STUDY OF CUTANEOUS DISORDERS IN GERIATRIC AGE GROUP

by

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M. D

in

Dermatology, Venereology and Leprosy

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LIST OF ABBREVATIONS

BI	-	Bacterial infections
CA	-	Cherry angiomas
CB	-	Candidal balanitis
CD	-	Contact dermatitis
DPN	-	Dermatosis papulosa nigra
ED	-	Eczematous disorders
FF	-	Fissure feet
FI	-	Fungal infections
IGH	-	Idiopathic guttate hypomelanosis
IN	-	Intertrigo
L	-	Lentigines
LSC	-	Lichen simplex chronicus
N	-	Neoplasms
NE	-	Nummular eczema
P	-	Pruritus
PD	-	Pigmentary disorders
PDE	-	Photodermatitis
PI	-	Parasitic infestations
PO	-	Pompholyx
PSO	-	Psoriasis
PV	-	Pityriasis versicolor

SC	-	Senile comedones
SD	-	Stasis dermatitis
SDE	-	Sebborheic dermatitis
SK	-	Sebborheic keratosis
SE	-	Solar elastosis
SP	-	Senile purpura
S	-	Skin tags
TC	-	Tinea capitis
TCO	-	Tinea corporis
TCR	-	Tinea cruris
TF	-	Tinea faciei
TM	-	Tinea manuum
VBD	-	Vesiculobullous disorders
VI	-	Viral infections
X	-	Xerosis

ABSTRACT

Background:

Increase in life expectancy due to better and improved medical facilities leads to increase in number of individuals among geriatric age group who are exposed to various environmental factors for a longer duration of time. This leads to increase in various dermatological and associated systemic diseases in geriatric age group. The studies conducted on geriatric dermatoses are sparse.

Objective:

To study the various patterns of cutaneous disorders in geriatric age group.

Methods:

A total of 550 consecutive geriatric patients were examined in a hospital based, cross-sectional study between the period of October 2008 to May 2010.

Results:

Of 550 patients, 302 (55%) were males and 248 (45%) were females. There were 456 (82.9%) patients belonging to age group 65-74 years, 84 (15.2%) belonging to age group 75-85 years and only 10 (1.8%) were above 85 years of age. Among photoaging skin changes seborrhoeic keratosis 207 (37.6%) and senile comedones 207(37.6%) were noted more in males. Cutaneous diseases like fungal, bacterial and viral infections, parasitic infestations, eczema, psoriasis, pigmentary disorders are noted more in males. Among different age groups, fissure feet 7 (70%), parasitic infestations 2 (20%), malignancies 1 (10%) were noted more above 85 years of age. Eczematous disorders 61 (13.3%) were more common in the age group of 65- 74 years. Among infections, fungal

infections 47 (8.5%) were commonly noticed. Among eczematous disorders, photodermatitis 26 (36.6%) was the commonest disorder noticed. Psoriasis was present in 12 (2%) of patients. Vesiculobullous disorders were noticed in 3 (0.5%) of patients. Cutaneous drug reactions were noted in 2 (0.3%) of patients. Basal cell carcinoma was the only cutaneous malignancy seen in 2 (0.3%) patients. Associated systemic ailments were observed in 107 (19.4%) patients. Hypertension 44 (8%) was the commonest followed by diabetes 41 (7.4%).

Conclusion:

The present study provides data on various cutaneous disorders in elderly based on gender, different age groups and associated comorbid conditions. Health promotion and education of these disorders can reduce the morbidity in elderly individuals. The database demonstrates the need for further studies addressing clinical, etiological and therapeutic issues of major dermatoses affecting the quality of life of elderly.

Key Words : Cutaneous disorders, geriatric age group

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INTRODUCTION

The geriatric population consists of persons over 65 years of age. The skin of elderly differs from that of adult in several ways. All skin components and regions alter in the process of intrinsic aging. The intrinsic changes are governed by genes. Among the extrinsic factors more than 90% of age associated problems on exposed skin are caused by ultraviolet (UV) radiation i.e photoaging.

The overall functions of skin declines with age. Decline is noted in cell replacement, sensory perception, thermal regulation and chemical clearance. Decrease in sweat, sebum and vitamin D production also occurs. Immune response is compromised. Incidence of neoplasm increases and there is greater susceptibility to skin infections. Wounds heal more slowly due to combination of decreased immune and inflammatory response, collagen degradation and decreased synthesis.

Both the epidermis and the dermis become thinner on the sites unexposed from sunlight as the age advances. The cell turnover is halved between the third and seventh decades of life. The epidermis thins from 4-5 cells thick at the age of 20 to approximately 3 cells thick at the age of 80. The individual keratinocytes also shrink with age. Sebum and sweat secretion rates, pigment production and immunological defenses are all decreased in the elderly.

These age-associated functional decrements are widely and reasonably presumed to contribute to clinical skin disease, as well as contribute to systemic health problems of the elderly. It is difficult to draw a line between the physiological and the true pathological changes of old age. The most characteristic signs of physiological changes of aging skin are- atrophy, laxity, wrinkling, sagging, dryness, multiplicity of pigmented and other blemishes, sparse grey hair and longitudinal

ridges of the nails. Aging of skin is a highly complex process and intrinsic aging must be separated from the effects of environmental factors.

Commonest problem related to old age is localized or generalized pruritus. Any clinical type of eczema, bullous disorders, leg ulcers, herpes zoster, benign and malignant skin tumors, ectoparasitic infestations such as scabies etc, can occur in elderly individuals.

Diseases of the aged are becoming increasingly important, as there is gradual increase in the life expectancy in the last few decades due to better and improved medical facilities. Increase in life expectancy leads to increase in number of individuals among geriatric age group who are exposed to various environmental factors for a longer duration of time. This leads to increase in various dermatological and associated systemic diseases in geriatric age group. Hence it is important to know the patterns of dermatoses prevalent among geriatric population. The identification and treatment of these conditions is important for the practicing clinician. However, studies on geriatric dermatoses, conducted in India are sparse.

Hence, this study has been undertaken to know the incidence of different cutaneous lesions among elderly in India.

OBJECTIVE OF STUDY

To study the various patterns of cutaneous disorders in geriatric age group.

REVIEW OF LITERATURE

Globally, as of now, 10% of the world's population is elderly and it is expected to increase to 21% in 2051. It is likely to increase from the current 600 million to 1.97 billion in 2051. India is the second largest country in the world, with 76 million elderly persons above 60 years of age.[1] The increasing number and proportion of elderly will have a direct impact on the demand for health services. We have several subspecialties within the field of dermatology like pediatric dermatology, dermatosurgery and cosmetic dermatology to name a few. But rarely does anyone mention geriatric dermatology. May be it just doesn't have the glitz and glamour of some of these other areas, but definitely it deserves more attention, and soon. The incidence of skin complaints in this group tends to be higher than in any other age group. The incidence of common skin findings in the elderly are listed in **Table 1.**

Table 1:
Epidemiology:
Incidence of common skin findings in the elderly[2]

Diseases	Male %	Female %	Total %
Cherry angiomas	31.5	18	49.5
Seborrheic keratosis	24	13.5	37.5
Naevus cell naevi	22	10.5	32.5
Fissured soles	20	10	30
IGH	17	7.5	24.5
Acrochordons	11	9	24.5
Vitiligo	7.5	11.5	19
Fungal infections	9.5	9	18.5
LSC	8	4	12
Lentigines	6.5	5.5	12
Comedones	9	2.5	11.5
Psoriasis	6.5	4	10.5
Senile purpura	4	5	9
Callosities	5	4	9
Contact dermatitis	3.5	4	7.5
Xerosis	4.5	2.5	7
Telangiectasia	2.5	3.5	6

NOTE: IGH-Idiopathic guttate hypomelanosis. LSC - Lichen simplex chronicus.

An epidemiological study conducted in Mumbai, India has shown that, pruritus was the commonest complaint observed. Certain conditions like cherry angiomas, seborrhoeic keratosis, fissured soles, idiopathic guttate hypomelanosis etc. have higher incidence in males than females.[2]

The five most frequently encountered diseases in elderly patients observed in study conducted in India are eczematous dermatitis, fungal infections, pruritus, bacterial and viral infections. The most common disorders in males were fungal, bacterial and viral infections, disorders of feet, cutaneous ulcers and vesiculo-bullous diseases whereas in females immune-rheumatologic diseases and disorders of mucous membrane are commonly noticed in a study conducted in Turkey. Infestations were common in spring and summer, fungal infections were common in summer, pruritus in autumn was more common.[3]

AGING OF THE SKIN:

The most immediate, and perhaps most telling, evidence of an individual's age is the appearance of skin, hair and nails.[4] Aging is the progressive, intrinsic, time-dependent deterioration of an organism's structural or functional integrity. Skin aging is a continuous process that affects skin function and appearance. However, not everybody ages at same speed. It is generally agreed that certain individuals look "old for their age" or "young for their age". Intrinsic, environmental, and lifestyle factors contribute to the pace of skin aging. Chronic sun exposure has been identified as one of the most important environmental injuries leading to acceleration and aggravation of skin aging.[5]

Types of aging:

1. Intrinsic /chronological or endogenous aging:

Intrinsic aging is defined by the clinical, histological and physiological decrements that occur in sun-protected skin of older individuals, affecting rate of epidermal turnover, clearance of chemical substances from the dermis, dermal thickness and cellularity, thermoregulation, rate of re-epthelialization after wound healing, mechanical protection, immune responsiveness, sensory perception, sweat and sebum production.[6]

Intrinsic aging refers to the skin changes that result as a consequence of longevity. It exists in its purest form in non-sun exposed (photoprotected) body sites. Clinically, intrinsically aged skin is atrophic, which may result in prominence of vasculature, transparent quality and loss of elasticity.[6] Endogenous skin aging can be viewed on the non-UV exposed skin of the body and can be considered as model of the aging process taking place in internal organs.

2. Extrinsic aging/exogenous/photoaging/dermatoheliosis:

Photoaging-the most recognized form of extrinsic aging of the skin is caused by effects of frequent and cumulatively prolonged sun exposure, specifically UV radiation, superimposed on intrinsic aging. In contrast to intrinsically aged skin, photo aged skin is characterized not only by an exaggeration of the changes above but also by the presence of qualitatively different changes induced by sun exposure. Photo damage is the specific damage produced in tissue by single or repeated exposure to ultraviolet light. Photo aging is believed to account for the vast majority of the skin's age-related cosmetic as well as clinical problems.[6]

Photodamage, refers to the physical and morphologic alterations secondary to solar UV exposure and is the main component of photoaging. Typically, the skin

becomes coarse, pebbly and rough and shows hyperpigmentation, hypopigmentation and telangiectasias, as well as fine and coarse wrinkles.[7] Environmental factors may affect the structural and functional integrity of the skin as people age. These changes may add to or exacerbate the alterations related to intrinsic aging. Affects more individuals with type I and type II. Both intrinsic (chronological) and extrinsic (photoaging) contribute to the cutaneous aging.

Factors that affect skin aging include: [8,9,10,11,12]

- Smoking
- Air pollution
- Low relative humidity.
- Frequent hot bathing and vigorous toweling.
- Low ambient temperature
- Ionizing and non-ionizing irradiation
- Natural deleterious gases by depletion of ozone layer
- Invasion by pathogenic bacteria and viruses
- Mechanical stress.
- Systemic illness.

Up to 40% of changes that contribute to the aged appearance are the result of non genetic factors. Aging skin undergoes progressive degenerative change. Structural and physiologic changes that occur as a natural consequence of intrinsic aging combined with the effects of a lifetime of ongoing cumulative extrinsic damage and environment insult can produce a marked susceptibility to dermatologic disorders in the elderly. Topical tretinoin treats photoaging after it has occurred and also retards or prevents photoaging.[13]

THEORIES OF AGING:[14]

THE NEUROENDOCRINE THEORY: This theory elaborates on wear and tear by focusing on the neuroendocrine system. As age advances the hypothalamus loses its precision regulatory ability and the receptors which uptake individual hormones become less sensitive to them. Accordingly, the secretion of many hormones decline and their effectiveness is also reduced due to the down-grading receptors.

The free radical theory or oxidative stress theory: It postulates that free radicals, which are normally produced by intracellular metabolic pathways, progressively and cumulatively contribute to the demise of the cell. Free radicals can induce cross-linking of macromolecules and thus impeding nutrient diffusion, can inactivate or alter enzymes, fragment DNA, peroxidize lipids and may contribute to carcinogenesis.

THE MEMBRANE THEORY OF AGING**:** According to this theory, it is the age related changes of the cells ability to transfer chemicals, heat and electrical processes that impair it. As age advances the cell membrane becomes less in lipids. This impedes its efficiency to conduct normal function. There is deposition of lipofuscin in the skin, brain, heart and lungs.

THE HAYFLICK LIMIT THEORY/CELLULAR SENESCENCE THEORY: According to this theory, cells possess a “biological clock” ,which signals the end of heir replicative life span, and as a consequence, they can’t be stimulated to enter the S1 phase off cell cycle by physiological mitogens, arresting at the G1 phase.

Structural changes of elderly skin:

The features of chronological and photoaged skin are listed in **Table 2** and **Table 3**

Epidermis: In many areas of skin, the epidermis becomes thinner with age. hotoaged epidermis is initially thickened but eventually atrophies. Loss of rete ridges and consequent flattening of dermal-epidermal junction is a hallmark of intrinsically aged skin. Loss of vertical polarity in epidermal cells and irregularity of the epidermal cell alignment are also common features in chronically sun-damaged skin. Melanocytes are decreased in number in intrinsically aged epidermis. In contrast, in chronically sun exposed, photoaged skin there is an increase in number of melanocytes.[15]

Dermis: The solubility and the turnover rate of collagen decreases with aging. The changes in collagen quantity and structure are exacerbated in photodamaged skin. Collagen formation in the papillary dermis diminishes by 56% in actinically damaged skin when compared with skin subjected only to chronological aging. Individual elastic fibers in chronologically aged skin can show diminished numbers and diameters, and they can exhibit fiber fraying and increased tortuosity. [16] Degradation and remodeling of collagen take place in the papillary dermis accompanied by deposition of other matrix components, predominantly abnormal elastic fibers.[17] The typical fine, netlike organization of elastic fibers in the young papillary dermis disappears with age, and this network is replaced with thin, disorganized sheets of elastic fibers in the dermis of aged skin. Chronic photodamage results in a gradual decrease in the number and size of dermal vessels over several decades of sun exposure, most likely due to degenerative changes of the dermal extracellular matrix.[18] A marked loss in dermal nutritional vessel density and surface area for exchange is a feature of both chronologic aging and photoaging.[19] Facial wrinkles, accentuation of forehead lines and nasolabial folds, drooping of eyelids and fan

shaped radiating lines around the mouth accompany loss of elasticity, thickening and lower water content of the skin.

Eccrine glands are reduced in number and function in aged skin. Sebaceous glands become hyperplastic and larger. Sebaceous gland hyperplasia presents clinically as giant comedones.[20] The pathophysiological process of photoaging and skin aging derive largely from aberrant regulation of a multitude of finely tuned molecular mechanisms, which have evolved to maintain the structural integrity of skin connective tissue.[21] Graying of scalp and beard hair is due to reduction (gray hair) or loss (white hair) of melanin from hair follicles and is an unambiguous manifestation of advancing years. Nails grow more slowly in the elderly and are characterized by brittleness and longitudinal beaded ridging.

Features of photoaged skin include dryness, freckling, IGH, wrinkling, inelasticity, elastosis, sebaceous hyperplasia, purpura, stellate pseudo scars, premalignant and malignant lesions etc.[22]

There are 4 types of photoaging according to the Glogau classification:[20]

Type 1 represents early photoaging with mild pigmentary changes and minimal wrinkles. It occurs between the age 20-30 years.

Type 2 represents early to moderate photoaging with “wrinkles at rest”, early senile lentigines and palpable keratosis. It occurs between 30-40years of age.

Type 3 represents advanced photoaging with visible keratoses, dyschromias, telangiectasias and “wrinkles at rest.” This occurs at the ages of 50 years or later.

Type 4 represents severe photoaging, wrinkles throughout with no normal skin, yellow grey color of the skin and skin malignancies. This type occurs in the 6^{th} or 7^{th} decade.

Table 2: Features of chronological and photoaged skin

FEATURE	INTRINSIC AGING	EXTRINSIC AGING
Clinical appearance	Smooth, unblemished Loss of elasticity	Nodular, leathery, blotchy coarse wrinkles
Skin surface marking	Maintenance of normal geometric patterns	Markedly altered and often effaced
Epidermis		
Thickness	Thinner than normal	Acanthotic in early stages,atrophy in end stages
Proliferative rate	Lower than normal	Higher than normal
Basal keratinocytes	Modest cellular irregularity	Marked heterogeneity, numerous dyskeratoses
Keratinization	Unchanged	Unchanged
Stratum corneum	Normal thickness: 'basket-weave' pattern.	Heterogeneity: 'basket-weave' and compact patterns.
Dermo-epidermal junction	Loss of rete pegs,flat:modest reduplication of lamina densa	Loss of rete pegs,flat:extensive reduplication of lamina densa.
Dermis		
Grenz zone	Absent	Prominent
Elastin	Elastogenesis,followed by elastolysis-'moth-eaten'fibres	Marked elastogenesis followed by massive degeneration-dense accumulation in fibres.
Lysozyme	Modest deposition	Increased deposition
Collagen	Modest change in bundle size and organization	Moderate change in bundle size
Microvasculature	Normal architecture	Abnormal deposition of basement membrane like material
Inflammatory cells	No evidence of inflammation	Perivenular,histiocytic-lymphocytic infiltrate

Table 3: Clinical findings in skin due to aging and to habitual exposure to sun [23]

STRUCTURE	ELDERLY SKIN	CHRONICALLY SUN-DAMAGED SKIN
Epidermis	Thin	Keratosis-solar(actinic),cheilitis,disseminated superficial actinic porokeratosis
Dermis	Thin,inelastic,wrinkled	Yellowish-brown dotted or plaque like thickening(solar elastosis)
Hypoderm	Fold formation	None
Blood vessels	Cherry angiomas, venous lakes	Diffuse telangiectasia, erythema, brown pigmentation on side of neck, senile purpura **(Fig. 1)**
Melanocytes	Solar lentigo	Mottled irregular areas of hypopigmentation and hyperpigmentation. Solar keratosis.
Hair	Gray, alopecia	None
Sebaceous glands	Sebaceous hyperplasia	Solar comedones, nodular cutaneous elastoides
Eccrine glands	Dry skin	None
Nails	Onychoschizia,onychorrhexis, onychogryphosis	None

Functions of human skin which decline with age are listed in **Table 4.**

Table 4: Functions of human skin that decline with age [24]

• Barrier function • Cell replacement • Chemical clearance • DNA repair • Epidermal hydration • Immune responsiveness • Mechanical protection	• Sebum production • Sensory perception • Sweat production • Thermoregulation • Vitamin D production • Wound healing

The commonest cutaneous disorders seen in the eldery are listed in **Table 5**

Table 5: Cutaneous disorders in the elderly:

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Xerosis
Solar elastosis
Pruritus
Eczematous disorders
Cutaneous infections
Infestations
Papulosquamous disorders
Blistering disorders
Pigmentary changes of photoaging
Benign skin tumors
Premalignant and malignant tumors

XEROSIS: Xerosis is characterized by pruritic, dry, cracked and fissured skin with scaling.[25] Dryness is a frequent finding in both women and men over 65years of age. It is a normal finding in aging skin. Xerosis or dryness of the skin, affects at least 75% of this age group, thus making it the most common skin disorder in the elderly.[26] Dry skin occurs predominantly on the dorsal and lateral surfaces of the extremities, is

dependent on environmental conditions, and worsens during the dry winter months.[27] As the skin ages, it becomes drier and tends to become itchier.[25] The incidence and the severity increases with age. Dry skin results from abnormalities in the stratum corneum that occurs intrinsically with aging. Decreased moisture in the stratum corneum is secondary to an increase in stratum corneum transepidermal water loss (TEWL). The increased TEWL is secondary to a defect in the permeability barrier allowing excessive water to be lost to the atmosphere versus staying in the stratum corneum. The permeability barrier is affected by the reduction in stratum corneum lipid biosynthesis. The stratum corneum lipids are ceramides, triglycerides and fatty acids. These lipids are integral to the epidermis and function to prevent TEWL. Deficiency in any one of these may result in dry skin. The stratum corneum abnormality of decreased epidermal filaggrin leads to decreased natural moisturizing factor. It has strong humectant properties and maintains the hydration of the outermost layers of the stratum corneum.[26] This tendency is heightened by low relative humidity, frequent hot bathing and vigorous toweling, low ambient temperature, systemic illness.[25] The hallmarks of xerosis are aggregated desquamation of corneocytes appearing as fine white scales and a loss of elasticity, also describes as decreased mechanical flexibility of the stratum corneum.[27] Clinically with xerosis the outer layers of the skin become irritated, inflamed and itchy. The dull gray-white color of the skin is secondary to the lost cohesiveness of the stratum corneum and to roughness, which lead to the inability to refract light.[26] There is decreased sebaceous and sweat gland activity, this reduced activity predisposes the aged skin to moisture depletion.[25] A decreased aminoacid content, probably related to water binding in the stratum corneum, has been found in dry and aged skin. [27] There is decrease in epidermal proliferation and epidermal expression of basal and differentiation-related

keratins. Additionally, pre-existing disease states, therapies and medications make the aging individual more susceptible to xerosis. Some of these conditions include radiation, end-stage renal disease, nutritional deficiency, thyroid disease and neurological disorders with decreased sweating, anti-androgen medications, diuretic therapy, HIV and malignancies. Deficits in both skin hydration and lipid content play a key role in xerosis. Consequently, the skin's inability to retain moisture and provide an effective barrier directly impacts the development of xerosis in aging skin. Once the stage is set for xerosis development, the scenarios of flaking, fissuring, inflammation, dermatitis and infection develop. The xerotic vicious cycle needs to be broken to disable the process and prevent complications.[25]

In some patients surface texture of the skin assumes a cracked appearance resembling crazy paving. These cracks or fissures are present from epidermal water loss. This is known as eczema craquele or asteatotic eczema. Xerosis is characterized by pruritic, dry, cracked and fissured skin with scaling. If the skin splits and cracks deeply enough to disrupt dermal capillaries, bleeding fissures may occur. Pruritus occur leading to secondary lesions. Scratching and rubbing activities produce excoriations, an inflammatory response, lichen simplex chronicus. Subsequently, environmental allergens and pathogens can easily penetrate the skin, increasing the risk of allergic and irritant contact dermatitis, as well as infection.

