A CLINICAL STUDY OF GERIATRIC DERMATOSES

Dissertation Submitted to

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

APRIL - 2016

In Partial fulfilment of the regulations required for the award of

M.D. DEGREE

IN

DERMATOLOGY, VENEREOLOGY AND LEPROLOGY

BRANCH – XII

DEPARTMENT OF DERMATOLOGY

Coimbatore Medical College Hospital, Coimbatore

Declaration

DECLARATION

I, Dr. S. Anitha solemnly declare that the dissertation entitled "A clinical study of Geriatric dermatoses" was done by me in the Department of Dermatology and Venereology at Coimbatore Medical College Hospital during the period from August 2014 to July 2015 under the guidance & supervision of Dr.P.P.Ramasamy M.D.,D.D., Professor & Head of Department, Department of Dermatology and Venereology, Coimbatore Medical College Hospital, Coimbatore. The dissertation is submitted to Tamil Nadu Dr.MGR Medical University, Chennai towards the partial fulfilment of the requirement for the award of M.D., degree in Dermatology, Venereology and Leprology. I have not submitted this dissertation on any previous occasion to any university for the award of any degree.

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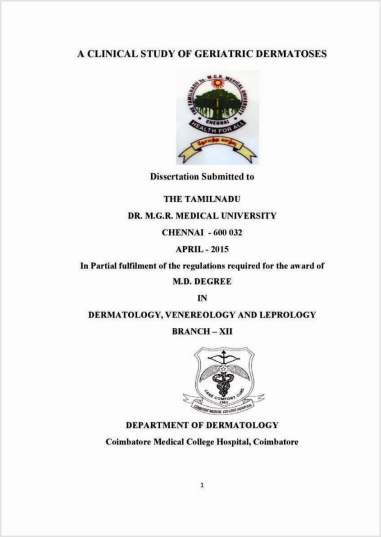

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ABBREVIATIONS

DNA	-	Deoxyribonucleic acid
ICAM-1	-	Intercellular adhesion molecule-1
UV	-	Ultraviolet
IL	-	Interleukins
VEGF	-	Vascular endothelial growth factor
NF κB	-	Nuclear factor kappa B
MMP	-	Matrix metalloproteinase
TNF – alfa	-	Tumour necrosis factor alfa
AP – 1	-	Activator protein – 1
RNA	-	Ribonucleic acid
TGF – β	-	Transforming growth factor – beta
LC	-	Langerhan's cell
HRT	-	Hormone replacement therapy
MRSA	-	Methicillin resistant staphylococcus aureus
HZ	-	Herpes zoster
HSV	-	Herpes simplex virus
KOH	-	Potassium hydroxide

SCC	-	Squamous cell carcinoma
HPV	-	Human papilloma virus
BCC	-	Basal cell carcinoma
5-FU	-	5- Fluorouracil
HIV	-	Human immunodeficiency virus
PUVA	-	Psoralen with ultraviolet A light
CO_2 LASER	-	Carbondioxide laser
Nd:YAG	-	Neodymium Yttrium Aluminium Garnet
IgG, IgA	-	Immunoglobulin G and A
LSEA	-	Lichen sclerosus et atrophicus

Introduction

INTRODUCTION

Ageing is a natural process. People aged 60 years and above are generally referred to as older population.[1] In India, there were 72 million elderly persons above 60 years of age as of 2001 and this number is likely to increase to 179 million in 2031 and further to 301 million in 2051, hence dermatologic care in geriatric population needs emphasis.[2]

Common skin disorders seen in the elderly are xerosis, pruritus, photoaging (dermatoheliosis), benign tumors like acrochodrens, seborrheic keratosis, cherry angioma and infections like herpes zoster, dermatophytosis, etc. Eczematous conditions like asteatotic eczema, stasis eczema, discoid eczema are common in elderly[3]. The dermatology practice of the future will see an increase in the number of geriatric patients and geriatric health care has become a major international issue.[4] In India, very few studies have been done to look into the cutaneous manifestations in the elderly people though several studies have been carried out in the west.[5] In this scenario, with life expectancy in India going up to 63.9 years in males and 66.9 years in females in 2004[6], this study was undertaken to study the spectrum of cutaneous manifestations and prevalence of physiological and pathological changes in the skin of elderly people.

Aims of the Study

AIM AND OBJECTIVES

AIM

To study the spectrum of various geriatric dermatoses among out patient population at Department of Dermatology, Venereology, Leprosy, Coimbatore medical college hospital , Coimbatore.

OBJECTIVES

- To describe the clinical pattern of various dermatological disorders in the elderly

- To know their incidence, factors contributing to it and their association with systemic disease.

Review of literature

REVIEW OF LITERATURE

Skin serves as a primary physical barrier between man and his environment.[7] It plays a major role in social and sexual interactions and it is a marker of systemic disease.[8] Ageing is the decline in the ability of an organism to maintain homeostasis in environment. This change in the ability to maintain the internal environment leads to a decrease in viability and an increase in vulnerability.[9] In addition hygeine, personal habits such as smoking and alcohol, socioeconomic status,nutrition, climate, color of skin, neurological or systemic diseases etc., contribute a role.[8]

Skin diseases are common and inevitable consequence of ageing. Moreover the clinical presentation is not as classical as they do in younger population. A lifetime solar exposure, along with intrinsic changes in the dermal structures predisposes to a variety of skin diseases.[9]

Cutaneous ageing includes 2 distinct entities:

1] Intrinsic ageing : it is an unavoidable change attributable to the passage of time alone . It has 2 categories : Those that occur within the tissues themselves , those that are the result of hormonal changes.

2]Photo ageing : it is the superimposition on intrinsic ageing with chronic sun exposure which are neither universal nor unavoidable.[10]

The ageing process :

Ageing occurs at all levels of biological organization , these are described in terms of morphological , physiological , biochemical , and molecular alterations . It is also obvious that ageing does not proceed at the same rate in all tissues.[7]

Important factor in ageing is functional decline in endocrine function . In women ovarian function will decline due to reduced secretion of ovarian hormones. This leads to marked changes in the bone metabolism that can be corrected by hormone replacement therapy . There is no overall change in the thyroid hormones but tissue utilization of thyroid hormone declines . There may be changes in metabolism of insulin which is reflected as altered glucose tolerance . Adrenal gland function is normal , no alteration in cortisol secretion and circadian rhythm is similar in both young and the old . Target tissue responses are significantly delayed . DNA synthesis and the cell division rate are reduced . With age there is reduction in the effectiveness of immune system and this is a reflection of changes in the thymus, other lymphoid organs.[7]

BIOLOGY OF AGEING

Ageing is a complex process that is due to progressive derangement of molecues over time.[11] Aging skin is particularly vulnerable to environmental and genetic factors.[12]

Intrinsic ageing

Genetic factors which control intrinsic ageing include:

a] telomere shortening

b] mutations in mitochondrial genome

d] the absolute length of the continuous cell cycle[13]

e] hormone systems

e] skin colour[12]

Gravitational forces or traction in skin can activate phospholipase A2, leading to generation of prostaglandins and leukotrienes. This results in cytokine release, neutrophil recruitment, free radical generation . Macrophage recruitment, cell movement out of blood vessels is facilitated by ICAM-1 synthesis duo to anoxia. In response to neurogenic inflammation, cutaneous neuropeptides regulate the expression of endothelial cells and cell surface adhesion molecules on immune cells. ICAM-1 expression is induced by non enzymatic glycosylation of blood vessels, a normal feature of ageing and accelerated diabetes mellitus. ICAM-1 is also ligand for lymphocyte function associated antigens. These triggers involve cyclical synthesis and expression of ICAM-1, leading to invasion of immune cells in the dermis which results in self sustaining inflammatory and ageing process.[11]

Photoageing

It is the aging caused by both direct and indirect effects of UV radiation. This results in damage to telomeres, generation of free-radicals and these effects are largely influenced by skin type.[14]

Generation of free radicals results in 50% of UV induced damage. Signs of photoageing like wrinkling, increased elastin and collagen damage are due to DNA damage induced by UV radiation.[14]

Damage to cellular DNA, lipids and proteins is caused by generation of free radicals is mainly by ultraviolet A, and to a lesser extent by ultraviolet B.[14] Marker for UVA damage which is found in dermal fibroblasts of photoaged skin is 4977 base pair deletion, it is found in mitochondrial DNA.The mitochondrion has the highest turnover of free-radicals in the cell and is responsible for cellular energy production. Mutations in its genome may be associated with the changes seen with photoageing because many of the genes involved in this process are encoded in mitochondrial DNA.[15]

Neutrophils increase oxidative damage through their production of free-radicals. This may be due to elevated levels of proinflammatory cytokines like IL-1, IL-6, vascular endothelial growth factor[VEGF] and tumour necrosis factor alfa. These are caused by transcription factor nuclear factor NF-κB which is activated by UV radiation. Amplification of the UV response is by attracting neutrophils that contain preformed neutrophil collagenase (MMP-8) and by stimulating the transcription of inflammatory cytokines. MMPs

expression is also increased by NF-κB. The process of angiogenesis, is assisted by reductions in thrombospondin-1, an angiogenesis inhibitor, and increases in platelet-derived endothelial cell growth factor, an angiogenesis activator and increased vascular endothelial growth factor(VEGF) levels. In elderly people photocarcinogenesis as well as telengiectasias in sun exposed areas are caused by UV induced changes in gene expression.

Epidermal growth factor, TNF-alfa and IL-1 receptors are activated in keratinocytes and fibroblasts within 15 minutes after UV exposure. This leads to the expression and also activation of the nuclear transcription factor activator protein 1(AP-1). Matrix metalloproteinases(MMPs) is the enzyme responsible for degradation of the extracellular matrix.This enzyme is regulated by transcription of AP-1. The major metalloproteinase responsible for collagen degradation is MMP-1which is found in both epidermal keratinocytes and dermal fibroblasts. It requires iron for its activation. The transcription factor NF-κB is also activated by UV radiation via an iron-dependent mechanism.[16] There is a dose-response relationship between UV exposure and MMP induction. Up-regulation of MMP can occur even with a minimal dose of UV, well below that required to produce erythema. These elevated levels of MMP can be maintained by minimal repetitive exposures to UV radiation, it may be equivalent to 5 to 15 minutes of exposure to midday sun every other day. Both local and systemic immunosuppression produced is by UV radiation. This is partly mediated by altered cytokine expression and also by DNA damage.[14]

Theories of ageing :

There are several theories about the aging process. These are :

a] Waste product theory : Most cells of the ageing show increased concentrations of lipofuscin [ageing pigment] particularly in heart and brain . Lipofuscin granule is heterogenous , irregular in shape and contains proteins, carbohydrates , lipids along with various lysosomal enzymes . It is caused by free radical induced damage .

b] Cross- linkage theory : there are several forms of collagen in mammalian skin such as Type I and Type III . Type I is found predominantly in adult , whereas Type III is found in higher concentrations in embryonic and young skin . There is also cross linking of DNA polymers with changes in the histone and non histone protein fractions . There is age related increase in the number and quantity of non – histone chromatin fractions in ageing tissues .

c] Free radical theory : Superoxide radical is a by-product of various enzyme reactions caused by UV light , ultrasound , X and gamma rays , toxic chemical and metal ions . Free radicals also cause lipid peroxidation . There are age related reductions in glutathione , glutathione reductase and superoxide dismutase from the blood cells , liver and eyes which are found to be protective against free radical induced damage .

d] Somatic mutation theory : Mutations in somatic cells have been implicated as the cause of ageing which is supported by the effect of ionizing radiation .

e] Errors in protein synthesis : Ageing cells are more prone to error during transcription due to insertion of incorrect aminoacids into proteins. If errors occur at the aminoacid residue there is only little effect on the cell, but if it takes place within the enzymes [various DNA and RNA polymerases] , the damage will be more serious.[7]

MORPHOLOGICAL ALTERATIONS IN THE AGEING SKIN

EPIDERMIS

Ageing epidermis shows slow recovery of barrier function in damaged stratum corneum.The most reliable histologic change is flattening of dermal epidermal junction with effacement of both the dermal papillae & epidermal rete peg.[12] This results in variability in epidermal thickness and individual keratinocytes size. There is decrease in epidermal fillagrin which results in dry and flaky skin especially over the lower limb.[10]

Increased susceptibility and fragility of the epidermis is due to atrophy of the stratum spinosum, thinning of the epidermis by 10%-50% and increased heterogeneity of size of basal cells.[17]

An endocrine function of human epidermis that declines with age is vitamin D production. There is decrease in DNA damage repair capacity and in addition antioxidant defense system decline with age. The epidermal repair

rate after wounding declines with age. Decrease in the number of enzymatically active melanocytes per unit surface area of skin, reducing the body's protective barrier against UV radiation. Increase in cellular mutability or tendency to become senescent is caused by combination of these changes.[10] There is widening of inter keratinocyte space in sun protected skin which is evidenced by electron microscopy. Also there may be reduplication of lamina densa and anchoring fibril in basement membrane zone.[12]

Dermis: Wrinkling of ageing skin is almost entirely the result of changes in dermis . The collagen per unit area of unexposed skin, bulk of the dermis decreases with age. This UV mediated down regulation of collagen synthesis is mediated by AP-1 and transforming growth factor beta (TGF-β). The elastic fibers are progressively reduced and the collagen bundles become disoriented and fragmented. There is disintegration of elastic fibres with age and after the age of 70 most fibres appear abnormal. These changes are due to reduced synthesis and elastolysis.[14]

Although substances enter aged skin more easily than young skin, they are removed more slowly into the circulation because of changes in the dermal matrix and reduction in the vasculature, resulting in alteration in permeability of skin.[18]

Increased biosynthetic activity is reflected by highly activated endoplasmic reticulum, fibroblasts become stellate shaped. There is steady increase in number of mast cells, mononuclear cell and neutrophils. In papillary

dermis there is loss of collagen I and III with an associated increase in matrix metalloproteins after the age of 70 years. The increased levels of mitogen activated protein kinases in aged skin produces oxidative damage. Reactive oxygen species and free radicals are the key drivers for degeneration.[12]

Eccrine glands: As a result of a combination of reduction in the number of glands and its output, spontaneous sweating on the fingertips declines in old age.[19] This leads to increase in core and skin temperature in elderly individuals. There is an age associated structural alteration in eccrine gland.[17] The impaired response of sweat glands to central and peripheral stimuli is due to diminished thermoregulatory ability in elderly.

Apocrine gland: Less odour is produced due to decrease in size as well as function of the apocrine gland.[10]

Sebaceous glands: Production of sebum is at greatest in early adulthood and gradually lessens in old age. Sebaceous glands increase in size inspite of their decreased output because turnover of sebaceous cells is slower in senility.[10] This along with diminished sweat gland output probably contributes to xerosis.

Nerves and sensation: Pacinian and Meissner's corpuscles decrease to l/3rd of their density.[20] Structural irregularities and variation in size is seen. Ageing often decreases sensory perception and increases threshold for pain.[21] Consequently, the elderly are predisposed to injuries such as thermal burns. These alterations have negative impact on the ability of the elderly to perform delicate maneuvers, compromising their ability to compete in the workplace.[1]

Hair: Hair has no vital function in humans, however its psychological functions are extremely important.[21] By the end of 5th decade, approximately half of the population has 50% gray hair and virtually everyone has some degree of graying due to deficient tyrosinase or loss of melanocytes from hair bulb.[12] With ageing the scalp hair becomes gray, finer and also the hair follicles decrease in number. On the scalp, the temples usually show graying first, followed by a wave of greyness spreading to the crown and later to the occipital area.

Loss of hair pigmentation is associated with a decrease and eventual cessation of tyrosinase activity in the lower bulb. Melanocytes are absent from the bulbs of white hairs although non-melanized melanocytes are still present in the outer root sheat.[22] Decrease in hair follicle density is due to atrophy and fibrosis. There in an increase in proportion of telogen hair follicles.

As people age, baldness occurs to some extent in all males and some females. It differs from androgenetic baldness in that it is characterized by modest reduction in size of the hair follicles. There is conversion of terminal hair to vellous hair with reduction in terminal to vellous hair ratio. Hair diameter, hair length and the percentage of hairs in the anagen phase is reduced.[23,24] Post-menopausal women hair loss is due to decreased estrogen-androgen ratio. In some facial areas, specifically ears and nose in males and upper lip, chin of females, small light colored vellous hairs are slowly

converted into larger, dark colored terminal hairs, resulting in considerable cosmetic compromise for the elderly.[7]

Nail

The elderly people show various age related changes and disorders affecting their nails.[25] Surface is lustreless, nail plate becomes thinner and is more prone to fracture. Lunula disappears, longitudinal ridging becomes evident. Nails are more brittle in elderly and are characterized by beaded ridging, called 'sausage links'. Brittleness is caused by reduction in lipophilic sterols and free fatty acids.[26] Cumulative effects of trauma and mechanical distortion results in thickened nails and onychomycosis. Nails appear dull and its colour varies from shades of yellow to opaque. White nails in elderly is termed as Neapolitan nails since it resembles the three bands of Neapolitan ice cream. Other changes are decrease in the rate of linear nail growth by approximately 50%, variation in contour of nails like platonychia and koilonychias. Transverse leukonychia and frictional longitudinal melanonychia are due to repeated trauma. There is age related longitudinal fissuring and splitting into layers. Common toe nail deformities are thickened toe nail and chronic fungal infection requiring therapy for longer duration with increase the risk of developing drug related adverse effect. Other changes are onychogryphosis, subungual corn (heloma), age related ingrowing toe nail. Myxoid pseudocyst is the commonest age related benign tumor.[27]

Wound Healing

Wound healing is retarded in the elderly. Epidermal turnover time is decreased compared with the young. Disorders with delayed wound healing like diabetes mellitus, peripheral vascular disease, hypoproteinemia are common among elderly. Dysregulated expression of MMPs and elevated proteinase activity are involved in disturbed wound healing.[17] Consequently re-epithelialisation of skin following trauma or dermabrasion takes nearly twice as long in the elderly. Reduced estrogen levels observed in postmenopausal women have been associated with disturbed tissue regeneration through impaired cytokine transduction, altered protein balance and uncontrolled inflammation.[28]

Angiogenesis

An age associated marked loss of dermal vessel density, and surface area have been described.

This explains why aged skin is characterized by, reduced UV induced erythema, decreased skin temperature, reduction of cutaneous vascular responsiveness and reduced nutrient supply which leads to ulceration.[17]

Vitamin D synthesis

The concentration of 7- dehydrocholesterol, showed 50% reduction from age 20 to 80. Factors that contribute to deficiency state are behavioral factors, including limited sun exposure and malnutrition.

Immune Function

Immunological decay occurs as people age in both the T cell and B cell mediated immunity, cell mediated immunity in particular undergoes significant deterioration.

The number of antigen presenting Langerhan's cells (LCs) significantly decreases from approximately 1200/mm^2 to 800/mm^2 in elderly with advancing age.

As a result the cells appear to be functionally impaired, which explains the diminished cutaneous immune function, increased propensity to infection [especially viral and fungal], and cancer in the elderly.

Function of human skin that decline with Age[29]

- Sebum Production
- Immune responsiveness
- DNA repair
- Vitamin D Production
- Thermoregulation
- Chemical Clearance
- Barrier Function
- Sensory perception
- Cell replacement
- Mechanical Protection
- Epidermal Hydration
- Sweat production

Major Histopathologic Changes in Normal Aged skin[30]

- ❖ Decreased interdigitations dermoepidermal junction
- ❖ Decreased vertical height of keratinocytes
- ❖ Increased surface area of corneocytes
- ❖ Decreased corneocyte adhesion
- ❖ Decreased number of melanocytes
- ❖ Decreased number of langerhans cells
- ❖ Decreased number of eccrine glands
- ❖ Decreased number and size of hair follicles
- ❖ Increased size of sebaceous glands
- ❖ Decreased papillary - capillary network
- ❖ Decreased dermal thickness
- ❖ Decreased subcutaneous tissue

There are many factors which contribute to the ageing process : These include menopause, alcohol intake and smoking.

Effects of menopause:

Menopause occurs around the age of 50 years. Estrogen derived from ovaries decreases since they become atrophic during menopause. After menopause most estrogen are derived from conversion of androstenedione of adrenals into estrone. The decrease of estrogen leads to atrophy of the vaginal epithelium. The pH of vagina increases and this leads to infections.[31] External genitals becomes atrophic due to loss of vuval subcutaneous fat. Pubic hair also

diminishes. Thinning of epidermis and dermis and loss of dermal elasticity may occur due to circulating estrogen levels.[32]

Menopausal flushing

It is described as a sudden feeling of intense heat in the face, neck and chest, often accompanied by discomfort and sweating. It typically lasts for 3to5 min. Some women develop palpitations, throbbing in the head and neck, headaches, waves of nausea and anxiety attacks.[33]

Estrogen therapy is most effective.[34] If it is contraindicated other drugs like progestins, clonidine, paroxetine, gabapentin, venlafaxine can be considered.[35]

Keratoderma climactericum

This term has been used to describe the appearance of tough skin on the palms and soles, especially around the heels. Formerly reported as a specific association with the menopause, the same changes are seen in men and women at other ages, many of whom are obese. It may therefore be a non-specific event. It may respond to systemic retinoids.[36]

Complications of HRT

HRT may be responsible for a number of cutaneous problems. Estrogen therapy may exacerbate, increased skin sensitivity, spider angioma, chloasma, darkening of naevi, the skin changes of porphyria cutanea tarda or lupus

erythematous, photosensitivity, pompholyx, erythema multiforme, urticaria and acanthosis nigricans.[37]

Role of Smoking in Aging skin

Smoking is associated with decreased water content in the stratum corneum and accelerated hydroxylation of estradiol leading to decreased estrogen in skin, which in turn contributes to dryness and atrophy. Smokers have increased incidence of skin cancer and severe photo aging like changes.[38]

But solar elastosis is restricted in the papillary dermis, in smoker's skin it occurs in both papillary as well as reticular dermis. This dermal elastosis is a result of increased elastase activity in neutrophils, chronic dermal ischaemia, decreased level of vitamin A, which reduce the capacity to quench free radicals and increase DNA damage.[39]

SKIN PROBLEMS IN THE ELDERLY

SENILE PRURITUS :

Also known as Willan's itch, is described by Robert Willan[1757-1812]. It is defined as an unpleasant sensation that leads to intensive scratching. Pruritus in aged is most commonly observed event.[40] The term senile pruritus or Willan's itch is most commonly due to unknown etiology.[41] It may be generalized or localised, with or without skin changes.

It is mandatory to look for primary cutaneous disease and systemic causes. In majority of cases it is due to xerosis. Infestations like scabies are particularly liable to miss. Bullous pemphigoid may present with non-specific (or) even no rash. In anogenital itching many of the skin changes may be concealed. Candidiasis in undiagnosed diabetes and classical sign of lichen scleroses like wrinkling, follicular indwelling could be altered by excoriation and inflammation. [42]

It is necessary to rule out systemic causes like renal failure, thyroid disorders, hepatic disorder, metabolic disorders like diabetes mellitus. It could be secondary to malignant neoplasm like lymphoma, leukaemia, haematological diseases like polycythemia vera. Adverse drug reactions can manifest as pruritus.[43]

Pathogenesis :

It is unclear. A neural mechanism was proposed by Kaposi, a subclinical neuropathy with increased threshold of touch and pain lead to central disinhibition of itch. Presence of anti basement membrane zone antibodies suggested that there may be an involvement of immunological mechanism.

Treatment: It includes frequent use of emollients. If no relief is obtained, then gabapentin, serotonin antagonists, UVB phototherapy may be given. Physical methods such as cutaneous field stimulation and acupuncture can attenuate pruritus in some patients. Patients are educated to avoid of excessive cold

exposure and frequent washing with soap .They are instructed to wear fully covered cloths.

Wrinkling :

The aged skin is very fragile, translucent, wrinkled and easily bruises. It is the commonest sign of ageing, defined as crease (or) furrows in the skin surface. They are also seen in congenital and acquired skin disorders. Histologically, there is epidermal thinning, decrease in chondroitin sulphate and deposition of abnormal elastic tissue in the papillary dermis. [44]

Wrinkling can be classified into three types morphologically. [45]

Crinkles :

This is a very fine wrinkling which occurs even in areas protected from sunlight, which may disappear when the skin is slightly stretched. They are caused by deterioration of vertical subepidermal fine elastic fibers.[46] Elastic fibres begin to deteriorate from the age of 30 onwards regardless of sun exposure.[47] Marked form of crinkles seen in mid-dermal elastolysis.

Glyphic Wrinkles

These are accentuated skin markings and occur in skin damaged by sunlight producing elastic degeneration. It is caused by elastotic degeneration caused by sunlight. It is present on sides and back of neck.

Linear Furrows:

These are long, straight and slightly curved grooves seen on the faces of elderly people. They include horizontal frown lines along the forehead, "Crows Feet" radiating from the lateral canthus of the eye, creases from the nose to the corners of the mouth.

