8-methoxy psoralen (8-MOP). UVA is produced from fluorescent tubes, mounted upright in a hexagonal shaped cabinet inside which the patient stands throughout the treatment. The spectrum of UVR emitted is 330-390 nm and peaks at 360 nm. Infrared rays are also emitted and it is essential to have a cooling fan so that the patient can tolerate up to ½ hour in the cabinet.

Method The patient takes 3-6 tablets of psoralen preferably with milk 2 hours before exposure. Tablet dosage is according to body weight . UVA is Calculated according to skin type in joules . There is little erythema with UVA; therefore the skin type chart has to be used. (To produce an erythema with UVA requires a dosage 1000 times greater than UVB.).

The dosage is recorded in Joules/cm2. An exposure meter is used to test the output and measures milliwatts/cm2; 1 mW/ cm2 = 1/1000 Joules/second.

Duration of treatment This may be 5 minutes at first for skin types I and II and progressed by 1 minute up to 15 minutes. It may start at 6 minutes and progress by 2 minutes up to 20 minutes

for skin type III and IV. It may start at 7 minutes and progress by 3 minutes up to 25 minutes for skin type V and VI. A record is kept of the total Joules count. This is essential because there is an undeniable risk of malignant melanoma in patients who have been exposed to between 1500 J and 2000 J. The patient attends three times a week until healing starts, and then frequency of treatment is reduced to twice weekly, once weekly, once per fortnight or monthly â€˜holding sessions.

Precautions/ dangers/ advice to patients on PUVA. Do not take psoralen on an empty stomach. There is a real danger of cataract; therefore protective goggles are essential during exposure. Polaroid sunglasses must be worn from the time of taking the psoralen to at least 12 hours after. The psoralen is excreted in 8 hours but the effect of photosensitizing continues. The physiotherapist should test the glasses with a Black ray meter; the glasses must screen 90% of UVA. Patients are advised to wear protective glasses out of doors for at least 24 hours after taking the psoralen and also whilst watching television, a VDU screen or in fluorescent lighting. The skin must be covered in bright sunlight and a hat worn for 24 hours after treatment. Stop using all ointments during PUVA. If the skin is dry simple oil or lubricating lotions may be used. Do not become pregnant or father a child- contraceptive measures are essential during PUVA treatment. A check up is essential every month after completing of treatment. During treatment if patient feels fainting; the physiotherapist must be called immediately.

ALOPECIA

Alopecia is defined as premature loss of hair. Some times leads to total loss of hair from the body.

Classification

- **Alopecia areata :** Loss of hair from scalp in patches.
- **Alopecia totalis :** Scalp hair loss along with eye brows.
- **Alopecia universalis :** Loss of hair all over the body.

Causes/ Etiology of alopecia areata

The condition is thought to be an autoimmune disorder in which the body attacks its own hair follicles and suppresses or stops hair growth. There is evidence that T cell lymphocytes cluster around these follicles, causing inflammation and subsequent hair loss. An unknown environmental trigger such as emotional stress or a pathogen is thought to combine with hereditary factors to cause the condition. There are a few recorded cases of babies being born with congenital alopecia areata; however, these are not cases of autoimmune disease because an infant is born without a fully developed immune system**.**

- **Age**: Generally affects under 30 years of age.
- **Predisposingfactors**:
  a) Poor health
  b) Heredity plays an important role
  c) Anxiety and fatigue.
- **Sex**: It affects both the sexes equally.

Pathological Changes

- Hair becomes weak from root and comes out of follicle.
- Atrophy of hair follicle occurs.
- Sebaceous glands becomes inactive or less active.

Clinical Features

- Insidious onset.
- Hair starts falling in clumps.
- White skin appears after the patches of hair falls.
- Baldness appears.

Prognosis

Growth of fine hair may be seen within two months. Majority of patients recover within a year. Sometimes patient may not recover. The new hair which appears can be pigmented, different from normal hair.

Physiotherapy Aims:

- To improve general health
- To improve nutrition to hair follicles

Means:

- To improve general health UVR treatment + Theraktin are given. Sub-erythema or doses of E1 are given for 5-8 minutes daily.
- Individual patches are treated by E2 and E3 doses of of UVR and Kromayer, twice a week.

Treatment should be continued for 2-3 months, and as the hair starts growing UVR must be stopped to that area. This should not be confused with balding. Sympathy and understanding is important in these cases.

VITILIGO

It is a chronic disorder that causes depigmentation patches in skin. It occurs when the melanocytes, the cells responsible for skin pigmentation, die or are unable to function. The term Vitiligo is probably derived from the latin word Vitilus - meaning calf and was first used by roman physician Celsus of 1st century AD,The characteristics white patches of disease resembled the white patches of a spotted calf in India. **Leukoderma** is a generic name for relatively or absolutely lightened in colour.

Incidence of Vitiligo

vitiligo disease affects 1-2% of the population and affects both males and females of all races. Vitiligo can begin at any age although about fifty percent of people who have Vitiligo developed it before they turned 25. Vitiligo is more noticeable to people with darker skin color.