SOLAR ELASTOSIS: Solar or actinic elastosis refers to the histologic alteration observed by light microscopy in the dermis of persons with long-term exposure to the sun. It is a degenerative change in the dermis caused by prolonged exposure to solar radiation. It is characterized clinically by yellowish discoloration and histologically by degeneration of elastic fibers. UVB wavelengths are most likely to cause solar elastosis. UVR induces the elastin gene transcriptional activity by a four fold increase

in elastin promoter activity and decreases the fibrillin 1 expression resulting in heavy deposition of elastic fibers, which are dystrophic and truncated. Increase of a lysine derived crosslink compound, desmosine, has been demonstrated in photoaged skin. On these altered elastic fibers, exogenous substances may deposit such as lysozyme, which correlates with basophilic degeneration of the fibers. This abnormal elastic tissue replaces the normal matrix composed mainly of collagen and is almost always separated by the Grenz zone. Solar elastosis is generally manifested as wrinkled, inelastic skin with a thickened leathery appearance and has an underlying yellowish hue owing to degenerative changes of the dermal elastic and collagen fibers admixed with overlying epidermal melanocytic dyspigmentation. These typical clinical findings have been referred to as Milian's citrine skin. Other signs of photoaging including atrophy, wrinkles, easy bruising, telangiectasias, lentigo and seborrhoeic keratosis often accompany this condition. This range of changes due to chronic sun damage has been called dermatoheliosis. Severity is dependent on the duration and intensity of sun exposure. Patients with fair skin are more susceptible, where as it is almost never seen in persons of color. Skin lesions of solar elastosis are most common on sun exposed sites such as face and forearms and form yellow dermal papules and plaques. Although usually photoaging manifests as Milian's citrine skin, the spectrum of visible changes occurring from solar elastosis may vary, depending on the anatomic site.

Variants of solar elastosis are listed in **Table 6.**

Table 6: Solar elastotic variants [28]

Milian's citrine skin
Striated beaded lines
Favre-Racouchot syndrome
Actinic comedonal plaque
Cutis rhomboidalis nuchae
Elastotic nodules of ear
Diffuse elastoma of Duberuilh
Actinic granuloma
Solar elastotic bands of forearms
Degenerative collagenous plaques of hands
Keratoelastoidosis marginalis
Digital calcific papular elastosis
Adult-onset colloid milium
Unilateral facial actinic elastotic plaque

Cutis rhomboidalis nuchae: It is clinical manifestation of severe sun damage to the normally redundant skin overlying the posterior nuchal area, in occupationally predisposed individuals. It appears as well defined furrows into an irregular rhomboidal pattern known as cutis rhomboidalis nuchae **(Fig 2)**. In severely affected individuals, cysts and comedones, presumably due to poor stromal support for the pilosebaeous units, also develop in the area.[28]

Cutaneous nodular elastoidosis with cysts and comedones (Favre-Racouchot syndrome): It usually affects the periorbital skin and upper lateral aspects of cheeks. Initially it manifests as patulous pilosebaceous orifices filled with keratinous debris superimposed on actinically damaged skin **(Fig 3)**. This condition occurs due to weakened dermal support around the pilosebaceous units caused by sun damage to the elastic and collagen fibers.

Elastotic nodules of the ear: These are discrete, semi translucent, asymptomatic papules approximately 5mm, which develop on the anterior crus of the helix.

Striated beaded lines: Develop in severely sun damaged skin overlying the anterior chest of men. Hyperplastic sebaceous glands, arranged in closely set parallel lines, are prominent against a background of markedly atrophic skin.

Stellate pseudoscars: These are multiangulated hypopigmented lesions that occur predominantly on the extensor forearms of elderly patients with severe actinic elastosis. They are usually preceded by hemorrhage (senile purpura), with or without erosion.

Actinic granulomas: These are annular lesions that develop on habitually sun-exposed skin, especially on the face. The condition probably represents a repair phenomenon within the sun damaged dermal connective tissue.[28] Three distinctive zones histologically have been described: peripheral zone of solar elastosis, the annulus or reactive zone and central or healed elastotic free zone.

Actinic comedonal plaques: Erythematous to bluish lesions consisting of nodules and plaques that have a distinctive pattern. Comedone -like structures are typically present and histologically the condition is characterized by dilated, keratin-filled follicular units within a dermal matrix of amorphous, sun-damaged collagen and elastic fibers.

Venous lakes: These are usually present over ear and lower lips with severe sun damage. Approximately 5mm in diameter, deep blue in color and disappear on diascopy. They may occur due to poor stromal support of already weakened, bulging blood vessels.[28]

PRURITUS: Pruritus or itch, may be the most common symptom of skin disease,particularly for the seventh and eighth decades. In elderly, pruritus often results initially from xerosis. It may frequently occur in the evening or at other quiet times.[25,26] Characteristic feature of pruritus include scratching and inflammation. Itching is thought to be induced by the effect of histamine and is mediated exclusively by peripheral nervous system. Itching evokes the desire to scratch. Scratching produces an immunologically-based inflammatory response.[25,29]

Senile pruritus is itch in an elderly patient in whom primary skin disease, xerosis, drug reaction and underlying systemic causes have been excluded.[30] There may be underlying neural mechanisms in causing itch as a result of peripheral nerve atrophy. Age related changes in the nerves lead to increased touch and pain thresholds, possibly due to subclinical neuropathy.

Idiopathic itch in elderly in some cases may be a result of this subclinical neuropathy. Loss of touch and nociceptive input might then allow central disinhibition of itch. This is analogous to the itch reported in the missing limbs of amputees, a phenomenon referred to as phantom itch. "Pseudophantom itch", due to subclinical neuropathy in the elderly, has been proposed as a mechanism for "senile pruritus".[29]

The itch-scratch cycle eventuates in lichenification, excoriations, infection and traumatic purpura.[26] The presence of primary lesions can establish the diagnosis. Excoriations are less frequent in dermal processes such as urticaria, despite severe itching. Lichenification is a secondary change that requires as much as 90 hours of scratching or as many as 140,000 scratches.[29] Pruritus can be psychogenic in origin. However, there are a number of dermatological and metabolic conditions that evoke pruritus. Many environmental situations that promote itching include stress, anxiety,

pollen, wool intolerance, pets and bedding. Dermatological conditions include infestations, infections, lichen planus, nodular prurigo, eczema and miliaria.

Localized pruritus without rash includes conditions like notalgia paresthetica, meralgia paresthetica, brachioradial pruritus, post-stroke pruritus, phantom itch, brain tumor or abscess, multiple sclerosis, transverse myelitis, diabetes mellitus. Generalized pruritus without diagnostic skin lesions is caused by many systemic conditions which are listed in **Table 7.**

Table 7: Systemic conditions in which generalized pruritus without diagnostic skin lesions [31, 32]

Metabolic and endocrine conditions	Thyroid diseases Carcinoid syndrome
Hematological disorders	Iron deficiency anemia,Polycythemia vera
Renal disease	Chronic renal failure
Hepatic	Biliary obstruction
Malignant conditions	Lymphoma,leukemia,solid tumors,myeloma
Psychogenic state	Delusion of parasitosis

Various drugs which cause generalized pruritus are listed in **Table 8.**

Table 8: Drugs that cause generalized pruritus [29]

Aspirin	Opioid agonists
Penicillins	Phenothiazines
Cephalosporins	Anticonvulsants
Sulfonamides	Benzodiazepines
Fluroquinolones	Antivirals
Antimalarials	Interferons
Qunidine	Gold
ACE inhibitors	Statins
Beta blockers	Anabolic steroids
Calcium channel blockers	Tamoxifen
Thiazide diuretics	Bleomycin
Sulfonylureas	Methoxsalen
Lithium	

ECZEMATOUS DISEASES OF ELDERLY: The geriatric population comprises a large percentage of the total patient population and the geriatric patient's chief complaint is often that of a pruritic rash or lesion heralding an eczematous disease. The overall prevalence of eczema is about 2.4%.Greater than 25% of all of the health care costs in an individual with eczema may be attributed to eczema and co morbid conditions.[33] The natural decline of the functional ability of the skin manifests clinically with physical changes of decreased turgor, thinning, dryness, ecchymosis and increased number of discrete lesions overall. Decreased amount of sebaceous glands and sebum production and diminished amount of hydration coupled with a reduction in the actual cellular numbers provide a template for extrinsic factors to induce further functional decline.[33]

In the early stages of an eczematous disease, the patient often presents with erythema, edema, scaling and crusting. Progression of the diseases process can lead to lichenification, spreading and pigmentation variation. The geriatric skin is especially vulnerable to the induction of the disease process and the decreased ability to combat and eventually resolve the process.[33]

The aged may suffer from any of the clinical types of eczema. Atopic dermatitis occasionally continues into old age or even appears for the first time. Common adult form of atopic dermatitis is hand dermatitis. It goes through several appearances or phases. In the beginning stages there is an erythema and edema which lead to vesiculation, crusting, excoriation, scaling and lichenification.

Asteatotic eczema or eczema craquele is an eczematous disorder that is virtually specific to the skin of the elderly, occurring against a background of generalized xerosis. It occurs mainly during the winter and is seen in patients with dry skin. General dry skin may be seen in patients with atopic eczema, chronic renal or liver failure, AIDS, myxedema and in malabsorption especially vitamin A or zinc. It occurs most often on the anterolateral portion of the lower legs. First the skin becomes dry and scaly and then red papules develop from scratching. The lesions may take on the appearance of cracked porcelain or "crazy paving'', which is a pattern of fissuring that develops when short vertical fissures connect with horizontal fissures.[34] Diuretics can predispose one to asteatotic eczema.

Eczema herpeticum is a combination of atopic dermatitis and the herpes simplex virus and is known as "Kaposi's varicelliform eruption''. It may be fatal if viremia occurs. About 10 days after exposure the patient develops the vesicles, usually in location where there was previously atopic dermatitis. Cases are seen at all ages, most commonly in the second and third decades.

Nummular eczema or discoid eczema is a form of constitutional eczema that seems more common in the elderly than in any other age group. It is characterized by single, non-specific circular or oval plaques of eczema with a clearly demarcated edge. Dry skin caused by low environmental humidity is associated with discoid eczema in elderly.

Stasis dermatitis affects a large portion of the elderly population. Venous or Gravitational eczema occur secondary to venous hypertension. It is characterized by bilateral circumferential dermatitis of the ankle and calf areas. The triad of alopecia, waxy appearance and yellow-brown pigment from hemosiderin deposition is diagnostic with or without pitting edema.[33] The elderly patients can have stasis dermatitis in places other than the lower extremities if they are not mobile. Places of chronic pressure such as buttocks, heels, forearms and any other site that rests on a solid surface may give rise to static integumental blood flow. It is often accompanied by dilatation or varicosity of the superficial veins, oedema, purpura, haemosiderosis, ulceration or small patches of white atrophic telangiectatic scarring (atrophic blanche). These changes are often modified by secondary contact dermatitis, infection and rubbing. Marked secondary lichenification and chronic lichen simplex are also often seen in older patients. Chronic stasis dermatitis appears lichenified and hyperpigmented and eventually lead to the "inverted champagne bottle" appearance of the leg. The disease can lead to increased susceptibility to ulceration or cellulitis. An acute exacerbation of stasis dermatitis can result in "id" reaction or autosensitization dermatitis, producing secondary, acute, papulovesicular, often symmetrical distribution on the extremities. [25, 35]

Seborrheic dermatitis is generally found in sebaceous rich tissue, such as the scalp and central face, more common in those confined to bed. The central nervous system may play a role in the severity of this disorder due to apparent increased incidence of this form of dermatitis with Parkinson's disease, quadriplegia, ischemic infarcts, Alzheimer disease and emotional stress. It may also be associated with diabetes mellitus.[25, 26] Association with HIV is seen in 20-85% of patients. It is characterized by flaky, scaly, greasy, erythematous, itchy rash. Commonly seen in the glabellar area between the eyebrows and nasolabial fold, postauricular area.[33]Especially in the obese patients, flexural pattern is often encountered, which may mimic intertrigo and flexural psoriasis.

CUTANEOUS INFECTIONS IN THE ELDERLY:

As people age they experience more illness and this applies in particular to skin infections. The integrity of the skin declines with age. The elderly are paradoxically more prone to both malnutrition and obesity, factors that facilitate the entry of pathogens into the skin. With the age the function of sweat glands decreases, the skin thins and the ambient moisture content of the skin declines.

Cutaneous infections in elderly are common due to: [36]

1) Compromised local cutaneous health predisposing to growth of infective organism.
2) Age associated decreased immune function. With advancing age, the immune system of animals and humans undergoes characteristic changes, usually resulting in decreased immune competence, termed immunosenescence.[37]
3) Underlying systemic disorders associated with decreased immune function.

Pathogenic states that provide the milieu for infection are more common in the elderly. In particular diabetes and neoplasms undermine the function of the immune

system. Diseases such as hyperlipidemia and hypertension decrease blood flow to the skin decreasing the ability of the elderly body to fight off infection. Decreased blood flow slows healing, increases xerosis and allows pathogens the opportunity to enter broken skin. Infections may be of bacterial, viral or fungal etiology.

Bacterial infections: A variety of Gram-positive bacterial infections are more common in the elderly than in younger persons. These include cellulitis, erysipelas, necrotizing fasciitis, folliculitis and furunculosis. [38] Impetigo and folliculitis in the elderly are caused by *Staphylococci*, in contrast to impetigo in the pediatric population, which is usually caused by *Streptococci*. Orbital cellulitis that is caused by *Streptococcus viridans* alone or in combination with Gram-negative bacteria is common in elderly. *Pseudomonas* cellulitis of the ear, an infectious process that affects elderly diabetic individuals, which is rare in younger population. Erysipelas, a β-hemolytic *Streptococcal* infection of the skin, is more common in the elderly and tends to spread more readily in this age group. Necrotizing fasciitis caused by a strain of *Streptococcus*, is more frequent in the elderly and is associated with increased morbidity and mortality in this age group.[39] The mortality rate of necrotizing fasciitis varies between 30%-50%.

Viral infections: Herpes zoster **(Fig 4)** is more common in old age, the relative incidence rising from 4 in 1000/year at age 55 years to 10 in 1000/year at age 90 years. It has been estimated that 25% of people over the age of 65 years develop herpes zoster at some time.[40] The incidence of herpes zoster is influenced by the age and immune status of the patient, but not the gender. The incidence continues to rise after the age of 60years.The lifetime risk is about 50% among those who survive to 85 years. Analysis of stressful life events did not reveal a definite relationship to risk of herpes zoster.

Post herpetic neuralgia (PHN) is the commonest complication occurring after zoster in the elderly. It is defined as persistence or recurrence of pain in an affected area for more than one month after the lesions have healed. The severity and duration of pain are age related, but it is not related to the degree of immunocompetence. The risk of PHN and the intractability of pain increases with age. Few children have PHN, whereas 27%, 47% and 73% of untreated adults over 55, 60 and 70 years of age, respectively, develop PHN. The incidence of PHN is as high as 20% in persons 60 years and older.[41] Early diagnosis and treatment of herpes zoster may minimize the intensity and duration of PHN. Whether or not skin lesions are present,systemic antiviral therapy should be initiated promptly, and is most efficacious when begun within 72 hours of the onset of symptoms.

Fungal infections: Fungal infections are among the most prevalent dermatologic conditions in the elderly, second only to benign and malignant tumors. They are caused by yeasts (*Malassezia, Candida*), dermatophytes (*Trichophyton, Microsporum*, *Epidermophyton*) and rarely nondermatophyte molds (*Scopulariopsis, Aspergillus*, *Fusarium*).

Onychomycosis is present in approximately 40% of patients after 60 years and tinea pedis is present in approximately 80% of this patient population. Although usually present for decades, **tinea pedis** may exacerbate with age. In elderly diabetic patients, interdigital tinea pedis may ulcerate and predispose to bacterial cellulitis, a presentation that is relatively rare in the young adult immunocompetent patient. Male gender and bedridden status are important risk factors for fungal infections. **Oral candidiasis** in elderly occurs due to predisposing factors like diabetes, vitamin deficiency or malnourishment, denture use, malignancy, immunosuppressive agents.

Cutaneous fungal infections and their causative organisms are listed in **Table 9**

Table 9: Cutaneous fungal infections and their common causes categorized by specific anatomic location: [42]

Anatomic location	Fungal infection	Pathogens
Scalp and face	Seborrheic dermatitis Tinea capitis	*Malassezia* species *Trichophyton tonsurans, Microsporum canis*
Oral mucosa	Thrush Atrophic candidiasis Angular chelitis	*Candida albicans*
Trunk	Tinea versicolor Tinea corporis **(Fig 5)**	*Malassezia globosa* *Trichophyton* species
Intertriginous sites	Intertrigo Perianal Erosio interdigitalis blastomycetica Tinea cruris	*Candida albicans* *Tinea rubrum*
Genitalia	Balanitis Vulvovaginitis	*Candida albicans*
Hands	Paronychia Tinea mannum	*Candida albicans* *Tinea rubrum*
Feet	Tinea pedis	*Tinea rubrum, T.mentagrophytes, E.floccosum*
Nails	Onychomycosis	*T.rubrum, C.albicans*, nondermatophyte molds

Because of impaired host defenses and a favorable environment at specific anatomic sites, there is an increased prevalence of seborrheic dermatitis, mucosal and cutanoeus candidiasis, tinea pedis and onychomycosis in the geriatric population compared with other age groups.

Parasitic Infestations: With the geriatric population on the rise, there are institutions for the elderly in all different formats, such as assisted-living facilities, nursing homes and so forth, being built up rapidly to accommodate. Because of these living arrangements, however, this population is at risk for infestations particularly scabies and pediculosis.

Scabies: scabies is an intensely pruritic and highly contagious infestation of the skin caused by *Sarcoptes scabiei*. Patients suffering from scabies have increased during the last 20 years. It causes major problems in nursing homes, particularly in debilitated patients. The risk factors for infection with scabies among nursing homes include age of the institution (more than 30 years), size of the institution (more than 120 beds) and the ratio of beds to health workers (>10:1). Transmission requires direct skin to skin contact with an infected person. A delayed type IV hypersensitivity reaction to the mites, their eggs, saliva and scybala occurs approximately within 2-6 weeks of infestation. This inflammatory reaction responsible for the intense pruritus, which is the hallmark of the disease. Patients show signs of infestation after about 2 weeks or more. The burrows are typically found on the hands, interphalangeal finger webs, wrists, waistline, anterior and posterior axillary folds. They can also be found on the external genitalia, nipples and buttocks. Scabies can exist in different forms. Nodular scabies comprise about 10% of the cases and usually resolves spontaneously after weeks or months. A distinctive highly contagious form of scabies, known as "Norwegian scabies" or "crusted or keratotic scabies", has a predilection for individuals who are immunocompromised (HIV disease, organ transplant recipients), aged, debilitated or mentally retarded. In regular scabies, on average the number of mites on a host at any one time is about 10 to 15 mites. In crusted scabies, the patient is infested with thousands to millions of mites. The skin manifestation is much more severe and it is usually not very pruritic. Anticipating secondary infection, such as *Staphylococcus aureus*, and potential epidemic outbreaks are critical when dealing with scabies. [43]

Pediculosis: Pediculosis is an infestation of the hairy parts of the human body. Lice are arthropods, which belong to the order Phthiraptera. There are two species of lice that infest humans; *Pediculus humanus* and *Phthirius pubis*. *P.humanus* is divided into subspecies, *P humanus capitis* (head louse) and *P humanus corporis* (body louse). Lice are transmitted through direct contact with bedding, brushes, clothing. Crowding is one of the risk factors for spreading the infection. Body lice have a strong association with poor socioeconomic level. Pubic lice are typically transmitted sexually and this frequently coexist with sexually transmitted disease.[43]

PAPULOSQUAMOUS DISORDERS IN THE ELDERLY:

The common papulosquamous disorders in elderly include psoriasis and lichen planus.

Psoriasis: It is a chronic, disfiguring, inflammatory and proliferative condition of the skin, in which both genetic and environmental influences have a critical role. Psoriasis has a bimodal distribution of age of onset. Those individuals with early onset appear, in general, to have more severe disease and are much likely to have an affected first degree relative with psoriasis. Males and females are equally affected by psoriasis. The bimodal peak in disease onset could be taken as evidence for the existence of two pathogenetically distinct forms of the disease. Type 1 is hereditary, strongly HLA associated particularly HLA –Cw6, early onset and more likely to be severe. Type 2 is sporadic, HLA unrelated, of late onset and usually mild. Drugs like lithium salts, antimalarials, non-steroidal anti-inflammatory drugs (NSAIDS), angiotensin-converting enzyme (ACE) inhibitors and the withdrawal of corticosteroids may exacerbate psoriasis. Photosensitive psoriasis is associated with skin type I, advanced age and female sex. Most forms of psoriasis present before the fourth decade, although pustular psoriasis of the palms and soles is extremely rare before adult life.

In old age, psoriasis may appear unexpectedly for the first time, either spontaneously or following a pre-existing skin disease.[44] Clinically characterized by well defined erythematous scaly plaques distributed bilaterally over extensor surfaces of the body particularly the elbows and knees, lumbosacral area, back and scalp **(Fig 6 & 7)**.

Nail changes are present in 25-50% of all cases. There is no sex predilection, but patients over 40 years of age are affected twice as often as those under 20 years. Although pitting is the most frequent changes seen, discoloration, subungal hyperkeratosis, onycholysis and splinter haemorrhages can occur. Patients with psoriasis are at an increased risk of developing malignancy, particularly non-melanoma skin cancer and lymphoproliferative cancers. In elderly drug-induced or drug-aggravated psoriasis, especially for patients receiving polypharmacy or with recent worsening or poor response to conventional therapy is common. Other frequently encountered forms of psoriasis in the elderly include psoriatic arthritis and its complications, inverse psoriasis and potentially life-threatening complications such as erythrodermic or acute pustular psoriasis, where early recognition and systemic therapy is critical. Topically applied medications, such as topical corticosteroids, salicylic acid, tar and dithranol preparations, calcipotriol and tazarotene, are the favored first-line therapeutic options in the elderly. Narrowband ultraviolet B phototherapy is also well established as a standard therapy for psoriasis. Systemic therapy with agents such as methotrexate, acitretin and cyclosporin should be judiciously reserved for severe, extensive cases in view of their lower therapeutic index in the elderly. Clinical severity of psoriasis increases among very elderly patients and is associated with different levels of skin-related quality of life impairment and psychological distress.