In youth the linear furrows caused by facial muscle contraction disappear due to elastic recoil, but in older people they are permanent.[45]

Cigarette smoking is a potent independent cause, cigarette face is described as pale, grey, wrinkled skin seen in heavy smokers who are five times more likely to be wrinkled. [48]

Pathogenesis is not very clear, but causative factors might include ischaemia due to vasoconstriction induced by nicotine, sympathetic nerve stimulation, increased platelet aggregation, decreased prostacyclin formation, decreased tissue oxygenation, increased tissue carboxy hemoglobin and reduced collagen deposition. The additive induction of MMP- 1 expression which causes wrinkling may be induced by both UVA and tobacco smoke.[49]

Senile xerosis and asteatotic eczema :

It is otherwise known as 'eczema craquele'. Skin of the aged feels dry to touch which may be due to the reduced water content of the stratum corneum without actual water loss.[50] It is worse in the winter known as winter eczema prurigo (or) pruritus hiemalis. The texture of the skin assumes a cracked

appearance resembling crazy paving (i.e. short vertical fissures connect the horizontal fissures) which is most pronounced on the legs. Frequent washing, central heating in cold climates also aggravates the problem by reducing atmospheric humidity. It is necessary to rule out the use of diuretics. This condition is extremely pruritic, responds well to application of mid potency topical steroids, emollients , avoidance of excessive use of soaps and frequent washing. [51]

Eczematous disease of Geriatric population

The aged skin can suffer from any clinical type of eczema.

a) Stasis eczema

It is seen on the lower extremities and is associated with long standing venous insufficiency. It is characterized by bilateral circumferential dermatitis of ankle and calf areas. The triad of alopecia, waxy appearance and yellow brown pigment from haemosiderin deposition is diagnostic with or without edema. Elderly patients can have stasis dermatitis in other sites, if they are not mobile, such as buttocks, heels, forearm or other sites that rest on solid surface which give rise to decreased blood flow and later develop into pressure ulcers.[52]

b) Nummular eczema or Discoid eczema :

This is much more common in elderly, characterized by coin shaped or oval lesions that appear anywhere in the body . This can be mistaken for tinea corporis, psoriasis, contact dermatitis or neoplasia. [52]

c) Seborrheic dermatitis

Flexural pattern is more often seen in these aged individuals who are confined to bed. This may be mistaken for intertrigo and flexural psoriasis. [53]

d) Atopic Eczema :

It may continue into old age (or) appears for the 1st time.

e**) Contact dermatitis of irritant or allegic origin :**

These are less common in elderly due to decreased occupational exposure and reduced immune reactivity that occurs with age.[48] resulting from local medications, such as aminoglycosides, lanolin, parabens, antihistamines and anaesthetics. Other sensitizers include rubber in gloves and shoes, plastics in hearing aids, spectacle frames, plants and hair dyes. [54]

Photoaging (Dermatoheliosis):

Photoaging changes are superimposed on usual intrinsic and chronological aging process of skin. Solar elastosis resulting from long term UV exposure is characterized by the accumulation of degenerative material,

staining positively with elastic tissue stains.Various entities described. These are:

1) Cutaneous nodular elastoidosis with cysts and comedones (Favre – Racouchot syndrome) : it is more common in men. Sites of predilection are upper lateral aspects of cheeks and periorbital areas. It presents as cystic nodules coalesce to form thickened yellowish plaque interrupted by numerous large comedones.[55] It is a manifestation of severe sun damage.

2) Stellate pseudoscars : In elderly patients with severe actinic elastosis extensor aspect of the forearm develops multiangulated hypopigmented lesions. These are usually preceded by hemorrhage.

3) Milian's citrine skin : It occurs in fair skinned individuals. Skin appears translucent due to epidermal atrophy along with solar elastosis. They are more prone for nonmelanomatous skin cancers especially squamous cell carcinoma.

4) Cutis rhomboidalis nuchae : It occurs on the back of neck as atypical rhomboid furrowed pattern. It occurs more commonly in sailors and farmers. It is a manifestation of severe sun damage.

5) Acrokeratoelastoidosis marginalis : it presents as a group of glistening papules in a linear arrangement along the margins of the hand.

6) Venous lakes : These are more common on the ears and lower lip, deep blue in color, 5 mm in diameter. It is the result of profound dermal elastosis resulting in poor stromal support, in an already weekened bulging blood vessel.[56]

Senile purpura (Actinic purpura, Bateman's purpura)

Purpura is defined as extravasation of blood into the skin and mucous membranes. It is a common finding in the elderly. It is due to loss of dermal connective tissue support resulting in increased fragility of dermal vessels. Lesions are most commonly seen over the dorsa of hands, forearms, following trivial trauma. The lesions dark red or purplish macules of varying sizes. It is diagnosed clinically by the sites of the lesions, associated atrophic changes, and by the absence of any general bleeding tendency. Primary amyloidosis is the rare cause of purpura in the elderly. It is due to deposition of amyloid material in and around dermal blood vessels. This is most marked in the body folds, especially around the eyes. Intake of aspirin, anticoagulants like warfarrin and heparin must be ruled out. It may resolve spontaneously after a few weeks. No treatment is needed for this benign disorder.[57]

Infectious diseases in elderly

A variety of infections including bacterial, viral, and fungal may occur commonly in the elderly population.

Bacterial infection :

Due to alteration in skin architecture and loss of barrier function both staphylococcus and streptoccal infections are seen frequently.[58] Impetigo, and folliculitis in elderly are caused by staphylococcus. It can be treated with penicillinase resistant semisynthetic penicillin or erythromycin. Distinct forms of cellulitis like orbital cellulitis or pseudomonas cellulitis of ear more common in elderly with diabetes. Erysipelas, a beta haemolytic streptococcal infection is more common in elderly and spreads more rapidly. Necrotising fasciitis is a rare cutaneous infection but is more frequent in elderly. Methicillin resistant staphylococcus aureus (MRSA) has become common in hospital and community acquired infections.

Viral Infection :

Because of decline in immunity as the age advances and reactivation of the virus which becomes latent in dorsal root ganglia herpes zoster is common in aged persons. Lesions are clear fluid filled grouped vesicles on an erythematous base in dermatomal distribution. Most frequently affected segments are thoracic and lumbar , particularly T3-L2[59] and ophthalmic division of trigeminal nerve. Few vesicles develop beyond the dermatome in some individuals which is more common in elderly.

Treatment : Antivirals like aciclovir in the dose of 800mg 5 times per day for 5 days or valacyclovir 1gm tds for 5days or famiciclovir - 0.5g tds for 7 days

can be given . Ideally the treatment is to be started within 3 days of the appearance of rash. But in old age it can be started even after 3 days. [58]

Post-herpetic Neuralgia

It is a commonest complication of HZ, defined as persistence or recurrence of pain in an affected area for more than 1month after the lesions have healed.[60] It is more common in patients more than 60yrs (50%).[61] Other risk factors include presence of prodromal pain, severe pain during the acute phase of HZ, greater rash severity, more extensive sensory abnormalities in the affected dermatome.[62] Spontaneous remission may occur over a period of time. It has 2 forms : a continuous as well as spasmodic shooting type. There may be pruritus, hyperaesthesia, allodynia, burning sensation, dysesthesia, anaesthesia. Pain may be relieved by application of cold water, ice cubes, topical capsaicin, topical lignocaine. If pain is severe systemic drugs like amitryptyline (25-75mg/day), carbamazepine (600-1000mg/day), phenytoin (300-400mg/day), lamotrigene (200-400mg/day), gabapentin 300mg/day can be tried.

Herpes simplex viral(HSV) infection : it is also common in these individuals. There are 2 antigenic types: HSV1 and HSV2. Herpetic gingivostomatitis is the most common form of HSV1 infection, HSV2 is responsible for genital infection. Recurrences are more common with herpes genitalis than with oropharyngeal herpes. Antivirals like acyclovir , valacyclovir , famiciclovir all have good safety profile.[8]

Molluscum contagiosum : may occur in elderly but immunocompromised state must be ruled out.[8] It is characterized by dome shaped umblicated papules transmitted by skin to skin contact. It can be treated by needling, cryotherapy, electrodessication and curettage.

Fungal infections :

Onychomycosis, tinea pedis, tinea cruris, candidiasis occur commonly in the aged population .

Onychomycosis : Toe nail onychomycosis is more common . It is caused by Trichophyton rubrum, mentagrophytes and Epidermophyton floccosum.[63] It most commonly presents with discolouration beginning at the free edge of the nail, lateral nail fold, subungual hyperkeratosis leading to separation of nail plate from the nail bed . It is usually asymptomatic in the elderly. The only problem it produces is,it is difficult to cut the nails with the ordinary scissors . Diagnoses can be confirmed by nail clippings for KOH mount , fungal culture . Finger nail onychomycoses is similar to that of toe nail onychomycoses . The nail plates become opaque and friable and has a yellowish colour . It can be treated with antifungal agents like terbinafine, itraconazole, fluconazole.[64]

Tinea corporis : It is most commonly caused by T. rubrum . Lesions on the trunk and limbs are typically annular , this appearance is due to elimination of the fungus from the cen1tre of the lesion as the infection spread at the margins . Diagnosis can be confirmed by scrapings from the edges of the lesion . It can be effectively treated with fluconazole, griseofulvin for 4-6 weeks .

Tinea cruris : It is uncommon in the elderly. It has a characteristic appearance which is often confused with intrtrigo, flexural psoriasis and sebhorreic dermatitis.Treatment includes both topical and systemic antifungal drugs.[8]

Tinea Pedis

Usually presents as scaling pruritic areas in the toe-webs and on the soles of the feet. It occurs much more frequently in men. Tinea pedis is probably usually acquired as a result of contact with infected squamous debris on the floors of showers and swimming baths. It produces chronic, fine, scaling on the soles of the feet, toe webs.

The diagnosis can be confirmed by taking scrapings of scaling, mounting in 10% KOH, and examining the material under the micro scope. Characteristic fungal mycelium can be seen. Tinea pedis usually responds to treatment with topical antifungal preparations like whitfield's ointment broad spectrum imidazoles – miconazole, clotrimazole, enconazole, sulconazole.

If topical therapy is not completely effective, oral antifungal drugs like fluconazole and griseofulvin for 6 weeks can be given.[65]

Scabies

Scabies is often misdiagnosed in the elderly. It is due to the fact that elderly may not present with extensive inflamed or excoriated lesions, they might present only with features of xerosis. The infestation is acquired as a result of close physical contact with another individual suffering from scabies ,

for example, holding hands, or sharing a bed with an infested person. Transient contact does not lead to transmission of the parasite.

The major symptom of scabies is itching, characteristically worse at night. The itching is thought to be due to hypersensitivity to mite faeces, though there may be some contribution from burrowing activity of the mites. The latent period is 4-6 weeks. The average number of adult mites on an infected individual is in the range of a dozen.

There are two principal components of the eruption in scabies – burrows and the scabies 'rash'. The burrows are tunnels made in the stratum coreneum by female mites. Each burrow is a few millimeters long, and is usually serpiginous. At one end of the burrow, the position of the actively burrowing mite, there is a tiny vesicle. Many burrows are partially or completely destroyed by scratching. Mites appear to burrow preferentially in areas of skin where there are few hair follicles. Typically burrows are found on the sides of fingers, in the web-spaces between the fingers, on the flexor aspect of the wrist, the palms of the hands, penis and scrotum, and on the margins of the feet.

The rash of scabies is an eruption of tiny inflammatory papules which are most profuse in the axillary regions, across the abdomen particularly around the umbilicus, and on the thighs. There are often secondary cutaneous changes. Scratching produces secondary eczematization and may introduce bacteria into the skin, resulting in folliculitis and/ or impetigo. In some patients, eczema is the major feature of their skin lesions, which are wrongly treated by steroids. It

can be diagnosed by scrape of the roof of the burrow with a scalpel blade, then transferred to a microscope mineral oil are 10% KOH as a mounting medium. [1]

Norwegian (crusted) Scabies

Norwegian scabies is so called because it was first described in Norwegian lepers in 1848. It differs from ordinary scabies in the clinical picture and in the number of mites present on the skin in which there are millions of mites.

The clinical picture may mimic hyperkeratotic eczema or psoriasis. Typically, an affected individual has thick, scaling plaques on the dorsa of the hands and fingers, the palms of the hands, extensor aspects of the elbows, the scalp and ears, and the soles of feet and toes. In some patients, most of the skin surface is involved. The nails are often affected, and show thickening of the nail plates and subungual hyperkeratosis.

Patients are frequently mentally recarded or suffering from dementia, this is more common in elderly.

Treatment

Gamma benzene hexachloride is an effective scabicide, and is non-irritant. This should be applied from neck to toes, and left on the skin for 8-12 hours then washed off. 5% permethrine is also commonly used . Single dose of oral ivermectin 200µg/kg body weight or two doses at an interval of two weeks can be given. [7]

Pediculosis

Generally uncommon in elderly, scalp itching is the usual symptom due to acquired hypersensitivity to the saliva of the louse. If the hair is never washed then plica polonica (matted hair) results. It can be treated with topical gamma benzene hexachloride or 1% permethrine .[7]

Neoplasms

Both benign and malignant neoplasms have been noticed in the elderly population with increased frequency.

Seborrheic Keratosis (Senile wart, basal cell papilloma)

Seborrhoeic warts are the most common benign skin tumour seen in the elderly. The cause is unknown but a familial tendency has been suggested.The lesions are multiple, most commonly on the upper trunk and face. They start as sharply defined brown macules and develop into slightly raised, brown to black, oval papules, or into polypoidal lesions with a stuck-on appearance. The colour is even but may vary from skin coloured, waxy-yellow, lightly pigmented or dark brown to balck depending on the amount of melanin present. Their size may vary from a few millimeters to several centimeters, their shape often ovoid, with the long axis along dermatomal lines. Their greasy appearance is not realted to seborrhea, the term seborrhoeic referring to their pattern of distribution.

It may mimic solar lentigo, lentigo maligna, pigmented solar keratosis, melanocytic naevi. The slight surface irregularity is the main clinical clue but biopsy confirmation is often necessary.

The eruptive pattern of numerous seborrhoeic warts (the Leser-Trelat sign) is indicative of associated internal malignancies like, gastrointestinal tract, breast and also leukemia, lymphoma and melanoma.[66]

Two clinical variants will be described: Dermatoses papulosa nigra and stucco keratosis.

Dermatosis papulosa nigra: It is found in about 35% of all adult blacks. They are located predominantly on the face, especially in the malar regions. They also occur on the neck and upper trunk. It consists of small smooth pigmented papules, except on the neck and trunk, where some of them may be pedunculated.[67]

Histopathology is similar to seborrheic keratosis . Most lesions are acanthotic type with more number of squamous cells, with only a few basaloid cells.[67]

Stucco keratosis: these are small gray-white seborrheic keratoses, measures 1-3 mm in diameter. They are symmetrically arranged on the distal portion of the extremities, especially over the ankles.[68]

Histopathology: It has the appearance of the hyperkeratotic type of seborrheic keratosis,with church spire pattern of upward extending papilla. Horn cysts and basaloid cells are usually absent.[69]

Histopathology of seborrheic keratosis : There is hyperkeratosis, acanthosis, lower border of the lesion is horizontal and sharply demarcated. The characteristic cell of seborrheic keratosis is the basaloid cell, slightly elongated with dark oval nucleus. These cells do not show any pallisading unlike those of basal cell carcinoma. Horn cysts are present which are round structures with central pink keratin similar to normal stratum corneum.

Histological subtypes : Six types recognized:

1. Irritated or Inverted follicular: occurs due to topical application of irritants . Squamous eddies are present in addition to typical features of seborrheic keratosis.[70] Occasional atypical mitotic figures, lichenoid infiltrate in dermis. It can cause diagnostic confusion with squamous cell carcinoma. However, the architecture of the lesion, lack of dermal penetration and striking horizontal lower border help in diagnosing seborrheic keratosis.
2. Adenoid are reticulate: Many cords of basaloid cells, which are often interlocking grow down from the dermis.
3. Hyper Keratotic: with gross hyperkeratosis, numerous digitate upward extensions of the papillae resemble church spires.

4. Acanthotic: It is the most common type in which hyperkeratosis and papillomatoses are often slight, but the epidermis is greatly thickened.
5. Clonal type: with basaloid cells occurring in nests or clones, which are separated from each other by normal keratonocytes. It represents an intrepdermal epithelioma of Borst-Jadassohn.[71]
6. Melanoacanthoma: Increased number of melanocytes and plenty of melanin within tumour cells.[72]

Treatment: Cryotherapy, electrodessication with curettage, Excision, laser ablation.

Skin tags (Fibroepithelial polyp, Acrochordon)

Skin tags are small firboepithelial polyps that are seen on the neck, axillae and groin of middle-aged and elderly people, increasing in frequency with age and obesity. They may be caused by simple outgrowths of the skin, pedunculated seborrhoeic warts or melanocytic naevi. They are often multiple, usually 1-4 mm in size rarely more than 3 cm. They are pigmented and multiple, primarily of cosmetic importance only. Skin tags are more numerous in some diseases such as acromegaly, acanthosis nigricans and polyposis coli.[73] Treatment includes electrodessication for small lesions, larger lesions should be excised.

Cherry angioma or Senile haemangiomata

Also known as campbell de Morgan spots or senile ectasia. They are dome shaped, bright red to purple in color, upto few cm in diameter. They commonly present over chest, trunk and upper limbs. Their number increases as the age advances. Sudden appearance of multiple lesions may accompany the development of an internal malignancy.[74] Histologically they are capillary hemangiomas.

Bowen's disease

It is a SCC in situ which occurs most commonly in fair skinned individuals. The highest incidence is in the sixth to eighth decade. Etiology includes exposure to chronic UVB radiation and arsenic, human papilloma virus (HPV) infection with oncogenic types like 16, 18, 39, 51,etc., metastases are rare.[75]

Both sun exposed and covered sites are involved.[76] It is usually a single lesion, well circumscribed, round to oval, pink to salmon red or dark red, scaly patch or plaque, few millimeters to several centimeters in diameter. Surface may become hyperkeratotic, crusted, fissured or ulcerated.

Histopathology : it is thought to arise from outer root sheath of the hair follicle or the epidermal cells of the acrotrichium. There is hyperkeratosis, parakeratosis and acanthosis, full thickness epidermal dysplasia with an intact basement membrane. Atypical keratinocytes with loss of polarity having a

"windblown" appearance.[77] Pagetoid cells are seen haphazardly throughout the epidermis. Moderate lymphocytic infiltrate is seen in the upper dermis.

Treatment: Simple excision for smaller lesions, topical or intralesional 5-FU, 5% imiquimod, curettage with electrodessication, cryotherapy**,** CO_2 laser ablation, photodynamic therapy are the various modalities used.

Due to the cultural habit of chewing betel nut and areca nut is common in India, many elderly suffer from oral sub mucosal fibrosis.[78] Bidi smokers, common in many aged individuals is another cause for lip cancer.

Actinic keratosis: It is a premalignant condition that is more common in 65 years of age or older.[79] Main risk factor is UVB exposure. It develops on the skin exposed to UVB. Common sites are the backs of the hands, forehead, and the ears. It presents as a rough, skin colored to reddish brown papule with adherent yellow brown scale, usually less than 1 cm in diameter. It may also manifest as cutaneous horn. Increase in size of the lesion, pruritus, ulceration and bleeding suggests transformation to SCC. The average rate of progression to invasive SCC is 8%.[80] It is also a risk factor for BCC and malignant melanoma.

Histopathology: There are five types: Hypertrophic, atrophic, pigmented, bowenoid and acantholytic.

There is hyperkeratosis, alternating with parakeratosis, and irregular acanthosis. Atypical cells which appear large, irregular in size and

shape,having pleomorphic nuclei with atypical mitotic figures are seen. Dyskeratotic cells are also seen. In the dermis, solar elastosis and mild infiltration of lymphocytes and plasma cells seen.

There are five types: Hypertrophic, atrophic, pigmented, bowenoid and acantholytic.[70]

Treatment: Medical therapies include, 5% imiquimod, topical or intralesional 5-FU, intralesional interferon. Cryotherapy, curettage, shave excision, electrosurgery are other modalities of treatment.

Actinic cheilitis : is a premalignant condition characterized by skin changes occurring on the lower lips. Excessive sunlight produces dryness, scaling, atrophy, and telangiectasia. Fissures, leukoplakia, and carcinoma may develop.

Keratoacanthoma (Molluscum sebaceum)

It is a common benign self- resolving tumour believed to arise from hair follicles. It is an abortive malignancy, rarely pregresses to SCC.[81] Ultraviolet radiation seems to be a major factor.It is three times more common in males. It has a peak incidence between 60 and 65 years. Contact with tar, mineral oil can also be a trigger. It is associated with carcinoma of the larynx, multiple internal malignancies, leukemia, HIV infection and it can occur in transplant recepients.[82]

Over 50% occur on the face, followed by the back of the hands, ears, forearms, neck and legs. It predominantly affects sun exposed surfaces.

Subungual, scrotal and peri anal lesions are rare. It is usually a solitary, pink or flesh colored, dome-shaped nodue with a central keratin plug.

It has 3 stages : proliferative stage, mature and involuting stage. In the early, proliferative stage, a rapidly growing skin- coloured, dome- shaped nodule is seen to develop a central crust. The mature keratoacanthoma is cup-shaped and filled with keratin, the whitish keratin being easily visible. Regression takes 3 months on average, the keratinous plug being ejected and the base healing with a small puckered scar. Acantholytic squamous cell carcinoma has a similar distribution and growth pattern, and is usually misdiagnosed clinically as a keratoacanthoma. There are several clinical variants: [81]

1) agglomerated form, 2) giant form- it grows upto 9cm or larger.

3) keratoacanthoma centrifugum marginatum- grows upto 20 cm, 4) keratoacanthoma involving oral and other mucous membranes like laryngeal mucosa, 5) Multiple eruptive keratoacanthomas of Ferguson-smith- it is autosomal dominant with onset during childhood and adolescence. 6) generalized eruptive keratoacanthoma of Grzybowski- it is associated with PUVA, radiotherapy, HIV, immunosuppression AND exposure to chemicals like mineral oil. 7) multiple familial keratoacanthomas of Witten and Zak- it has features of Grzybowski and Fergusion-smith. 8) plate shaped, pseudorecidive, occupational(tar), 9) distal digital keratoacanthoma- it is rare,it may be solitary or multiple. The onset is from 28-76 years of age with male

predominance. The lesion is painful, rapidly growing and invasive. It involves distal subungual tissue or proximal nail fold often leading to paronychia. It predominantly involves first three fingers especially the thumb. 10) keratoacanthoma in special situations: Muir-Torre syndrome- it is autosomal dominant with sebaceous tumors, keratoacanthomas, epidermal cysts occurring in association with gastrointestinal cancers and colonic polyps. It is also associated with Xeroderma pigmentosum, nevus sebaceous of Jadassohn.

The histological picture is dependent on whether the lesion has been removed during its proliferative, mature or involuntional stage. It is thought to arise from the hair follicle, which may explain the characteristic evolution of the lesion. The following features should be looked for: 1) the lesion is mostly exophytic and partly endophytic. 2) shape of the lesion is globular, well circumscribed, and has a cental cup shaped crater, filled with keratin and nuclear debris. 3) on one side of the invagination is an epidermal lip, which rises above the lesion as buttress. 4) there are large, atypical keratinocytes, may have mitotic figures, cells apper pale, pink and glassy. 5) squamous eddies and horn pearls may be present, causing confusion with well differentiated squamous cell carcinoma. 6) epidermis may show acanthosis and psoriasiform hyperplasia. Many polymorphonuclear cells may be seen within the epidermis. 7) dermis may show lymphoid infiltrate.[81]

Treatment: For solitary lesions surgical excision is desirable. Curettage with electrodessication, cryotherapy, intralesional triamcinolone, intralesional

chemotherapy with 5-Flurouracil(5-FU), Bleomycin, Interferronα-2a and methotrexate.Systemic therapy with retinoids, methotrexate, 5-FU, cyclophosphamide has been used for multiple lesions. For subungual lesions with underlying bone involvement amputation of the digit may be required.

Squamous Cell carcinoma

Squamous cell carcinoma (SCC) is a second most common form of malignant tumour that arises from the epidermis or its appendages. It frequently arises on the sun exposed skin of middle aged and elderly individuals. Lifetime risk of developing SCC is 7-11%.[83] It has high risk of metastases. Most SCCs arise from actinic keratoses(0.025%-16%).[80] It is more common in men.

The different clinical types of SCC are as follows:[7]

1) Keratotic or invasive SCC: it is a raised, firm, pink to flesh colored papule or plaque with smooth, verrucous, or papillated surface, and indistinct margins.

2) Nodular SCC: the lesion is more elevated with features similar to keratotic SCC.

3) Arsenic induced SCC: it develops on arsenical keratoses over the palms, soles and trunk or within Bowen's disease lesions on sun protected sites.

4) Radiation induced SCC: deveiops as an ulceration or erosion over radiation induced skin changes like hyperpigmentation, telengiectasia, dermatitis, or fibrosis.

5) Thermal induced SCC: it may develop on the background of erythema ab igne

6) SCC developing within chronic ulcers: Marjolin's ulcer is an example of SCC arising at the site of a chronic ulcers.

7) SCC of the mucosa: It may involve the lower lip secondary to actinic cheilitis. It also develops on thebackground of erythroplasia or leukoplakia. The common sites are the lateral aspect of the tongue, floor of the mouth and soft palate. Genital SCC may also develop over long-standing lichen sclerosus et atrophicus, leukoplakia, erythroplasia of Queyret.

8) Verrucous carcinoma: It is a low grade SCC, characterised by slowly enlarging, verrucous, cauliflower like growth. It rarely metastasizes. It may involve the sole (epithelioma cuniculatum), oral cavity (oral florid papillomatosis), anogenital region (giant condyloma acuminata of Buschke-Lowenstein) and scars(papillomatous cutis carcinoids).