Vitiligo Causes

The cause of vitiligo disease is not known, but doctors and researchers have several different theories. There is strong evidence that people with vitiligo inherit a group of three genes that make them susceptible to depigmentation. The most widely accepted view is that the depigmentation occurs because vitiligo is an autoimmune disease-a disease in which a person's immune system reacts against the body's own organs or tissues. As such, people's bodies produce proteins called cytokines that alter their pigment-producing cells and cause these cells to die. Another theory is that melanocytes destroy themselves. Finally, some people have reported that a single event such as sunburn or emotional distress triggered vitiligo; however, these events have not been scientifically proven as causes of vitiligo.

Vitiligo Symptoms

Vitiligo disease is characterised by the appearance of depigmented patches(milky white) on the skin, common in sun exposed areas like hands, feet, arms, face and lips. Other common areas include armpits,groin, around the mouth, eyes, nostrils, navel and genitals. Rarely the patches show slight erythema, but as a rule they show only depigmentation and sensivity to light, the hair may be white or black but in a particular lesion, when hairy areas are involved the hair may turn white.

Vitiligo generally appears in one of the three patterns:

- **Focal Pattern** : Depigmentation is limited to one or only few areas.
- **Segmental Pattern** : Depigmentation develops on only one side of the body.
- **Generalised Pattern** : Depigmentation develops on different parts of the body.

Diagnosis of Vitiligo Disease

Clinical Examination: Depigmented patch is usually the diagnostic features of Vitiligo disease. There may be a predisposing history of a rash/sunburn or trauma at the site of patch 2-3 months prior to the onset or History of auto-immune disease in the family. A biopsy of affected skin confirms Vitiligo.

Vitiligo Cure

Sometimes the best treatment for vitiligo is no treatment at all. In fair-skinned individuals, avoiding tanning of normal skin can make areas of vitiligo almost unnoticeable because the (no pigment) white skin, of vitiligo has no natural protection from sun. These areas are easily sunburned, and people with vitiligo have an increased risk to skin cancer. They should wear a sunscreen with a SPF of at least 30 should be used on all areas of vitiligo not covered by clothing. Avoid the sun when it is most intense to avoid burns.

Disguising vitiligo with make-up, self-tanning compounds or dyes is a safe, easy way to make it less noticeable. Waterproof cosmetics to match almost all skin colors are available. Stains that dye the skin can be used to color the white patches to more closely match normal skin color. These stains gradually wear off. Self-tanning compounds contain a chemical called **dihydroxyacetone** that does not need melanocytes to make the skin a tan color. The color from self-tanning creams also slowly wears off. None of these change the disease, but they can improve appearance. Micropigmentation tatooing of small areas may be helpful.

If sunscreens and cover-ups are not satisfactory, r doctor may recommend other treatment. Treatment can be aimed at returning normal pigment **(re-pigmentation)** or destroying remaining pigment **(depigmentation)**. None of the re-pigmentation methods are permanent cures.

Treatment of Vitiligo Disease in Children

Aggressive treatment is generally not used in children. Sunscreen and cover-up measures are usually the best treatments. Topical corticosteroids can also be used, but must be monitored. PUVA is usually not recommended until after age 12, and then the risks and benefits of this treatment must be carefully weighed.

Repigmentation Therapy

Topical Corticosteroids — Creams containing corticosteroid compounds can be effective in returning pigment to small areas of vitiligo disease. These can be used along with other treatments. These agents can thin the skin or even cause stretch marks in certain areas. They should be used under r dermatologist's care.

PUVA

PUVA is a form of repigmentation therapy where a type of medication known as psoralen is used. This chemical makes the skin very sensitive to light. Then the skin is treated with a special type of ultraviolet light call UVA. Sometimes, when vitiligo is limited to a few small areas, **psoralens** can be applied to the vitiligo areas before UVA treatments. Usually, however, psoralens are given in pill form. Treatment with PUVA has a 50 to 70% chance of returning color on the face, trunk, and upper arms and upper legs. Hands and feet respond very poorly. Usually at least a year of twice weekly treatments are required. PUVA must be given under close supervision by r dermatologist. Side effects of PUVA include sunburn-type reactions. When used long-term, freckling of the skin may result and there is an increased risk of skin cancer. Because psoralens also make the eyes more sensitive to light, UVA blocking eyeglasses must be worn from the time of exposure to psoralen until sunset that day to prevent an increased risk of cataracts. PUVA is not usually used in children under the age of 12, in pregnant or breast feeding women, or in individuals with certain medical conditions.

Narrow Band UVB (NBUVB)

This is a form of photo-therapy that requires the skin to be treated two, sometimes three, times a week for a few months. At this time this form of treatment is not widely available. It may be especially useful in treating children with vitiligo disease.

Read Questions and Answers about Vitiligo at National Institute of Arthritis and Musculoskeletal and Skin Diseases

Grafting

Transfer of skin from normal to white areas is useful for only a small group of vitiligo patients. It does not generally result in total return of pigment in treated areas.

Other Treatment Options

Other treatment options include a new topical class of drugs called **immunomodulators**. Due to their safety profile they may be useful in treating eyelids and children. **Excimer lasers** may be tried as well.

Depigmentation Therapy

For some patients with extensive involvement, the most practical treatment for vitiligo disease is to remove remaining pigment from normal skin and make the whole body an even white color. This is done with a chemical called **monobenzylether of hydroquinone** . This therapy takes about a year to complete. The pigment removal is permanent.