Lichen planus (LP): Lichen planus is an inflammatory, papulosquamous disorder affecting either or all of the skin, mucous membranes, hair and nail. The prevalence of LP is less than 1% worldwide.[45] Females appear to be more commonly affected than men. The average age of onset is the fourth decade in males and the sixth decade in females.[46] At least two-thirds of cases occur between the ages of 30 and 60 years of age.[45] The development of LP may be affected by seasonal or environmental factors. HLA-B8 is more common in patients with oral LP as a sole manifestation, and HLA-Bw35 is more strongly associated with cutaneous LP. The classic cutaneous lesion is a faintly erythematous to violaceous, flat-topped, polygonal papules distributed mainly on the flexor aspects of the extremities, associated with intense pruritus. A thin, transparent and adherent scale may be discerned atop the lesion. Fine, whitish puncta or reticulated networks referred to as Wickham striae are present over the surface of many well-developed papules.[45] Lichen planus of the oral cavity produces significant functional and symptomatic problems for elderly patients. Oral lesions often occur in the absence of concomitant skin lesions.

Unlike cutaneous lesions, oral lesions of lichen planus may become a chronic, recurring problem that is difficult to control. Rarely the disorder has a dramatic onset and presents as acute widespread scaling and erythema. Hypertrophic type is most frequently seen on the lower legs and forearms and tends to be persistent and very itchy. It may be more frequent in the elderly. A very rare complication of hypertrophic variant is squamous cell carcinoma.[47] LP runs a chronic course, over months to several years.

BLISTERING DISEASES IN THE ELDERLY: Blistering diseases in the elderly are a rare group of diseases that can be immune-mediated, drug induced or secondary to other systemic diseases. The autoimmune blistering diseases primarily affect

mucosal and cutaneous tissues. Common autoimmune blistering disorders in elderly in the order of frequency include bullous pemphigoid, cicatricial pemphigoid, linear IgA bullous disease, epidermolysis bullosa acquisita, pemphigus.

Pemphigus: Pemphigus is an autoimmune mucocutaneous blistering disease. Patients typically have autoantibodies which target specific proteins in the epidermal-epithelial cell membrane resulting in the loss of cell-cell adhesion. These lesions typically present as blisters and ulcers of mucosal and cutaneous tissues. The mean age of onset is usually the fifth decade. [48]

Pemphigus vulgaris is the most common form among the pemphigus family, accounting for approximately 70% of all cases of pemphigus. The disease usually begins in the oral cavity. It can be localized to it, may become generalized and involve 20% to 50% of the skin in severe disease. Involvement of ocular, nasal, pharyngeal, laryngeal, vaginal, penile and anal mucosa typically occurs in severe disease. Cutaneous lesions are typically present on the upper trunk, head, neck and intertriginous areas. Healing usually occurs without scarring, and can often be accompanied by postinflammatory hyperpigmentation.

Paraneoplastic pemphigus also known as “paraneoplastic autoimmune multiorgan syndrome”, described as a separate subtype of pemphigus associated with certain malignancies. It is a rare autoimmune mucocutaneous blistering disorder characterized by an associated neoplasm and the presence of unique antibodies directed at desmosomal plakins. The majority of patients are between the ages of 45 and 70 years. The commonest clinical feature which is also usually the earliest presenting sign, is recalcitrant stomatitis, seen as painful erosions and ulcerations of the oropharynx and vermilion border of the lips. It resemble pemphigus vulgaris or herpetic stomatitis. The skin lesions are polymorphous and pruritic and can resemble

erythema multiforme, toxic epidermal necrolysis, bullous pemhigoid or lichen planus.[49] The most common neoplasms include non Hodgkin's lymphoma, Chronic lymphocytic leukemia, Castleman's disease, thymomas, Waldenstrom's macroglobulinemia and bronchogenic carcinoma. It affects both mucosal and cutaneous tissues. There are up to one third of patients with this condition who do not have a detectable malignancy and often present many years after their previous malignancy was considered to be in remission.[48]

Drug induced pemphigus: The causes of drug-induced pemphigus are thiol drugs or SH drugs, whose molecules contain a sulfhydryl or thiol group in their chemical structure. D-Penicillamine is the commonest causative drug. Non-thiol drugs include penicillin, ampicillin, rifampicin, propranolol, phenytoin and phenobarbitone. Pemphigus vulgaris was the commonest form induced, with pemphigus foliaceous sen in only 15%.[49] The possible mechanisms vary from a direct toxic effect of the drug following systemic absorption to an allergic mechanism where pemphigus is induced by contact with plants and vegetables containing polyphenolic compounds.

Hailey-Hailey disease: It is also known as "benign familial pemphigus," is an autosomal-dominant genodermatoses. Although the age of onset and presentation is primarily seen in a younger age group, it is also seen in minority of patients in an older age group.[48] Lesions are most commonly distributed in the axilla, groin ,lateral parts of the neck , inframammary areas , antecubital and popliteal fossa, and perianal region. Exacerbating factors associated with Hailey-Hailey disease include heat, sweating and infections.

Bullous pemphigoid: Bullous pemphigoid is an autoimmune blistering disease with a mean age of onset of 65 years. The incidence is estimated in six to seven cases per million population per year with no gender predilection.[48] It is the most common

immune-mediated blistering disease affecting older patients. There is a relatively high incidence of anti-basement membrane zone antibodies in elderly individuals and are more susceptible to developing bullous pemphigoid. The disease is self-limited, lasting months to years, with recurrences following disease free periods in a minority of patients.[50] Untreated, it ranges in severity from mild to disabling, and the resulting prolonged loss of a effective cutaneous barrier may be fatal. It is characterized by large tense bullae on flexor surfaces of the extremities, axillae, groin and abdomen. The lesions can appear on erythematous or normal appearing skin. Approximately one-third of patients have oral blisters. Healing can occur with postinflammatory pigmentation changes. Bullous pemphigoid has also been associated with other autoimmune disorders, such as diabetes, psoriasis, pernicious anemia, rheumatoid arthritis, and multiple sclerosis. Malignancies of the gastrointestinal tract, urinary tract, lymphoreticular system, pancreas, genitalia, and breast may also be associated with the disease. The serum samples from elderly subjects possessed a relatively high incidence of anti-basement membrane zone antibodies. This observation of a specific immune defect in elderly individuals might explain that they are more susceptible to developing bullous pemphigoid.

Diagnosis is confirmed by skin biopsy. Immunofluorescent staining of perilesional skin is virtually pathgnomonic, showing linear deposition along the basement membrane zone of C3 in all patients and of IgG. Indirect immunofluorescent studies demonstrate antibasement-membrane zone antibodies of the IgG class in approximately 70% of patients. In pemphigoid, systemic corticosteroids and immunosuppressive agents are recommended.[48]

Cicatricial pemphigoid(CP): Also known as "mucous membrane pemphigoid," is a rare, chronic subepidermal blistering disease with a mean age of onset of 73 years.

Women are affected twice as frequently as men. There is no racial prediliction. The reported incidence varies from 0.87 to 1.16 cases per million people. Cicatricial pemphigoid usually affects mucosal sites and occasionally the skin. CP can often remain localized to the oral mucosa. Other areas of the oral cavity can also be involved including buccal mucosa, palate, tongue and lips. In some patients the predominant involvement is conjunctiva. Bullae and erosions heal with scarring. This scarring is often responsible for the major morbidity and irreversible sequelae associated with CP. It is a debilitating disease. Early diagnosis and treatment are necessary to prevent irreversible sequeale.[49]

PIGMENTARY CHANGES IN ELDERLY:

Uneven pigmentation is one of the major changes in the gross morphologic characteristics of aging skin and is much more marked in sun exposed areas. In epidermis, melanin pigments are good indicators of photoaging. Ultraviolet radiation plays a major role in the induction of melanocyte aging and may severely damage the melanocyte system of the skin, resulting in both hypermelanotic and hypomelanotic lesions.

Pigmentary changes of chronologic aging: In subjects older than 25 to 30 years, the number of dopa-positive melanocytes decreases with age by approximately 10% to 20% per decade. The number of melanocytic nevi increases from birth until young adulthood, followed by a relatively long periods of quiescence. Melanocytic nevi are rarely present in individuals older than 80 years and may involute after 50 years of age.

Graying of hair is a very common phenomenon that is usually irreversible. Senile graying is due to a decreased number of active melanocytes in the hair follicle and decreased activity of the remaining melanocytes.

Pigmentary changes of photoaging: Melanocyte density decrease with age in both sunexposed and protected areas, but the density is approximately 2-fold higher in skin with long-term sun exposure at all ages. Photoaged skin is characterized by mottled, irregular areas of pigmentation. The phenomenon is paradoxical with regard to the decrease in melanocyte density with age. The hyperpigmentation correlates with increase dopa-positivity of melanocytes. The mottled appearance may be due to the irregular distribution of melanocytes along the basement membrane, associated with a heterogenous distribution of melanosomes within the keratinocytes.[51]

Actinic or solar lentigines are very common benign lesions. The incidence of solar lentigines increase with age, affecting more than 90% of elderly. Both chronic and acute sun exposure are important in the pathogenesis of solar lentigines.[52] The macules are usually dark brown in color. They vary in size from a few millimeters to more than 1 cm in diameter. Solar lentigines may coalesce and have regular or irregular borders. They darken significantly after exposure to sunlight. Both chronic and acute sun exposures are important in the pathogenesis.

Solar lentigines may be extremely difficult to distinguish from other pigmented lesions of the elderly, especially lentigo maligna. In contrast to premalignant lesions, electron microscopic studies demonstrate a lack of melanosomal pleomorphism and atypical cytologic features of the melanocytes.[51]

Actinic keratosis are typically nonpigmented, and pigmented forms also exist named as spreading pigmented actinic keratoses. They occur on the face and are larger than 1 cm in diameter. Progressive lateral spread may result in large areas of extensive actinic damage.

Erythromelanosis interfollicularis of the neck and face is a common disorder characterized by a triad of erythema, follicular papules and

hyperpigmentation. The pale yellowish follicular papules lead to skin that resembles a plucked chicken. Between the papules, the erythema is due to melanin pigmentation. The lesions involve the sides of the neck and spare the shaded submental area, consistent with the causative role of long-term sun exposure.

Idiopathic guttate hypomelanosis (IGH) is an acquired leuckoderma, characterized by discrete, round or oval porcelain white macules of approximately 2-5mm in diameter, which increase in number with aging **(Fig 8)**. It was seen in only 20% of patients between ages of 20 and 30 but in 80% of patients over the age of 70.[53,54] Frequency in women is slightly higher than in men. The lesions are generally found in a photodistribution and tend to occur in chronically sun damaged skin.[54] It causes esthetic concerns in patients because it is found specifically on the sun exposed surfaces of the forearms and legs. Lesions are found most commonly on the arms and in descending order of frequency the legs, trunk and face. It is a disorder with multifactorial etiology, its pathogenesis may depend on various factors such as patient age and sun-exposure. The dopa-positive melanocytes and melanin content were found to be reduced in the lesions of idiopathic guttate hypomelanosis.[55]

Stellate peudoscars appear as small spots preferentially located on the back of the hands and forearms. They are common, occuring mainly after 70 years of age and develop in patients with diffuse atrophy and severe photoaging.

COMMON BENIGN SKIN TUMORS:

Cherry angioma (Campbell de Morgan's spot): Cherry angiomas are acquired vascular lesions that occur in upto 50% of adults. The lesions tend to appear most often on the trunk and extremities and can be up to several millimeters in diameter. They are round, bright to dark red non blanching vascular papules **(Fig 9)**. They first occur in early adulthood and increase in number with age.[56] They are asymptomatic.

They are composed of dilated and congested capillaries and venules.[57] The etiology is unknown, however eruption of multiple lesions following exposure to various chemicals, including mustard gas has been reported. They are also associated with exposure to bromides, solid organ transplantation, chronic graft-versus-host disease, cyclosporine therapy, 2-butoxyethanol and argon laser therapy. They are treated for cosmesis. Options include laser treatment, electrodessication of typical lesions and excision of larger lesions.[58,59]

Sebaceous hyperplasia (Senile hyperplasia): Sebaceous hyperplasia is common in middle aged and elderly persons. In patients with rare familial forms. The condition begins during puberty. It may also be seen in heart transplant recipients due to the process of dysplastic epithelial proliferation.[60] It consists of soft, yellow, dome - shaped papules, some of which are centrally umbilicated. Commonly occurring on the forehead, cheeks and nose, most lesions are 2 to 4 mm in diameter, but can also be up to 5cm in size. It can also occur on vulva. Except for cosmesis, the condition has no clinical significance, however the lesions are sometimes confused with early basal cell carcinomas. With its characteristic mosaic appearance, the surface of sebaceous hyperplasia is generally less uniform than that of basal cell carcinoma.[56]

Histologically, sebaceous hyperplasia consists of enlarged lobules of mature sebaceous glands with a central dilated duct. Although the etiology is unknown, solid organ transplant recipients taking cyclosporine and patients receiving hemodialysis are at increased risk. Treatment options include electrodesiccation, laser therapy and topical bichloroacetic acid.

Seborrhoeic keratosis: Seborrhoeic keratosis are common benign skin lesions in older individuals. Occasionally solitary, they usually present as multiple brown papules or plaques. There is a familial predisposition with a probable autosomal dominant inheritance for the development of seborrhoeic keratosis. Higher prevalence is noticed in sun exposed areas. They occur only on hair bearing skin sparing the mucosal surfaces, the palms, and the soles. The face, neck and trunk especially the upper back and the extremities are commonly affected. Lesions are typically sharply marginated, and include macules, papules or plaques depending on their stage of development. They vary in color, from brown to black. They may be flat, verrucous, polypoid or pedunculated **(Fig 10)**. Keratotic plugging with follicular prominence contributes to a stippled or velvety surface and gives a stuck-on appearance**(Fig 11)**.[57] Individual lesions grow larger than 5cm, but most lesions usually measure approximately 0.5-1cm in diameter. Lesions may become inflamed from rupture of small horn pseudocysts or trauma. Traumatized or inflamed lesions are often erythematous and crusty and they may be painful or pruritic. The **Leser-Trelat sign** is the abrupt appearance and rapid increase in size and number of multiple seborrhoeic keratoses as a result of underlying malignancy.[61,62] Usually associated with adenocarcinoma of stomach, colon, lung or breast. Most of them appear on the back and chest, followed by the extremities, face, abdomen, neck, axillae and groin.[63] Sometimes the eruption is described as appearing in a "Christmas tree" or "splash" pattern, rain drop pattern, although this may occur with eruptive seborrhoeic keratosis unassociated with malignancy.[64] Pruritus accompanying the sign occurs in almost half the patients. Malignant acanthosis nigricans is most frequently seen with the sign of Lesar-Trelat and is evident in about 35% of patients. Regression typically takes place when the underlying condition is treated or resolves. Differentiating between

seborrheic keratosis and melanoma is a clinical challenge. Both have variable dark colors, the potential for large size, and irregularity. A key differentiating feature is that a melanoma tends to vary more in color, such as brown, blue, black, gray and red, where as seborrheic keratosis tends to be rougher than that of melanomas, which is smooth, yet often friable. Treatment of seborrheic keratosis is indicated for cosmetic reasons, to decrease irritation or to rule out malignancy. Most common methods used are cryosurgery, curettage and excision.

Dermatosis papulosa nigra (DPN): Dermatosis papulosa nigra consists of multiple dark brown to black small papules on the face of darkly pigmented individuals. It is considered as a variant of seborrheic keratosis. It has a strong familial predisposition. Lesions most commonly appear in the peri-adolescent years, progressing through different stages of development and peaking in the sixth decade. Women are twice as likely to be affected. It is characterized by the presence of multiple hyperpigmented, sessile, smooth-surfaced papules measuring from 1 to 5mm in size, occasionally pedunculated lesions are seen **(Fig 12)**. The papules are distributed symmetrically on the face, especially on the malar areas and the forehead. They may also be found on the neck, chest and back. They gradually increase in size and number over time and do not regress.[65]

Acrochordons: Acrochordons or skin tags are derived from ectoderm and mesoderm and represent a hyperplastic epidermis. They are found in 25% of persons and increase in number with age. Obesity is a predisposing factor. The axilla, neck and inguinal regions are the most common sites. They are usually pedunculated but can also be sessile. They range in size from less than 1mm to 1cm in diameter and are skin-colored or brown.[57]

Keratoacanthoma: Keratoacanthomas are rapidly growing lesions that occur primarily on sun-exposed skin in elderly. The majority of lesions involve the face and upper extremities although they frequently occur on the lower extremities, especially in women. They begin as papular lesions, enlarging over 2 to 4 weeks to a size of 2 cm or more. The papule often develops an umbilicated keratinous core. After 4 to 6 months, the lesion involutes with expulsion of the core, often leaving a hypopigmented scar. They are usually solitary, but multiple lesions may be present. Possible causative agents include ultraviolet light, human papillomavirus and prolonged contact with coal tar derivatives. Biopsy and treatment are recommended, because of histological similarity to squamous cell carcinoma and possible scarring after involution. Total excision is the preferred treatment for most solitary keratoacanthomas. For smaller lesions, electrodesiccation and curettage is adequate.[56]

Epidermoid cyst: Epidermoid cysts, also kown as inclusion cysts or epidermal inclusion cysts are round and mobile, ranging in size from a few millimeters to several centimeters. The cyst is filled with keratin and lined with stratified squamous epithelium. They commonly occur on the back, face and chest and communicate with the skin through a small, round, keratin filled plug. The cysts may remain small for years or may grow rapidly. Rupture of the cyst wall into the dermis initiates an inflammatory response. Indications for excision include cosmesis, pain and recurrent infection.[57]

PREMALIGNANT AND MALIGNANT SKIN TUMORS IN THE ELDERLY:

Premalignant keratinocytic lesions in elderly include actinic keratoses, arsenical keratoses, thermal keratoses, chronic radiation keratoses, viral keratoses, Bowen's disease, plasma cell balanitis of Zoon, erythroplasia of Queyrat, leukoplakia,

erythroplakia. Malignant conditions in elderly include squamous cell carcinoma, basal cell carcinoma and malignant melanoma.

Actinic keratosis: Actnic keratosis is the most common precancerous skin lesion. It occurs primarily in light skin types. It develops as a result of cumulative effect of solar radiation on the exposed skin in susceptible individuals, usually occurring after the age of 65.[62] Asiatic race are much less prone to developing solar keratosis owing to their greater cutaenous melanin protection. More common among males. These are proliferations of transformed, neoplastic keratinocytes that are confined to the epidermis and induced by exposure to UV radiation in sunlight.[66] The initial damage takes place in the DNA and most of the UV-induced lesions in the DNA are repaired. Neoplastic transformation occurs in keratinocytes that have been exposed to UV radiation and is due to primarily mutations in the p53 gene.[67] These mutations appear to induce activation of oncogenes and or inactivation of cell growth and apoptosis. In time, these cells proliferate in the epidermis and eventually extend into the dermis, at which metastatic spread can occur. Cytologic atypia is visible in early stages and is identical to that seen in metastatic lesions or in squamous cell carcinoma in the dermis. While these remain confined to the epidermis, the lesions that they cause are termed as actinic keratosis, but when they extend more deeply to involve the papillary and or reticular dermis, they are termed squamous cell carcinoma. They occur on sun-exposed areas such as the face, lower lip, bald scalp, neck, arms and hands. They appear as rough, scaly papules and plaques range in size from 1mm to 2.5cm or more in diameter **(Fig 13)**. They may be best diagnosed by running the hand over the skin surface. In their earliest stages, they are more easily felt than seen, having a sandpaper like texture.[68] They may also serve as a marker for people at increased risk for development of malignant melanoma. Actinic keratosis result ultimately in

invasive squamous cell carcinoma. Indeed 1-10% of actinic keratosis develop into squamous cell carcinoma. The most common treatment options are cryotherapy with liquid nitrogen, 5-fluorouracil, 5% imiquimod cream, 3% diclofenac gel and curettage.[69]

Squamous cell carcinoma in situ (Bowen's disease): It affects both skin and mucous membranes and has the potential to progress to invasive squamous cell carcinoma. It may occur at any age in adults, but is rarely seen in individuals younger than 30 years. The typical patient with Bowen's disease is older than 60 years. It can be found on any body site, including both sun-exposed and non-sun exposed regions of the body, although there is predilection for sun-exposed surface such as the head and neck and for lower legs of women. A number of factors have been implicated in the development of Bowen's disease, including history of significant sun exposure, arsenic exposure, ionizing radiation, immunosuppression, HPV 16 infection. It typically presents as a discrete, slowly enlarging, pink to erythematous thin plaque with well demarcated, irregular borders and overlying scale or crust. Individual lesions may measure up to several centimeters in diameter. Clinical variants include intertriginous Bowen disease and mucosal type of Bowen disease. Clinically, it is often mistaken for superficial basal cell carcinoma, patches of dermatitis, psoriasis or lichen planus. The presence of Bowen disease is a marker for a high risk of developing a subsequent non melanoma skin cancer. Treatment includes curettage, cryosurgery, 5-fluorouracil, 5%imiquimod, laser ablation, radiotherapy.[70]

Plasma cell balanitis of Zoon: It is an idiopathic benign disorder of uncircumcised male genitalia in the middle aged, though an analogous condition has been described on the vulva, mouth, lips and epiglottis. This chronic, reactive, principally irritant, mucositis brought about by a dysfunctional prepuce with friction playing a part. This

condition presents as a circumscribed, persistant plaque with a shiny smooth surface on a glans penis. The plaque is moist with a glistening appearance an has minute red specks (cayenne-pepper spots). The lesion is usually asymptomatic and single. It has to be differentiated from erythroplasia of Queyrat, which clinically has a velvety surface and shows features of squamous cell carcinoma in situ on histopathology. Circumcision is curative. Topical corticosteroids cause mild improvement, but the lesions usually recur following discontinuation of treatment.