Etiology includes UV exposure mainly UVB, PUVA, ionizing radiation like grenz ray, gamma rays, or X- rays. Chronic arsenic exposure, immunosuppression, chronic inflammatory diseases such as long standing oral

ulcerative lichen planus, chronic osteomyelitic sinus tracts, lupus vulgaris, hidradenitis suppurativa and epidermolysis bullosa also predispose to SCC ,Naevus sebaceous, porokeratosis, epidermal naeuvs, very rarely, seborrhoeic keratosis may lead on to SCC.[84]

Xeroderma pigmentosum, albinism and epidermodysplasia verruciformis are among the genoderamtoses predisposing to multiple SCC but patients with these conditions will usually present in younger age group.[85]

Histopathology: 1) down growths of epidermis into dermis and subcutis, connection of tumour masses to epidermis. 2) the cells are large, polymorphic, dyskeratotic cells appear pinkish, individual cell keratinisation is seen. 3) nuclei are large atypical with large nucleoli and show mitotic figures. 4) tumour masses extend deep into subcutis, pushing and compressing appendages, infiltrating vessels and nerves. 5) horn pearls are an important feature.They are large, well formed in well differentiated squamous cell carcinoma. 5) acantholysis due to inadequate formation of desmosomes may lead to cleft formation. 6) mononuclear inflammatory infiltrate in dermis.[86]

Treatment : Electrodessication and curettage is indicated for small(less than 10 mm) lesions. Excision is done for tumours larger than 3 cm.[87] Other options are laser, photodynamic therapy. Radiotherapy is more suited to medium- sized lesions around the eye and generally is used more in the elderly because of less concern with the cosmetic result, and general lack of fitness of the patient for surgical intervention.[7]

Basal cell carcinoma

Basal cell carcinoma (BCC) is a most common malignant tumour of the skin.[63] It is more common in adult males especially in the elderly. Male to female ratio is 3:2. It occurs on areas of chronic sun exposure. It is locally invasive, rarely metastasizes. It is composed of cells similar to those in the basal layer of the epidermis and its appendages.[88] It is recognized by the pearly waxy papules, with central depression, a rolled out or thready translucent border with erosion or ulceration, bleeding, crusting and telengiectasia.[89]

Etiology includes ultraviolet radiation (mainly UVA to some extent UVB), ionizing radiation(X-ray and grenz ray), arsenic exposure, immunosuppression(HIV, organ transplants),[90] previous history of nonmelanoma skin cancers.

CLINICAL TYPES OF BCC

1) Nodular / Noduloulcerative (Rodent ulcer) : it is the commonest type (60-80 %) , characterized by a pearly waxy papule with central depression , a rolled out thread translucent border , with erosion or ulceration , bleeding , crusting and telengiectasia over the surface .

2) Micronodular BCC : Lesions are flat to raised and infiltrative occurring on the back .

3) Pigmented BCC : pigmentation may be gray to black .

4) Superficial BCC : It presents as an scaly erythematous plaque with rolled out margin . There may be superficial erosions and crusting . It forms 10 – 30% of BCC. It has to be differentiated from psoriasis, bowen's disease or eczema . It is more common in HIV infection .[91]

5) Morphoeaform BCC : It presents an illdefined, whitish to yellowish, flat to depressed, scar like, indurated plaque with indistinct borders

6) Fibroepithelial BCC : It presents as an solitary to multiple, sessile to pedunculated, pink firm nodules, on the back

Histopathology

Nests of basaloid cells arranged in a pallisading manner with retraction artifact.The tumour masses always surrounded by a dermal stroma on which it is dependent for survival. This is the reason for its inability to metastasize. Cells lie in groups or nests may form horn cysts. Mild infiltrate may be seen surrounding the tumour masses.[92]

Treatment

Currettage with electrodessication, cryosurgery, excisional surgery, radiotherapy and Moh's micrographic surgery are the modalities utilized.[93] Medical treatment with topical and intralesional 5-FU, 5% imiquimod cream(applied at bed time to the treatment area, including 1 cm of skin surrounding the tumour, 5 days a week for 6 weeks), interferon α-2b 1.5

million units injected intrlesionally 3 times a week for 3 weeks. Lasers like CO_2, Nd:YAG and pulsed dye lasers have been used to ablate the lesions.[94] Photodynamic therapy is useful for superficial tumors (less than 3 mm thicknes), simultaneous treatment of multiple tumors and in immunocompromised patients.[95]

Malignant melanoma

Malignant melanoma of the skin is a tumour arising from cutaneous melanocytes, melanocytic naevi or blue naevi. There are four types of malignant melanoma: superficial spreading melanoma, nodular melanoma; lentigo maligna melanoma, and acral lentignous melanoma. Particularly lentigo maligna melanoma, may occur in the elderly population and present as brownish or black plaques, especially over the face with irregular borders and irregular pigmentation.[7] Acral lentiginous melanoma is more common in India. It is frequently seen on the soles than on the palms.[96]

Superficial spreading melanoma is the most common type in light skinned people.[97] Sites of predilection are the back in men and the legs in women. It presents as a pigmented macule or patch with an irregular border that gradually develop into a thin plaque . Nodular melanomas begin as nodules without going through a phase of macular pigmentation.[98] They occur most frequently on trunk , followed by head and neck and legs . Some nodules may be amelanotic showing little or no pigmentation these may be difficult to diagnose clinically .

Histopathology : Shows proliferation of atypical melanocytes predominantly along the demoepidermal junction. Atypical melanocytes with marked pleomorphism is seen . These atypical melanocytes may migrate along the sweat ducts . There may be nests of atypical melanocytes at the junction and individual melanocytes in the epidermis above the basal layer upto the stratum corneum.[99]

Treatment : They are treated by surgical excision.The recommended margin is 0.5 cm for in situ lesions, 1 cm for melanomas upto1 mm thick ,1to 2 cms for melanomas upto 1-4 mm thick, 2-3 cms for melanomas more than 4 mm in thickness.[100]

Pigmentation abnormalities

The most obvious senile changes in skin are pigmentation abnormalities.

Senile lentigens (Solar lentigo): It is the major clinical features of photoaged skin in Asians. It is light brown in colour. It appears after chronic sun exposure. It is more than 1 cm in diameter, commonly seen over back of hands and face.The lesions increase in number with age. Lesions may fade, persist indefinetly or rarely transform into lentigo maligna, lichenoid keratosis or seborrheic keratosis.

Histopathology : There is Epidermal atrophy, in between epidermal atrophy rete ridges are elongated, club shaped or tortuous and show small, budlike extensions. The elongated rete ridges, especially in their lower portion

composed of deeply pigmented basaloid cells intermingled with melanocytes. Mild collections of a perivascular mononuclear cell infiltrate is seen in the upper dermis. No junctional activity or atypical melanocytes observed.[101]

Treatment : Preventive measures includes avoidance of excessive sun exposure and daily use of sunscreens. It may be treated with cryotherapy or electodessication.

Freckles (Ephelides)

These are small (less than 5 mm in diameter), circumscribed, poorly marginated, pale brown macules that appear exclusively on the sun exposed skin. Freckling is due to increased amount of melanin.There is no increase in number of melanocytes.[102]

Idiopathic Guttate hypotmelanosis

Also known as disseminate lenticulate leukoderma. It is due to reduced number of melanocytes. They appear well circumscribed, like porcelain white chip inverted into the skin. They vary in size from few millimeters to 0.5 cm. It is most frequently found in anterior surface of legs and forearms. Other sites are chest, trunk and abdominal wall. Two clinical types. 1.Actinic type (seen in sunexposed sites), 2. non actinic type (seen in non sunexposed sites e.g. trunk).[103]

Differential diagnosis includes vitiligo, Tinea versicolor, post inflammatory hypopigmentation. Histopatholgy shows deficient epidermal

melanin, decreased number of melanocytes, incompletely melanised melanosomes.[104] Dopa reaction is reduced are absent.

Treatment : Reassurance.

Bullous Disorders

Blistering disorders like bullous pemphigoid and cicatricial pemphiogid are common in the elderly.

Bullous pemphigoid

It is generally a disease of elderly, the median age of onset ranges from 60 to 75 years.[105] It is more common in men. Although bullae are the most characteristic feature of bullous pemphigoid, they are not always present initially and the process may begin with a rather non-specific phase. This initial period or 'prepemphigoid is generally associated with intense irritation, which may precede the development of visible skin lesions by several months. Later, large, tense blisters develop both on an erythematous and on normal skin. There may or may not be mucosal involvement. Histopathology shows subepidermal bullae with numerous eosinophils.[106]On direct immunofluoroscence there is linear deposition of IgG, C3 to the epidermal basement membrane zone.

Treatment includes topical steroids with topical antibiotics for mild disease .systemic drugs like prednisolone, azathioprine, intravenous immunoglobulin are used in more severe form of the disease .

Cicatricial pemphigoid :

It is also seen in elderly (the peak incidence is between the age of 60 and 80 years).[107] It is more common in women. It is a chronic subepidermal blistering disease of the mucosa that may involve the skin. It usually results in permanent scarring of the affected area, particularly conjunctiva. Oral mucosa is more commonly involved (85%), followed by, ocular, nasal, nasopharyngeal, laryngeal, esophageal and genital mucosa in decreasing order. Histopathology shows sub epidermal bulla, direct immunofluorescence shows linear deposition of IgG (mostly) and IgA (insome) to the epidermal basement membrane zone.[108]

Treatment : For mild to moderate disease topical steroids alone or in combination with oral tetracycline, nicotinamide can be used . In moderate to severe disease systemic corticosteroids are the mainstay of treatment along with azathioprine , cyclophosphamide, intravenous immunoglobulin, dapsone can be used.

Dermatitis Herpetiformis

Even though it is common in young adults it is also seen in elderly.It is intensely pruritic, chronic, recurrent papulo vesicular disease. The eruption is

symmetrical, polymorphic, consisting of erythematous, urticarial, papular, vesicular or bullous lesions. It is associated with gluten – sensitive enteropathy. Histopathology shows deposition of granular pattern of IgA in the papillary dermis, papillary tip microabscess.[109]

Treatment : Dapsone is the mainstay of treatment. Gluten free diet mandates avoidance of wheat, barely, rye, oats

Linear IgA disease

It is a chronic acquired subepidermal disease of children and adults with cutaneous and mucosal involvement. Histopathology shows linear deposition of IgA antibody on the basement membrane zone. Adult form is most common after the age of 60yrs.[110] There is characteristic annular lesion with blistering around the edge.

Treatment includes topical and systemic steroids, dapsone, azathioprine, tetracycline can be used.

Drug eruptions

Elderly patients are generally on several medications due to the multiple medical disorders. Sudden onset of skin lesion in a patient without any prior skin disease should always raise the suspicion of drug eruption.[111] The most common eruptions in elderly population are exanthematous eruptions; they take the form of maculopapular, morbilliform, or erythematous lesions. The other presentations could be vasculitis, fixed drug eruptions, erythema multiforme,

urticaria, contact dermatitis, purpura, and photodermatitis. Fixed drug eruption may present as rounded single erythematous or bullous lesions, which typically recur at the same spot upon rechallenging with the same medication. Drug-induced vasculitis appears as purpuric maculopapular eruption mainly on limbs and may be accompanied by systemic symptoms such as fever, myalgia and fatigue. The loss of dermal collagen and fat coupled with vascular fragility may predispose the elderly to traumatic purpuric lesions. Many elderly are on different medications that may cause thrombocytopenia, leading to purpura.

Erythema multiforme is due to hypersensitivity reaction characterized by target-like lesions on the extremities and trunk accompanied by systemic symptoms. It may take the form of minor or major form. The latter may present as more serious life-threatening Stevens-Johnson syndrome or toxic epidermal necrolysis. Medical history and physical examination are also important in reaching the correct diagnosis. The standard treatment is discontinuation of the causative therapeutic agent and substitution with another drug.[112]

Nutritional disorders

Chronic illnesses, malabsorption, impaired metabolism, immobility, depression, eating disorders, social deprivation, drugs, and poverty make the elderly susceptible to a variety of nutritional disorders. The cutaneous signs of chronic ill health may include hair loss, hyperpigmentation of the skin, and dry and brittle nails. Women with advanced age are particularly prone to

deficiencies in vitamins B_{12}, A, C, and D, calcium, iron, zinc, and other trace elements, with consequent skin changes.

Iron deficiency results in an iron deficiency anemia, manifested by pallor, weakness, irritability, palpitations, sore tongue, angular stomatitis, loss of hair, and koilonychia. Zinc deficiency may be acquired as found in liver and pancreatic disease or in malabsorption. An eczematous eruption is seen in areas of trauma, such as knees, ankles and elbows. Seborrheic dermatitis-like lesions are seen on the face. Hair growth is sparse, and total alopecia may occur. Zinc supplementation leads to rapid improvement and serum alkaline phosphatase levels return to normal. Vitamin C deficiency resulting in scurvy manifests as small perifollicular hemorrhages. Niacin deficiency results in pellagra. Appropriate nutritional intake and excluding possible systemic diseases that may cause vitamin and trace elements deficiencies and palliative care is the standard of treatment in nutritional deficiencies.[113]

Psychodermatological disorders

Psychodermatological disorders seen in the older population include delusion of parasitosis, lichen simplex chronicus, neurotic excoriations and prurigo nodularis.

Delusion of parasitosis

It is the commonest monosymptomatic hypochondriacal psychosis and it usually occurs in females. They are socially deprived, hard of hearing and

visually impaired. They bring with them bits of skin scales, insect parts, other specimens to prove their existence- match box sign. Sometimes their spouse starts believing these delusions[folie-a deux-craziness]. Scabies, diabetes, hypothyroidism, Grover's disease, pellagra, polycythemia vera and multiple sclerosis, central nervous system dysfunction should be ruled out.[114]

Treatment: It is difficult to treat, antipsychotics like pimozide [4-6mg] is the most effective drug,[115] olanzepine [2.5-5mg] may be tried.

Lichen simplex chronicus [neurodermatitis circumscripta]

It presents with lichenified red plaque due to repeated rubbing or scratching with the dominant hand. It is common in elderly. It involves shins, forearms, palms and back of neck[known as lichen nuchae]. It may also involve perianal and vulval skin. Postulated mechanism is intense itching and consequent scratching that is responsible for lichenification, with accentuation of criss cross skin markings.[116] Histopathology shows hyperkeratosis, acanthosis, elongation of rete ridges, vertically oriented collagen fibres in the thickened papillary dermis. Response to corticosteroids is very poor, intralesional steroids can be tried.

Prurigo nodularis

It presents with erythematous or hyperpigmented , scattered, discrete keratotic nodules on the extremities. Etiology includes emotional stress.[117] It may be seen in association with underlying metabolic disorder (hepatic

dysfunction, uremia, anemia, myxedema), neoplastic diseases (Hodgkins disease), HIV infection. The number of calcitonin gene related peptide and substance P-immunoreactive nerve fibre bundles in the nodules is increased.[118] Lesions present as nodules with surrounding hyperpigmentation, warty surface, which may be eroded, scaly, or crusted. Lesions usually seen on the extensor surface of the limbs. Psychological approach, topical steroids, cryotherapy, doxepin cream, habit reversal therapy are helpful.[119]

Other disorders seen in elderly individuals are:

These are leg ulcers, psoriasis, lichen planus, lichen scleroses et atrophicus and erythroderma.

Leg ulcers

An ulcer is defect of epidermis and dermis produced by sloughing of necrotic tissue. It is a common problem in the elderly. It is due to chronic venous insufficiency, neuropathy due to diabetes , leprosy or tabes dorsalis and trauma.It is also seen in those with impaired mobility due to cerebrovascular accidents, chronic infections such as leprosy, tuberculosis, syphilis.

Lichen planus

It is an inflammatory, papulosquamous disorder affecting skin, nails, hair, mucous membranes. It is more common in females especially in 6^{th} decades. The classical lesions are pruritic, erythematous to violaceous, flat-topped, polygonal papules and plaques distributed mainly on the flexor aspect

of the extremities. It is associated with intense pruritus. Genital lichen planus is more common in elderly females.[120] It is essential to exclude lichenoid drug eruptions due to drugs in elderly individuals.

Psoriasis

It can occur at any age. There is a bimodal age of onset, the first peak at 15-20 years, second one at 55-60 years.[121] It presents as chronic, symmetrical, well defined erythematous, dry red scaly papules and plaques situated most commonly over extensor aspects of extremities, scalp, palms and soles. Involvement of joints, nails is also common. Treatment includes topical keratolylitcs, emollients. Systemic drugs like methotrexate may be considered , if hepatic and renal fuctions are normal.

Lichen sclerosus et atrophicus (LSEA): (white spot disease, lichen albus)

It is a chronic inflammatory dermatoses with relapsing and remitting course, that causes significant discomfort and morbidity. It is most commonly seen in adult women, but also in men and children. Any skin site may be affected but it is more common in the anogenital area (85-98%) with intractable itching and soreness, progression destructive scarring is common. Extra genital LSA is less common (15-20%).

It has a bimodal peak in incidence from prepubertal children to post menopausal age group in women, male to female ratio of 10:1 to 6:1.[122] The exact etiology is unknown. Probable etiological factors include, hormonal

influence, chronic friction or rubbing, infection with spirochete Borrelia, oxidative stress, familial tendency. There is a strong association with autoimmune disorders like alopecia areata, vitiligo, thyroid disease and pernicious anemia . In females It is characterized by porcelain white atrophic papules coalescing into plaques that may extend to involve the vulval and perianal area in a figure of eight configuration. Overlying skin may show telengiectasia, purpura, erosions, fissuring or ulceration. It usually occurs on the labia majora, labia minora and clitoris. Vagina and cervix are almost always spared. It may lead to introital narrowing ('keyhole' or hourglass), loss labia minora, clitoral burying and obstruction to urinary flow. There is 4-5% risk of developing squamous cell carcinoma, in vulval LSA.

In men lesions are usually seen on the glans and prepuce. It can result in phimosis, paraphimosis, painful erections and urinary obstruction. The end stage is balanitis xerotica obliterans which is characterized by depigmented, thickened and contracted prepuce, fixed over the glans and not retractable.

Extragenital lesions are more common over upper trunk, sites of pressure, flexor aspect of wrist, around the neck, periumblical area, buttocks and thighs.

Histopathology shows hyperkeratosis with follicular plugging, atrophy of stratum malphigi with hydropic degeneration of basal cells, pronounced dermal edema and homogenization of collagen in the upper dermis and lymphocytic infiltrate in the mid dermis.[123]

Treatment: Ultrapotent topical steroids (halobetasol propionate or 0.05% clobetosol propionate), emollients, topical tacrolimus or pimecrolimus, topical calcipotriol, topical retinoids can be used. Circumcision in men, cryotherapy, CO_2laser, systemic drugs include oral antimalarials, antibiotics like ceftriaxone or penicillin may be tried.[124]

Erythroderma

It is a generalized inflammatory skin disorder manifesting with erythema and scaling affecting more than 90% of the body surface area. It can be classified as primary erythroderma arising on normal looking skin due to an underlying systemic disorder or drugs. Secondary erythroderma arises from preexisting dermatoses. It is more common in males.

Etiology includes psoriasis (it is the commonest cause in adults), eczemas (atopic eczema, seborrheic dermatitis, stasis eczema, phytophotodermatitis), infections like dermatophytosis, norwegion scabies, HIV, lymphoma, leukemia, drugs like phenytoin, carbamazepine, beta blockers, cimetidine, gold salts, lithium, omeprazole, pemphigus foliaceous, idiopathic.

Clinical features include erythma and scaling involving more than 90% of body surface area.[125] Large scales are seen in acute cases and smaller one in chronic cases. There may be enlargement of lymph nodes, it is called dermopathic lymphadenopathy,which should not be mistaken for lymphomas. Systemic manifestations include fever or hypothermia, malaise, chills and bodyache. There may be high output cardiac failure.

Underlying causes must be ruled out. Drug induced erythroderma and erythroderma arising from preexisting dermatoses has good prognosis.

Recently described entities :

Senescent actinic depigmentation of scalp was recently described. Predominantly in men, but rarely seen in women, with androgenic alopecia in persons aged older than 65 years.[126] Patients present with hypopigmented and depigmented macules on the scalp. Yoga sign is another recently described condition that is seen in people sitting cross legged on hard floors without carpets[127].

Materials and methods

MATERIALS AND METHODS

STUDY DESIGN

This is a descriptive study of new patients attending outpatient clinic in the Department of Dermatology, Venereology, Leprosy at Coimbatore Medical college hospital, Coimbatore.

Time period: August 2014- July 2015.

INCLUSION CRITERIA

All patients aged more than 60 years and above attending the outpatient clinic were included .

EXCLUSION CRITERIA

Severely ill and immunocompromised individuals.

METHOD OF COLLECTION OF DATA

- Informed consent was taken from all the patients prior to the examination.
- Detailed history including the duration of the disease, site of involvement, occupation, leisure activities and demographic details were taken.
- Thorough systemic & dermatological examination will be done.
- Investigations like complete blood count, liver function test, renal function test, random blood sugar was done.

- Other investigations like KOH mount, Tzanck smear, skin biopsy, immunofluorescence was done for all relevant cases or if the diagnoses could not be arrived clinically.

- All these data were recorded in a proforma, tabulated and analyzed statistically

Observation of Results

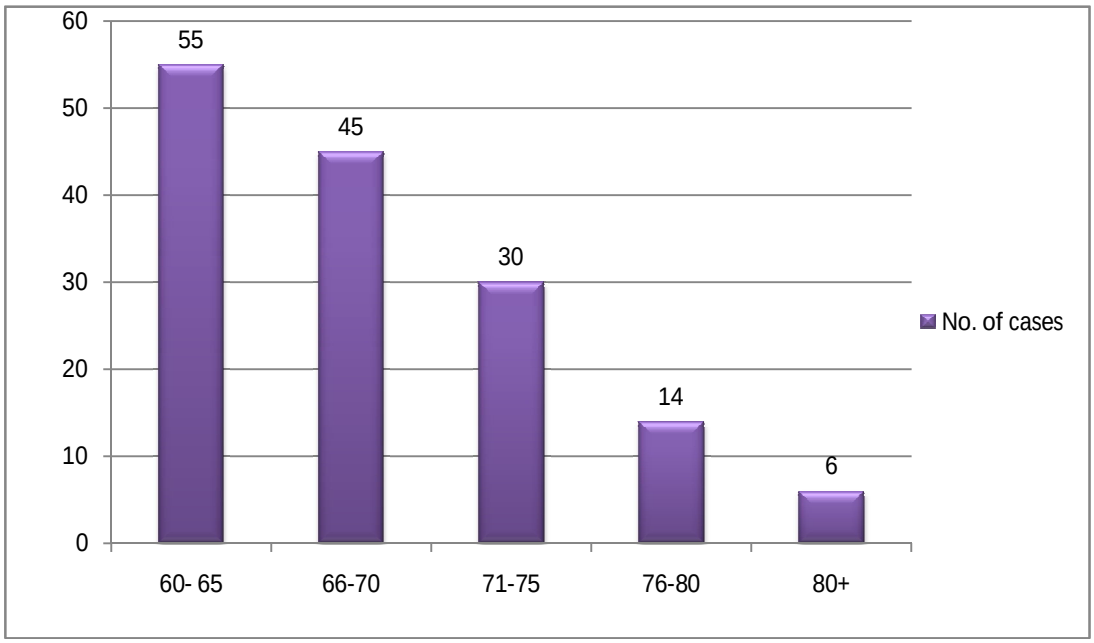
OBSERVATION AND RESULTS

A total of 150 cases with age above 60 years attending OPD of Coimbatore Medical College Hospital were included in the study

TABLE 1 : AGE DISTRIBUTION

Age Groups	Number of Patients	Percentage (%)
60-65	55	37%
66-70	45	30%
71-75	30	20%
76-80	14	9%
80+	6	4%
Grand total	**150**	**100**

CHART 1: AGE DISTRIBUTION (N=150)

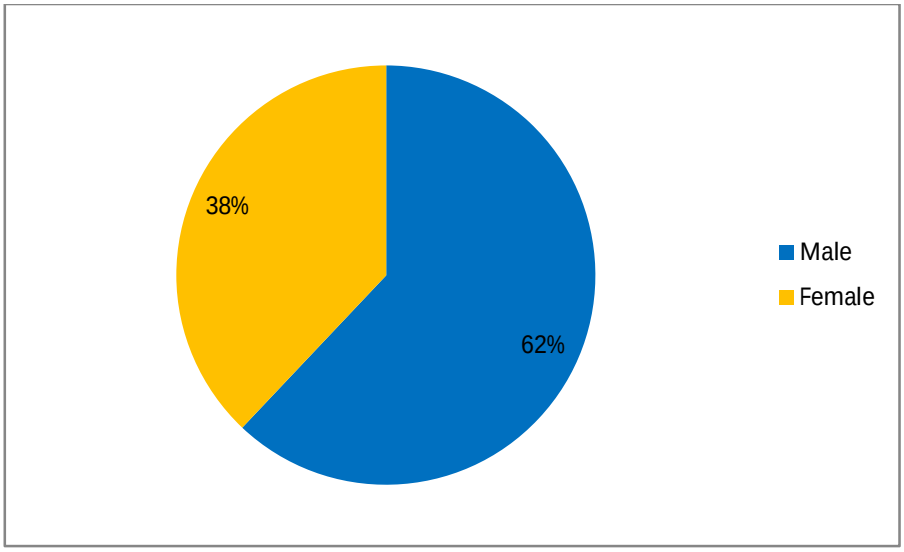


Maximum number of patients in this study belonged to age group of 60-65 years (37%), followed by 66-70 years (30%). The eldest patient was of 91 years.

TABLE 2 : SEX DISTRIBUTION

Sex	Number of Patients	Percentage (%)
Male	93	62
Female	57	38
Grand total	**150**	**100**

CHART 2: SEX DISTRIBUTION



Among 150 patients there were 93 males (62%), and 57 females (38%) in our study. Male to female ratio was 1.63:1.

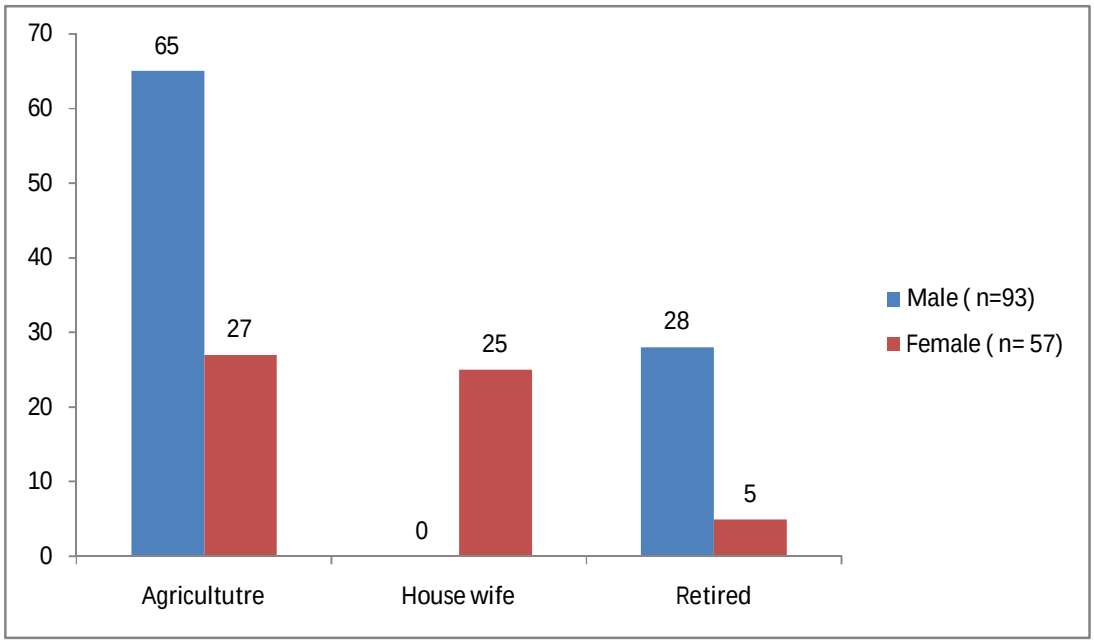
TABLE 3 : OCCUPATION DISTRIBUTION

Type	Number of Patients	Percentage (%)
Agriculture	92	61.3
Houswewife	25	16.7
Retired	33	22.0
Total	**150**	**100**

TABLE 4 : OCCUPATION DISTRIBUTION BY GENDER

Type	Female	Male	Total
Agriculture	27	65	92
Housewives	25	-	25
Retired	5	28	33
Total	**57**	**93**	**150**

CHART 3: OCCUPATION DISTRIBUTION BY GENDER

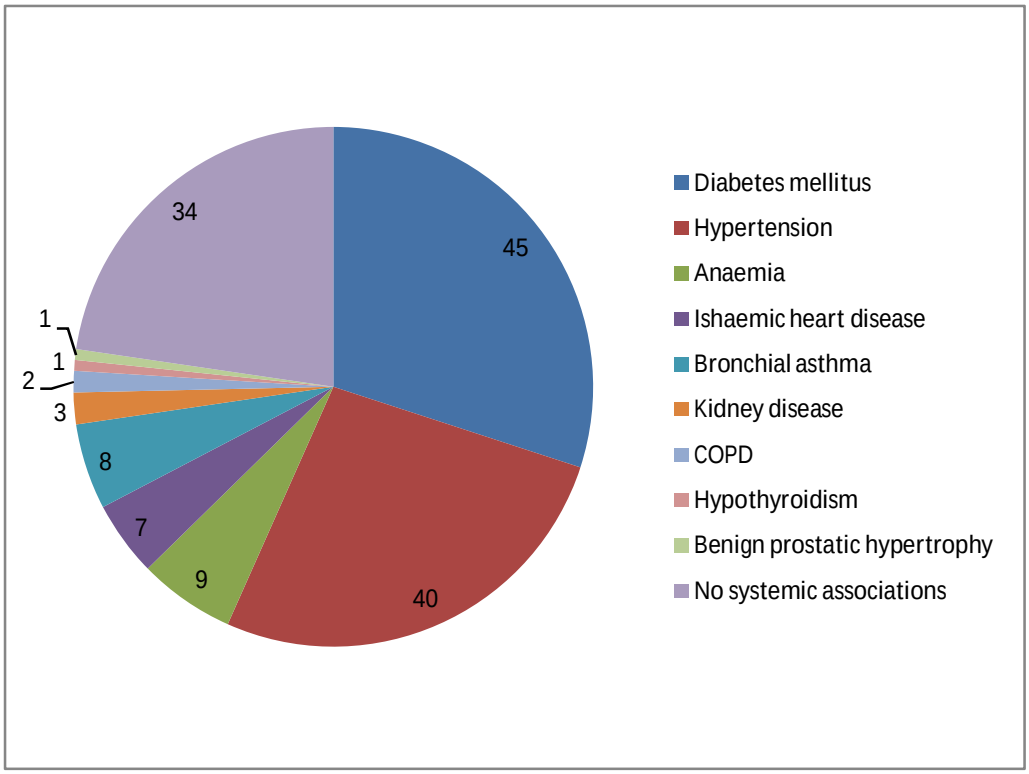


In our study among 150 patients, 92 were agricultural workers (61.3%), of which 65 were males, 27 were females, 25 females were housewives, 33 were retired (22.0%) of them there were 28 males and 5 females. Thus most patients in our study belong to agriculture work and most of the female patients were housewives.

TABLE 5 : ASSOCIATED SYSTEMIC DISEASES

Associated Systemic Diseases	No. of Cases	Percentage (%)
Diabetes mellitus	45	30%
Hypertension	40	26.7%
Anaemia	9	6%
Ishaemic heart disease	7	4.7%
Bronchial asthma	8	5.4%
Kidney disease	3	2.1%
COPD	2	1.3%
Hypothyroidism	1	0.6%
Benign prostatic hypertrophy	1	0.6%
No systemic associations	34	22.6%
Total	**150**	**100%**

CHART 4: ASSOCIATED DISASES

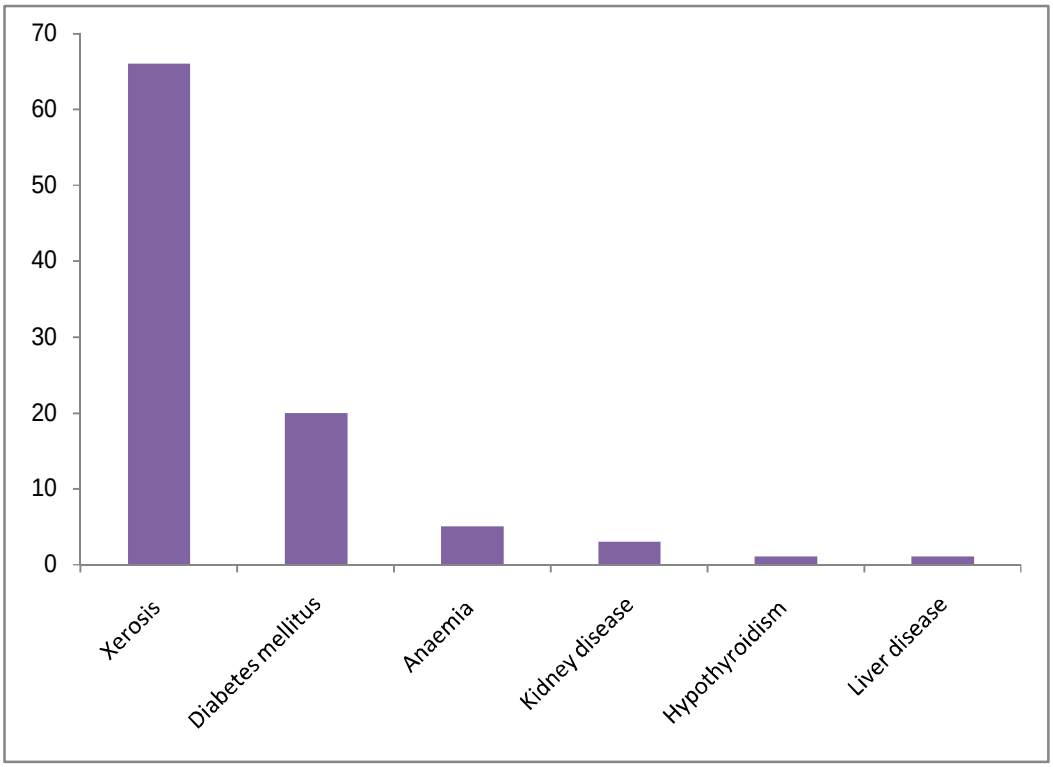


In our study diabetes mellitus was the commonest associated disease seen in 45 cases (30%), followed by hypertension in 40 cases (26.6%), both diabetes mellitus and hypertension was seen in 10 cases, anaemia in 9 cases (6%), bronchial asthma in 8 cases (5.3%), ischaemic heart disease in 7 cases (4.7%), kidney disease in 3 cases (2%), COPD (chronic obstructive pulmonary disease) in 2 cases (1.3%) hypothyroidism and benign prostatic hypertrophy in 1case each (0.6%),

TABLE 6 : GENERALISED PRURITUS

No	Conditions associated with GP	No of Cases	Percentage (n=96)
1	Xerosis	66	68.7%
2	Diabetes mellitus	20	21%
3	Anaemia	5	5.2%
4	Kidney disease	3	3.1%
5	Hypothyroidism	1	1%
6	Liver disease	1	1%
Total		**96**	**100%**

CHART 5 : GENERALISED PRURITUS

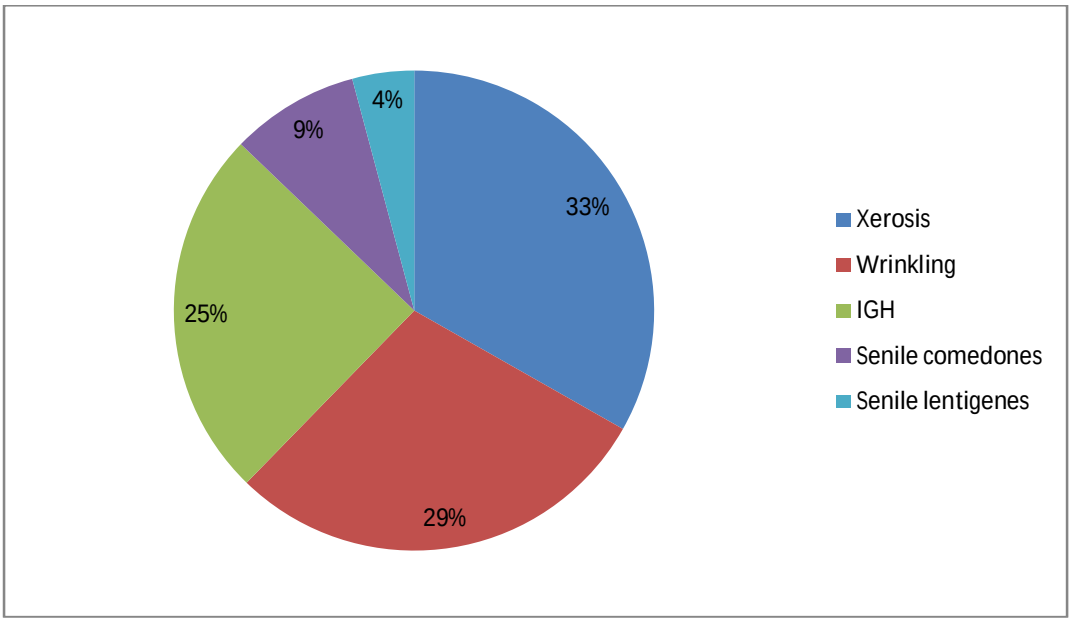


Generalized pruritus was seen in 96 cases in our study, of which xerosis was most commonly associated with generalised pruritus in 66 cases (68.7%), diabetes mellitus in 20 cases (21.0%), anaemia in 5 cases (5.2%), kidney disease in 3 cases (3.1%), hypothyroidism in 1 case (1.0%), and liver disease in 1 case (1.0%), thus senile pruritus was commonly associated with xerosis in this study.

TABLE 7 : PHYSIOLOGICAL SKIN CHANGES WITH AGING

Skin Changes	Number of Patients	% of Total n=289 Patients
Xerosis	96	33.2%
Wrinkling	84	29.1%
IGH	72	25%
Senile comedones	25	8.6%
Senile lentigenes	12	4.1%
Total	**289**	**100%**

CHART 6 : PHYSIOLOGICAL SKIN CHANGES WITH AGING



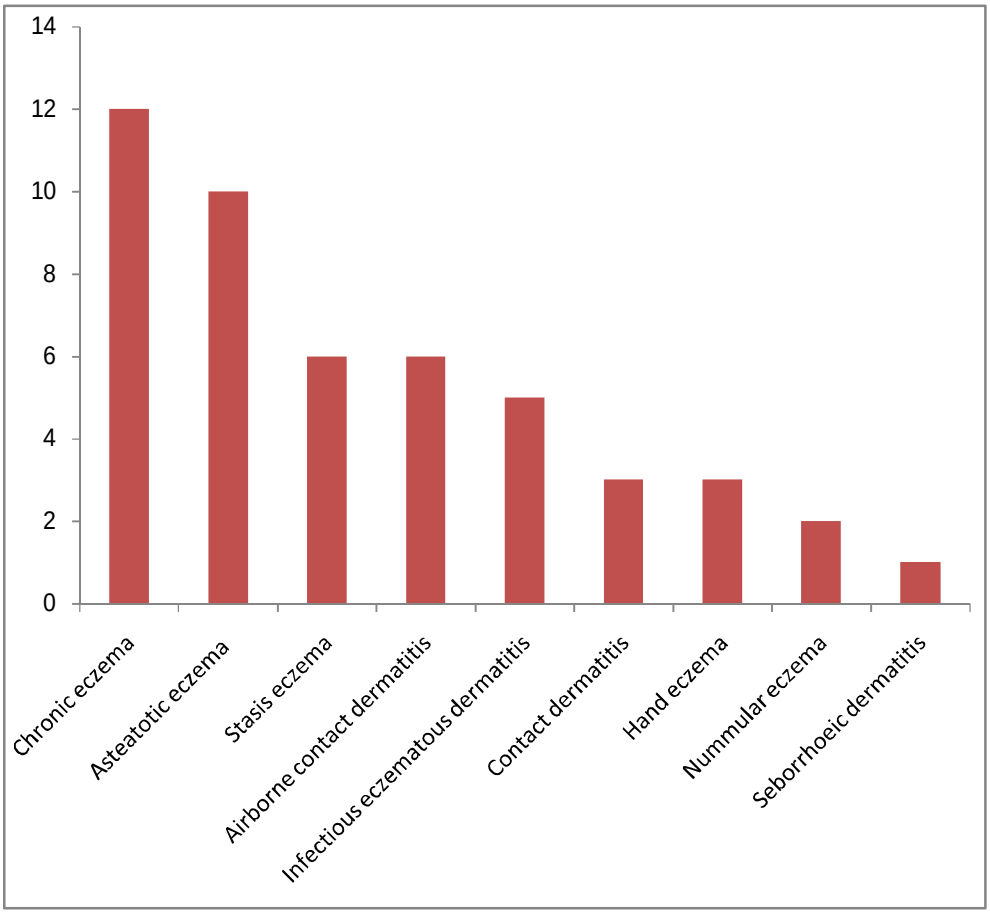
Xerosis was seen most commonly in this study in 96 cases (33.2%), followed by wrinkling in 84 cases (29.1%), IGH in 72 cases (25.0%), senile comedones 25 cases (8.6%), and senile lentigenes in 12 case (4.1%). In some of these cases combination of above findings were seen.

TABLE 8: PATHOLOGICAL SKIN CHANGES

ECZEMATOUS CONDITIONS

Type of Eczematous Conditions	No of Cases	Percentage (%) n=48
Chronic eczema	12	25%
Asteatotic eczema	10	20.85%
Stasis eczema	6	12.5%
Airborne contact dermatitis	6	12.5%
Infectious eczematous dermatitis	5	10.41%
Contact dermatitis	3	6.25%
Hand eczema	3	6.25%
Nummular eczema	2	4.16%
Seborrhoeic dermatitis	1	2.08%
Grand Total	**48**	**100%**

CHART 7 : ECZEMATOUS CONDITIONS



In our study among the 48 cases of eczematous conditions, chronic eczema was seen in 12 cases (25%), followed by asteatotic eczema in 10 cases (20.85%), stasis eczema was seen in 6 cases (12.5%), airborne contact dermatitis in 6 cases (12.5%), infectious eczematous dermatitis (10.41%), contact dermatitis and hand eczema in 3 cases each (6.25%), nummular eczema in 2 cases (4.16%), and seborrheic dermatitis in 1 case (2.08%).

TABLE 9 : PAPULOSQUAMOUS DISORDERS

Type of Papulosquamous Disorders	No of Cases
Psoriasis	12
Lichen planus	5
Total	**17**

In our study among the 17 cases of papulosquamous disorders psoriasis was seen in 12 cases and lichenplanus in 5 cases.

TABLE 10 : TYPES OF INFECTION

Type of Infections	Sub Type	No. of Cases	Percentage (%)
Fungal Infections	**Fungal Total**	**28**	**54.9**
	Dermatophytosis	23	82.14
	Candidiasis	5	17.8
Bacterial Infections	**Bacterial Total**	**12**	**23.54**
	Cellulitis	3	25
	Folliculitis	4	33.33
	Furuncle	5	41.66
Viral Infections	**Viral Total**	**11**	**21.56**
	Herpes zoster	8	72.72
	Viral warts	3	27.27
Grand Total		**51**	**100**

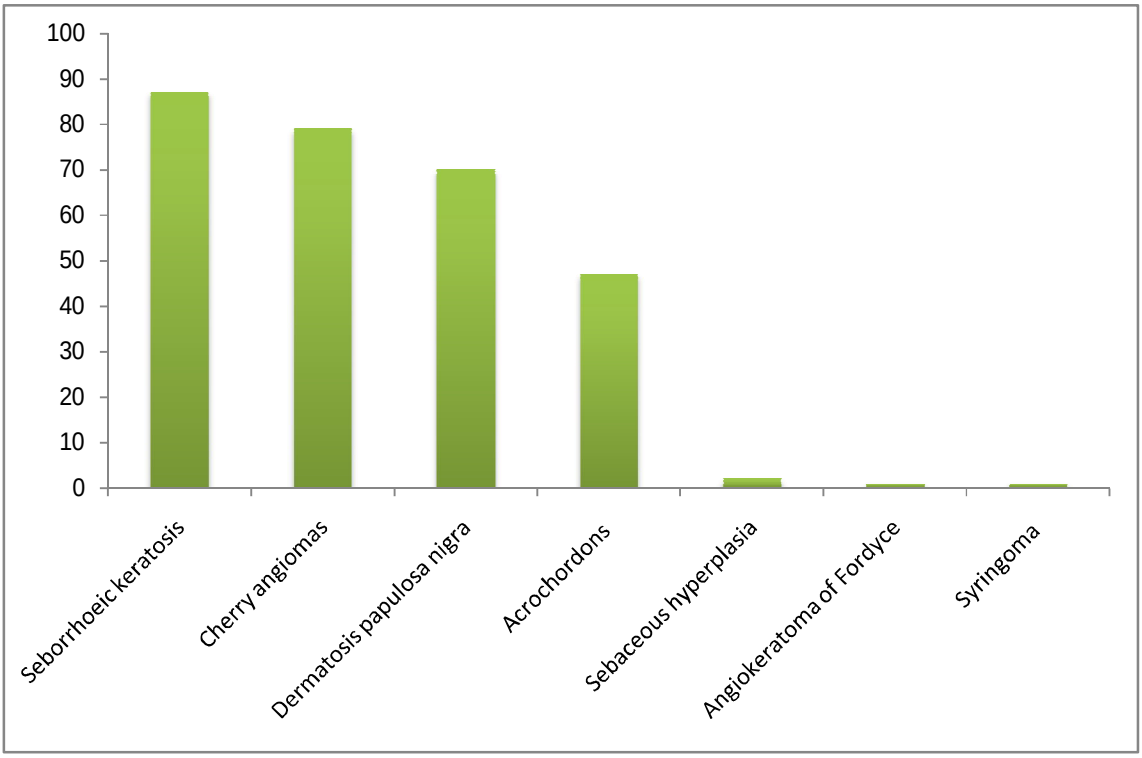
In our study infections were seen in 51 cases, of which fungal infections was the most common finding seen in 28 cases (54.9%). Bacterial infections were seen in 12 cases (23.54%) and viral infections in 11 cases (21.56%).

Among the 28 cases of fungal infections dermatophytosis was seen in 23 cases (82.14%), and candidiasis in 5 cases (17.8%). Of the bacterial infections cellulitis was seen in 3 cases (25%), folliculitis in 4 cases (33.33%), furuncle in 5 cases (41.66%). Among the viral infections herpes zoster in 8 cases (72.72%) and viral warts in 3 cases (27.27%).

TABLE 11 : BENIGN TUMORS OF SKIN

No.	Condition	No of Cases	Percentage (%) N=287
1	Seborrhoeic keratosis	87	30.31
2	Cherry angiomas	79	27.52
3	Dermatosis papulosa nigra	70	24.4
4	Acrochordons	47	16.3
5	Sebaceous hyperplasia	2	0.68
6	Angiokeratoma of Fordyce	1	0.35
7	Syringoma	1	0.35
Total		**287**	**100**

CHART 8 : BENIGN TUMORS OF SKIN



Among the 287 benign skin lesions seen in our study the most common was seborrheic keratoses with 87 cases (30.31%), followed by cherry angiomas in 79 cases (27.52%), dermatosis papulosa nigra in 70 cases (24.4%), combined features of above findings were seen in some cases. Acrochordons in 47 cases (16.3%), sebaceous hyperplasia in 2 cases (0.68%), angiokeratoma of fordyce and syringoma in 1 case each (0.35%).

TABLE 12 : PREMALIGNANT AND MALIGNANT TUMORS

Types	Sub Type	No of Cases
Pre Malignant Condition	Bowens disease	1
Malignant Tumors	Basal cell carcinoma	3
	Squamous cell carcinoma	1
Grand Total		**5**

Among the premalignant conditions 1 case of Bowen's disease was seen. The most common malignant condition seen in our study was basal cell carcinoma seen in 3 cases,and squamous cell carcinoma of lips was seen in 1case.

TABLE 13 : BULLOUS DISORDERS

Types	No of Cases
Bullous pemphigoid	7
Pemphigus vulgaris	3
Grand Total	**10**

Bullous pemphigoid was seen most frequently among the bullous disorders in 7 cases, pemphigus vulgaris in 3 cases.

TABLE 14 : PSYCHOCUTANEOUS DISORDERS

Types	No of Cases	Percentage (% N=19
Delusional parasitosis	10	52.64
Lichen simplex chronicus	8	42.10
Prurigo nodularis	1	5.26
Grand Total	**19**	**100**

Among the psychocutaneous disorders delusional parasitosis was seen in 10 cases (52.63%), lichen simplex chronicus in 8 cases (42.10%) and prurigo nodularis in 1 case (5.26%).

TABLE 15 : MISCELLANEOUS CONDITIONS

S.No	Condition	No of Cases	Percentage (%) N=39
1.	Amyloidosis(macular, lichen)	10	25.65
2.	Leg ulcers	8	20.5
3.	Vitiligo	5	12.84
4.	Chronic urticaria	4	10.25
5.	Acrokeratoelastoidosis marginalis	3	7.69
6.	Granuloma annulare	3	7.69
7.	Colloid milium	2	5.13
8.	Lichen scleroses et atrophicus	2	5.13
9.	Perforating dermatoses	1	2.56
10.	Pyogenic granuloma	1	2.56
Total		**39**	**100**

Among the 39 miscellaneous skin conditions we have seen macular and lichen amyloidosis in 10 cases (25.65%), leg ulcers in 8 cases (20.5.%),vitiligo in 5 cases (12.84%), chronic urticaria in 4 cases (10.25%), granuloma annulare and acrokertoelastoidosis marginalis in 3 cases each (7.69%), colloid milium and lichen scleroses et atrophicus in 2 cases each (5.13%), perforating dermatoses and pyogenic granuloma in 1 case each (2.56%). We couldn't find incidence of granuloma annulare, colloid milium, perforating dermatoses and pyogenic granuloma in various literatures.

TABLE 16 : NAIL CHANGES IN OUR STUDY

S.No	Condition	No of Cases	Percentage (%) N=193
1	Loss of luster	93	48.18
2	Longitudinal ridging	52	27.0
3	Subungual Hyperkeratoses	12	6.21
5	Beau's lines	3	1.55
6	Pitting	3	1.55
7	Onychomycosis	8	4.14
8	Thickening	22	11.39
	Total	**193**	**100**

Out of 193 nail changes loss of luster was the commonest nail change seen in 93 cases (48.18%), followed by longitudinal ridging in 52 cases (27%), nail plate thickening in 22 cases (11.39%), subungual hyperkeratosis and in 12 cases (6.21%), onychomycosis in 8 cases (4.14%), beau's lines and pitting in 3 cases each (1.55%). In some of these cases combinations of finding was seen.