Erythroplasia of Queyrat: It is an in situ squamous cell carcinoma affecting the mucosal surfaces of the penis in uncircumcised males. It presents as a well demarcated, shiny, velvety, bright red plaque. It occurs in uncircumcised men between the ages of 20 and 80 years, although the majority of cases are seen between the 3rd and 6th decades. Risk factors include poor hygiene, accumulation of smegma, heat, friction, trauma and genital herpes simplex virus infection. Affected males complain of localized pain, pruritus, difficulty in retracting the foreskin over the glans, bleeding and crusting. Lesions persist and enlarge slowly, and they typically have been present for several years. Several treatment options are available, including excision, Mohs micrographic surgery, CO2 laser ablation, topical 5-FU, and topical imiquimod.[69]

Squamous cell carcinoma (SCC): Squamous cell carcinoma is a malignant neoplasm derived from suprabasal epidermal keratinocytes.[70] In most cases it evolves from precursor lesions of actinic keratoses and Bowen's disease. It represents a broad spectrum of disease ranging from superficially invasive cancers to highly infiltrative, metastasizing tumors. It is strongly associated with advanced age and a sharp increase in incidence is seen after age of 40 years. Increased exposure to UV radiation and greater longevity are possible causes for the increase in disease. It is twice as common

in men as in women, probably as a result of greater lifetime UV exposure in men.[71]Persons with fair complexion, red hair, blue eyes are at greater risk. It arises on sun-exposed areas such as head, neck and dorsal hands. In black populations it arises most often on site of pre-existing inflammatory skin condition, burn injuries or trauma.[72] A firm, flesh colored or erythematous, keratotic papule or plaque is most common, they also may be pigmented. They may also present as an ulcer, smooth nodule or a thick cutaneous horn. Margins may be indistinct. With enlargement, there is usually increased firmness and elevation. Progressive tumor invasion results in fixation to underlying tissues. Clinically, verrucous or scaly lesions has to be differentiated from benign conditions like wart, seborrheic keratosis, actinic keratosis, melanocytic nevus, pyogenic granuloma, eccrine poroma. Treatment includes non-excisional ablative techniques, Mohs surgery, conventional surgical excision, radiation therapy.

Basal cell carcinoma (BCC): BCC is a malignant neoplasm derived from non keratinizing cells that originate in the basal layer of the epidermis. BCC is the most common cancer in humans.[73] It is more common in elderly individuals. The tumor characteristically develops on sun-exposed skin of lighter skinned individuals, more common on nose. Risk factors include UV exposure, light hair and color and inability to tan. Individuals with sun-sensitive skin, timing and character of solar exposure may be more important than cumulative dose in predicting adult BCC risk.[74] Patients with BCC are at increased risk for melanoma but not for other internal malignancies. BCC usually develops on sun exposed areas of the head and neck but can occur anywhere on the body. Nodular and infiltrative BCC occur most commonly on the sun exposed areas of the face and neck. Superficial BCC is frequently detected on the non sun exposed trunk.[75] Features include translucency, ulceration, telangiectasias and the

presence of a rolled or waxy border **(Fig 14)**. BCC clinical subtypes include nodular, pigmented, superficial, sclerosing, fibroepithelioma of Pinkus. BCC resembles conditions like dermal nevus, appendageal tumor, SCC, dermatofibroma, scar, seborrhoeic keratosis, skin tag, trichoepithelioma and fibroma. Management includes surgical excision, Mohs micrographic surgery, curettage and electrodesiccation, cryosurgery, topical 5% imiquimod cream and 5-fluorouracil.

Malignant melanoma: It occurs among all adequately studied racial and ethnic groups. Its incidence is much lower compared to non melanoma skin cancers but has been rising in fair-skinned populations through out the world for several decades. The risk factors for melanoma occurring in an individual include a combination of constitutional predisposition(skin color, tendency to freckle, family history of melanoma, presence of a large number of naevi, increasing age) and exposure to environmental factors (UV light). There is a strong association between sun exposure during critical periods of early life and subsequent risk of melanoma during adulthood.[76,77] Suspicion of malignant melanoma in any pigmented lesion follows the "ABCD" rule: Asymmetry, Border irregularity, Color variation or dark black color, Diameter greater than 6mm. Hence, any change in the shape, color or size of a pigmented lesion should arouse suspicion of cancer. The ugly duckling sign refers to a melanotic lesion that is atypical beyond the context of surrounding naevi. Malignant melanoma can occur anywhere on the skin and is often seen on the back, head and neck in males, lower extremities in females, and the palms and soles in blacks. Melanoma is the leading cause of death from skin disorders and the mortality rates from melanoma have been increasing during the past half century. It accounts for 79% of all skin cancer deaths.[78] Early detection is vital, as the prognosis depends on the depth of penetration of the lesion. A lesion that is clinically suspicious for melanoma

should ideally undergo an excisional biopsy with narrow margins (2mm). An incisional biopsy should be reserved for cases in which tumor is too large to be excised.

METHODOLOGY

Source of Data:

A hospital based, cross sectional study was conducted in the department of Dermatology, Venereology and Leprosy, B.L.D.E.U's Shri.B.M.Patil Medical College Hospital and Research Centre, Bijapur. The study was carried out on 550 consecutive patients, aged 65 years and above who attended the Dermatology OPD and referred from inpatient wards of other departments. The study was conducted during the period of October 2008 to May 2010.

METHOD OF COLLECTION OF DATA:

INCLUSION CRITERIA:

Geriatric patients over 65 years of age irrespective of gender were included in the study.

METHODS:

In all the subjects included for the study, demographic data regarding name, age, sex, occupation, socioeconomic status and address were recorded. A complete clinical examination of skin, nail, hair and mucous membrane was carried out. General physical examination and systemic examination was performed. In the subjects with cutaneous disorders, a detailed history of the illness, regarding duration, onset, evolution, symptoms and recurrence was recorded. Relevant investigations such as complete haemogram, absolute eosinophil count, biochemical tests, cytology and a skin biopsy was performed whenever required.

STATISTICAL ANALYSIS:

The observations pertaining to parameters under study among the geriatric age group are expressed in percentage. Diagramatic representation of affected patients based on gender and different age groups were included in this study. Z test is applied for testing differences of proportion based on gender. F test is applied for testing differences of proportion based on different age groups and χ^2 is applied for testing association of skin diseases with co morbid conditions.

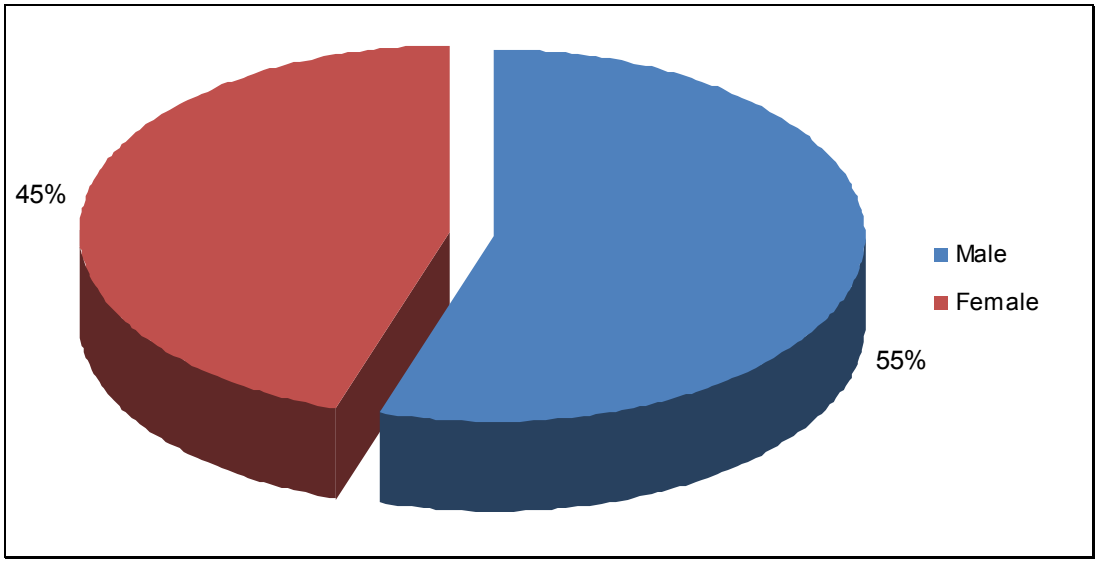
RESULTS:

Table 10 : Gender distribution in geriatric patients

Sex	Total	Percentage (%)
Male	302	55
Female	248	45

Graph: 1

Gender distribution in geriatric patients



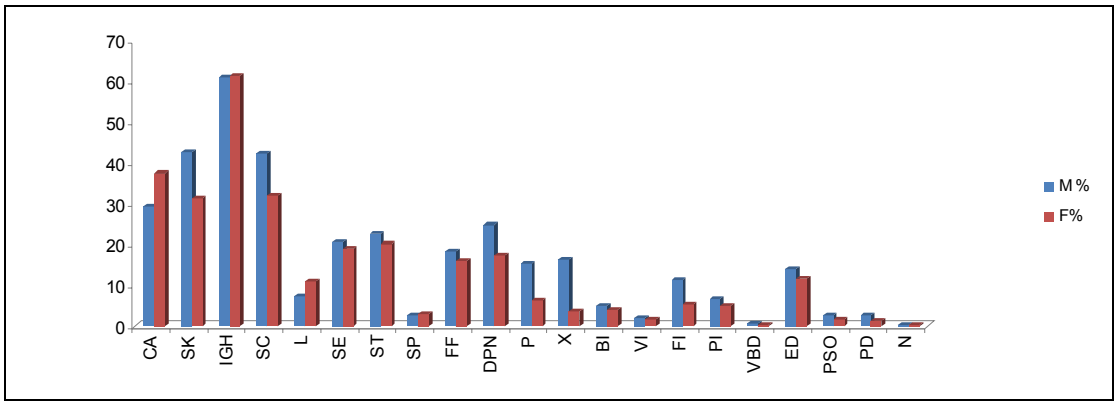
Of 550 elderly patients males were 302 (55%) and females were 248 (45%).

Table 11 : Distribution of skin findings in the elderly

Diseases	Male		Female		Total	
	No.	%	No.	%	No.	%
Cherry angiomas	89	29.47	93	37.5	182	33.091
Seborrheic keratosis	129	42.715	78	31.452	207	37.636
IGH	184	60.927	152	61.29	336	61.091
Senile comedones	128	42.384	79	31.855	207	37.636
Lentigines	22	7.2848	27	10.887	49	8.9091
Solar elastoses	62	20.53	47	18.952	109	19.818
Skin tags	68	22.517	50	20.161	118	21.455
Senile purpura	8	2.649	7	2.8226	15	2.7273
Fissure feet	55	18.212	40	16.129	95	17.273
DPN	75	24.834	43	17.339	118	21.455
Pruritus	46	15.232	16	6.4516	62	11.273
Xerosis	49	16.225	9	3.629	58	10.545
Bacterial infections	15	4.9669	10	4.0323	25	4.5455
Viral infections	6	1.9868	4	1.6129	10	1.8182
Fungal infections	34	11.258	13	5.2419	47	8.5455
Parasitic infestations	20	6.6225	12	4.8387	32	5.8182
Vesiculobullous disorders	2	0.6623	1	0.4032	3	0.5455
Eczematous disorders	42	13.907	29	11.694	71	12.909
Psoriasis	8	2.649	4	1.6129	12	2.1818
Pigmentary disorders	8	2.649	3	1.2097	11	2
Neoplasms	1	0.3311	1	0.4032	2	0.3636

Graph: 2

Incidence of skin findings in the elderly



Of all the cutaneous disorders in elderly, idiopathic guttate hypomelanosis (IGH) was most commony seen in 336(61%), among which 184 (60.9%) were males and 152 (61.2%) were females followed by seborrheic keratoses which was seen in 207 (37.6%), among which 129 (42.7%) were males and 78 (31.4%) were females. Senile comedones were seen in 207(37.6%), 128 (42.3%) males, 79 (31.8%) females.

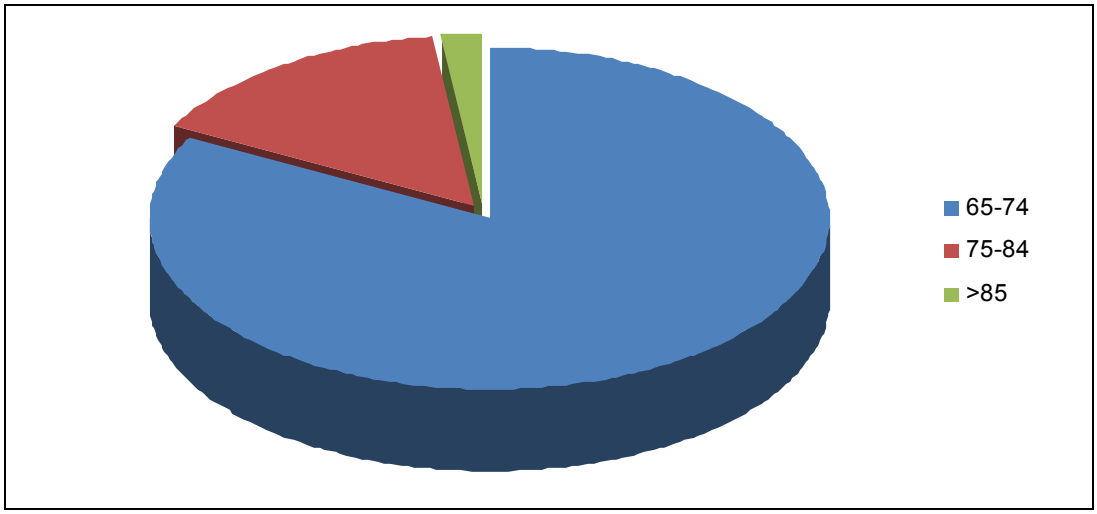
In males fungal infections P=0.005(<0.05), were significantly more common than females.

Table 12 : Distribution of patients in different age groups

Age group	Total	Percentage (%)
65-74	456	82.9
75-84	84	15.2
>85	10	1.81

Graph : 3

Distribution of patients in different age groups



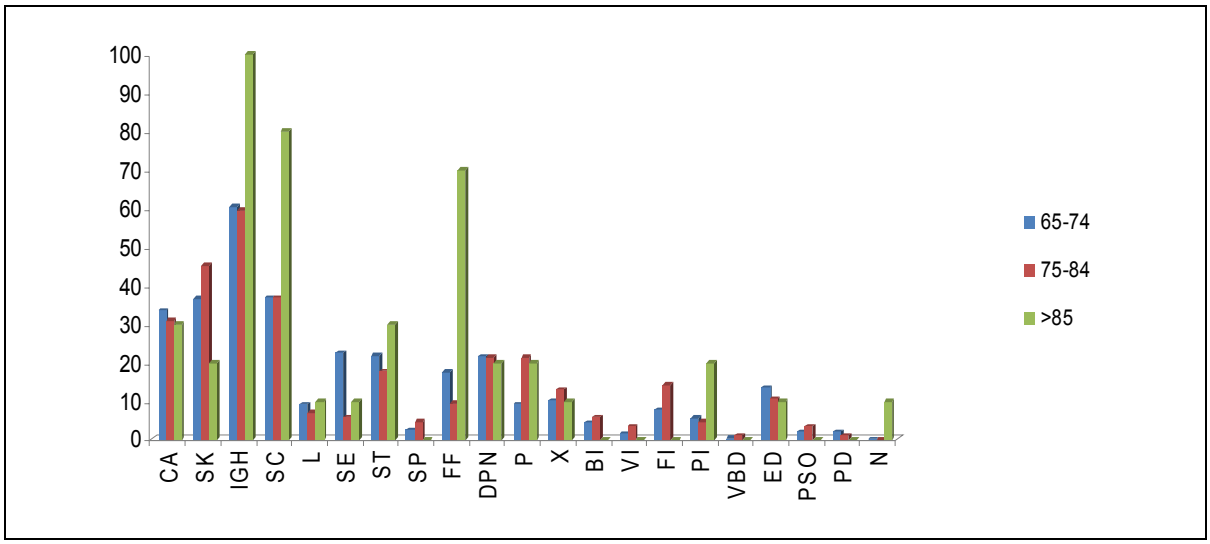
Of the 550 patients, there were 456(82.9%) patients belonging to the 65-74 years age group, 84 (15.2%) belonging to 75-84years and only 10(1.81%) patients were above 85 years.

Table 13 : Distribution of skin findings according to age group

Skin findings	65-74 years		75-84 years		>85 years	
	No.	%	No.	%	No.	%
Cherry angiomas	153	33.553	26	30.952	3	30
Seborrheic keratosis	167	36.623	38	45.238	2	20
Idiopathic guttate hypomelanosis	276	60.526	50	59.524	10	100
Senile comedones	168	36.842	31	36.905	8	80
Lentigines	42	9.2105	6	7.1429	1	10
Solar elastoses	103	22.588	5	5.9524	1	10
Skin tags	100	21.93	15	17.857	3	30
Senile purpura	11	2.4123	4	4.7619	0	0
Fissure feet	80	17.544	8	9.5238	7	70
Dermatosis papulosa nigra	98	21.491	18	21.429	2	20
Pruritus	42	9.2105	18	21.429	2	20
Xerosis	46	10.088	11	13.095	1	10
Bacterial infections	20	4.386	5	5.9524	0	0
Viral infections	7	1.5351	3	3.5714	0	0
Fungal infections	35	7.6754	12	14.286	0	0
Parasitic infestations	26	5.7018	4	4.7619	2	20
Vesiculobullous disordes	2	0.4386	1	1.1905	0	0
Eczematous disorders	61	13.377	9	10.714	1	10
Psoriasis	9	1.9737	3	3.5714	0	0
Pigmentary disorders	10	2.193	1	1.1905	0	0
Neoplasms	1	0.2193	0	0	1	10

Graph: 4

Distribution of skin findings according to age group



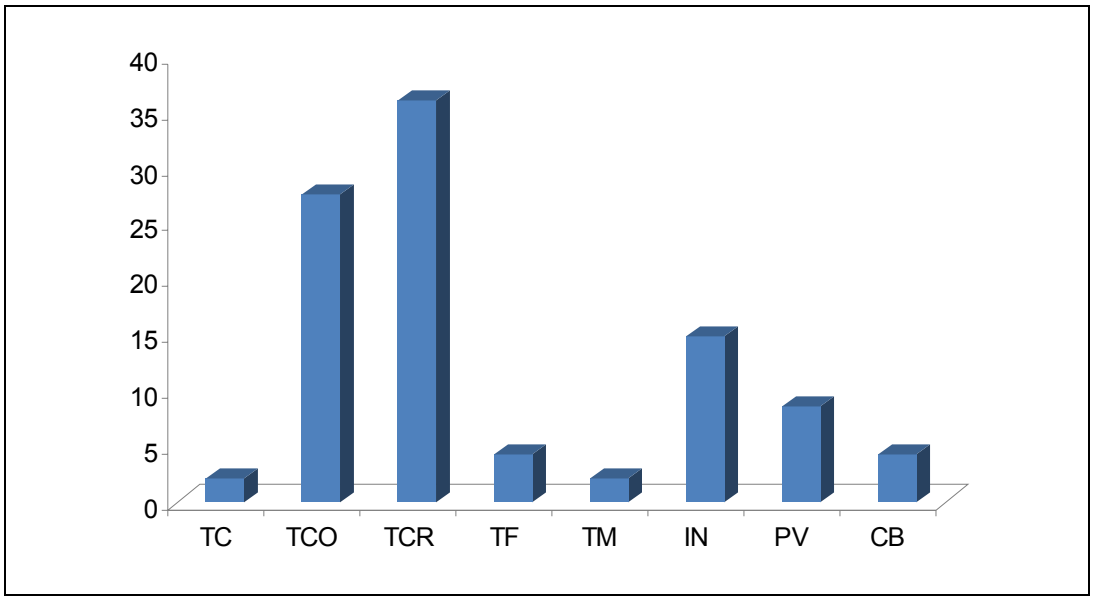
Among the age group 65-74, idiopathic guttate hypomelanosis 276 (60.5%) is the commonest cutaneous disorder seen followed by senile comedones 168 (36.8%) and seborrhiec keratosis 167(36.6%). Among the age group 75-84, IGH 50 (59.5%) is the commonest finding seen followed by seborrhiec keratosis 38(45.2%). In patients with above 85 years, IGH 10(100%) is the commonest finding, followed by senile comedones 8(80%). There is statistically significant association of these findings with age group P=0.00<0.05.

Table 14 : Distribution of fungal infections in elderly

Fungal infections	Total	Percentage(%)
Tinea capitis	01	2.12
Tinea corpris	13	27.6
Tinea cruris	17	36.1
Tinea facei	02	4.2
Tinea manum	01	2.1
Intertrigo	07	14.8
Pityriasis versicolor	04	8.5
Candidal balanitis	02	4.2

Graph: 5

Distribution of fungal infections in elderly



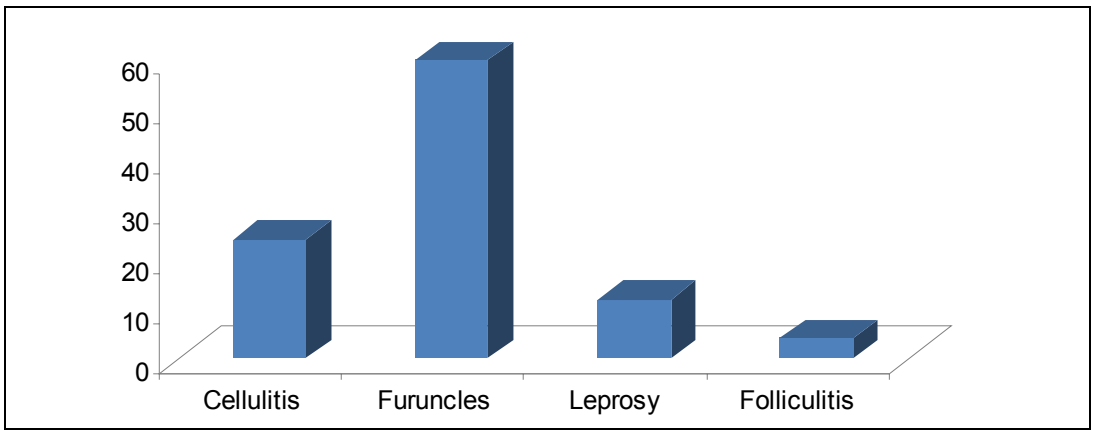
Among the fungal infections tinea cruris 17(36%),I s the most common infection, followed by tinea corporis 13(27.6%).