TABLE 17 : HAIR CHANGES IN ELDERLY

No	Condition	No of Cases
1	Diffuse hair loss in females	37
2	Androgenetic alopecia in males	70

In our study graying of hair was seen in all cases 150 (100%). Among 57 females diffuse hair loss was seen in 37 cases (64.91%) and out of 93 males androgenetic alopecia was seen in 70 cases (75.26%). One case of plica polonica of hair was seen in our study.

Color Plates

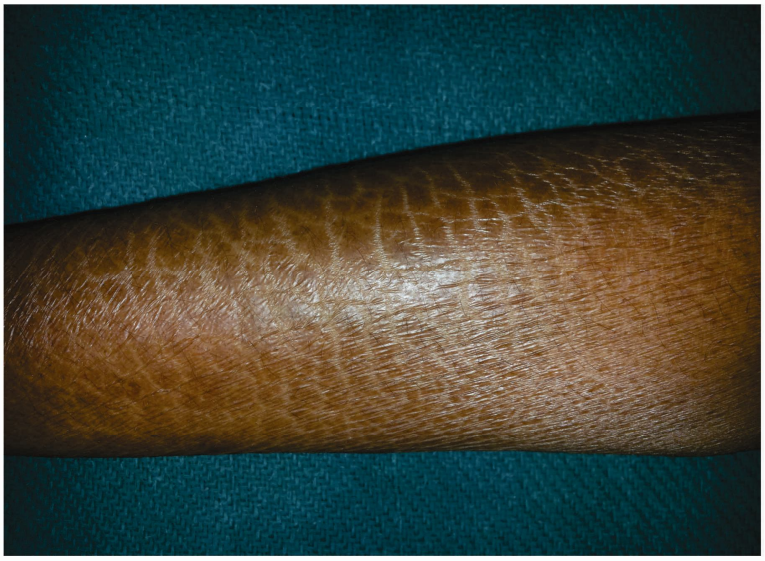


Fig : 1 Xerosis

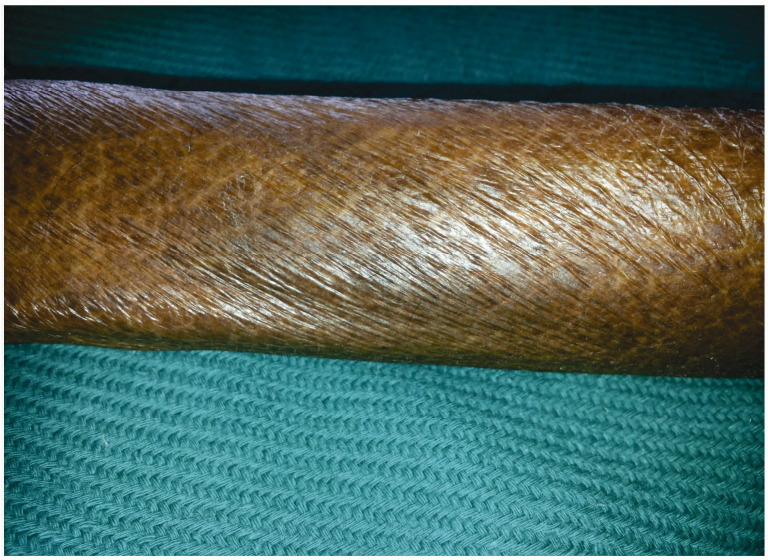


Fig : 2 Wrinkling

Fig : 3 Crow's feet

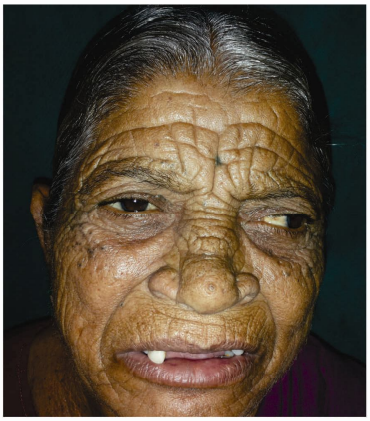


Fig : 4 Glyphic wrinkles

Fig : 5 Senile Comedones

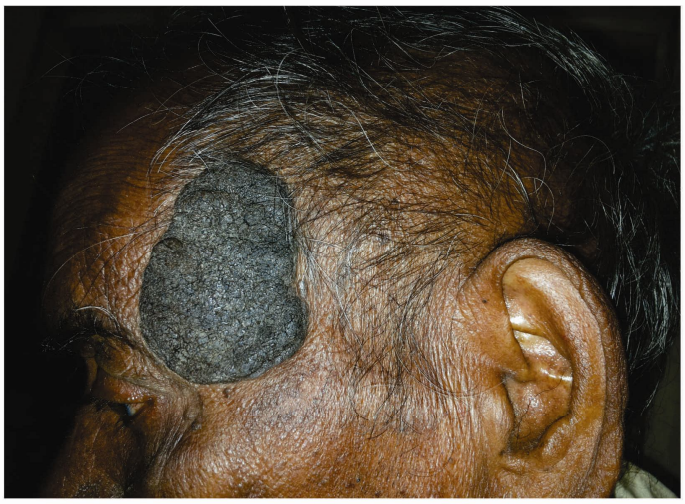


Fig : 6 Seborrhoeic Keratosis

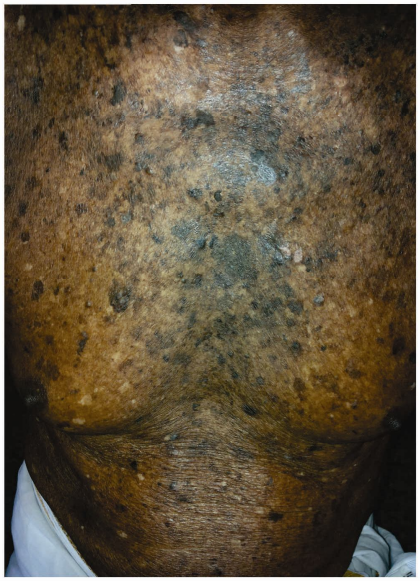


Fig : 7 Seborrhoeic Keratosis with IGH

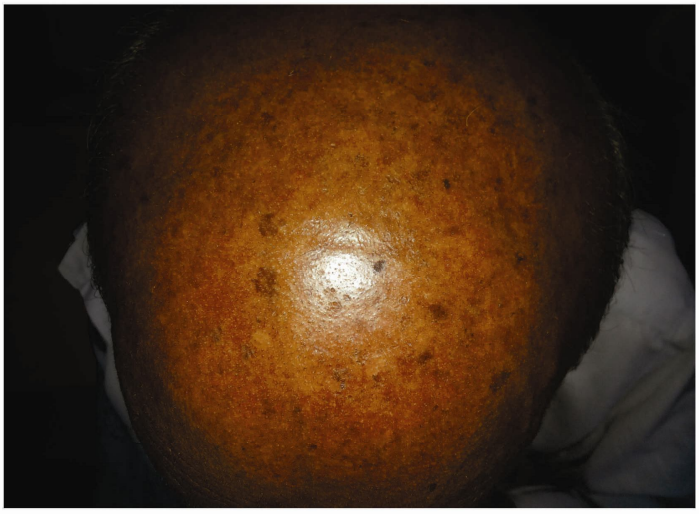


Fig : 8 Senile Lentigenes

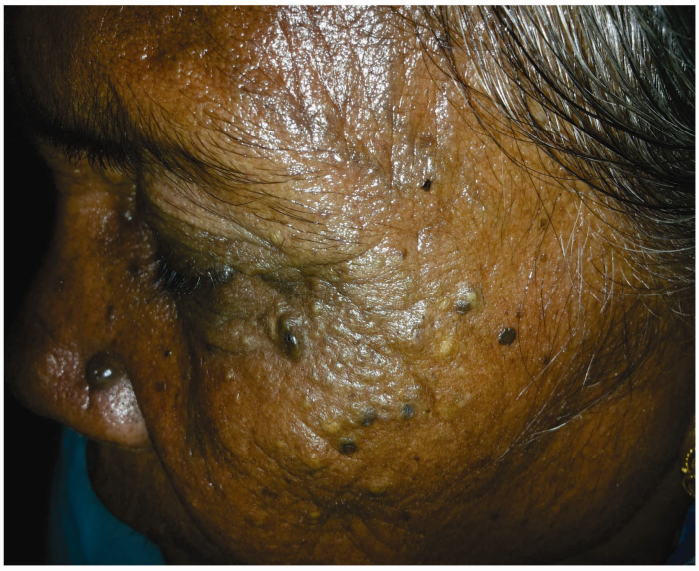


Fig : 9 Dermatosis papulos nigra

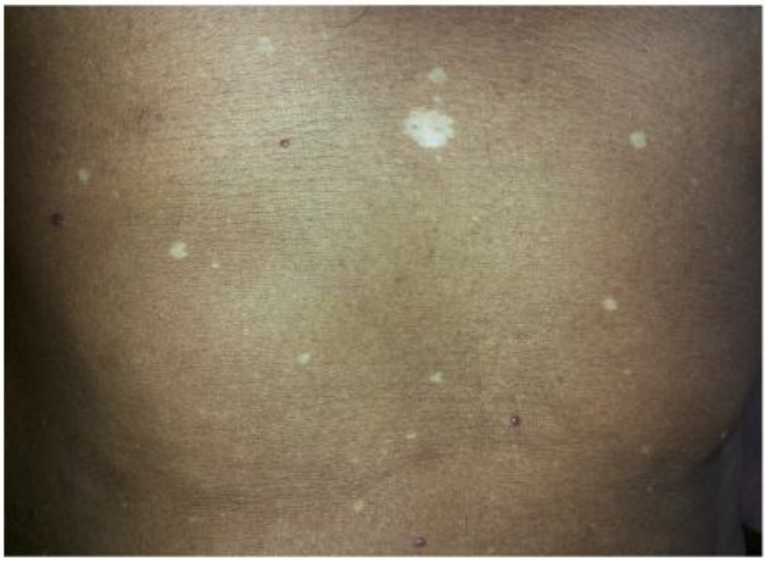


Fig 10 : IGH

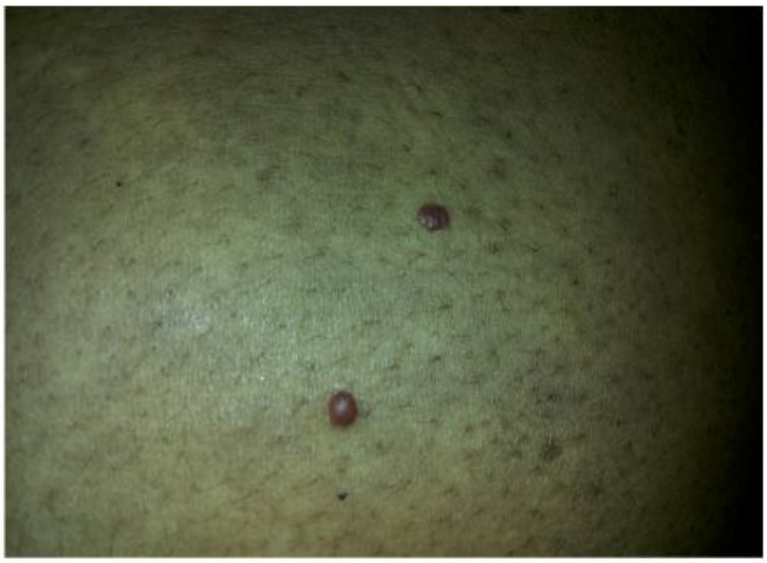


Fig 11 : Cherry angiomas

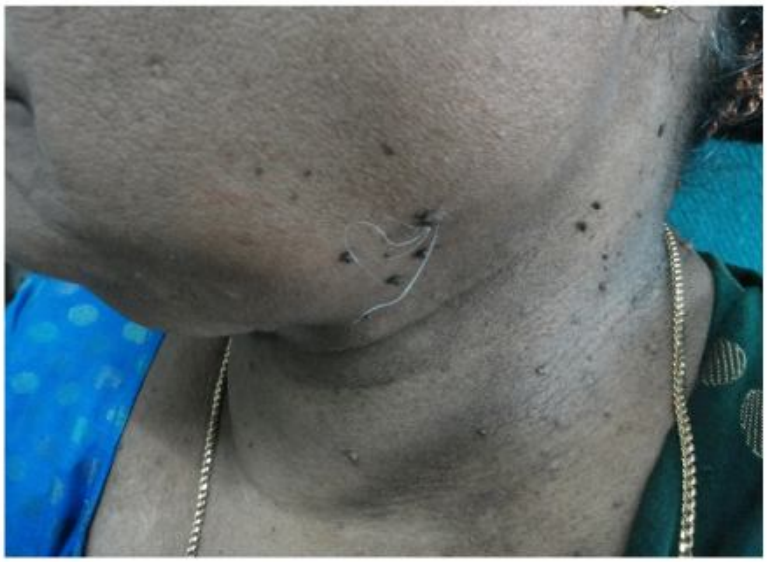


Fig 12 : Acrochordons

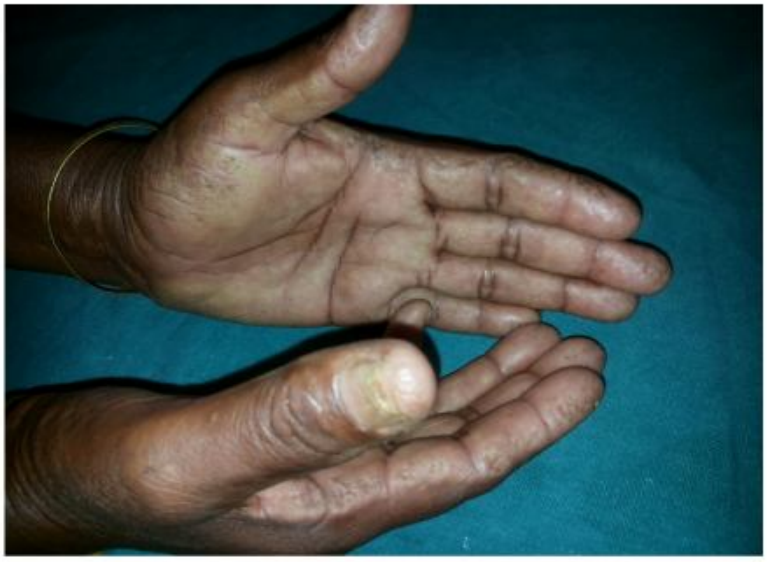


Fig 13 : Acrokeratoelastoidosis marginalis

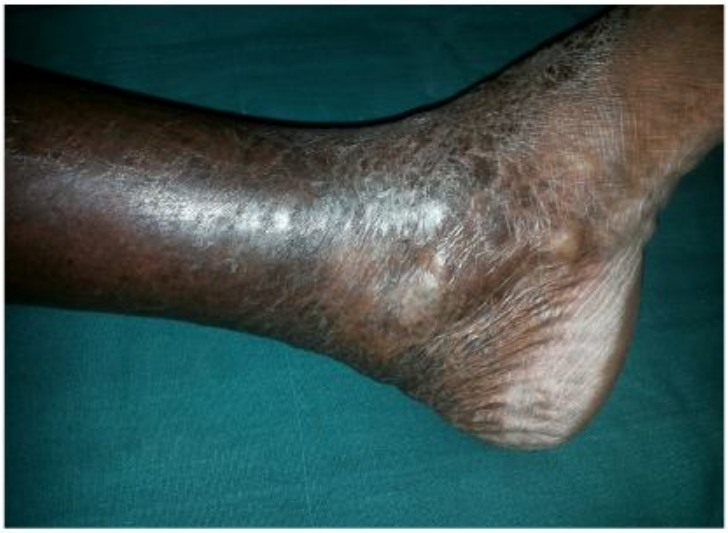


Fig 14 : Chronic Eczema

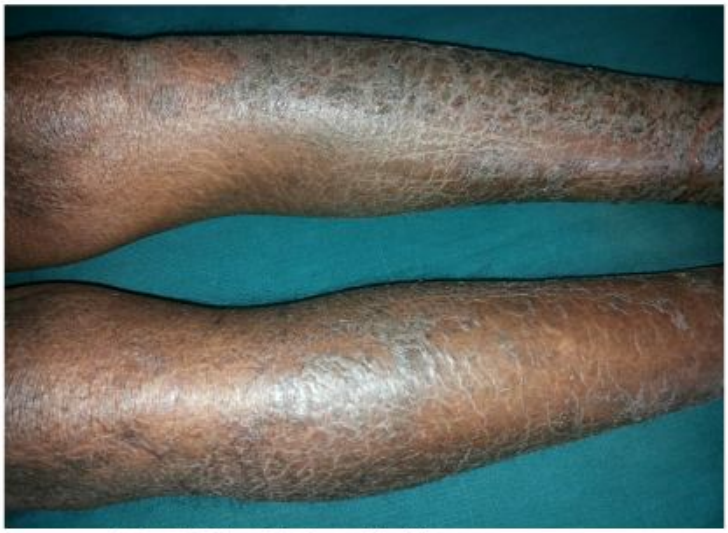


Fig 15 : Asteatotic eczema

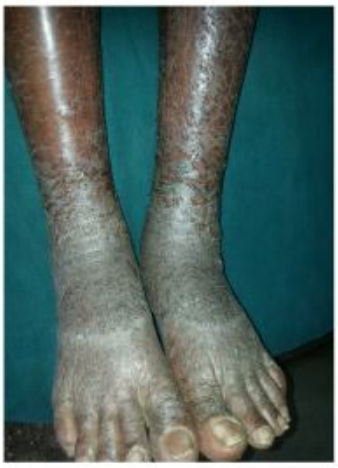


Fig 16 : Infections Eczematous dermatitis

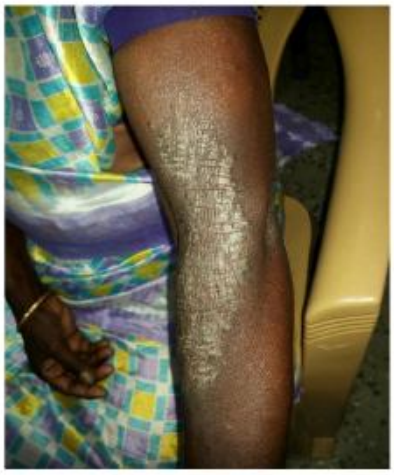


Fig 17 : LSC

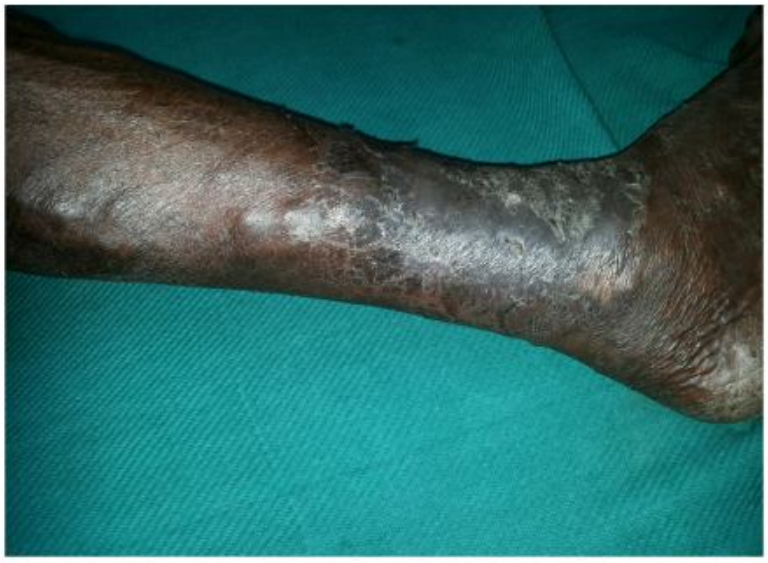


Fig 18 : Stasis Eczema

Fig 19 : Contact Dermatitis to Eye Drops

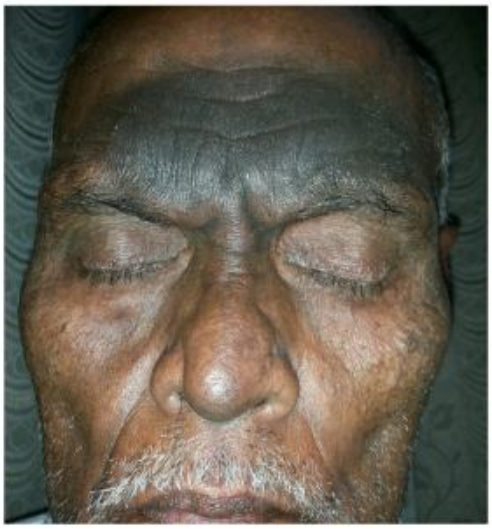


Fig 20 : Contact Dermatitis to kumkum

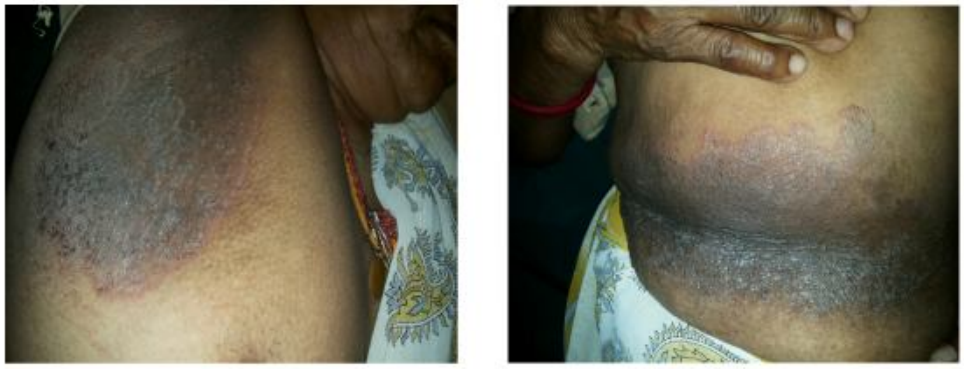


Fig 21 : Tinea corporis

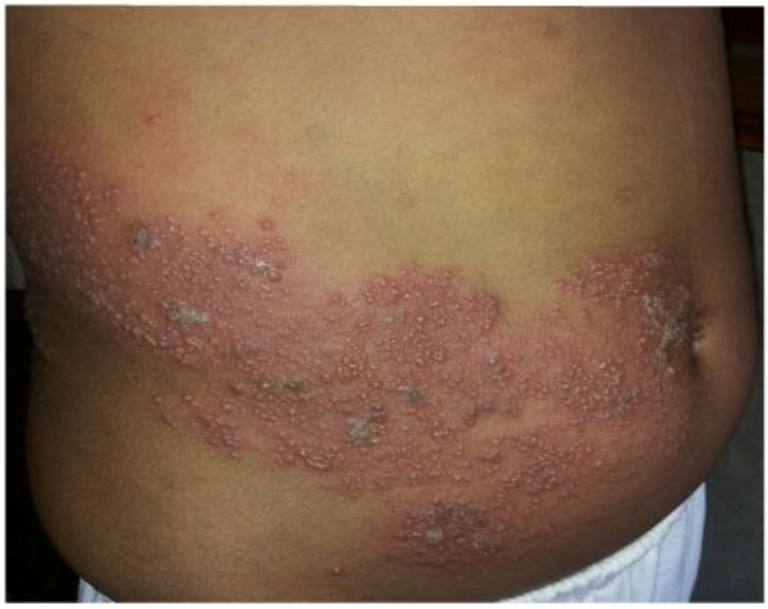


Fig 22 : Herpes zoster

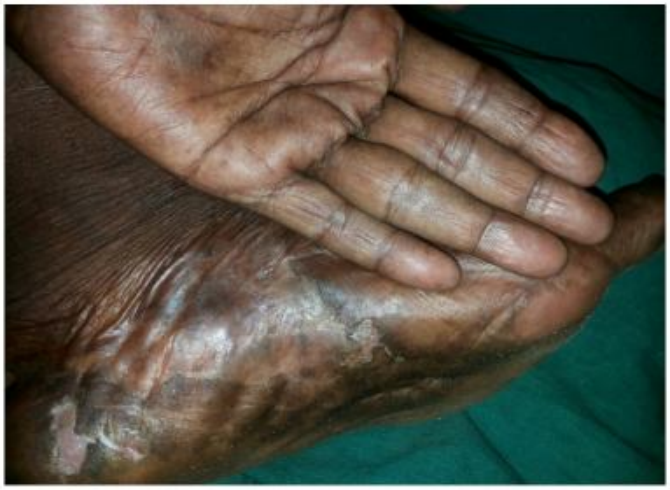


Fig 23 : Palmo Plantar Psoriasis

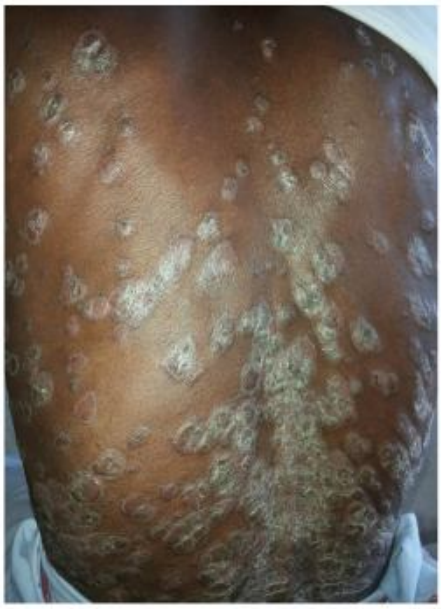


Fig 24 : Chronic Plaque Psoriasis

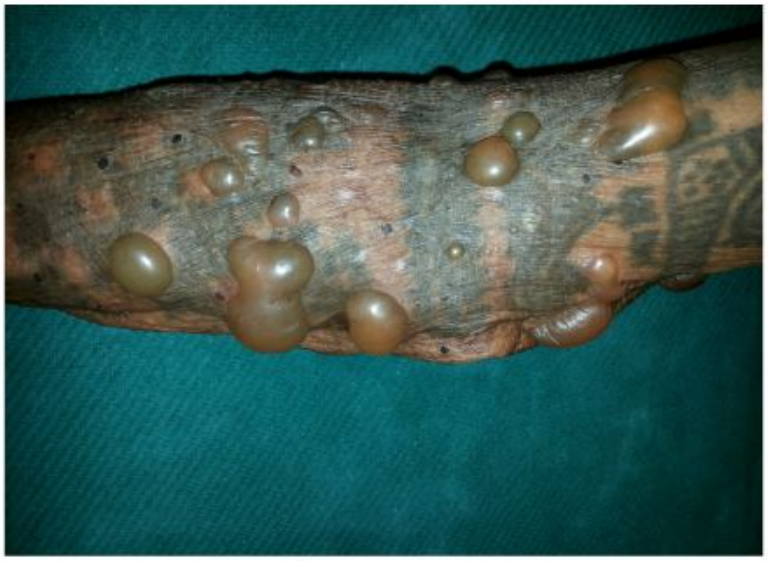


Fig 25 : Bullous Pemphigoid

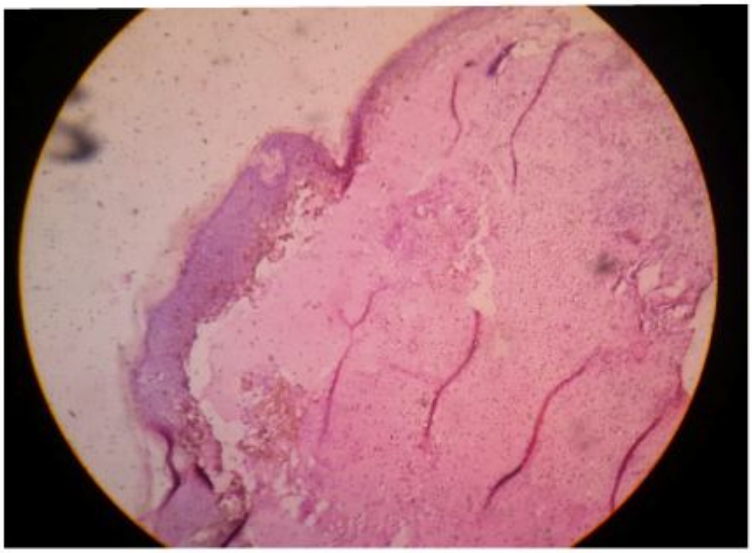


Fig 26 : Sub epidermal bulla (HPE)