Table 15: Distribution of bacterial infections in elderly

Bacterial infectons	Total	Percentage (%)
Cellulitis	06	24
Furuncle	15	60
Leprosy	03	12
Folliculitis	01	04

Graph: 6

Distribution of bacterial infections in elderly



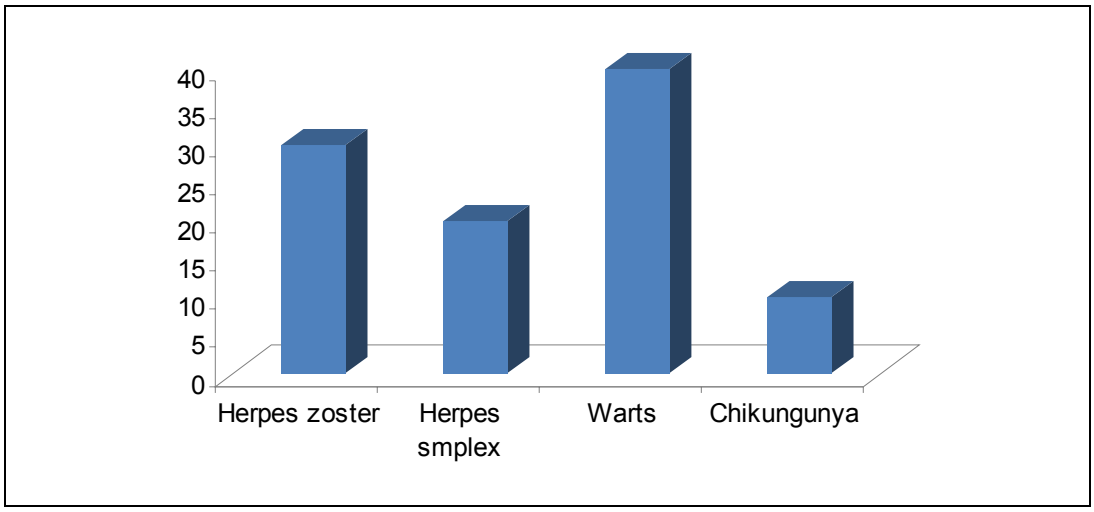
Among bacterial infections, furunculosis 15(60%) is the commonest infection seen followed by cellulitis 06(24%).

Table 16 : Distribution of viral infections in elderly

Viral infections	Total	Percentage(%)
Herpes zoster	03	30
Herpes simplex	02	20
Warts	04	40
Chikungunya	01	10

Graph: 7

Distribution of viral infections in elderly



Among viral infections, commonest infection seen is warts 04(40%), followed by herpes zoster 03(30%).

Table 17: Distribution of vesiculobullous disorders in elderly

Vesiculobullous disoders	Total	Percentage(%)
Pemphigus vulgaris	02	66.6
Bullous pemphigoid	01	33.3

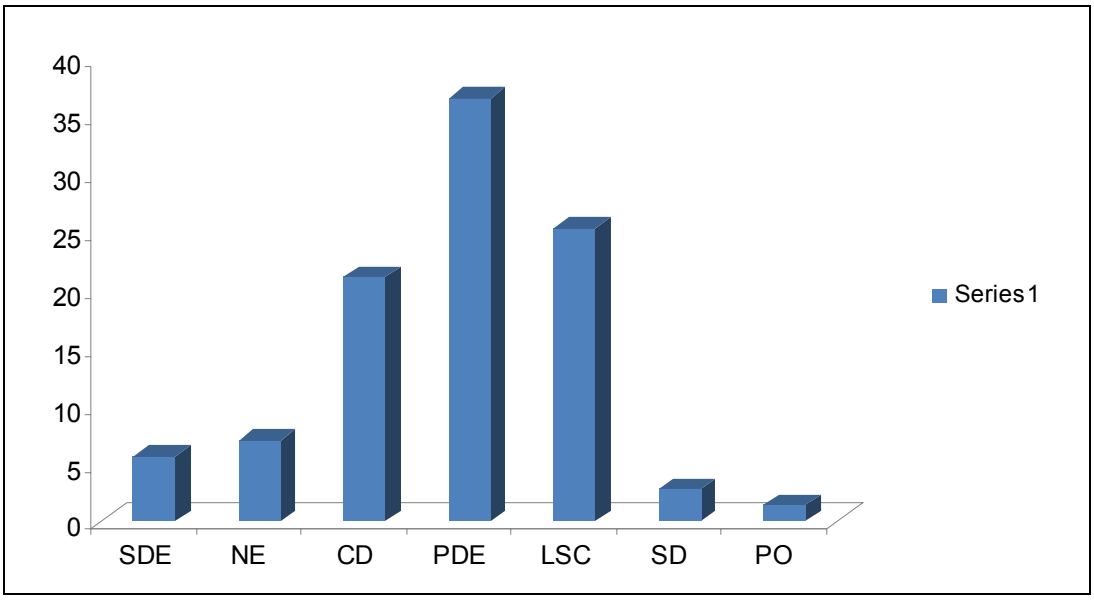
Among vesiculobullous disorders, pemphigus vulgaris was seen in 02(66.6%) patients followed by bullous pemphigoid in 01 (33.3%).

Table 18 : Distribution of eczematous disorders in elderly

Eczematous disorders	Total	Percentage(%)
Seborrhiec dermatitis	04	5.6
Nummular eczema	05	7.04
Contact dermatitis	15	21.1
Photodermatitis	26	36.6
Lichen simplex chronicus	18	25.3
Stasis dermatitis	02	2.8
Pompholyx	01	1.4

Graph : 8

Distribution of eczematous disorders in elderly



Among patients with eczematous dermatosis, photodermatitis 26(36.6%) is the commonest disorder followed by lichen simplex chronicus 18(25.3%) and contact dermatitis 15(21.1%).

Seborrhiec keratosis 207(37.6%) is the commonest neoplasm seen followed by cherry red angioma 182(33.09%) and dermatosis papulosa nigra 118(21.4%). Basal cell carcinoma is the only malignant condition seen in 2 (0.3%) patients.

. Pruritus P=0.00(<0.05) and xerosis P=0.00(<0.05) were significantly associated with anemia.

DISCUSSION

The elderly population, aged 65 or older, constitutes a large and rapidly growing segment of the population. As India is passing through the last phase of demographic transition, the life expectancy among elderly at age 60 was estimated at 16 years for males and 18 years for females and at age 70, it was 10 years for males and 12 years for females.[1] The rapid demographic shift has created challenges to the health care system. Because the burden of dermatologic diseases in the elderly is increasing and substantial, and as their dermatologic demands are largely unmet, it is important for health care providers to note the pattern of geriatric skin disorders.

GENDER DISTRIBUTION OF CUTANEOUS DISORDERS IN THE ELDERLY:

All the 550 elderly patients included in this study had one or the other cutaneous lesions. In the present study, there were 302 (55%) males and 248 (45%) females. Males outnumbered the females, which is comparable to that of previous studies.[2,3,79,,80,81] Certain photoaging skin changes like seborrhoeic keratosis (42.7%) and senile comedones (31.8%) are noted more in males. Similar findings have been noted in a study conducted by Pathange VS (24.5%) from India.[2] Among 550 patients 43.6% of patients were agricultural labours.

Among cutaneous diseases fungal infections ($P=0.005<0.05$) have been noticed more in males than females which is statistically significant as comparable to that of previous studies.[3,80,82] This variation in gender may be due to the more contact with environmental and physical factors, differences in clothing and more physical activities in males who have more active outdoor life. There was no statistically significant difference noted in gender among certain disorders like bacterial and viral infections, parasitic infestations, vesiculobullous disorders and malignant tumors.

AGE DISTRIBUTION OF CUTANEOUS DISORDERS IN THE ELDERLY:

The frequency of some disorders change as the age advances. Photoaging skin changes like idiopathic guttate hypomelanosis and senile comedones have been noted more in patients above 85 years than earlier age groups, as these conditions are common in chronically sun damaged skin.

The frequency of pruritus (21%) and xerosis (13%) is noted more in later age groups. As the age advances, there is decrease in sebum secretion, increase of dry skin which may be responsible for more number of patients complaining of pruritus and xerosis in the advanced age group. Similar findings have been reported in previous study.[3] Loss of cutaneous barrier due to scratching or dryness predisposes the patient to bacterial infections. This signifies the importance to educate elderly in minimizing xerosis by repeated application of emollients. Anemia and underlying systemic diseases like diabetes mellitus, chronic renal failure, and malignancies should be ruled out in patients with pruritus and xerosis. In the present study, there is statistically significant association of pruritus and xerosis with anemia (P 0.00<0.05).

There is increase in frequency of parasitic infestations above 85 years of age. Lack of personal hygiene in patients of this age group who are more dependent on others may predispose them to infestations. There is need to educate them about personal hygiene and overcrowding in order to prevent the infestations.

Increase in incidence of malignancy (10%) has been seen in patients above 85 years of age. As the age advances there is increase in cumulative effect of sun exposure and carcinogens and impaired capacity to repair DNA, which may be the reason for increase in frequency of malignancy in advanced age group. This finding is comparable to that of previous study.[3]

Patients with age group of 65 - 74 years showed statistically significant increase of eczematous disorders as there is more contact with allergens and environmental conditions in this group. Similar finding was reported in previous study.[3]

Fissure feet (70%) has been noted more above 85 years. Impairment of skin barrier function, improper foot care and geographical location with its environmental effects may be the cause of increased frequency of this condition.

Table 19 : Common cutaneous disorders in elderly as compared with other studies

Skin disorders	Present study (%)	Grover S[81] (2009) (%)	Yalcin B [3] (2006) (%)	Smith DR[82] (2002) (%)	LiaoYH[80] (2001) (%)	Chopra A[79] (1999) (%)	Patange VS[2] (1995) (%)
IGH	61	76.5	-	-	-	-	24.5
Seborrhoeic keratosis	37.6	43	-	-	-	24.2	37
Senile comedones	37.6	6.5	-	-	-	-	11.5
Eczema	12.9	39	20.4	7.3	58.7	2.8	-
Fungal	8.5	35	15.8	61.6	38	5.6	18.5
Bacterial	4.5	3.5	7.3	-	8.6	4.7	-
Viral	1.8	5	6.7	-	12.3	0.9	-
Bullous disorders	0.5	0.5	1.5	-	0.8	3.6	-
Psoriasis	2	-	-	-	3.9	5.6	10.5
Malignancy	0.3	-	-	0.3	2.1	0.5	-

Note: IGH-idiopathic guttate hypomelanosis.

SKIN CHANGES OF PHOTOAGING:

Among the photoaging skin changes, idiopathic guttate hypomelanosis (61%) is the commonest finding noted followed by seborrhoeic keratosis (37%) and senile comedones(37%). This variation from other studies conducted by Grover et al [81] and Pathange VS[2] may be due to different ethnic, regional, occupational and environmental variations. Cutaneous malignancies like basal cell carcinoma and malignant melanoma may arise from seborrhoeic keratosis and they also have association with underlying malignancies like adenocarcinoma of stomach or colon, and care must be taken to critically evaluate any rapidly growing, symptomatic or unusual lesions.[83]

INFECTIONS:

Fungal infections (8.5%) were more common among infections as compared to that of other studies followed by bacterial (4.5%) and viral infections (1.8%).[2,3,79,80,82] The common fungal infection noticed was tinea cruris (36%). This may be due to differences in clothing and difficulty in maintaining hygiene in elderly.[84] Furunculosis (60%) is the commonest bacterial infection noted which is comparable that of study conducted by Liao YH.[80] There was no statistically significant association of these infections with diabetes mellitus($P=0.095>0.05$). Viral warts (40%) are the commonest viral infections noted. In elderly, there is decrease in personal care, epidermal turnover and immunologic functions which may be responsible for the higher rate of infections. Loss of cutaneous barrier due to xerosis and repeated scratching and also underlying conditions like diabetes mellitus may predispose patients to infections.

ECZEMATOUS DISORDERS:

Eczematous disorders are noticed in 12.9% of patients in the present study. The variation with other study conducted by LiaoYH[80] may be due to various racial, environmental and occupational differences. Photodermatitis (36%) is the commonest dermatitis noticed in the present study with aggravation during summer **(Fig 15)**. This may be due to chronic exposure to the sun, altered barrier function and faulty antigen removal. This is followed by lichen simplex chronicus (25%). Xerosis in elderly leads to repeated scratching or rubbing which causes lichenification of skin. Contact dermatitis (21%) in elderly may be due to increased chance of exposure and entry of various allergens and irritants due to impaired skin barrier. This condition is predominant in agriculturists in the present study which increases the risk of exposure to contact allergens. Seborrheic dermatitis is seen in 5.6% of patients. Seborrheic dermatitis increases throughout late phase of life, may be due to immune system impairment, mood depression syndromes, overall morbidity and comorbidity, age related loss of self-sufficiency.[85] In the present study 50% of patients were associated with diabetes mellitus. Systemic corticosteroids and immunosupressants which are used in eczematous disorders should be given with caution as there will be impaired immune response and renal clearance in elderly.

PAPULOSQUAMOUS DISORDERS:

Among the papulosquamous disorders, psoriasis is noticed in 12(2%) of the patients which is less comparable to that of previous studies.[2,79,80] This is followed by lichen planus (0.3%) and pityriasis rubra pilaris (0.18%). Psoriasis is associated with numerous comorbidities that have a major impact on severely affected patients. Besides psoriatic arthritis, other diseases such as metabolic syndrome, cardiovascular diseases, reduced quality of life, depression, malignancy are of major importance.[86,87]

In particular, patients with severe forms of psoriasis are at a higher risk of developing cardiovascular diseases and myocardial infarction. An increased prevalence of concomitant diseases leads to an increased intake of medications that could affect the onset, severity and course of psoriasis. Medications used to treat psoriasis may influence comorbidities and complicate the management of severely affected patients.[88]

VESICULOBULLOUS DISORDERS:

The frequency of vesiculobullous disorders (0.5%) was less compared to study conducted by Yalcin B.[3] Bullous disorders noted were Pemphigus vulgaris 2(66.6%), bullous pemphigoid 1 (33.3%). These disorders may be associated with comorbid conditions like diabetes mellitus, pernicious anemia, rheumatoid arthritis, multiple sclerosis and underlying malignancies.[48] Diabetes mellitus (33.3%) was associated with vesiculobullous disorders in the present study. Medications used for vesiculobullous disorders like systemic corticosteroids and immunosuppressives may increase the risk of underlying conditions. Elderly with vesiculobullous disorders should be evaluated for these comorbid conditions and managed with utmost care especially with respect to the clinical course and their long term prognosis.

CUTANEOUS DRUG REACTIONS:

The common cutaneous drug reactions (0.3%) noted in the study was fixed drug reaction and maculopapular rash. This is less compared to studies conducted by Yalcin B [3] and LiaoYH.[80] The commonest group of drugs noted in the present study were non steroidal antiinflammatory drugs.The elderly are particularly at increased risk of adverse drug reactions attributed to polypharmacy and physiological changes affecting the pharmacokinetics and pharmacodynamics of many drugs or poor compliance due to cognitive impairment or depression.[89] An important risk factor for

developing adverse drug reaction is the previous occurrence of it. Re-exposure to offending drugs due to poor documentation can cause the patient to experience the same adverse drug reaction again, thus emphasizing the importance of accurate documentation of reaction at the time of the event and providing relevant information to the elderly patient about it helps to prevent further occurrence.

OTHER CONDITIONS:

The other cutaneous disorders noticed in the study were urticaria (0.5%), cutaneoussma ll vessel vasculitis (0.18%), lichen sclerosus et atrophicus **(Fig16)** (0.3%), amyloidosis (0.18%), keloid (0.3%), cutaneous horn (0.18%).

CUTANEOUS MALIGNANCIES:

The greater incidence of malignancy in advancing age may be due to increase exposure to sunlight which increases the risk of carcinoma. Basal cell carcinoma 2 (0.3%) is the only malignant condition noticed in this study with occurrence on face which signifies the role of sun exposure as risk factor. In other reported studies from India this condition was not seen, however malignant melanoma (0.4%) was seen in one study.[79] In a study conducted by Liao YH BCC was noted in 29.8% of cases.[80] This variation may be due to the racial and cultural differences. Skin with more innate pigmentation provides a more efficient protection against sunlight than fair skin.[80] BCC is generally a disorder of white individuals, in whom there is a lifetime risk of 28%-33% of developing this malignancy. It typically occurs in areas of chronic sun exposure.[90]

NAIL CHANGES:

The commonest nail change noticed was longitudinal ridging (21.4%). This was the common nail finding noticed in previous study conducted in India by Grover S.[81] This is due to altered turnover rate of the matrix cells in the elderly.[91]

COMORBID CONDITIONS:

Associated systemic ailments were noticed in 19.4% of patients. Hypertension (8%) was the commonest followed by diabetes (7.4%) as comparable to that of study conducted by Grover S.[81]

CONCLUSION

Elderly patients are at risk of developing photoaging and chronological age related skin lesions. The former manifests as variety of benign skin tumors. The latter manifests as xerosis and pruritus. The associated co-morbid conditions in already susceptible elderly patients may prone to infections and eczematous conditions.

Thus cutaneous disorders cause considerable morbidity in elderly individuals. Health promotion and education about photoprotection, hydration of skin and management of co-morbid conditions can do much to reduce the risks of these disorders in elderly individuals. The database demonstrates the need for further studies addressing clinical, etiological and therapeutic issues of major dermatoses affecting the quality of life of elderly.

SUMMARY

A hospital based, cross-sectional study on cutaneous disorders in 550 elderly patients was conducted from October 2008 to May 2010. Cutaneous disorders in elderly based on gender, different age groups and associated comorbid conditions were analyzed.

Following are the salient observations:

- Male : Female ratio was 1.2:1
- Most of the patients 456(82.9%) were in age group of 65-74 years.
- Photoaging skin changes like, seborrhoeic keratosis 207(37.6%) and senile comedones 207(37.6%) were noted more in males. Cherry red hemangiomas 182(33.09%) and lentigenes 49(8.9%) were more in females.
- Cutaneous diseases like fungal infections 47(8.5%) are noted more in males.
- Eczematous disorders 61(13.3%) were more common in the age group of 65-74 years.
- Fissure feet 7(70%), parasitic infestations 2(20%), malignancies 1(10%) were noted more above 85 years of age.
- Among infections fungal infections 47(8.5%) were commonly noticed infections.
- Photodermatitis 26(36.6%) was the commonest eczematous disorder noticed.
- Among the papulosquamous disorders psoriasis is noticed in 2% of the patients.
- Pemphigus vulgaris 2(66.6%) was the commonest disorder noted followed by bullous pemphigoid 1(33.3%) among vesiculobullous disorders.

- Basal cell carcinoma was the only cutaneous malignancy seen in 2 (0.3%) patients.
- Associated systemic ailments were noticed in 107(19.4%) patients. hypertension 44(8%) was the commonest followed by diabetes 41(7.4%).
- Anemia 11(2%) is significantly associated with xerosis 58(10.5%) and pruritus 62(11.2%) respectively.

The present study provides data on various cutaneous disorders in elderly based on gender, different age groups and associated comorbid conditions. Health promotion and education of these disorders can reduce the morbidity in elderly individuals. The database demonstrates the need for further studies addressing clinical, etiological and therapeutic issues of major dermatoses affecting the quality of life of elderly.

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SCHEME OF CASE TAKING

Department of Dermatology, Venereology and leprosy

B.L.D.E.A'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR

SL.NO Date OP/IP. No

Name Age Sex-M/F

Socioeconomic status Address

Occupation

Cutaneous diseases

1. Complaints :
2. Duration:
3. Type of lesions
4. Distribution of lesions:
5. Severity and associated symptoms:
6. Others
7. Systemic diseases – 1 2 3 4

Duration

Treatment

Duration

Drugs 1.

2.

8. Personal history:

9. Family history:

General Physical Examination:

Pallor Icterus Clubbing Cyanosis

Pedal edema Lymphadenopathy

Vitals: BP- Pulse

Systemic Examination

RS: CVS: CNS: Abdomen:

Mucous membrane:

Nail changes:

Hair changes:

Investigations

Blood- Hb TC DC ESR

Peripheral smear

Platelet count

Urine Sugar Albumin Microscopy

RBS

Absolute eosinophil count

Liver function tests

Renal function tests

Thyroid function tests

SAMPLE INFORMED CONSENT FORM BLDEA'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR-586 103

RESEARCH INFOMED CONSENT FORM

TITLE OF THE PROJECT :- A SURVEY OF CUTANEOUS DISORDERS IN GERIATRIC AGE GROUP.

PG GUIDE :- DR. ARUN C. INAMADAR

PG STUDENT :- DR. S.SUDHA

PURPOSE OF RESEARCH :-

I have been informed that this project will study the various patterns of Dermatological problems in geriatric age group and the variations in the frequency depending on age and gender.

BENEFITS:-

I understand that my participation in this study will help the investigator to understand the disease better and will help in the management of the disease.

PROCEDURE :-

I understand that relevant history will be taken and I will undergo detailed clinical examination after which necessary investigation will be done whenever required.

RISK AND DISCOMFORTS:-

I understand there is no risk involved and I will experience minimal pain during the procedures performed.

CONFIDENTIALITY :-

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs, videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION :-

I understand that I may ask more questions about the study at any time concerned. The researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION :-

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that the researcher may terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician if this is appropriate.