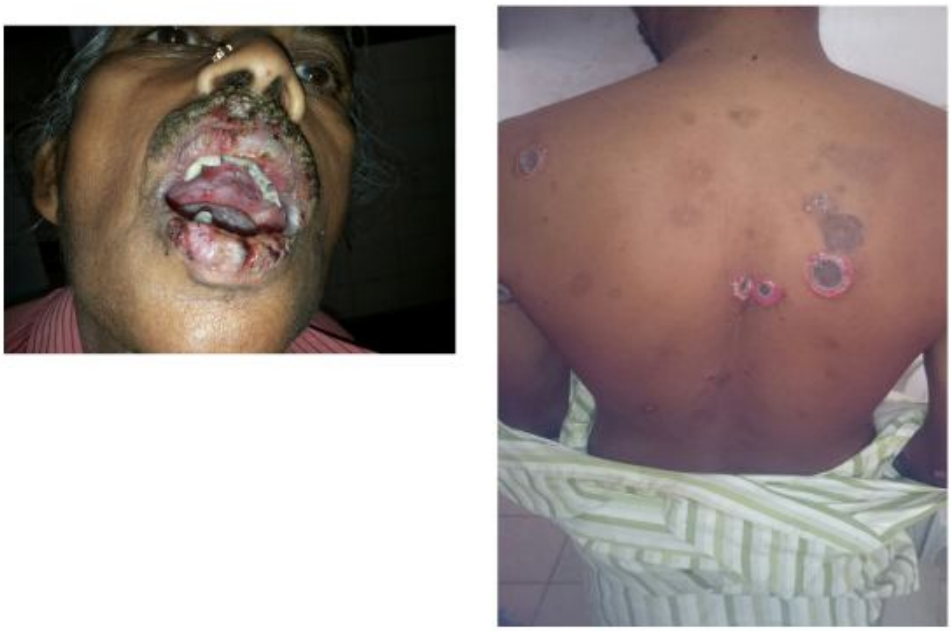


Fig 27 : Pemphigus Vulgaris

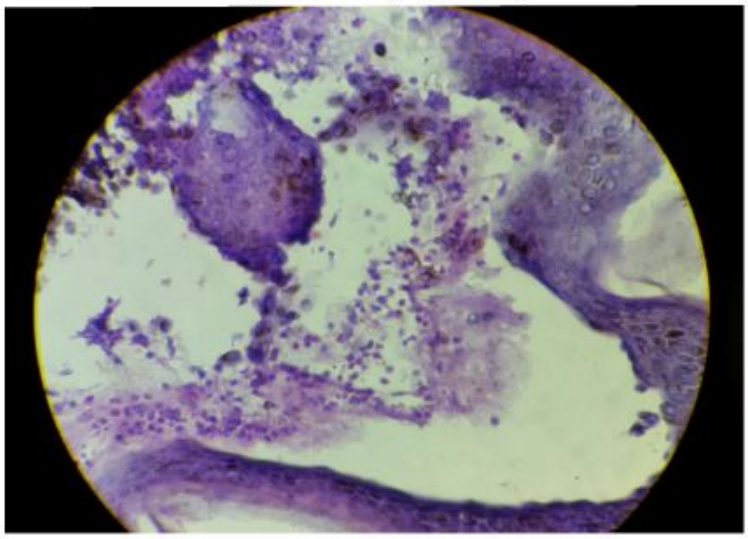


Fig 28 : Intra Epidermal Bulla (HPE)

Fig 29 : Squamous Cell Carcinoma of Lip

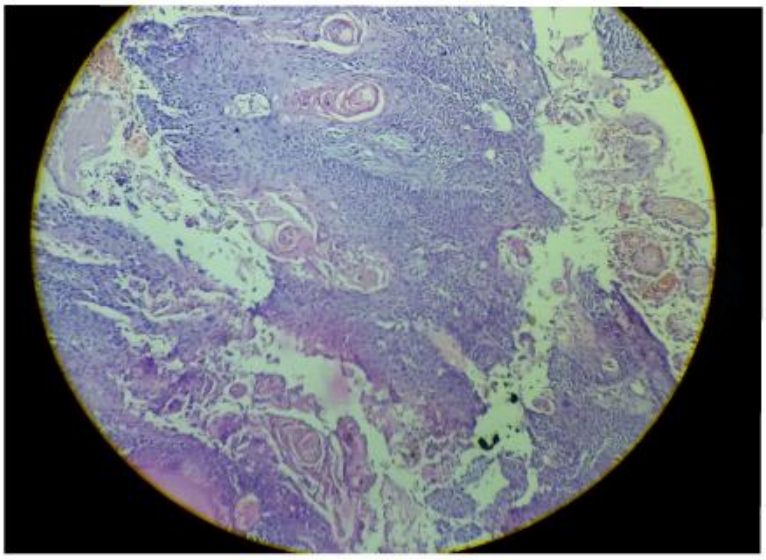


Fig 30 : HPE Showing keratin pearls

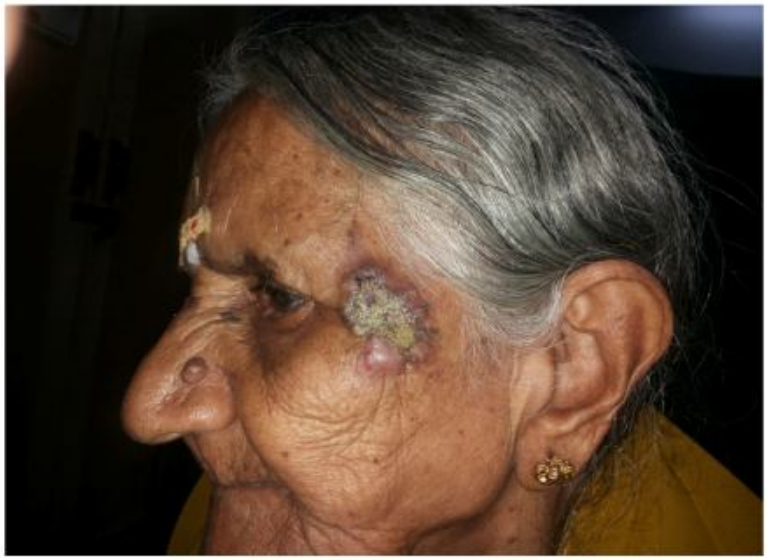


Fig 31 : Basal cell carcinoma

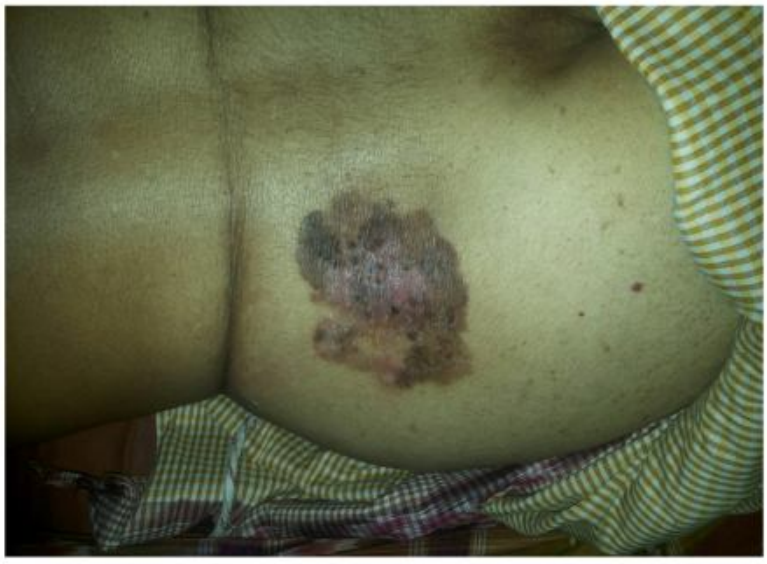


Fig 32 : Bowen's Disease

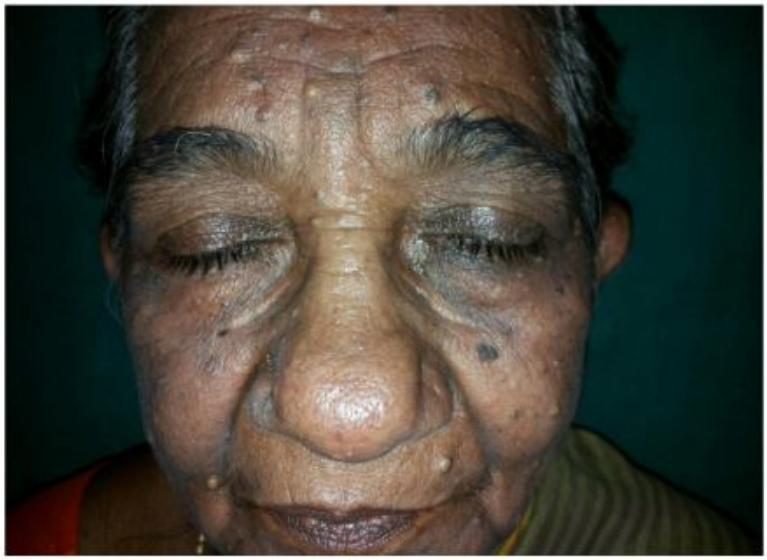


Fig 33 : Colloid Milia

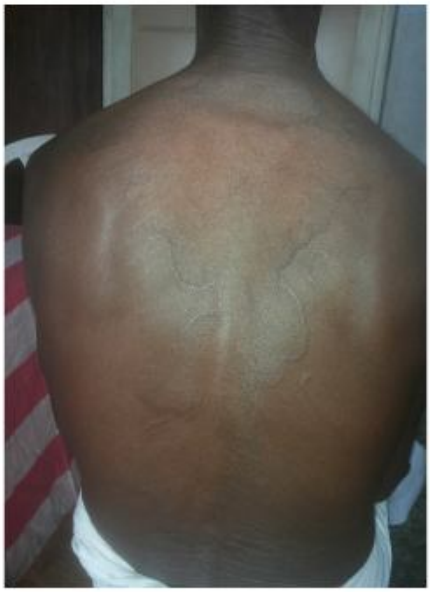


Fig 34 : Granuloma annulare

Fig 35 : Vitiligo

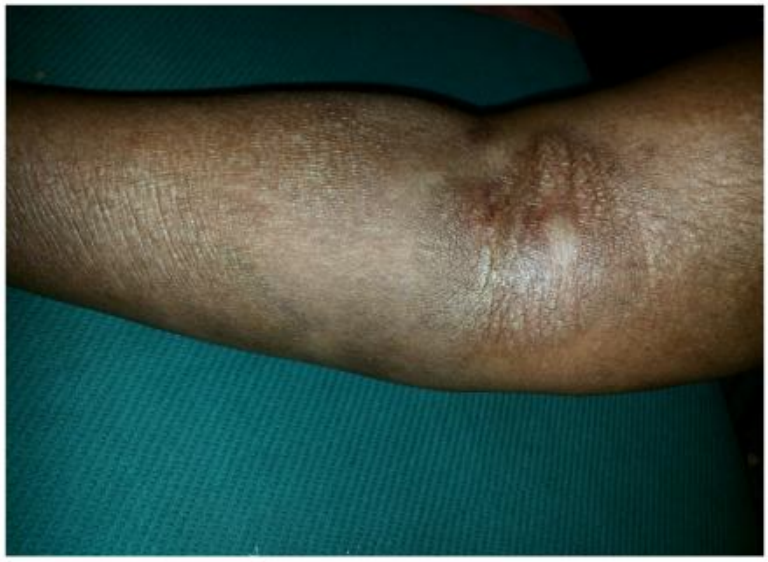


Fig 36 : LSEA

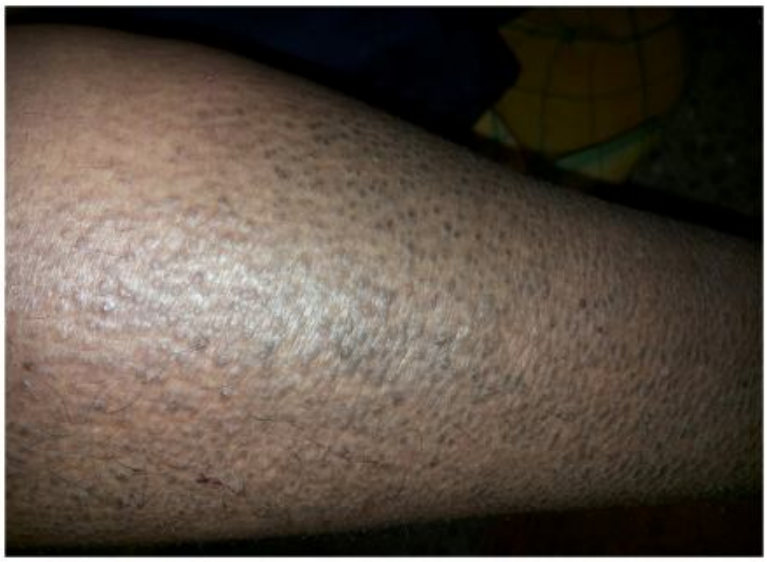


Fig 37 : lichen amyloid

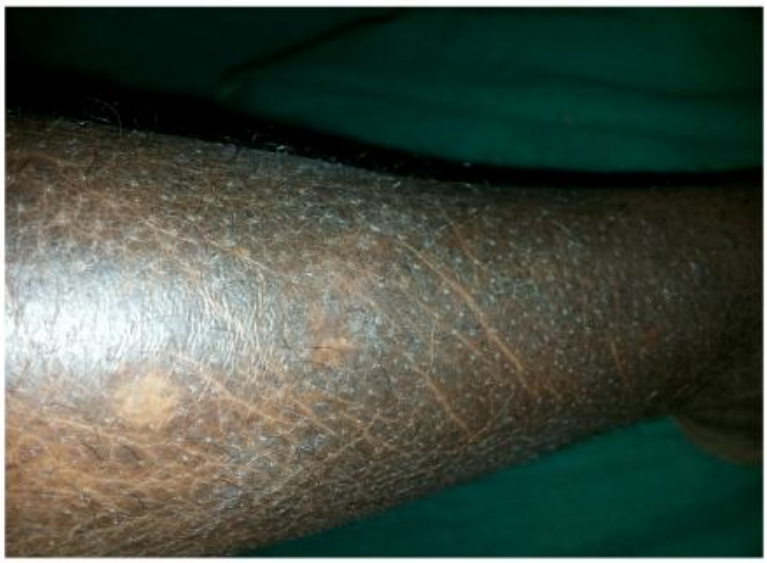


Fig 38 : Macular amyloid

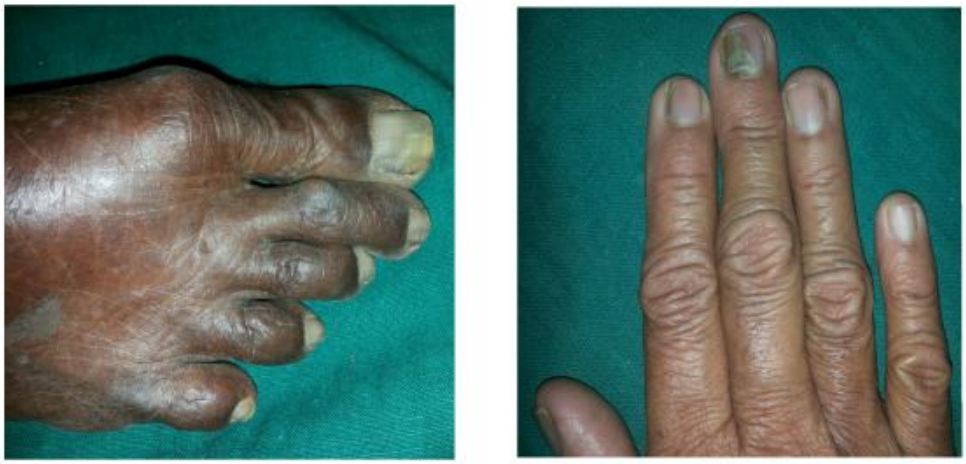


Fig 39 : Onychomycosis

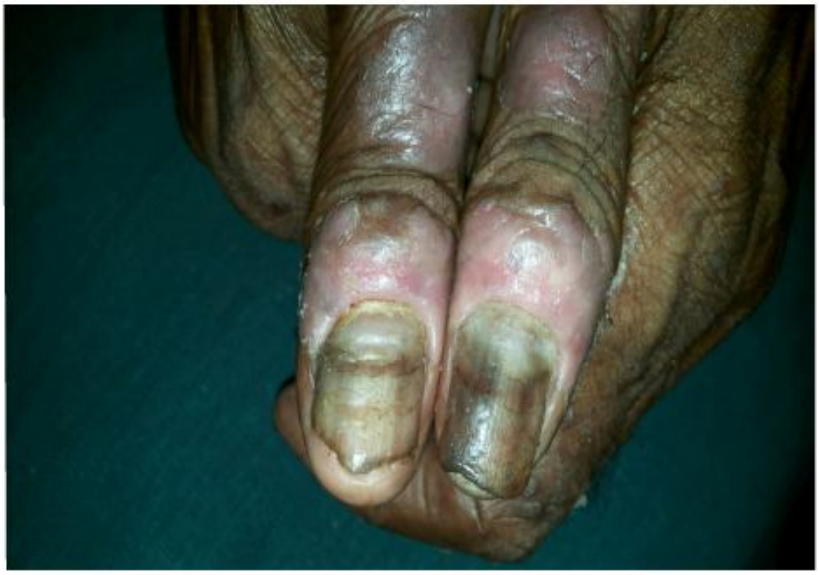


Fig 40 : Pitting and Beau's lines

Fig 41 : Plica Polonica

Fig 42 : Gray hair

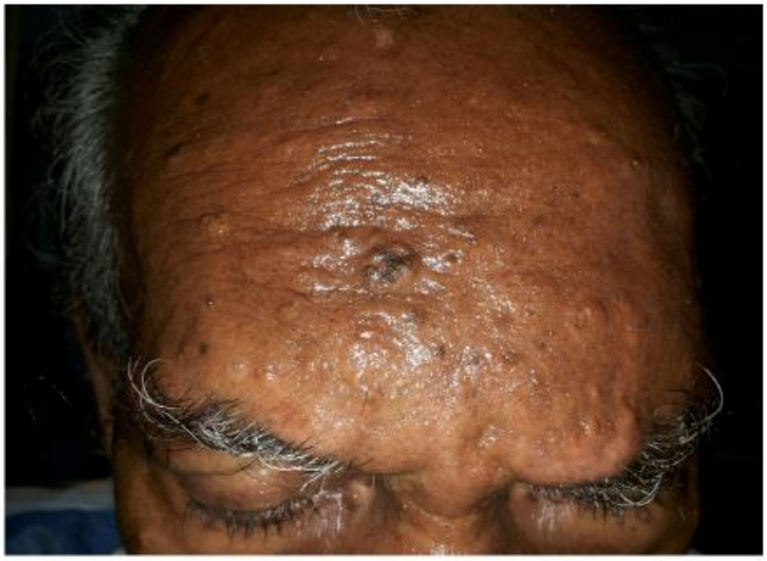


Fig 43 : Sebaceous hyperplasia

Fig 44 : Angiokeratoma of Fordyce

Discussion

DISCUSSION

Ageing is defined as an irreversible process, beginning or accelerating at maturity, which results in an increased range or number of deviations from an ideal state. Old skin is dry and rough, atrophic, wrinkled, unevenly pigmented, shows loss of elasticity, and is prone to develop a number of tumors.[3] Ageing skin has susceptibility to dermatological disorders due to the structural and physiological changes that occur as a consequence of intrinsic and extrinsic ageing.

1. NUMBER OF CASES IN RELATION TO AGE AND SEX

In this study, a total of 150 patients varying in age from 60-91 years were examined. Of these, 93 patients (62%) were males and 57 (38%) were females. The eldest patient was 91 years of age. Male to female ratio was 1.63:1 Patange and Fernandez[128] studied 200 cases in an OPD setting, aged 55-85 years of age, of which 63% were males and 37% were females. Our study was comparable to Patange and Fernandez study. Priya Cinna and Thappa[129] did a hospital based descriptive study on 500 elderly (males aged 60 years and above and females aged 50 years and above). In their study female predominance was seen with female to male ratio 1.34:1. Out of 500, 213 were males (42.6%) and 287 (57.4%) were females.

Verbov[130] examined 170 consecutive patients aged 60-90 years in an OPD. Beauregard and Gilchrist[131] studied 68 patients aged between 50-91 years. Droller[132] made a study on random cases of 476 individuals. Out of this, 192 were men and 284 were women, all patients between 60-90 years of age. Weismann et al[133] studied 494 residents of a Danish home for the aged between 55 and 106 years age. Tindall and Smith[134] studied 163 volunteers, all above 64 years of age.

2. ASSOCIATED CONDITIONS

Among 150 cases in this study, 116 had associated systemic illness. This is higher than found in other studies. Diabetes mellitus was the commonest association seen in 45 (30%), followed by hypertension in 40 cases (26.7%).

Patange and Fernandez[128] observed associated systemic ailments in 30% of cases. In a study by Priya Cinna and Thappa,[129] Diabetes (28.9%) and Hypertension (25.5%) were the commonest associated condition which is similar to our study. Beauregard and Gilchrest[131] described 89.7% of patients to have major medical illnesses, of which 88.2% were on medication.

3. SYMPTOMATOLOGY

Among 150, generalized pruritus was seen in 96 cases (64%), of which xerosis was associated with generalised pruritus in 66 cases (44%), diabetes mellitus in 20 cases (13.3%), anaemia in 5 cases (3.3%), kidney disease in 3 cases (2%). hypothyroidism in 1 case (0.6%), liver disease in 1 case (0.6%), Patange and Fernandez[128] noticed generalized pruritus in 78.5% of cases, Priya Cinna and Thappa[129] observed in 49.6% of cases, of whom 29.8% were associated with xerosis. Beauregard and Gilchrist[131] found in 29% patients.

Incidence of generalized pruritus in our study was lower than that of Patange and Fernandez but comparable to the study conducted by Priya Cinna and Thappa and Beauregard and Gilchrist

4. SKIN CHANGES WITH AGEING

Ageing skin is particularly vulnerable to environmental insults due to the structural and physiological changes that occur as a consequence of both intrinsic and extrinsic ageing.

a) Xerosis

It literally means dry skin. It is called as asteatotic eczema, when it is associated with eczematous changes. It was a common finding in our study and observed in 96 patients out of 150 (64%).

Chopra et al[135] noticed in 108 (50.8%) cases. Beauregard and Gilchrist[131] found in 85% of cases. Tindall and Smith[134] observed an incidence of 77%. Our study was similar to Chopra et al observation.

b) Wrinkling

This was a next most common finding in our study and observed in 84 patients out of 150 (56%). This incidence was lower than found in other studies. Beauregard and Gilchrist[131] observed in (95.6%). Patange and Fernandez[128] do not mention the incidence of wrinkling. Tindall and Smith[134] in (94%) and Priya Cinna and Thappa[129] reported an incidence of 100% in their study. In our study most of the wrinkling was observed on sun exposed areas like face, neck, forearms, dorsa of hands.

c) Idiopathic guttate hypomelanosis

In our study among 150 cases IGH was seen in 72 (48%). This observation was higher than seen in other studies. Priya Cinna and Thappa[129] found it in 26% (130 cases). Beauregard and Gilchrist[131] in 24.4% of cases. In our study most lesions are observed in non sun exposed areas like chest and shins of lower limb.

d) Senile comedones

Among 150, it was found in 25 cases (16.6%) in this study which was comparable to the study conducted by Patange and Fernandez[128], who reported an incidence of 11.5%. Priya Cinna and Thappa[129] found it in 23 cases (4.6%). Grover and Narasimhalu[136] seen in 13 cases (6.5%).

e) Senile lentigenes

In our study senile lentigenes was found in 12 cases out of 150 (8%).It was comparable to the study conducted by Patange and Fernandez[128] who observed an incidence of 12%. Beauregard and Gilchrist[131] noticed it in 70.6%. Tindall and Smith[134] reported an incidence of 51%. Racial influence could be the cause for lower incidence seen in our study.

4) PATHOLOGICAL SKIN CHANGES

In this study following pathological skin conditions were observed : Eczematous conditions, Papulosquamous disorders, infections, benign skin tumours, premalignant and malignant skin tumours, bullous disorders, psychocutaneous disorders, drug reactions and miscellaneous skin changes.

a) Eczematous conditions

Among 150 cases, eczematous conditions were found in 48 (32%) cases in our study. Of these asteatotic eczema was found in 10 cases (6.6%). Stasis eczema in 6 cases (4%), airborne contact dermatitis in 6 cases (4%), infectious eczematous dermatitis in 5 cases (3.3%), contact dermatitis in 3 cases (2%) and seborrhoeic dermatitis in 1 case (0.6%).

Patange and Fernandez[128] found contact dermatitis in 7.5%. Priya Cinna and Thappa[129] observed an incidence of eczema in 24.2%. Beauregard and Gilchrist[131] reported an incidence of contact dermatitis in 11.8%, stasis eczema in 5.9%, seborrheic dermatitis in 10. 5% Verbov[130] noticed an incidence of

24.7%. Weismann et al[133] study showed an incidence of seborrheic dermatitis in 7%, stasis dermatitis in 6.9% and contact dermatitis in 3.8%. Johnson[137] observed contact dermatitis in 2% and seborrehic dermatitis in 3.6%.

Incidence of eczema in our study was higher than found in other studies. Our study was similar to Johnson study in contact dermatitis and to Beauregard and Gilchrist observation in stasis eczema.

a) Papulosquamous disorders:

It includes psoriasis and lichen planus in this study.

In this study psoriasis was seen in 12 cases out of 150 (8%),which was similar to that seen by Patange and Fernandez[128] who observed it in 10.5% of cases. Tindall and Smith[134] found in 7 cases (3.5%). Beauregard andGilchrist[131] reported an incidence of 2.9%.

Out of 150, lichen planus was seen in 5 cases in our study (3.3%). Our study was comparable with a study by Sahoo, Singh et al[138] who reported 5% incidence of lichen planus.

b) Infective conditions

Among 150 cases, infective conditions were seen in 51 patients (34%) in this study, out of these fungal infections were seen in 28 cases (18.6%), bacterial infections were seen in 12 cases (8%), viral infections in 11 cases (7.3%).

Among the fungal infections dermatophytosis was seen in 23 cases (15.3%) and candidiasis was seen in 5 cases (3.3%). Among bacterial infections cellulitis was seen in 3 cases (2%), folliculitis in 4 cases (2.6%) and furuncle in 5 cases (3.3%). Among viral infections herpes zoster was seen in 8 cases (5.3%) and 3 cases (2%) of viral warts were seen.

Patange and Fernandez[128] have observed infective dermatoses in 34.5% of the total dermatoses. Of these, fungal infection was seen in 17.5%, bacterial infection in 8.5% and viral infections in 5%. Beauregard and Gilchrist[131] found dermatophytosis in 17.7%. Priya Cinna and Thappa[129] noticed in 46.8%, of which fungal infections were seen in 34.4%, bacterial infections in in 0.8%, viral infections in 0.6%. Tindall and Smith[134] observed dermatophytosis in 79%, but they have not mentioned about other infective conditions. Johnson[137] found 12.7% incidence of dermatophytosis.

Infective conditions of our study was comparable with the findings of Patange and Fernandez. The incidence of dermatophytosis in this study is similar to the study conducted by Patange and Fernandez and Beauregard and Gilchrist.

c) Benign tumours of the skin

Out of 150 cases seborrheic keratoses was seen in 87 (58%) cases. Cherry angioma in 79 (52.6%) cases, dermatosis papulosa nigra in 70 (46.6%) cases, achrochordons in 47 (31.3%) cases.

Patange[128] found seborrheic keratoses in 37.5%, cherry angioma in 46.5%, and achrochordons in 24.5% of cases. Priya Cinna and thappa[129] noticed seborrheic keratosis in 253 cases (50.6%), cherry angioma in 36 (7.2%) cases and acrochordons in 49% and sebaceous hyperplasia in 1.6% of cases.

Beauregard and Gilchrist[131] found seborrhic keratoses in 61.2%, cherry angioma in 53.7% cases and dermatosis papulosa nigra in 58.8% of cases. Tindall and Smith[134] observed seborrheic keratoses in 88% and cherry angioma in 75% of cases. Grover and Narasimmhalu[136] reported seborrheic keratosis in 43% and cherry angioma in 63% of cases.

The incidence of seborrheic keratosis, dermatosis papulosa nigra and cherry angioma were comparable to that of Beauregard and Gilchrist observation. Incidence of sebaceous hyperplasia was comparable to Priya Cinna and Thappa study. Incidence of angiokeratoma of Fordyce and syringoma are not mentioned in any of these studies.

d) Premalignant and malignant tumours

Among the premalignant conditions 1 case (0.6%) of Bowen's disease was seen in our study. The reason is predominant skin type of our population is IV/V, which is resistant to UV light induced damage.

There were 3 cases (2%) of basal cell carcinoma and 1 case (0.6%) of squamous cell carcinoma were seen in this study among the malignant tumours (2.6%). It is comparable to the study conducted by Priya Cinna and Thappa[129] who reported 5 cases of malignancy (1%). Verbov[130] found higher (13.5%)

incidence of malignancy and Beauregard and Gilchrist[131] observed 4.4% of skin cancer.

e) Bullous disorders

Among 150, bullous disorders were seen in 10 (6.6%) cases, out of which bullous pemphigoid was seen in 7 cases (4.6%) and pemphigus vulgaris seen in 3 cases (2%).

Priya Cinna and Thappa[130] found pemphigus vulgaris in 9 cases (1.8%), which is similar to our study.They have reported incidence of bullous pemphigoid in 8 cases (1.6%). Chopra et al [135] observed 4 cases of bullous pemphigoid (1.8%).

f)Psychocutaneous disorders

Out of 150, total number of of psychocutaneous disorder in this study was 19 cases (12.6%), lichen simplex chronicus was seen in 8 cases (5.3%) delusional parasitosis in 10 cases (6.6%) and prurigo nodularis was seen in 1 case (0.6%).

Incidence of lichen simplex chronicus in a study conducted by Chopra et al[135] was 13.5%. Patange and Fernandez[128] observed lichen simplex chronicus in 12% of cases. Chopra[135] et al found delusional parasitosis in 6 cases (8%), which was comparable to our study. The incidence of other psychocutaneous disorders in our study was lower than that found in other studies.

f) Miscellaneous skin conditions

In our study, cutaneous amyloidosis (macular, lichen) was seen in 10 cases (6.6%), leg ulcers seen in 8 cases (5.3%), chronic urticaria was seen in 4 cases (2.6%), vitiligo in 5 (3.3%)cases, granuloma annulare and acrokeratoelastoidosis marginalis in 3 cases each (2%), each of colloid milia and lichen scleroses et atrophicus in 2 cases each (1.3%), 1 case (0.6%) each of perforating dermatoses and pyogenic granuloma were seen.

Weismann and Krakauer et al[133] found 2.2% incidence of leg ulcers. Priya Cinna and Thappa[129] reported an incidence of 0.2% for colloid milia and 0.9% of acrokeratoelastoidosis marginalis in their study. We couldn't find incidence of other conditions in previous studies.

HAIR CHANGES

Graying of hair was seen in nearly all cases. In our study 64.91% of females showed diffuse thinning of hair.Androgenetic alopecia was seen in 70 out of 93 males (75.26%). Interestingly one case of plica polonica (matted hair) was seen in our observation.

In a study conducted by Patange [128] male pattern baldness was seen in 20 cases and diffuse hair loss in 94 females in their study of 200 patients. Priya Cinna and Thappa[129] found graying in 97.2% males and 90.9% females. Diffuse hair thinning was seen in 67.24% males and androgenetic alopecia was seen in 55.39% males by Chopra[135] et al observation.

NAIL CHANGES

Loss of luster was the commonest nail finding in our study seen in 93 cases (62%), longitudinal ridging was found in 52 cases (34.6%), nail plate thickening was seen in 22 (14.6%) cases and onychomycosis was noticed in 8 cases (5.3%).

Patange and Fernandez[128] observed loss of luster in 20.5%. Priya Cinna and Thappa[129] found loss of luster in 50.8%, vertical ridging in 24% and onychomycosis in 22.5%. Grover and Narasimmhalu[136] noticed loss of luster in 64%, vertical ridging in 72.5%, onychomycosis in 12% . Our study was comparable with Priya Cinna and Thappa and Grover and Narasimmhalu in loss of luster. Incidence of longitudinal ridging, beau's lines, pitting was comparable to Priya Cinna and Thappa study, (24%), (0.2%), (0.4%) respectively. Incidence of onychomycosis was lower than found in other studies.

Summary

SUMMARY

- In this study a total of 150 patients aged 60 years and above attending the OPD of Coimbatore Medical College Hospital were included.

- Maximum number of patients in this study belonged to 60-65 years (37%), followed by 66-70 years (30%). The eldest patient was of 91 years.

- There were 93 males (62%), 57 females (38%), in this study. Male to female ratio was 1.63:1.

- Most of the males had agriculture work and most of the females were housewives.
- Out of 150 cases 116 cases had associated systemic diseases. Diabetes mellitus was the commonest associated systemic disease seen in 45 cases (30%), followed by hypertension in 40 cases (26.7%).

- Among 150, generalized pruritus was the commonest symptom seen in 96 (64%) cases, of which 66 cases (44%) were associated with xerosis.

- In our study out of 150 cases, xerosis of the skin was one of the commonest finding seen in 96 cases (64%). Wrinkling was seen in 84 cases (56%), IGH in 72 cases (48%), senile comedones in 25 cases (16.6%) and senile lentigenes in 12 cases (8%).

- Among the pathological skin disorders eczematous conditions were seen in 48 out of 150 cases. Of these chronic eczema was the common finding among the eczematous conditions seen in 12 cases (8%).
- In our study among the papulosquamous disorders, psoriasis was seen in 12(8%) among 150 cases and lichen planus in 5 cases (3.3%).

- Among 150 cases infectious diseases were seen in 51cases (34%) in this study, of these fungal infections were common seen in 28 cases (`18.6%), bacterial infections in 12 cases (8%), viral infections in 11 cases (7.3%). Of the 28 cases of fungal infections, dermatophytosis was seen in 23 cases (15.3%), candidiasis in 5 cases (3.3%). Among the bacterial infections cellulitis was seen in 3 cases (2%), folliculitis in 4 cases (2.6%), furuncle in 5 cases (3.3%). Among viral infections herpes zoster was seen in 8 cases (5.3%) and viral warts in 3 cases (2%).

- Out of 150 the most common benign tumour was seborrheic keratosis in this study seen in 87 cases (58%), cherry angioma in 79 cases (52.66%), dermatosis papulosa nigra in 70 cases (46.66%), achrochordons in 47 cases (31.33%), sebaceous hyperplasia in 2 cases (1.33%) and angiokeratoma of Fordyce and syringoma in 1 case (0.6%) each.

- In this study among 150, 1 case (0.6%) of premalignant tumour, Bowen's disease was seen. Among the malignant tumours 3 cases (2%) of Basal cell carcinoma and 1 case (0.6%) of squamous cell carcinoma was seen.

- Among 10 cases of bullous disorders, bullous pemphigoid was seen in 7 (4.6%) cases and pemphigus vulgaris in 3 cases (2%).

- Among 19 cases of psychocutaneous disorders, delusional parasitosis was seen in 10 cases (6.6%), lichen simplex chronicus in 8 cases (5.3%) and prurigo nodularis in 1case (0.6%).

- In this study among the miscellaneous skin conditions (out of 150) chronic urticaria was seen 4 cases (2.6%), amyloidosis (both macular and lichen) in 10 cases (6.6%), leg ulcers in 8 cases (5.3%), vitiligo in 5 cases (3.3%), granuloma annulare and acrokeratoelastoidosis marginalis in 3 cases each (2%), colloid milia and lichen scleroses et atrophicus in 2 cases each (1.3%), perforating dermatoses and pyogenic granuloma in 1 case each (0.6%).

- In our study among 150, loss of luster was the commonest nail change seen in 93 cases (62%), followed by longitudinal ridging seen in 52 cases (34.6%), nail plate thickening in 22 cases (14.6%) and subungual hyperkeratosis in 12 cases (8%).

- Graying of the hair was seen in all cases. Out of 57 females diffuse hair loss was seen 37 cases (64.91%) and out of 93 males androgenetic alopecia was seen in 70 cases (75.26%). One case of plica polonica of hair seen.

- In this study 1 case of phenytoin induced drug rash was seen.

Conclusion

CONCLUSION

- In our study commonest age group was 60-65 years.
- Majority of the patients were males in this study.
- Most of the males had agriculture work and majority of the females were housewives.
- Commonest associated systemic disease was diabetes mellitus in our study.
- In our study commonest symptom was generalized pruritus and it was frequently associated with xerosis.
- In our study most common finding was xerosis, followed by wrinkling, IGH, senile lentigenes and senile comedones.
- Various pathological changes seen were eczematous conditions, papulosquamous disorders, infections, bullous disorders, benign and malignant tumors, psychocutaneous diseases and drug reactions.
- Commonest nail change was the loss of luster.
- Commonest hair change was graying of hair.

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PROFORMA

A CLINICAL STUDY OF CUTANEOUS MANIFESTATION

IN GERIATRIC AGE GROUP

Name: **Date:**

Age:

IP No. /OP No:

Sex:

Occupation:

Address:

Hospital: Coimbatore Medical College Hospital, Coimbatore.

Socioeconomic status: High/Middle/Low

Marital status: Married / single

Chief complaints:

History of presenting illness:

Past history:

H/O similar complaints in past

H/O Diabetes Mellitus/Hypertension/Asthma/Epilepsy/Drug allergy

H/O Leprosy/ autoimmune disease / Psoriasis / Connective tissue disease / Rheumatoid Arthritis / Infections.

H/O any other skin disease in past

Drug history:

Personal history:

Diet, Appetite, Sleep, Bowel & Bladder, Habits

Family history:

General physical examination:

BP: mmHg PR: /min Weight: kg

pallor /edema/cyanosis /clubbing /icterus /lymphadenopathy

Systemic examination:

Cardiovascular System:

Respiratory System:

Per Abdomen:

Central Nervous System:

Mucocutneous examination:

1.Erythematous/Non erythematous

Macules/Patch/ Papules /Plaques/ Vesicles/ Bulla

Wheals/ Nodules/ Excoriations /Crusting
Pigmentation :

Other skin lesions:

Localised/Generalised

Single/multiple

2. Sites: Exposed/Covered part

Face/Neck/Arm/Forearm/Back/Trunk/

Abdomen/Thigh/Legs/Genitilia

3. Pattern: Scattered/ Linear/Grouped/ Zosteriform

4. Spared Area:

5. Pre existing Skin Disease:

6. a) Xerosis

b) Tumors

c) Infections/Infestation

d) Eczema/Contact Dermatitis

e) Pigmentary disorders

f) Papulosquamous disorders

g) Metabolic disorder:

h) Disorders of Keratinization

i)Vesico Bullous disorders

j) Miscellaneous:

k) Urticaria

l) Pruritus

m) Other findings

n) Nail Changes

o) Hair Changes

p) Mucosal lesions: Oral mucosa:

Genital mucosa:

Investigations:

Complete haemogram with peripheral smear study, ESR, liver function tests, blood urea, random blood sugar, serum creatinine, lipid profile, serum electrolytes, thyroid profile, and stool for occult blood

Urine routine:

Dermatological procedures:

Skin scrapping and nail clipping for KOH mount to rule out fungal infection.

Tzanck smear:

Woods lamp:

Pus for culture and sensitivity:

Skin biopsy and direct immunofluorescence:

Diagnosis:

Treatment:

KEY TO MASTER CHART

1) **Sex:** M- Male, F- Female.

2) **Occupation:**

Agri	-	agriculturist
Ret	-	Retired
HW	-	Housewife

3) **Systemic diseases:**

HYP	-	Hypertension
DM	-	Diabetes mellitus
A	-	Anaemia
IHD	-	Ischaemic heart disease
H	-	Hypothyroidism
KD	-	Kidney disease
COPD	-	Chronic obstructive pulmonary disease
BPH	–	Benign prostatic hypertrophy

4) **Skin changes in elderly:**

XE	-	Xerosis,
WR	-	Wrinkling
IGH	-	Idiopathic guttate hypomelanosis
SL	-	Senile lentigenes
SC	-	Senile comedones
SK	-	Seborrheic keratosis
DPN	-	Dermatosis papulosa nigra
CA	-	Cherry angioma
AC	-	Acrochordons

5) **GP- Generalised pruritus**

6) **Pathological skin changes**

E - Eczematous conditions

CE - Chronic eczema

AE - Asteatotic eczema

SE - Stasis eczema

ABCD - Airborne contact dermatitis

IE - Infectious eczematous dermatitis

CD - Contact dermatitis

HE - Hand eczema

NE - Nummular eczema

Infections :-

FI - Fungal infections

BI - Bacterial infections

VI - Viral infections

PSQ - Papulosquamous disorders

PSO - psoriasis

LP - Lichen planus

B - Bullous disorders

BP - Bullous pemphigoid

PV - Pemphigus vulgaris

PSY - Psychocutaneous disorders

DP - Delusional parasitosis

LSC - Lichen simplex chronicus

PN - Prurigo nodularis.

T - Tumours:

SEB	-	Sebaceous hyperplasia
AF	-	Angiokeratoma of Fordyce
SY	-	Syringoma.

M - Miscellaneous conditions:

AM	-	Amyloidosis;
LU	-	Leg ulcers;
VO	-	Vitiligo;
CU	-	Chronic urticaria
AEM	-	Acrokeratoelastoidosis marginalis
GA	-	Granuloma annulare;
CM	-	Colloid milia;
PD	-	Perforating dermatoses;
PG	-	Pyogenic granuloma.

7) **N - Nails**

LL	-	Loss of luster
LR	-	Longitudinal ridging
SH	-	Subungual hyperkeratosis
OM	-	Onychomycosis
BL	-	Beau’s lines
TH	-	Thickening
P	-	Pitting

8) **H - Hair**

AGA	-	Androgenetic alopecia
DHL	-	Diffuse hair loss

ABBREVIATIONS

DNA	-	Deoxyribonucleic acid
ICAM-1	-	Intercellular adhesion molecule-1
UV	-	Ultraviolet
IL	-	Interleukins
VEGF	-	Vascular endothelial growth factor
NF κB	-	Nuclear factor kappa B
MMP	-	Matrix metalloproteinase
TNF – alfa	-	Tumour necrosis factor alfa
AP – 1	-	Activator protein – 1
RNA	-	Ribonucleic acid
TGF – β	-	Transforming growth factor – beta
LC	-	Langerhan's cell
HRT	-	Hormone replacement therapy
MRSA	-	Methicillin resistant staphylococcus aureus
HZ	-	Herpes zoster
HSV	-	Herpes simplex virus
KOH	-	Potassium hydroxide

SCC	-	Squamous cell carcinoma
HPV	-	Human papilloma virus
BCC	-	Basal cell carcinoma
5-FU	-	5- Fluorouracil
HIV	-	Human immunodeficiency virus
PUVA	-	Psoralen with ultraviolet A light
CO_2 LASER	-	Carbondioxide laser
Nd:YAG	-	Neodymium Yttrium Aluminium Garnet
IgG, IgA	-	Immunoglobulin G and A
LSEA	-	Lichen sclerosesus et atrophicus

Appendices

PROFORMA

A CLINICAL STUDY OF CUTANEOUS MANIFESTATION

IN GERIATRIC AGE GROUP

Name: **Date:**

Age:

IP No. /OP No:

Sex:

Occupation:

Address:

Hospital: Coimbatore Medical College Hospital, Coimbatore.

Socioeconomic status: High/Middle/Low

Marital status: Married / single

Chief complaints:

History of presenting illness:

Past history:

H/O similar complaints in past

H/O Diabetes Mellitus/Hypertension/Asthma/Epilepsy/Drug allergy

H/O Leprosy/ autoimmune disease / Psoriasis / Connective tissue disease / Rheumatoid Arthritis / Infections.

H/O any other skin disease in past

Drug history:

Personal history:

Diet, Appetite, Sleep, Bowel & Bladder, Habits

Family history:

General physical examination:

BP: mmHg PR: /min Weight: kg

pallor /edema/cyanosis /clubbing /icterus /lymphadenopathy

Systemic examination:

Cardiovascular System:

Respiratory System:

Per Abdomen:

Central Nervous System:

Mucocutneous examination:

1.Erythematous/Non erythematous

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2. Sites: Exposed/Covered part

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Abdomen/Thigh/Legs/Genitilia

3. Pattern: Scattered/ Linear/Grouped/ Zosteriform

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c) Infections/Infestation

d) Eczema/Contact Dermatitis

e) Pigmentary disorders

f) Papulosquamous disorders

g) Metabolic disorder:

h) Disorders of Keratinization

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o) Hair Changes

p) Mucosal lesions: Oral mucosa:

Genital mucosa:

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Complete haemogram with peripheral smear study, ESR, liver function tests, blood urea, random blood sugar, serum creatinine, lipid profile, serum electrolytes, thyroid profile, and stool for occult blood

Urine routine:

Dermatological procedures:

Skin scrapping and nail clipping for KOH mount to rule out fungal infection.

Tzanck smear:

Woods lamp:

Pus for culture and sensitivity:

Skin biopsy and direct immunofluorescence:

Diagnosis:

Treatment:

ANNEXURE – II

CONSENT FORM

Dr.S. Anitha Post Graduate student in the Department of Dermatology, Venereology and Leprosy, Coimbatore Medical College Hospital is conducting a study on "A CLINICAL STUDY OF GERIATRIC DERMATOSES". The study and test procedures were explained to me clearly. I here by give my consent to participate in this study and to give blood sample and to take biopsy if needed. The data obtained here in may be used for research and publication. I also allow the researcher to use my clinical photograph in this study for publication.