INJURY STATEMENT :-

I understand that in the unlikely event of injury to me resulting directly from my participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____

_____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent to participate as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

KEY TO MASTER CHART

M: Male

F: Female

CA: Cherry angiomas

SK: Seborrheic keratosis

IGH: Idiopathic guttate hypomelanosis

SC: Senile comedones

L: Lentigines

SE: Solar elastosis

ST: Skin tags

SP: Senile purpura

FF: Fissure feet

DPN: Dermatosis papulosa nigra

P: Pruritus

X: Xerosis

BI: Bacterial infections

VI: Viral infections

FI: Fungal infections

PI: Parasitic infestations

ED: Eczematous disorders

PSO: Psoriasis

PD: Pigmentary disorders

N: Neoplasms

SD: Systemic diseases

BCC: Basal cell carcinoma

DM: Diabetes mellitus

HTN: Hypertension

A: Asthma

AN: Anemia

TB: Tuberculosis

MASTER CHART

Sl. No	NAME	AGE	SEX	CA	SK	IGH	SC	L	SE	ST	SP	FF	DPN	P	X	BI	VI	FI	PI	ED	PS0	PD	N	SD
1	S.R.Bendi	80	M	+	+	+	+			+										+				
2	Sangawwa	65	F	+						+			+											
3	Yallappa	70	M			+	+		+			+				+								
4	Chandrasekar	65	M	+		+		+												+				
5	Gurawwa	65	F	+				+								+								
6	Rukamawwa	65	F		+	+	+					+								+				
7	Neelanagamma	70	M	+		+	+		+											+				
8	Rukamawwa	65	F		+	+	+					+								+				
9	Kallappa	80	M	+	+		+		+			+		+	+									
10	Lakshman	65	M	+	+				+			+	+									+		
11	Neelamma	70	F			+			+				+							+				
12	Jamunabai	70	F			+		+	+			+												
13	Danawwa	65	F		+	+	+				+		+											
14	Gurawwa	70	F	+			+		+	+														
15	Basappa	76	M		+							+	+					+						DM
16	Iramma	68	F	+			+		+	+				+	+									
17	L.L Rathod	68	M	+	+	+	+	+						+	+									
18	Pandit navi	65	M			+	+		+			+		+	+									
19	Sharanamma	65	F	+		+		+	+							+								
20	Bibilabai	68	F				+	+	+	+		+							+					
21	Ganagawwa	70	F	+	+	+			+															
22	Paramanna	65	M	+			+		+			+							+					
23	Kallappa	65	M		+	+	+			+							+							
24	Channappa	74	M	+		+	+									+								
25	Jayadevi	90	F	+		+			+			+							+					
26	Basavangoud	65	M			+	+		+			+												
27	Ningappa	83	M		+		+		+					+	+									
28	Khazı	75	M			+	+		+													+		
29	Monappa	80	M		+	+	+		+				+								+			
30	S.B Hiremath	73	M	+	+	+						+	+					+						
31	Gurulingawwa	70	M	+	+				+			+	+	+	+									
32	Martusab	65	M	+	+				+			+						+						DM
33	Bhimsha	65	M	+	+	+	+		+	+		+							+					
34	Gouramma	65	F	+		+			+													+		
35	Kashibai	70	F			+			+		+	+												
36	Humupawar	65	M	+			+		+															HTN
37	Sahadev	70	M		+	+						+												
38	N.S Gouda	70	F	+	+	+			+											+				
39	Hajeppa	66	M			+	+	+	+												+			
40	Basawwa	80	M	+					+				+							+				
41	Nanagouda	65	M	+		+	+	+	+											+				
42	Hipparigi	65	F		+	+			+	+			+							+				

43	Sahabee	80	F			+			+			+	+											DM
44	Mallappa	74	M	+	+	+							+											
45	Parasappa	65	M	+	+	+			+			+		+	+									
46	Kasibai	68	F	+		+		+	+															
47	Shankarappa	72	M	+		+	+		+		+	+		+	+									
48	Gurubasappa	65	M	+		+			+			+												
49	Kattimani	65	M	+		+	+		+					+	+									
50	kayanappa	85	M		+	+		+	+			+	+	+	+						+			
51	Satagouda	71	M				+	+	+			+		+	+									
52	Hiremath	73	M		+	+	+	+	+			+				+								
53	Byaed	73	M		+	+	+		+	+		+									+			
54	Hanumanth	70	M			+	+		+					+										
55	Gurappa	68	M			+	+	+	+	+				+	+									
56	Nimbaz	65	M		+	+	+	+												+				DM
57	Chandran	70	M	+		+	+					+		+	+									
58	Neelapagouda	73	M			+	+					+												
59	Susilabai	65	F	+		+	+	+															+	
60	Ramanna	88	M	+		+	+		+	+		+									+			
61	Ittasbai	70	F	+		+		+	+											+				
62	C.S Biradar	66	M		+	+						+	+					+						
63	Ranganath	78	M	+	+	+			+	+				+	+	+								
64	M.B Ganagchari	65	M		+	+			+			+						+						
65	Basamma	65	F	+		+	+																	
66	Chandappa	65	M		+	+	+						+											
67	Kashibai	75	F		+	+			+															
68	Nagamma	65	F			+	+		+															
69	Keshavrao	67	M				+	+	+				+		+									
70	Kashibai	70	F	+	+	+			+														+	
71	Pradani	68	M	+		+		+	+							+								
72	Khandoba	65	M			+	+		+	+				+										
73	Ratnarati	80	F	+		+	+	+										+						
74	Nimbewwa	65	F	+	+	+						+												
75	Droupathi	65	F	+		+		+													+			
76	Bsagondappa	65	M	+		+	+														+			
77	Bhimappa	66	M		+	+	+		+					+										
78	Chandrabai	65	F			+		+	+															
79	Shantilal	73	M	+	+	+															+			
80	Janardhan	69	M	+		+			+												+			
81	Neelamma	70	F	+		+			+												+			
82	Shevanta	70	F		+							+										+		
83	Kasturibai	65	F			+			+												+			
84	Bhimarayal	66	M	+		+	+						+			+					+			
85	Shettappa	85	M						+															
86	Gurulingappa	70	M		+		+																BCC	
87	Putlabai patil	65	F	+		+			+															
88	Gourabai	70	F	+					+			+												
89	Sharanrao	80	M				+		+	+								+						

90	Guragouda	70	M	+		+			+										+					
91	Danamma	65	F	+	+			+											+					
92	Danamma	65	F	+	+			+											+					
93	Shakuntala	65	F					+	+											+				
94	Biradar B.M	67	M	+		+												+						
95	Abdula	80	M						+				+	+										
96	Shivappa	72	M	+		+	+						+			+								
97	Gangabai	65	F					+	+															HTN
98	Seetawwa	66	F			+							+											HTN
99	Sanwanna	65	F						+				+											
100	Chandrabai	65	F				+		+															
101	Mahappa	70	M						+		+	+												
102	Rajakbee	70	F	+		+							+											
103	Parwati	80	F	+					+															
104	Irubai	67	F			+			+					+										AN
105	Somanagouda	68	M			+	+																	
106	Shankarappa	65	M				+		+					+	+									
107	Iramma	92	F			+	+					+		+	+									
108	Yallawwa	70	M	+		+	+		+															
109	Gurappa	65	M	+		+			+				+											
110	Saipanam	70	F		+		+		+	+														
111	Shibalawwa	70	F			+			+			+	+											
112	Motubai	65	F			+	+		+															HTN
113	Neelawwa	65	F	+					+	+			+											
114	Taramma	70	F			+	+		+															
115	Jaituna bai	68	F	+		+						+												
116	Hirabai	71	F	+		+			+			+												HTN
117	Ratnabai	68	F		+							+		+	+									DM
118	R.M Patil	75	M	+		+		+					+					+						
119	Jayamma	68	F			+			+		+		+	+										DM
120	Dattatraya	69	M			+			+													+		
121	Panchappa	78	M			+	+		+					+	+									
122	Appasab	78	M		+				+										+					
123	Neelawwa	65	F	+					+	+			+											
124	Taramma	70	F			+	+		+															
125	Jjaitunabi	65	F	+		+						+	+						+					
126	Hirabai	71	M	+		+			+			+												HTN
127	Dattatraya	69	M			+			+													+		
128	Panchappa	78	M			+	+		+					+	+									
129	Appasab	78	M		+				+										+					
130	Ramesh	71	M	+					+									+						
131	Basangouda	75	M	+		+														+				
132	Bhagirati	68	M			+	+							+										HTN
133	Shantabai	65	F	+	+							+		+										
134	Mahadev	65	M		+		+			+										+				A
135	Bheemappa	65	M	+						+										+				
136	Basappa	70	M							+			+											DM

137	Ramappa	67	M		+				+				+											DM
138	Dundappa	70	M			+	+			+														DM
139	Siddaraya	75	M				+		+	+														
140	Sidappa	67	M			+	+														+			DM
141	Shankawwa	80	F			+	+			+														
142	Basayya	66	M							+			+											DM
143	Saybee	68	F						+				+											
144	Yellawa	65	F		+	+			+															
145	Rachamma	65	F			+	+		+															
146	Kenchamma	80	M						+	+														
147	Kashibai	70	F		+				+				+											
148	Gangabai	65	F		+				+	+														
149	Irubai	67	F		+	+							+											
15O	Munalingma	65	F						+				+											HTN
151	Ramarao	66	M		+	+				+														DM
152	Ushnappa	80	M			+							+	+										
153	Shrishail	65	M		+	+				+														TB
154	Mallappa	75	M							+				+										A
155	Sharanamma	65	M			+	+			+														
156	Ansuya	68	F		+	+																		TB
157	S.D Hugar	71	M		+					+				+										
158	Lalsab	65	M	+			+																	
159	Mahadev	65	M	+									+											
160	Shantabai	65	F		+				+												+			HTN
161	Ishwarappa	80	M			+	+																	
162	Sharabasapa	68	M				+			+										+				
163	Kallappa	95	M			+	+		+											+				
164	Sharanappa	65	M				+		+	+		+												TB
165	Parvatamma	65	F						+			+												
166	Neelabai	70	F			+							+	+										
167	Indumati	70	F	+		+															+			
168	Manohar	74	M	+	+				+												+			HTN
169	Rehamtabee	95	F				+			+														
170	Tarabai	65	F	+					+				+											
171	Vital	69	M			+						+	+											
172	Mabubu	65	F				+			+		+												
173	Tulasibai	70	F				+		+															HTN
174	Rachawwa	65	F	+	+	+							+											
175	Chandramma	85	F	+		+	+																	
176	Bhojusingh	75	M				+		+															
177	Ramappa	70	M				+		+			+												
178	Girijabai	85	F	+					+															BCC
179	Durgawwa	69	M					+				+	+											
180	Jayawantawwa	65	F	+	+	+																		
181	Shivamma	65	F	+						+														
182	Siddappa	80	M		+	+			+															

183	Manappa	65	M		+		+		+																
184	Yallappa	65	M			+			+																DM
185	Shivamma	65	F	+			+																		
186	Bhimawwa	69	F			+	+																		
187	Shivamma	65	F	+	+																				
188	Apparaya	75	M		+	+	+																		HTN
189	Sidappa	75	M		+	+	+																		
190	Chandrabagawa	70	F	+					+																
191	Hanumawwa	65	F			+	+		+																
192	Seetabai	65	F		+	+				+		+													
193	Mallawwa	65	F	+		+			+																
194	Ragawwa	65	M				+		+																
195	Boramma	65	F					+				+	+												
196	Hanappa	65	M			+	+																		
197	Bhimawwa	70	F				+	+																	
198	Chandee	65	F			+			+				+												
199	Gouramma	65	F			+		+	+																
200	Chandrabai	65	F			+			+			+													
201	Mahadevi	65	F	+			+		+																
202	Irawwa	80	F	+		+			+																
203	Gangawwa	70	F	+		+						+													DM
204	Davalamma	65	F			+	+																		
205	Paridasab	65	F	+		+														+					
206	Ravatappa	65	M		+		+					+				+									
207	Siddappa	68	M			+	+						+							+					
208	Rangamma	66	F	+				+		+														+	
209	R.C Joshi	68	M						+		+										+				DM
210	Hasansab	70	M		+	+							+			+									
211	S.I.Pattar	74	M	+			+			+															HTN
212	Harubai	70	F		+	+	+																		
213	Margabai	80	M			+		+																	
214	Sonabai	65	F			+			+																
215	Suratabai	65	F						+	+															
216	Kallawwa	65	M	+			+		+																
217	Basu	80	M			+				+															
218	Sheetabai	70	F	+			+			+															
219	Babusab	65	M			+	+																		
220	G.H Metri	70	M		+			+				+	+												
221	Revathi	75	F		+						+			+	+										
222	Ningappa	65	M	+		+				+							+								DM
223	Krishnaraj	68	M		+							+							+						
224	Bouramma	78	F						+	+					+					+					HTN
225	Savitramma	76	F	+				+			+					+									
226	Mantappa	68	M		+				+							+									
227	Ramakanth	78	M		+		+			+	+			+	+										DM
228	Gouramma	68	F	+			+	+											+	+					
229	Jatteppa	70	M	+		+			+	+	+						+								

230	Hanumanth	78	M		+	+			+			+				+				+				
231	Shivamma	76	F	+			+	+				+												DM
232	Tamanna	78	M		+	+			+							+	+							HTN
233	Shantabai	65	M			+	+						+					+						
234	Ratnamma	65	F			+			+									+						DM
235	N.K Badiger	72	M		+		+													+				
236	Pakirappa	65	M					+	+				+											
237	Lingareddi	65	M			+			+				+											
238	Motuai	66	F			+			+				+									+		DM
239	Bhimawwa	69	M			+				+														
240	Basalingamma	65	F			+				+			+											
241	Channabasappa	70	M		+	+							+											
242	Kallappa	75	M			+				+														
243	Ganappagouda	75	M						+	+														
244	Sidappa	90	M							+			+											
245	Sidramayya	65	M	+	+	+																		
246	Sidappa	80	M		+				+															
247	Jayamma	65	F						+	+														
248	Chandramma	70	F	+					+	+			+											
249	Gangabai	70	F			+			+															
250	Mallamma	65	F			+			+															
251	Sharanawwa	65	F		+	+			+															
252	Ratnabai	65	F		+				+															
253	Nagawwa	65	F		+								+											
254	Jayantawwa	65	F	+	+																			
255	Balawwi	70	F	+		+																		
256	Halemma	75	F			+	+		+															
257	Abdul	67	M			+			+					+	+									AN
258	Sangawwa	70	F		+				+															
259	Shivamma	65	F	+		+																		
260	Huchappa	65	M		+	+	+																	
261	M.B Biradar	65	M		+		+				+													HTN
262	Sonabai	65	F	+			+																	
263	Lakshmibai	68	F			+							+											
264	Neelamma	70	F			+				+			+											
265	Neelawati	69	F							+		+												
266	Sushilabai	65	F			+	+		+															
267	Suvarna	65	F	+					+															
268	Ramabai	65	F			+	+		+															
269	Tulasawwa	80	F			+			+				+											HTN,DM
270	Shivamma	70	F	+									+											A
271	Yamunawwa	90	F			+	+		+			+												
272	Kashiba	88	F			+	+																	DM
273	Hirabai	78	F	+		+																		
274	Chanappa	65	M		+	+			+									+						
275	Ratnabai	74	F			+				+	+	+												DM
276	Daramma	72	F	+		+	+																	

277	Satnawwa	70	F			+			+																
278	Gurubai	65	F	+			+																		
279	Sharantawwa	70	F						+			+													
280	Bangarawwa	65	F				+		+																
281	Shankarmma	66	F						+			+													DM
282	Kamalabai	65	F	+					+												+				
283	Appasab	65	M			+	+																		
284	Basappa	68	M	+		+										+									
285	Mahadevappa	72	M		+				+																
286	Jeevagi	68	M		+	+																+			
287	Sidamma	65	F		+	+								+	+										DM
288	Biradar	65	M		+		+																		
289	Devamma	65	F			+			+												+				
290	Lakshmibai	65	F		+		+																		DM
291	Gurubasappa	65	M	+	+																				
292	Gurappa	65	M		+	+						+													
293	Shribavi	65	M					+	+		+														DM
294	Sanappa	70	M			+							+												
295	Madappa	80	M	+									+												
296	Makansab	65	M						+				+												
297	Madappa	80	M	+	+	+																			
298	Jamunabai	70	F	+	+																				
299	Gurawwa	70	F		+	+																			HTN
300	Danawwa	65	F					+				+													
301	Sahadev	70	M						+				+												HTN
302	Hunnu pawar	65	M			+			+				+												
303	Lakshman	70	M		+	+			+																
304	Kashibai	70	F	+			+																		
305	Gouramma	75	M	+	+																				
306	Yallawwa	70	F	+	+																				
307	Shrisai	65	M			+			+																
308	Kasturibai	65	F	+	+	+																			
309	Unnu	65	M		+				+			+	+												HTN
310	Hemabai	65	F			+			+																
311	Yamunabai	65	F			+				+			+												
312	Yallawwa	65	F		+				+		+														DM
313	Gurawwa	65	F		+								+												
314	Rewabai	75	F	+								+	+												
315	Bourawwa	68	F	+		+																			DM
316	Doddawwa	65	F		+		+																		A
317	Sidramayya	65	M	+			+			+		+													
318	Rehutappa	65	M							+															
319	Chandu	68	M	+								+	+												
320	Valubai	65	F	+		+	+																		
321	Roopabai	65	F			+			+																
322	Jannabai	65	F	+			+		+																
323	Sangappa	66	M							+			+												

324	Jannabai	65	F		+	+			+															
325	Sangappa	66	M		+	+			+															
326	Gangabai	65	M			+	+																	
327	Kantabai	70	F		+	+						+												
328	Tipawwa	70	F	+			+																	
329	Ameena	80	F		+	+																		
330	Nanagouda	65	M	+		+				+		+									+			
331	Khazi	75	M		+								+									+		
332	Shivagangawwa	70	M		+				+															
333	Ningappa	83	M		+	+			+					+	+									
334	S.B jalawadi	65	M			+	+		+					+	+									HTN,DM
335	A.S Basawappa	66	M		+					+			+											
336	Prajwal	75	M	+		+	+																	DM
337	Siddappa	73	M	+	+	+		+											+					
338	Revappa	65	M	+			+		+															DM
339	K.K Kulkarni	72	M				+	+	+				+									+		
340	Borawwa	75	F		+							+												
341	Nagamma	65	F				+		+															
342	S.B Matapati	65	M			+	+							+										AN
343	Janatabai	65	F			+			+															
344	Shivalingappa	68	M		+	+																		HTN
345	Yamunabai	70	F		+	+															+			
346	Ahimad rasul	70	M		+	+																		
347	Ramappa	68	M		+	+													+					
348	Govindappa	75	M			+	+							+	+									
349	Dharabasab	72	M			+	+										+							
350	Hirabai	82	F						+	+			+											DM
351	Ratnabai	75	F	+	+	+																		
352	Bandakka	70	F	+		+	+		+															
353	Sumitra	68	F		+	+																		
354	Rachamma	70	F			+			+															
355	Sundaramma	65	F			+	+		+															
356	Chndrawwa	70	F			+						+												HTN
357	Nagamma	65	F			+	+																	
358	Sidappa	66	M			+	+																	
359	Appasab	68	M	+		+	+																	
360	Shankarapa	73	M			+	+		+															
361	H..J Karanal	65	M		+	+	+																	
362	Sidramappa	65	M							+			+											
363	Sharangouda	65	M			+			+															
364	Basappa	65	M			+	+																	
365	Ramu	65	M				+																	
366	Sidanna	66	M		+	+						+							+					
367	S.S Pattar	74	M		+	+	+																	
368	Vasanth kumar	65	M		+	+	+															+		
369	Danawwa	65	F	+		+																		
370	Gurawwa	65	F		+	+																		

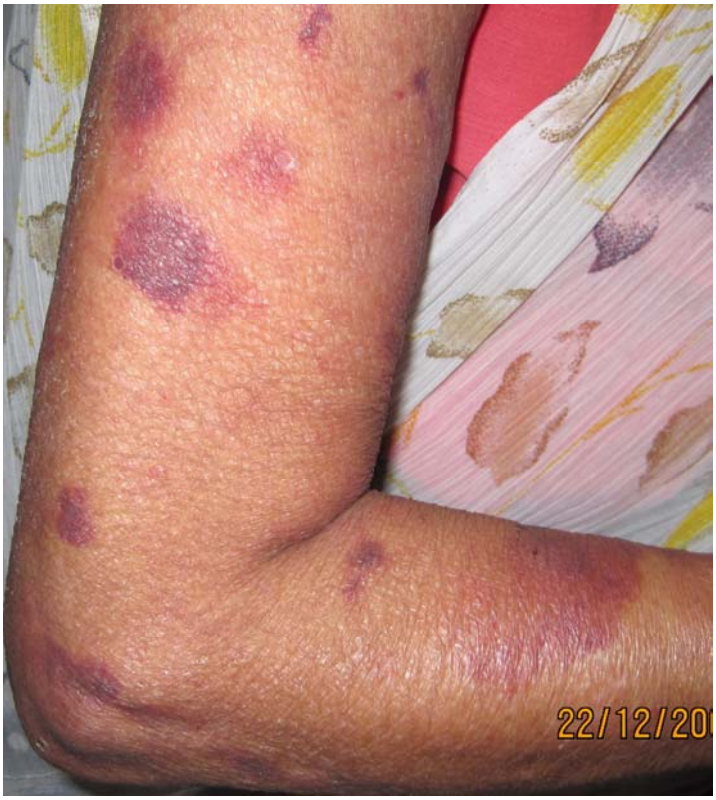
371	Lakshmibai	70	F							+		+													
372	Gangawwa	65	F			+	+																		
373	Kalawwa	65	F				+		+				+												
374	Gourawwa	65	F	+		+				+															HTN
375	Mallawwa	65	F			+	+																		
376	Borawwa	70	F	+			+					+													
377	Vasanth	65	M			+	+		+																
378	Jayawwa	65	F		+	+																			
379	Shantabai	65	F				+		+				+												
380	Bagawwa	70	F		+	+			+																
381	Irappa	65	M			+			+				+												
382	Bhimabai	65	F				+		+			+													A
383	Bouramma	65	F	+		+	+																		HTN
384	Akkawwa	65	F			+	+		+																
385	Shivappa	77	M		+	+	+					+													A
386	Rukmini	65	F	+				+		+											+				
387	Hajma	65	M	+		+						+	+												DM
388	Kalawati	65	F				+		+			+													HTN
389	Amarawati	68	F	+			+		+				+												
390	Kashibai	66	F			+	+		+	+															HTN
391	Parasuram	65	M			+				+				+	+										AN
392	Lalabi	68	F					+		+		+									+				
393	Sidramappa	68	M	+		+						+									+				HTN
394	Abdul	75	M			+	+																+		
395	Tanaji	65	M			+						+	+												
396	Kallamma	65	F	+						+				+	+										AN
397	Sharabai	67	F			+				+															
398	Prakash	68	M							+		+	+												
399	Saliya	65	F		+					+												+			
400	Abdul rajak	65	F		+	+	+															+			
401	Muragamma	70	F	+		+																+			
402	B.D Kumbar	70	M		+					+															
403	Bupan	65	M							+		+										+			
404	Siddalingawwa	65	F		+	+	+							+	+										
405	D.C More	65	M			+				+											+				
406	Nimbawwa	65	F			+			+													+			
407	Shranamma	65	F		+	+				+													+		
408	Madiwalappa	75	M		+	+								+	+										
409	Ramagandappa	70	M		+							+											+		
410	Sushila rathod	65	F	+		+			+												+		+		
411	Yankappa	74	M			+			+	+															
412	Rasul	65	M			+				+												+			
413	Jijabi	65	F			+	+							+	+										
414	Somalingappa	65	M		+					+															
415	Bhimappa	68	M	+						+		+		+	+										
416	S.G Arakeri	65	M			+	+			+													+		
417	Gangabai	68	F		+	+	+															+			

418	Kasturibai	68	F	+		+			+															
419	V.S Nuragoud	65	M			+	+														+			
420	Kabula	65	M	+		+								+	+									
421	Yamunabai	65	F			+			+	+														
422	Bibikatiya	65	F			+				+														
423	Girijamma	65	F	+		+								+	+									
424	Davalsab	72	M	+	+							+										+		
425	V.M Gabbur	65	M						+	+										+				
426	Kirucharan	70	M			+						+		+	+									
427	Gourawwa	68	F				+		+												+			
428	Umakanth	68	M		+	+														+				
429	Kudarsha	65	M		+	+															+			
430	Sidanna	68	M		+	+	+														+			
431	Nimbanna	65	M		+	+			+							+								
432	Shantabai	68	F			+			+							+								
433	Mallamma	68	F	+	+	+																		
434	Dhareppa	65	M	+					+							+								
435	Gurubai	65	F	+	+		+														+			
436	Shakuntala	66	F		+	+			+												+			
437	Yallamma	68	F		+	+																		
438	Mallappa	65	M	+		+											+							
439	Sharmilabai	68	F			+				+				+	+									AN
440	Gangamma	70	F		+	+			+											+				
441	Siddaganda	75	M		+	+												+						
442	Babugouda	68	M		+				+									+						
443	G.M Arkeri	68	M	+	+		+														+			
444	P.S Kodakal	68	M			+			+					+	+									
445	Madanppagouda	70	M		+	+				+							+							
446	Shakuntala	68	F		+	+	+														+			
447	S.B Hiramath	74	M		+	+												+						
448	Ramappa	75	M		+	+			+									+						
449	P.C Bidri	87	M		+	+												+						
450	Abdul	77	M			+	+																	
451	V..S Kulkarni	80	M		+				+	+				+	+									
452	Vittappa	78	M	+	+		+							+	+									HTN
453	Ashok	68	M	+		+			+													+		
454	Mallanna	65	M		+	+				+												+		
455	S. B Inchageri	65	M						+	+		+						+						
456	Sayawwa	66	F	+	+	+																+		
457	Gurappa	65	M		+	+				+											+			
458	Dundawwa	70	F		+		+			+				+										AN
459	Chandrashekar	65	M		+		+			+								+						
460	Siddappa	68	M			+						+								+				
461	A.S Kushal	70	M		+		+			+				+	+									
462	I.M Shindagi	65	M		+	+	+														+			
463	Lakshmibai	68	F			+				+		+		+	+									
464	Kubu	68	M		+	+				+								+						

465	M.BChakramani	70	M	+	+	+												+						
466	Basalingappa	65	M		+		+													+				
467	A.M Patil	68	M		+		+					+				+								
468	Rajamma	66	F			+				+									+					
469	Ningamma	65	M		+	+												+						
470	Halakatti	72	M		+		+			+				+										AN
471	Gurugidan	68	F		+		+			+										+				
472	Shivasankarapa	71	M			+	+					+								+				
473	Biradar	68	M		+					+						+								
474	Sugalabai	76	F		+		+															+		
475	Shivasiddappa	65	M				+		+				+								+			
476	Jevagi	68	M				+		+				+							+				
477	Balavantrayya	70	M				+			+										+				
478	Jyaneshwar	80	M		+	+							+				+							
479	Neelamma	70	F		+		+			+														
480	Neelabai	70	F		+		+			+				+	+									
481	Indumati	70	F		+			+				+								+				
482	Siddanagouda	65	M		+		+													+				
483	Shantabai	65	F		+		+			+										+				
484	Ishwarppa	80	M				+		+															HTN
485	Shivamma	80	F		+		+		+											+				
486	M.B Biradar	68	M	+	+		+							+										AN
487	Sonubai	65	F						+	+			+					+						
488	Halamma	75	F	+	+	+														+				
489	Sangappa	70	M		+	+				+			+							+				
490	Bagappa	68	M		+					+			+							+				
491	Neelabai	66	F			+			+															
492	Janatabai	66	F	+						+												+		
493	Rantappa	66	M		+	+	+											+						HTN
494	Basappa	66	M						+	+								+						
495	S.C Patil	66	M		+	+		+													+			
496	Ramappa	68	M	+		+	+									+								
497	Shivaji	67	M	+	+	+														+				
498	G.R Warad	84	M			+	+			+										+				
499	S.B Hulikeri	66	M	+	+		+																	
500	M.B Biradar	68	M	+		+	+											+						
501	S.S Hiremath	67	M				+						+				+							HTN
502	Parasappa	65	M		+	+			+											+				
503	Abdul karim	70	M	+			+						+							+				HTN
504	Motiram	75	M		+	+	+		+					+	+									
505	H.S Biradar	72	M			+							+		+					+				
506	G.G Dayagol	65	M			+	+											+						DM
507	Sangappa	75	M			+	+						+							+				DM
508	Shivalingappa	73	M	+						+			+	+	+									HTN
509	Lakshman	75	M	+									+	+	+									
510	Rangawwa	80	F	+		+			+															
511	Venkanna	72	M			+				+														H,DM

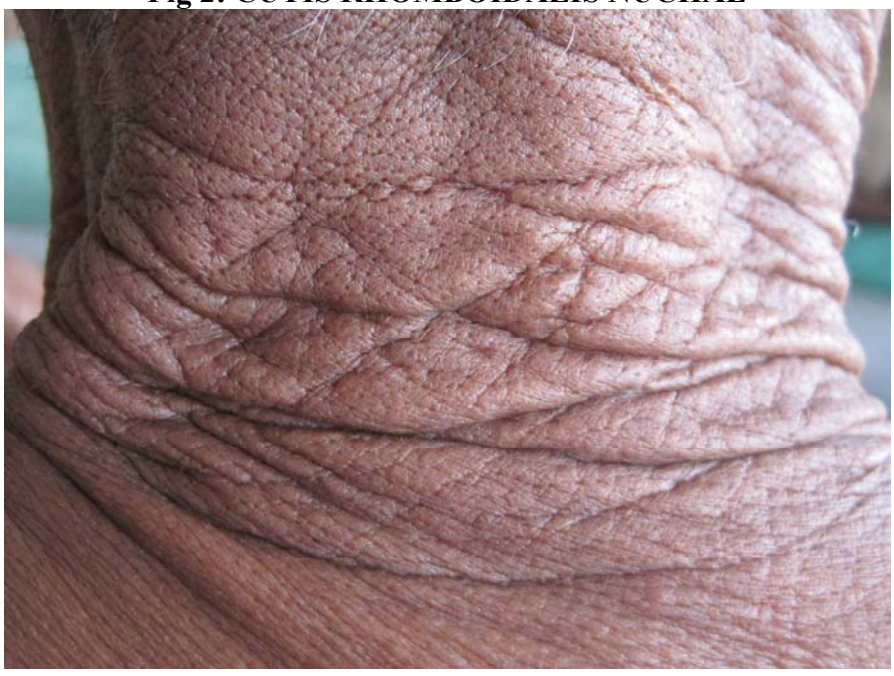
512	Appasab	65	M	+		+							+							+				
513	Dhanamma	65	M		+								+							+				
514	S.B Banashetti	66	M			+							+			+								
515	Ramanagouda	65	M		+								+		+	+								
516	Munnappa	78	M	+			+	+															+		
517	Neelawwa	68	F		+	+																			HTN
518	Ningamma	75	F	+		+																			
519	Nagawwa	85	F			+	+																		
520	Ningawwa	80	F	+		+			+																
521	Sundaramma	80	F			+							+												
522	Bhagawwa	65	F	+			+																		
523	M.S Biradar	65	M	+		+														+					HTN
524	Shivappa	80	M		+	+	+														+				
525	Mansur badani	82	M		+	+								+	+			+							
526	Muttappa	65	M	+	+								+								+				
527	Basamma	72	F		+	+		+							+										HTN
528	Shantawwa	65	F			+	+						+		+										AN
529	Siddanagouda	75	M		+				+		+		+												HTN
530	Basalingamma	65	F		+			+		+			+		+										DM
531	Iranagouda	75	M	+					+				+												
532	Guranna	68	M			+	+		+										+						
533	Mahadevappa	65	M		+	+									+										DM
534	Badrayya	65	M	+		+				+			+												
535	Shangappa	65	M			+							+												
536	Siddappa	70	M	+		+			+				+		+										
537	Chandu rathod	68	M						+	+			+		+										
538	Borawwa	66	F	+	+	+				+			+												HTN,DM
539	Shankarappa	66	M				+						+			+					+				
540	Shivayya	70	M		+					+			+					+							A
541	Shankarappa	68	M	+		+	+		+						+										AN
542	Kasappa	65	M	+	+	+	+																		
543	Siddappa	65	M		+	+											+								HTN
544	Shankarappa	68	M	+	+		+						+												HTN
545	Mahadevappa	66	M	+	+								+												
546	Yashwanth	72	M	+		+							+					+							
547	Nagappa	65	M	+		+						+	+		+			+							
548	Krishnabi	70	F		+	+																			HTN,DM
549	Shantabai	68	F			+							+												HTN
550	Bheemaraya	65	M		+	+	+	+					+												

Fig 1: SENILE PURPURA



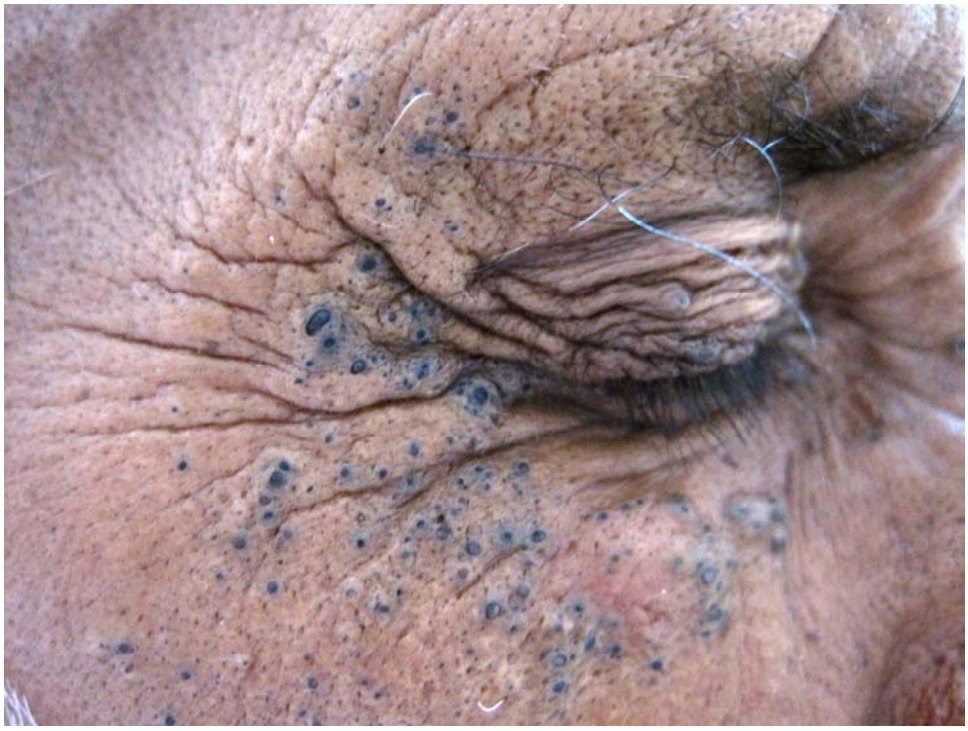
PURPURIC MACULES ON UPPERLIMB OVER WRINKLED SKIN

Fig 2: CUTIS RHOMBOIDALIS NUCHAE



RHOMBOIDAL PATTERN ON NAPE OF NECK

Fig 3: FAVRE-RACOUCHOT SYNDROME



MULTIPLE COMEDONES ON SUNDAMAGED SKIN OVER PERIORBITAL AREA

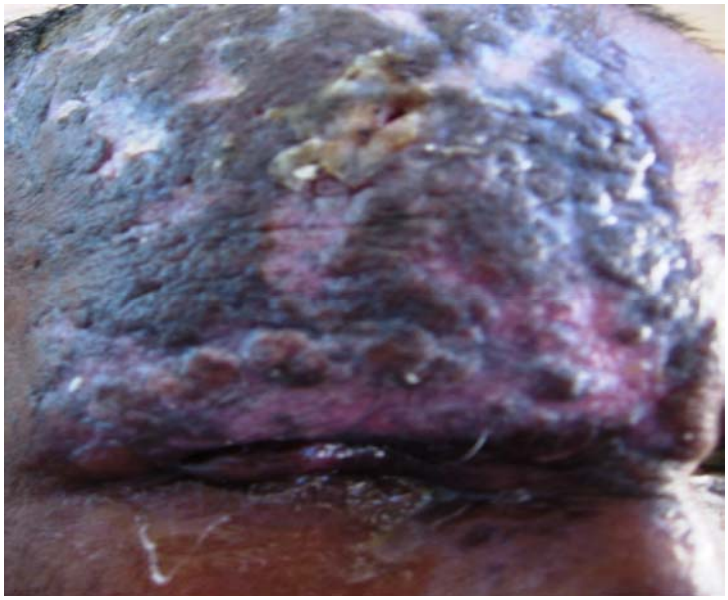
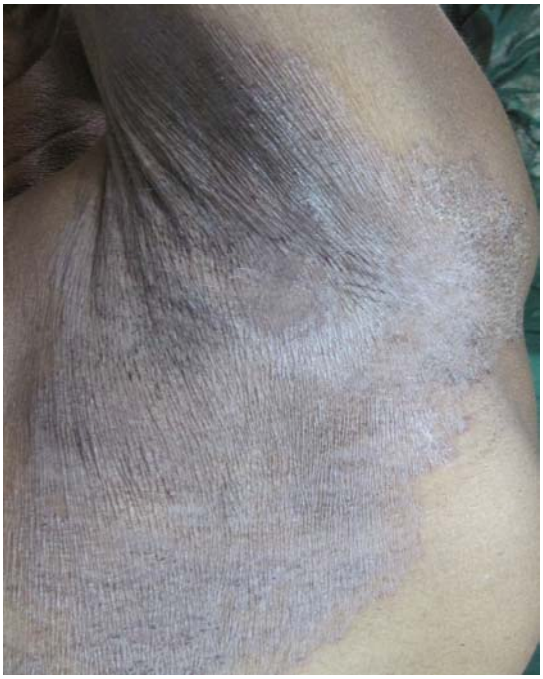


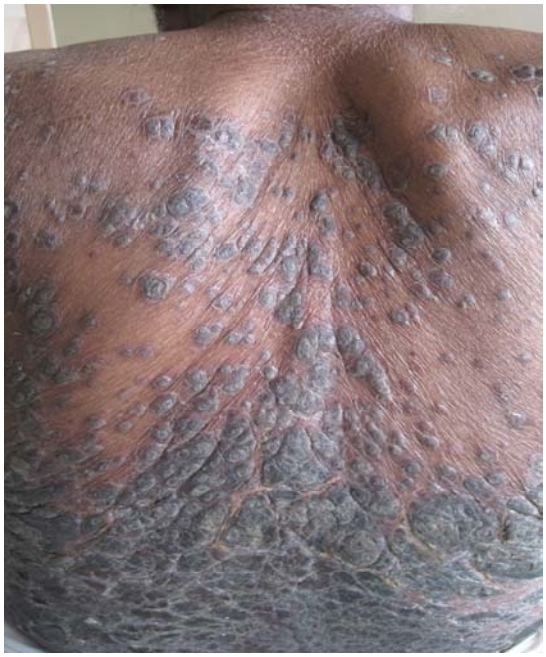
Fig 4: HERPES ZOSTER OPHTHALMICUS WITH SUPERADDED BACTERIAL INFECTION

Fig 5: TINEA CORPORIS



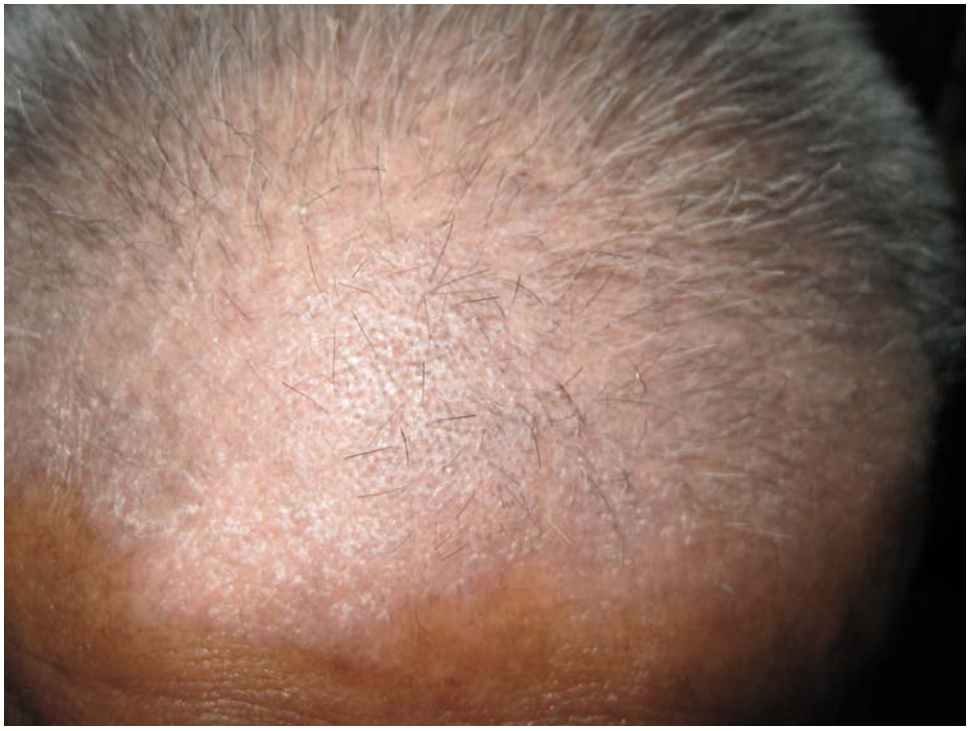
SCALY PLAQUE WITH CENTRAL CLEARING & WELL DEFINED BORDER SUGGESTIVE OF DERMATOPHYTE INFECTION