Place :

Date :

Signature of the patient

(Name:)

xggpj y;go t k;

bgah; :

ghypdk; :

Kfthp : taJ :

muR nfhit kUj;Jtf;fy;Y}hpa[y;njhy;neha;kUj;Jt Jiwapy; gl;l nkwgogg[gapYk;khz th;**kU. rh.mdpjh** mth;fs; "**nfhaKj;J}h; kUj;Jt fy;Y}hp kUj;Jtkidapy; Kjpnahh;fspd;njhy;neha; gwwpa** " Ma;tpy; nkwnfhs;Sk; bra;Kiw kwWk; midj;J tputu';fisak;nfl;Lf;bfhz;L vdJ renjf';fis bjspthpLj;jpf; bfhz;nld;vdgij bjhptpj;Jf;bfhs;fpnwd;

ehd; ,e;j Ma;tpy; KG rkkjj;Jld; Ra rpe;jidaplDk; fye;J bfhs;s rkkjpf;fpnwd;

,e;j Ma;tpy; vd;Dila midj;J tpgu';fs; ghJfhf;fggLtJld; ,jd; Kotpfs; Ma;tpjHpy; btspapLggLtjpy; Mlnrgid ,y;iy vdgij bjhptpj;Jf bfhs;fpnwd; vej neuj;jpy; mej Ma;tpypUe;J ehd; tpyfpf;bfhs;s vdf;F chpik czL vdgijak;mwpntd;

,lk;: ifbahggk;/ nuif

ehs;:

Master chart

MASTER CHART

SL. NO	Name	Demographics			Systemic Diseases			Physiological Skin Changes									Pathological Skin Changes										
		Age	Sex	OCC	HYP	DM	SD Others	XE	WR	IGH	SL	SC	SK	CA	DPN	AC	GP	E	Infections			PSQ	B	PSY	T	M	N	H
																			FI	BI	VI							
1	Namachivayam	60	M	Agri	-	+	-	+	+	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	LL	AGA
2	Ponnusamy	63	M	Agri	+	-	-	+	-	-	-	-	+	+	+	+	+	CD	-	-	+	-	-	DP	-	-	LL,LR	AGA
3	Saraswathy	65	F	Agri	+	-	A	-	+	+	-	-	+	-	+	-	-	-	-	-	-	-	BP	-	-	-	LL,TH	DHL
4	Palanisamy	78	M	Agri	-	+	IHD	+	+	+	-	-	+	+	-	-	+	IE	+	-	-	-	-	-	-	-	OM	AGA
5	Ashirbatham	67	M	Ret	+	+	KD	+	-	+	-	-	+	-	+	+	+	-	-	-	-	-	-	-	SEB	PD	LL	-
6	Subramani	72	M	Agri	-	-	-	+	+	+	-	-	-	+	+	-	+	CE	-	+	-	-	-	-	-	-	LR	-
7	Ramani	62	M	Agri	-	+	-	+	+	-	+	-	+	-	+	-	-	-	-	-	-	PSO	-	-	-	-	LL	-
8	Amaravathi	68	F	Agri	+	-	-	-	+	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	BCC	-	SH,TH,LL	-
9	Sudalaivadivu	75	M	HW	-	-	-	+	+	+	-	-	+	+	-	-	+	-		-	+	-	-	LSC	-	-	LL	DHL
10	Rangasamy	60	M	Agri	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	LU	LL	AGA
11	Krishnan	74	M	Ret	-	+	-	+	+	+	-	-	-	+	-	+	+	-	-	-	-	-	-	DP	-	-	LL	AGA
12	Balakrishnan	64	M	Agri	+	-	-	+	-	-	-	-	+	-	+	-	+	-	+	-	-	-	-	-	-	AM	LR,TH	AGA
13	Sampoornam	69	F	Ret	-	-	BA	+	+	-	-	-	+	+	-	-	+	AE	-	-	+	-	-	-	-	-	SH,TH	AGA
14	Vasantha	73	F	HW	+	-	-		+	+	+	-	-	-	+	-	+	-	-	-	-	-	-	LSC	-	-	LL	DHL
15	Kathiresan	65	M	Ret	-	+	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	PSO	-	-	-	-	LL	-
16	Ruckumani	66	F	Agri	-	-	-	+	-	-	-	+	-	-	+	-	+	-	-	-	-	-	-	-	-	VO	LR,TH	DHL
17	Lakshmi	79	F	HW	+	-	-	-	+	+	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	AM	LL,SH,TH	DHL
18	Thiruma	61	M	Agri	-	+	-	+	-	-	-	-	+	-	+	-	+	HE	-	-	+	-	-	-	-	-	LL	AGA
19	Gurusamy	72	M	Agri	-	+	-	-	+	+	+	-	-	+	-	+	-	-	-	-	-	-	BP	-	-	-	LR	AGA
20	Sankaran	60	M	Agri	-	-	BA	-	-	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	AF	-	LL	AGA
21	Anburaj	82	M	Agri	-	-	-	+	+	+	-	-	-	+	-	-	-	CE	+	-	-	-	-	-	-	-	LR,TH	AGA
22	Kaliammal	68	F	Agri	+	+	-	-	-	+	-	-	+	+	+	+	+	SD	-	+	-	-	-	-	-	-	LL	DHL
23	Fathima	84	F	HW	-	-	A	+	+	+	+	-	-	-	+	-	+	AE	-	-	-	-	-	-	-	CM	LL	-
24	Dasan	63	M	Ret	-	+	-	+	-	-	-	+	-	-	+	-	-	-	-	-	-	LP	-	-	-	-	LL	-
25	Gandhi	62	F	HW	+	+	-	+	-	-	-	-	+	+	-	+	+	NE	-	-	-	-	-	-	-	-	LR	DHL
26	Marudhamuthu	70	M	Agri	-	+	-	-	+	+	-	-	-	+	+	-	+	SE	-	+	-	-	-	-	-	-	LR	AGA
27	Pushparani	72	F	HW	-	-	-	+	+	-	+	-	+	-	+	-	-	-	-	-	-	PSO	-	-	-	-	LL,VR	-
28	Mary	64	F	HW	+	-	H	-	-	-	-	-	+	+	-	+	-	-	-	-	-	-	BP	-	-	-	LR	DHL
29	Subbammal	68	F	Agri	-	-	-	+	-	-	-	-	+	-	+	-	-	CE	-	-	-	-	-	-	-	-	LR	DHL
30	Vijayalakshmi	78	F	Agri	-	+	-	-	+	+	-	-	+	+	-	-	+	-	+	-	-	-	-	-	-	-	LL	-
31	Imam Roother	65	M	Ret	-	-	-	+	-	-	+	+	-	+	-	+	-	-	-	+	-	-	-	-	-	VO	LL	AGA
32	Chelladurai	75	M	Agri	+	-	IHD	-	+	+	-	-	-	+	-	+	+	-	-	-	+	-	-	PN	-	-	TH,LR	AGA
33	Gopal	63	M	Agri	-	-	-	+	-	-	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	GA	LL	AGA
34	Abdul Kaboor	66	M	Ret	-	+	-	-	-	-	-	+	+	-	+	-	+	-	-	-	-	-	-	-	-	AM	LR	AGA
35	Kannammal	86	F	Agri	-	-	-	+	+	+	-	-	+	-	+	-	+	ACD	-	-	-	-	-	-	-	-	LR	-
36	Pappu	60	F	HW	+	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	PV	-	-	-	LL	DHL
37	Sarasu	80	F	HW	-	-	-	+	+	+	-	-	-	+	-	+	+	CE	-	+	-	-	-	-	-	-	LL	-
38	Subbaiyan	75	M	Agri	-	+	BA	-	+	+	-	-	-	+	-	-	+	-	-	-	-	-	-	LSC	-	-	OM	AGA
39	Dharmalingam	65	M	Agri	-	-	-	+	-	-	-	+	+	-	+	-	+	-	-	-	-	-	-	-	-	CU	TH,LR	AGA
40	Nataraj	69	M	Agri	+	-	-	+	+	-	-	-	+	-	+	-	+	CE	-	+	-	-	-	-	-	-	LL	AGA

SL. NO	Name	Demographics			Systemic Diseases			Physiological Skin Changes									Pathological Skin Changes											
		Age	Sex	OCC	HYP	DM	SD Others	XE	WR	IGH	SL	SC	SK	CA	DPN	AC	GP	E	Infections			PSQ	B	PSY	T	M	N	H
																			FI	BI	VI							
41	Hanifa	73	F	Ret	-	-	IHD	-	+	+	+	-	-	+	-	-	-	ACD	-	-	+	-	-	-	-	-	LL	AGA
42	Mymoon beevi	64	F	HW	-	+	-	+	-	-	-	+	-	-	+	-	+	-	-	-	-	-	-	-	-	AM	LL	AGA
43	Arukkani	77	F	Agri	-	-	A	-	+	+	-	-	+	+	-	-	+	-	-	-	-	-	-	DP	-	-	LR	DHL
44	Rajammal	61	F	Agri	-	+	-	+	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	BCC	-	LR	DHL
45	Murugesan	67	M	Agri	+	+	KD	+	-	-	-	-	-	+	-	-	+	-	-	+	-	-	-	-	-	LU	LR	-
46	Ramalingam	86	M	Ret	-	-	-	+	+	+	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	-	SH	-
47	Sivaraj	91	M	Agri	-	+	-	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	DP	-	-	TH,SH	-
48	Rangan	63	M	Agri	+	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	PG	LL	AGA
49	Pappathi	70	F	HW	-	-	A	-	+	+	-	-	-	-	-	+	-	-	-	-	-	PSO	-	-	-	-	LL	DHL
50	Maran	74	M	Agri	-	+	-	+	+	+	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	LR	-
51	Raman	65	M	Agri	-	+	-	+	-	-	-	-	+	-	-	-	-	IE	-	-	-	-	-	-	-	LU	LL	AGA
52	Alfons	63	M	Ret	-	-	-	-	+	-	-	-	+	-	+	-	+	-	+	-	-	-	-	-	-	-	LL	AGA
53	Jayamani	67	F	Agri	+	-	-	+	-	+	-	-	+	-	+	-	+	AE	-	-	+	-	-	-	-	-	LL,LR	DHL
54	Deviammal	72	F	HW	-	-	A	-	+	+	-	-	-	+	+	+	+	-	-	+	-	-	-	-	-	-	LR	-
55	Kumarasamy	65	M	Agri	-	+	-	+	-		-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	LU	LR,TH	AGA
56	Vargish	68	M	Agri	+	-	-	-	-	+	-	-	+	-	+	+	-	-	-	-	-	-	BP	-	-	-	LL,LR	AGA
57	Saminathan	74	M	Ret	-	-	-	+	+	+	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	LL,TH	AGA
58	Durairajan	62	M	Agri	-	+	-	+	-	-	-	+	-	+	-	+	+	-	-	+	-	-	-	-	-	LU	LL,TH,SH	AGA
59	Jaganathan	69	M	Agri	+	-	-	+	+	-	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	LL	AGA
60	Subban	61	M	Agri	-	-	-	-	+	-	-	-	-	+	-	+	-	-	-	-	-	PSO	-	-	-	-	LL	AGA
61	Ruckumani	70	F	HW	-	-	-	-	+	+	-	-	+	-	+	-	+	-	-	-	-	-	-	DP	-	-	LL	DHL
62	Mariammal	79	F	Agri	-	+	BA	+	+	+	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	VO	LL	Plica polonica
63	Saroja	82	F	HW	-	-	-	+	+	+	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	AM	TH,LL	DHL
64	Samsudeen	64	F	Agri	+	+	-	-	-	-	+	-	-	+	-	+	-	CE	-	-	-	-	-	-	-	-	LL	-
65	Samikkannu	70	M	Ret	-	-	-	+	+	-	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	LL	-
66	Yusain	75	M	Ret	-	+	-	-	+	+	-	-	-	+	-	+	+	-	-	-	-	-	-	LSC	-	-	LR	AGA
67	Kuppusamy	61	M	Agri	-	-	-	-	-	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-	-	AEM	LL	AGA
68	Ramasamy	72	M	Agri	+	+	-	+	+	-	-	-	+	-	-	+	-	SE	-	-	-	-	-	-	-	-	LL	-
69	Muniammal	65	F	HW	-	-	A	+	+	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	LSA	LL	DHL
70	Mohan	67	M	Agri	-	+	BA	+	-	-	-	-	+	+	-	-	-	AE	+	-	-	-	-	-	-	-	OM,TH	AGA
71	Venkatachalam	73	M	Agri	-	-	-	+	-	+	-	-	+	-	+	-	-	SE	-	-	-	-	-	-	-	-	LR	AGA
72	Baby	63	F	Agri	-	-	BA	+	-	-	-	+	-	+	-	-	-	-	-	-	-	PSO	-	-	-	-	TH,LL	DHL
73	Chinnathayi	68	F	HW	-	+	-	-	-	-	+	-	+	-	-	-	+	-	-	-	-	LP	-	-	-	-	LL	-
74	Muthammal	80	F	HW	-	-	IHD	-	-	+	-	-	+	+	-	-	-	CE	+	-	-	-	-	-	-	-	LL	DHL
75	Malaichami	61	M	Agri	+	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	LSC	-	-	LR	AGA
76	Ganesan	70	M	Agri	-	-	-	+	-	+	-	-	-	+	-	-	+	-	-	-	-	-	-	DP	-	-	LR	AGA
77	Sundharamani	76	F	HW	-	+	-	+	+	+	-	-	-	-	-	-	-	SE	+	-	-	-	-	-	-	-	LR,TH	-
78	Petchiammal	62	F	HW	+	-	-	-	+	-	-	+	-	+	-	-	+	-	-	-	-	-	-	-	-	CU	LL	-
79	Rajamani	62	F	Agri	-	+	-	+	-	-	-	-	-	+	-	+	+	CE	-	+	-	-	-	-	-	-	OM,TH	DHL
80	Prakasam	68	M	Agri	-	-	COPD	+	+	+	-	-	+	-	+	-	+	-	-	-	-	+	-	-	BW	-	TH,LL	AGA

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		Age	Sex	OCC	HYP	DM	SD Others	XE	WR	IGH	SL	SC	SK	CA	DPN	AC	GP	E	Infections			PSQ	B	PSY	T	M	N	H
																			FI	BI	VI							
81	Basheer	73	M	Ret	-	+	A	+	+	+	-	-	+	+	-	+	+	-	-	-	-	LP	-	-	-	LU	LL	AGA
82	Janakiammal	64	F	Agri	+	-	-	+	+	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	VO	LL	AGA
83	Marimuthu	67	M	Agri	-	-	-	+	+	+	-	-	+	+	-	-	+	ACD	+	-	-	-	-	-	-	-	LL	AGA
84	Arumugam	74	M	Ret	-	+	-	+	+	+	-	-	+	-	+	-	+	IE	+	-	-	-	-	-	-	-	LR	AGA
85	Subbarayan	63	M	Agri	-	+	-	-	-	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	GA	TH,LL	AGA
86	Vellaigounder	69	M	Agri	+	-	-	-	+	+	-	-	+	-	+	-	+	ACD	+	-	-	-	-	-	-	-	LR,LL	-
87	Natchimuthu	61	M	Agri	-	-	-	+	-	-	-	+	-	+	-	+	+	-	-	-	-	-	-	-	-	AEM	LR,TH	AGA
88	Maniammai	67	F	HW	-	+	-	+	+	-	-	-	+	+	+	-	+	-	-	-	-	-	-	-	-	LSA	LL,LR	DHL
89	Theerthammal	60	F	Agri	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	PV	-	-	-	OM,LL	DHL
90	Dhanalakshmi	69	F	HW	+	-	-	+	+	+	-	+	+	-	+	-	+	ACD	+	-	-	-	-	-	-	-	LL	DHL
91	Kanniammal	65	F	Agri	-	+	-	+	+	-	-	-	+	-	+	-	+	-	+	-	-	-	-	DP	-	-	LR	DHL
92	Rajangam	70	M	Agri	-	-	BA	+	+	+	-	-	+	+	-	-	-	AE	-	-	+	-	-	-	-	AM	SH,TH	AGA
93	Musthafa	75	M	Ret	+	+	-	-	+	+	-	-	+	-	+	-	-	CD	+	-	-	-	-	-	-	-	OM,LL,LR	AGA
94	Perumal	61	M	Agri	-	+	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	PSO	-	-	-	CU	LL,TH	-
95	Konaimman	68	F	Ret	-	-	-	-	+	+	-	-	+	+	+	-	+	-	-	-	-	PSO	-	-	-	-	LR,LL	-
96	Suseela	63	F	Agri	-	-	-	+	-	-	-	-	-	+	-	+	+	CE	-	-	-	-	-	-	-	-	LR	DHL
97	Krishnamoorthi	66	M	Agri	+	-	-	+	+	+	-	-	+	-	+	-	+	-	-	+	-	-	-	LSC	-	-	LR	AGA
98	Anthony	64	M	Agri	-	-	-	+	-	-	+	-	-	+	+	-	-	-	-	-	-	-	-	-	SCC	-	LL	AGA
99	Rajendran	72	M	Agri	-	-	IHD	+	+	+	-	-	+	+	+	-	-	-	-	-	-	-	BP	-	-	LU	LL	AGA
100	Balan	65	M	Agri	+	-	-	+	-	-	-	-	-	+	-	+	+	SE	+	-	-	-	-	-	-	-	OM,LR	-
101	Sivaraman	68	M	Agri	-	+	BA	-	+	+	-	-	+	-	+	-	+	-	-	-	+	-	-	LSC	-	-	LL,TH	AGA
102	Muthaiyan	70	M	Agri	-	-	-	+	+	+	-	-	+	-	+	-	-	AE	+	-	-	-	-	-	-	-	LR,LL	AGA
103	Parasuraman	64	M	Agri	+	-	-	+	-	-	-	-	-	+	-	+	-	HE	+	-	-	-	-	-	-	-	SH,TH,LL	-
104	Annammal	66	F	HW	-	-	-	-	-	-	-	-	+	+	-	-	+	AE	-	+	-	-	-	-	-	-	LL	DHL
105	Guruvammal	63	F	Agri	-	+	-	+	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	LL	DHL
106	Alamelu	67	F	HW	+	-	-	-	+	+	-	-	+	+	-	+	+	NE	-	-	-	-	-	-	-	AM	LL	DHL
107	Valliammal	69	F	HW	-	-	-	+	+	+	-	-	-	+	-	+	+	-	-	-	-	-	-	DP	-	-	LR	-
108	Deivanai	67	F	HW	-	+	-	+	-	-	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	VO	LR	-
109	Veerammal	70	F	Agri	+	-	-	+	+	+	-	+	-	+	-	+	+	-	+	-	-	-	-	-	-	-	LR	-
110	Kandhasamy	72	M	Agri	-	+	IHD	-	+	+	-	-	+	-	+	-	+	-	+	-	-	-	-	-	-	-	LL,TH	AGA
111	Srinivasan	61	M	Agri	-	+	-	+	-	-	-	+	-	+	-	-	-	-	-	-	-	PSO	-	-	-	CM	LR,TH	AGA
112	Abdullah	68	M	Ret	+	-	KD	+	+	-	-	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-	LL,TH	AGA
113	Selvam	60	M	Agri	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	PV	-	-	-	BL,LL	AGA
114	Ramamoorthy	66	M	Agri	-	+	-	-	+	-	-	-	-	+	-	+	+	-	-	-	+	-	-	-	-	AEM	LL	AGA
115	Veerasamy	62	M	Agri	-	-	-	+	-	-	-	-	+	-	+	-	-	-	-	-	-	LP	-	-	-	-	LL	AGA
116	Karuppaiyah	67	M	Ret	-	-	-	+	-	-	-	+	-	+	-	-	+	CD	+	-	-	-	-	-	-	-	LL	AGA
117	Asha Begum	75	F	HW	+	+	A	+	+	+	-	-	+	-	+	-	+	-	-	-	-	-	-	DP	-	-	LL,TH	-
118	Chinniammal	64	F	Agri	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	BP	-	-	-	LR	DHL
119	Karunakaran	70	M	Ret	-	+	-	+	+	+	-	-	+	-	+	-	+	CE	+	-	-	-	-	-	-	-	LR	AGA
120	Adhimoolam	74	M	Ret	-	-	-	-	+	+	-	-	+	-	+	-	+	AE	+	-	-	-	-	-	-	-	LL	AGA
121	Kamatchi	65	F	Agri	-	-	-	+	-	-	-	+	-	-	+	-	-	-	-	-	-	PSO	-	-	-	AM	LL	DHL

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		Age	Sex	OCC	HYP	DM	SD Others	XE	WR	IGH	SL	SC	SK	CA	DPN	AC	GP	E	Infections			PSQ	B	PSY	T	M	N	H
																			FI	BI	VI							
122	Mani	69	F	Agri	-	+	-	-	+	+	-	-	-	+	-	-	+	-	+	-	-	-	-	-	-	-	LR	-
123	Narayanan	72	M	Ret	-	-	IHD	-	+	+	-	-	+	-	+	-	-	-	-	-	-	-	BP	-	-	-	BL,LL	-
124	Chidhambaram	62	M	Agri	-	-	-	+	-	-	-	-	+	+	-	-	+	-	-	-	-	-	-	-	BCC	-	LR	AGA
125	Perumathal	66	F	Agri	-	+	-	+	+	+	-	+	-	+	-	-	-	IE	+	-	-	-	-	-	-	-	LR	DHL
126	Muniappan	64	M	Agri	-	-	-	-	-	-	-	-	+	+	-	-	+	-	+	-	-	-	-	-	-	-	OM,TH	AGA
127	Balasubramani	73	M	Agri	-	+	BA	-	+	-	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-	LL,SH,TH	AGA
128	Ammakannu	60	M	Agri	+	-	-	+	-	-	-	-	+	-	+	-	-	-	-	-	-	LP	-	-	SY	-	LL,TH	-
129	Varadharajan	75	M	Ret	-	-	-	+	+	+	-	-	+	+	-	+	+	-	-	-	-	-	-	-	-	LSC	LL,SH,TH	AGA
130	Madhammal	68	F	Agri	-	+	A	+	-	-	-	-	-	+	-	-	+	-	+	-	-	-	-	-	-	-	LR	DHL
131	Neelambigai	72	F	Ret	+	-	-	-	+	+	-	-	+	+	-	+	+	-	-	-	-	-	-	-	-	AM	LL	DHL
132	Elumalai	78	M	Agri	-	-	COPD	+	+	+	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	-	SH,TH	AGA
133	Rahman	62	M	Ret	-	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	PSO	-	-	-	-	LL	AGA
134	Gurunathan	74	M	Ret	-	-	-	+	+	+	-	-	-	-	-	+	+	-	-	-	-	-	-	DP	-	LU	LL	AGA
135	Vedhamoorthy	66	M	Ret	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	PSO	-	-	-	-	LL	AGA
136	Chinnasamy	73	M	Agri	+	-	-	+	+	+	-	-	-	+	-	+	+	-	+	-	-	-	-	-	-	-	LR	AGA
137	Ellammal	76	F	HW	+	-	-	+	+	+	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	LL,LR	DHL
138	Sadhik Basha	65	M	Ret	-	+	-	+	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	CU	SH,TH,LL	-
139	Kamsala	79	M	Ret	+	-	-	-	+	+	-	-	+	+	+	-	+	-	-	-	-	-	-	-	-	AM	LL	DHL
140	Samuvel	70	M	Ret	-	-	-	+	+	+	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	LR	-
141	Isabellah	75	M	Ret	-	+	-	+	+	+	-	-	-	+	+	-	+	ACD	-	-	-	-	-	-	-	-	LL	-
142	Aandal	80	F	Ret	+	-	-	-	+	+	-	-	+	-	-	+	+	-	-	-	-	-	-	LSC	-	-	OM,LL	DHL
143	Muniandi	74	M	Agri	-	-	-	+	+	+	-	-	-	+	+	-	-	SE	-	-	-	-	-	-	-	-	LL	AGA
144	Veeramani	64	M	Agri	-	+	-	-	-	-	-	+	+	+	-	-	+	-	+	-	-	-	-	-	-	-	LL	AGA
145	Velankanni	67	F	Agri	-	+	-	+	-	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	GA	LR	DHL
146	Pandurangan	77	M	Agri	+		-	+	+	-	-	+	-	-	+	-	-	CE	-	-	-	-	-	-	-	-	LL	-
147	Chokkalingam	72	M	Ret	-	-	BPH	-	-	-	-	+	+	+	-	+	+	AE	-	-	+	-	-	-	-	-	LL	AGA
148	Manickam	69	M	Agri	-	+	-	+	-	-	-	+	-	+	+	-	-	IE	-	-	-	-	-	-	-	-	LL	-
149	Kannaiyan	80	M	Agri	+	-	-	+	+	+	-	-	+	-	+	-	-	CE	-	-	-	-	-	-	-	-	LL	AGA
150	Marudhamalai	63	M	Agri	-	-	-	+	-	-	-	+	+	-	-	-	+	AE	-	-	-	-	-	-	-	-	LL	-

KEY TO MASTER CHART

1) **Sex:** M- Male, F- Female.

2) **Occupation:**

Agri	-	agriculturist
Ret	-	Retired
HW	-	Housewife

3) **Systemic diseases:**

HYP	-	Hypertension
DM	-	Diabetes mellitus
A	-	Anaemia
IHD	-	Ischaemic heart disease
H	-	Hypothyroidism
KD	-	Kidney disease
COPD	-	Chronic obstructive pulmonary disease
BPH	–	Benign prostatic hypertrophy

4) **Skin changes in elderly:**

XE	-	Xerosis,
WR	-	Wrinkling
IGH	-	Idiopathic guttate hypomelanosis
SL	-	Senile lentigenes
SC	-	Senile comedones
SK	-	Seborrheic keratosis
DPN	-	Dermatosis papulosa nigra
CA	-	Cherry angioma
AC	-	Acrochordons

5) **GP- Generalised pruritus**

6) **Pathological skin changes**

E	-	Eczematous conditions
CE	-	Chronic eczema
AE	-	Asteatotic eczema
SE	-	Stasis eczema
ACD	-	Airborne contact dermatitis
IE	-	Infectious eczematous dermatitis
CD	-	Contact dermatitis
HE	-	Hand eczema
NE	-	Nummular eczema

7) **Infections :-**

FI	-	Fungal infections
BI	-	Bacterial infections
VI	-	Viral infections

8) PSQ - Papulosquamous disorders

PSO	-	psoriasis
LP	-	Lichen planus

9) B - Bullous disorders

BP	-	Bullous pemphigoid
PV	-	Pemphigus vulgaris

10) PSY - Psychocutaneous disorders

DP	-	Delusional parasitosis
LSC	-	Lichen simplex chronicus
PN	-	Prurigo nodularis.

11) T - Tumours:

SEB	-	Sebaceous hyperplasia
AF	-	Angiokeratoma of Fordyce
SY	-	Syringoma.

12) M - Miscellaneous conditions:

AM	-	Amyloidosis;
LU	-	Leg ulcers;
VO	-	Vitiligo;
CU	-	Chronic urticaria
AEM	-	Acrokeratoelastoidosis marginalis
GA	-	Granuloma annulare;
CM	-	Colloid milia;
PD	-	Perforating dermatoses;
PG	-	Pyogenic granuloma.

13) N - Nails

LL	-	Loss of luster
LR	-	Longitudinal ridging
SH	-	Subungual hyperkeratosis
OM	-	Onychomycosis
BL	-	Beau's lines
TH	-	Thickening
P	-	Pitting

14) H - Hair

AGA	-	Androgenetic alopecia
DHL	-	Diffuse hair loss