Fig 6: PSORIASIS VULGARIS



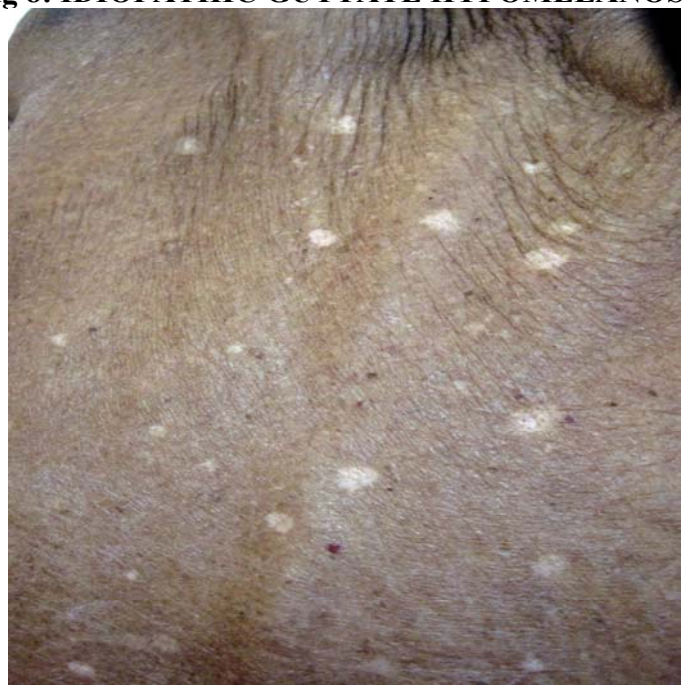
WELL DEFINED SCALY PLAQUES ON BACK

Fig 7: SCALP PSORIASIS



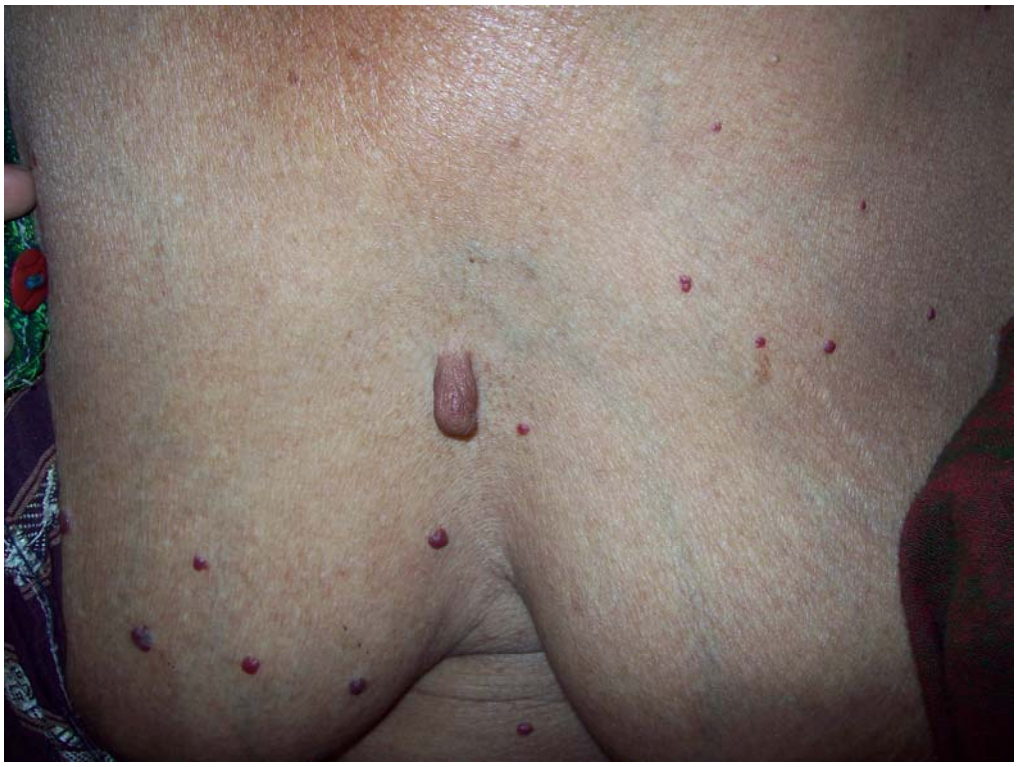
WELL DEFINED ERYTHEMATOUS SCALY PLAQUE ON SCALP

Fig 8: IDIOPATHIC GUTTATE HYPOMELANOSIS



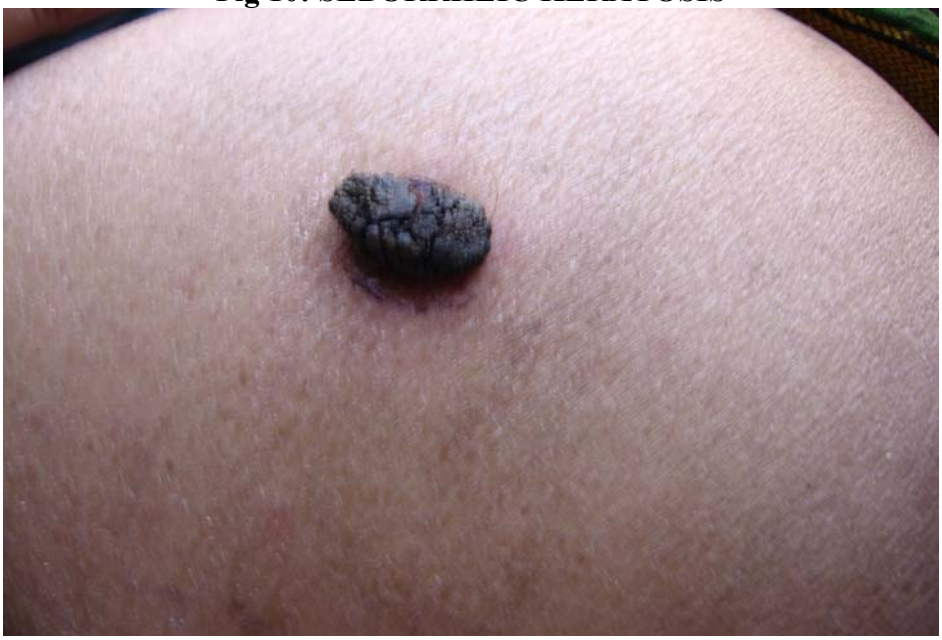
MULTIPLE PORCELAIN WHITE MACULES ON BACK

Fig 9: CHERRY ANGIOMAS



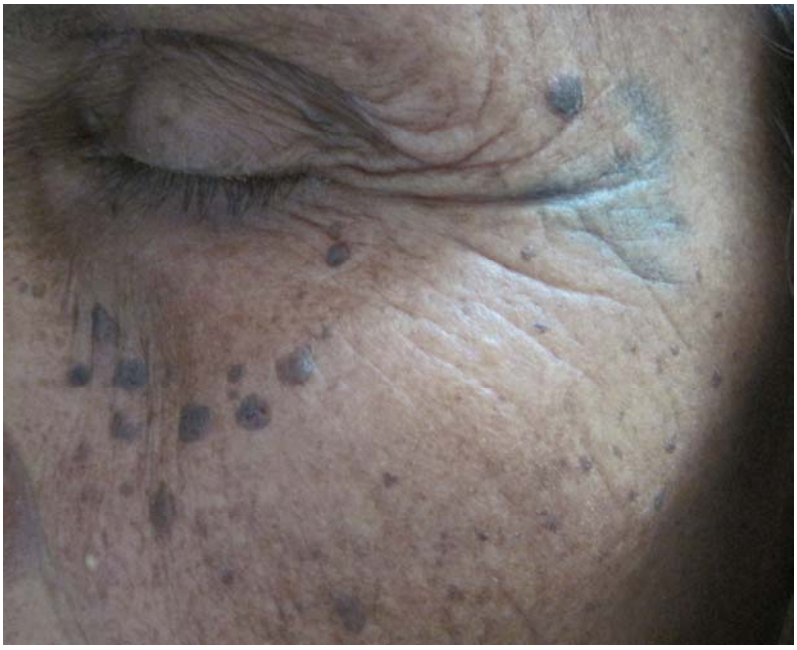
BRIGHT RED PAPULES ON CHEST

Fig 10: SEBORRHEIC KERATOSIS



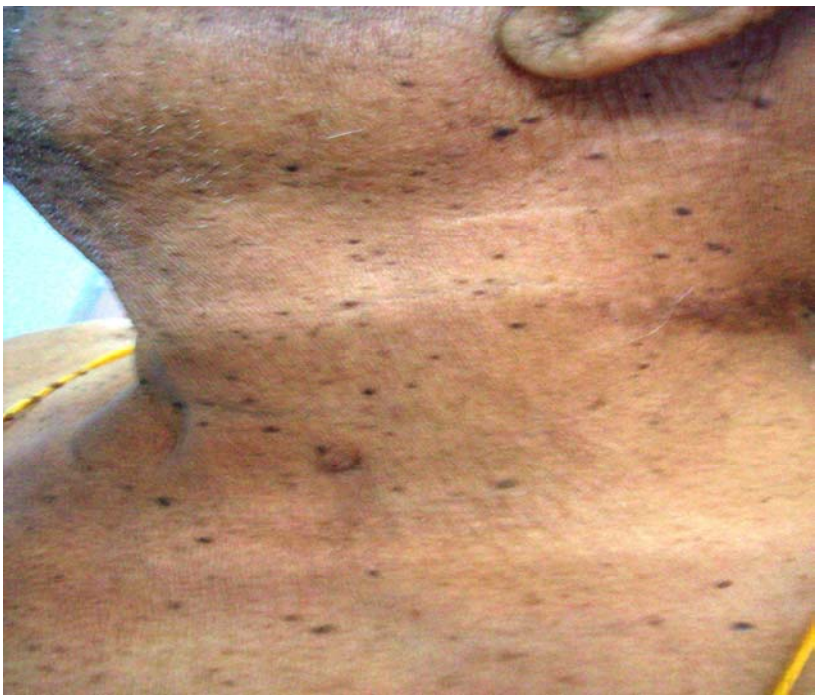
SINGLE HYPERPIGMENTED PAPULE WITH HYPERKERATOTIC SURFACE

Fig 11: SEBORRHEIC KERATOSIS ON FACE



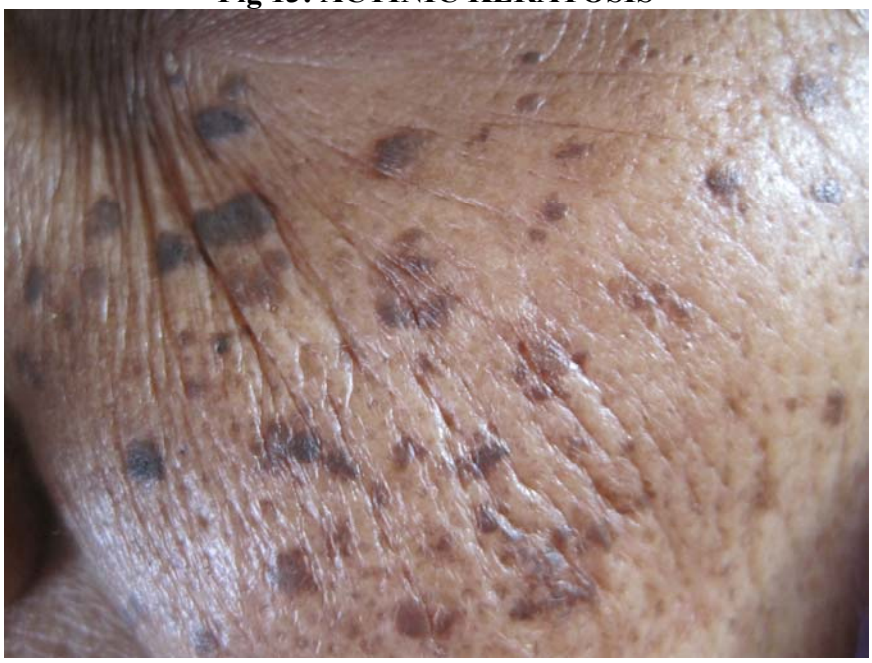
MULTIPLE PIGMENTED PAPULES WITH STUCK ON APPEARANCE

Fig 12: DERMATOSIS PAPULOSA NIGRA



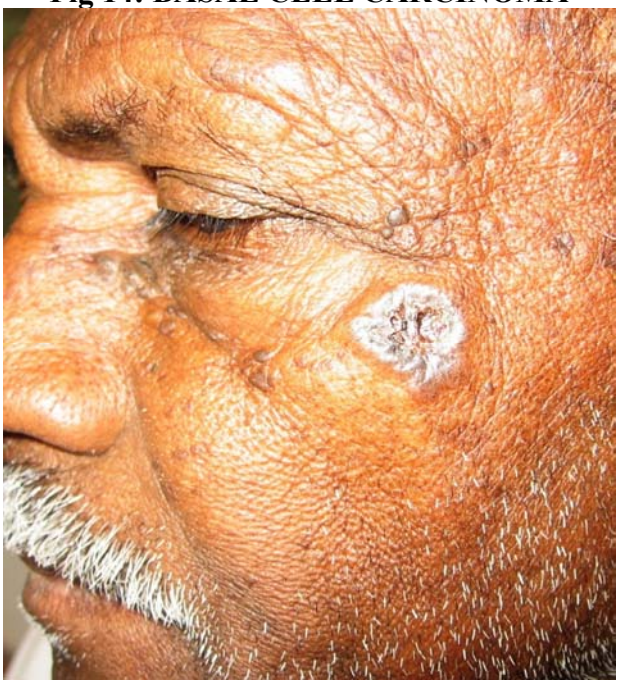
MULTIPLE BLACK SESSILE PAPULES ON NECK

Fig 13: ACTINIC KERATOSIS



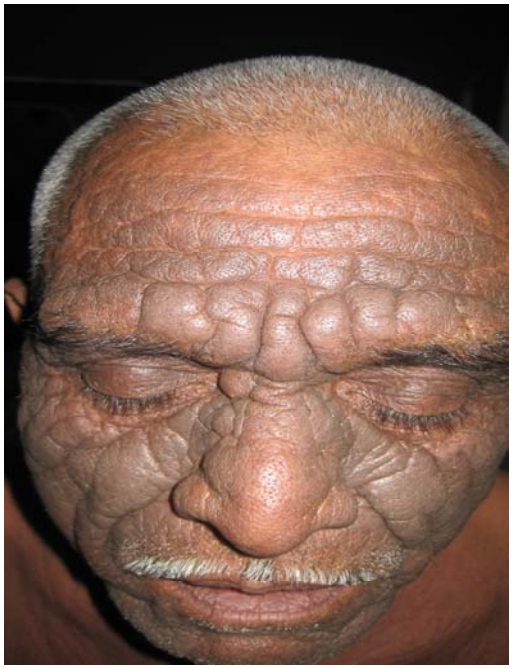
MULTIPLE HYPERPIGMENTED DRY KERATOTIC PAPULES ON FACE

Fig 14: BASAL CELL CARCINOMA



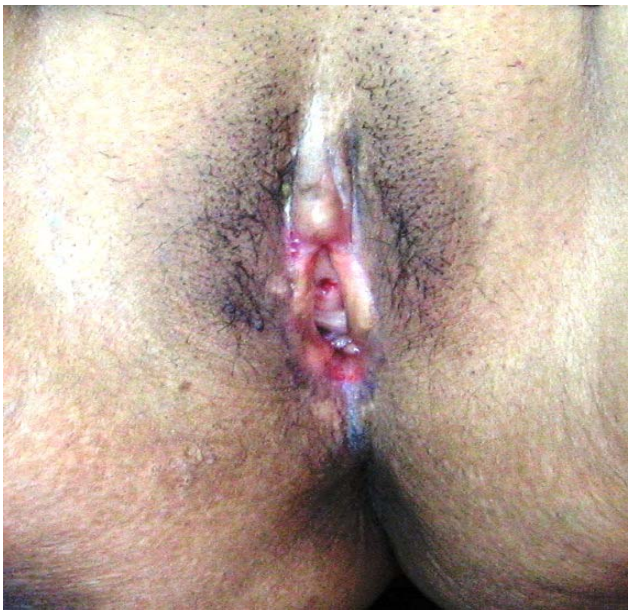
NODULOULCERATIVE PLAQUE WITH WAXY BORDER OVER FACE

Fig 15: ACTINIC RETICULOID



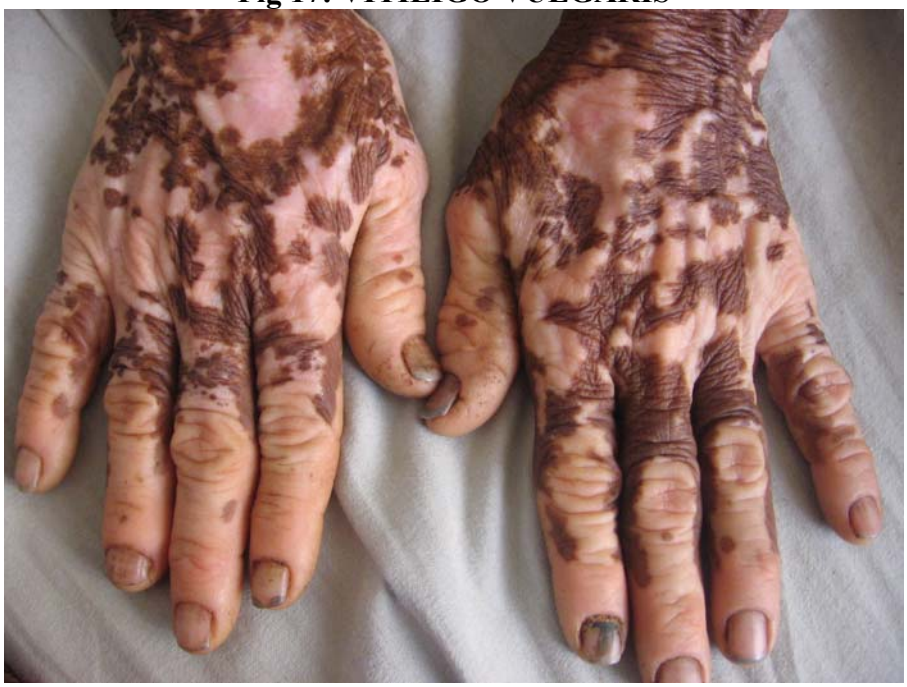
LICHENIFIED AND INDURATED PLAQUES ON FOREHEAD AND CHEEKS

Fig 16: LICHEN SCLEROSUS ET ATROPHICUS



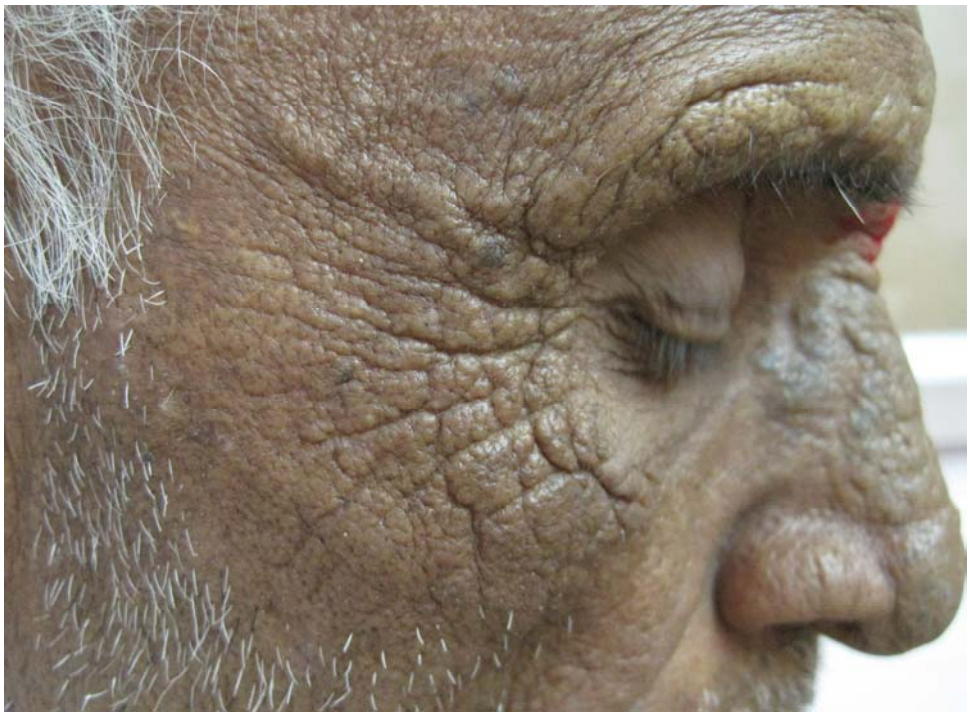
WHITE ATROPHIC SKIN ON LABIA MINORA

Fig 17: VITILIGO VULGARIS



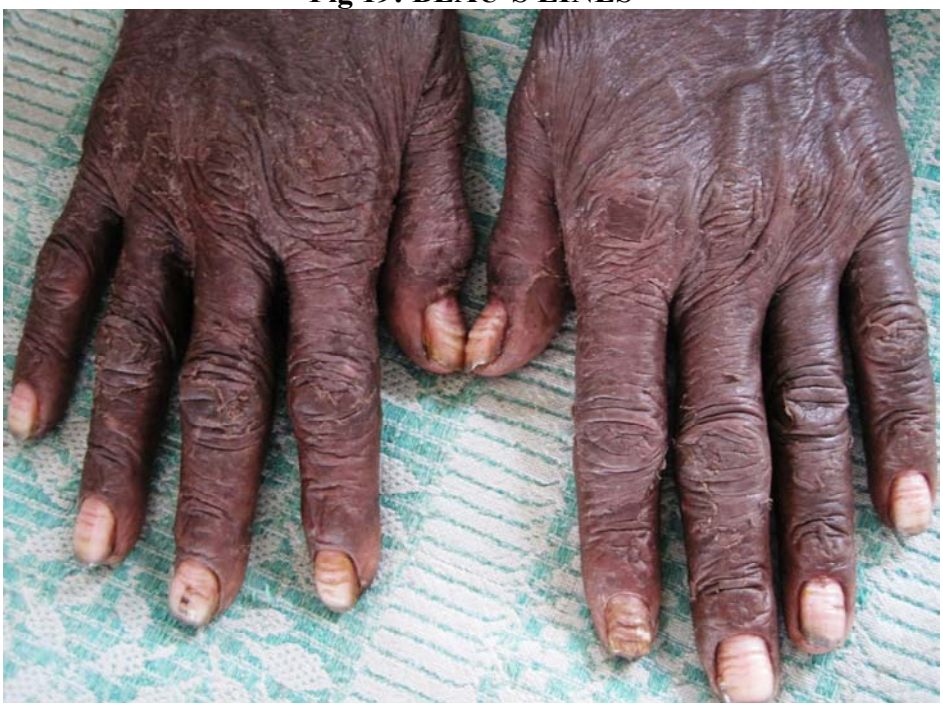
DEPIGMENTED PATCHES ON DORSUM OF HANDS

Fig 18: CROW FEET



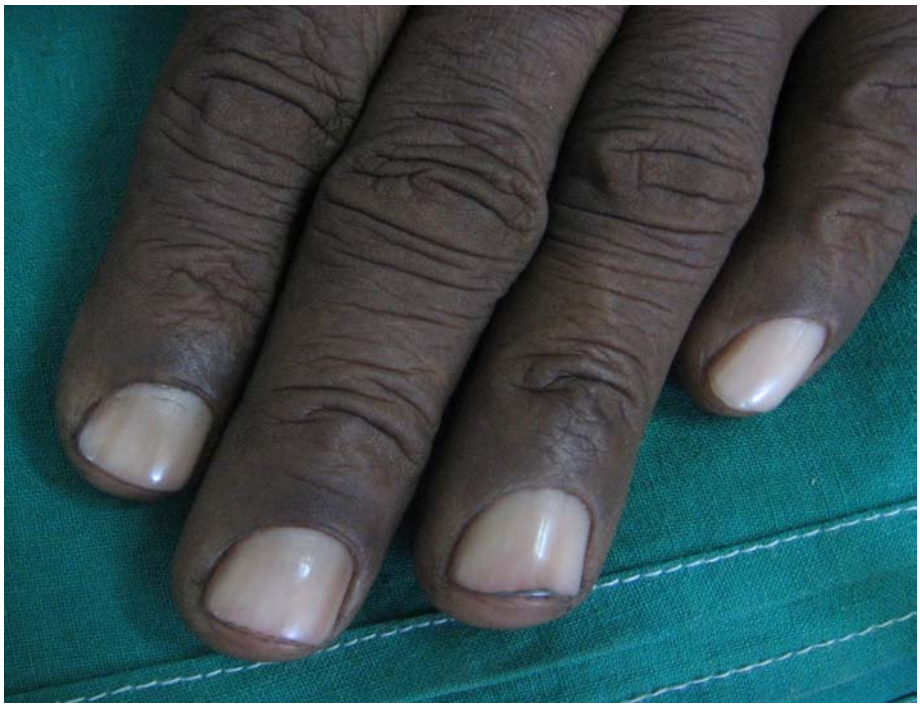
WELL DEFINED FURROWS OF PERIORBITAL AREA

Fig 19: BEAU'S LINES



TRANSEVERSE GROOVES OVER FINGER NAILS

Fig 20: SHINY NAILS



SHINY NAILS WITH SMOOTH SURFACE