CLINICO-PATHOLOGICAL STUDY OF CUTANEOUS TUMOURS OF HEAD AND NECK

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INTRODUCTION

Skin is a complex and the largest organ in the body. Because of its complexity a wide range of diseases can develop from the skin including tumors from surface epidermis, epidermal appendages , dermal & subcutaneous tissue. The vast diversity of these lesions combined with a body of descriptive data, often overlapping (clinical,histological) produces confusion in the area of nomenclature and difficulty in diagnosis.

Tumours of skin are histopathologically diverse group of entities which have common localized proliferation of cells resulting in clinically discrete lesions. They may be divided into a number of categories, reflecting their different biological behaviour. These include hamartomas, benign tumours, premalignant and malignant conditions.

This study of tumours of skin has been undertaken to find out the frequency of benign and malignant growths. The study has been limited to the cases attending the Dermatology Department, chengalpattu Government Hospital, chengalpattu.

Most of the tumours whether benign or malignant are symptomless but are cosmetically unacceptable. The distinction between benign and malignant neoplasm are rather more difficult to define when they appear in skin than when found elsewhere and histopathological examination is frequently required to

establish a definitive diagnosis. Diagnosis of any skin tumors can be done by correlating clinical features and histological features, which can be supported by histochemistry, immunohistochemistry and electron microscopy.

Thus, the study of skin tumors is perhaps more intriguing, fascinating, challenging and at times even frustrating than any other tumors.

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REVIEW OF LITERATURE

More than 100years ago, the noted pathologist Rudolph Virchow portrayed the skin as a protective covering for more delicate and functionally sophisticated internal viscera [(1)]

During the past three decades, scientific inquiry have demonstrated skin to be a complex organ in which precisely regulated cellular and molecular interactions govern many crucial responses to our environment. Factors affecting the delicate homeostasis that exists among the skin cells results in conditions as diverse as wrinkles, hair loss, blisters, rashes and even life threatening cancers and disorders of immune regulation [(2)]

Definition-**TUMOUR**

Tumour or Neoplasm defined by Sir Rupert Willis [(3)] as an abnormal mass of tissue the growth of which exceeds and is uncoordinated with that of normal tissues and persists in the same manner even after the cessation of the stimuli that evoked the change or responses.

CLASSIFICATION

Tumours on the whole may be divided into benign and malignant depending upon the histological features and certain biological behaviors.

Benign tumours show a high degree of structural differentiation usually composed of relatively well differentiated cells. The growth is slow, rarely shows

limited degree of infiltration, does not usually metastasize and may cease to grow spontaneously. Malignant tumours show structural abnormalities such as abnormal size, shape, nucleus to cytoplasm ratio, rapid growth with atypical mitotic figures and they infiltrate the surrounding tissues at the expense of the normal structures and frequently metastasize.

The histological differences between the two may at times be rather difficult as the benign tumours may show the some irregularities as that are found in malignant ones. On the other hand, metastasis may not occur in all malignancies.

More over new growths whose cells show definite malignant qualities may remain localized and may even heal spontaneously leaving the pathologist and clinician at loggerheads. For example keratoacanthoma one of the most rapidly growing skin tumours is benign but show malignant histopathological features. Next is basal cell epithelioma which is slow growing locally aggressive malignant tumour that ordinarily does not metastasize.

Genetic and environmental factors may play a role in the development of certain tumours.

Diagnosis of tumours has to be based on the history, clinical picture, histopathological examination with routine stains of haematoxylin and eosin and special stains and if needed histochemistry, fluorescent techniques and electron

microscopic studies. In this study the history, clinical picture and histopathology have been the diagnostic procedures adopted.

CLASSIFICATION OF SKIN TUMOURS

Tumors can arise from all the structures of epidermis, dermis or subcutaneous tissues and can be divided accordingly. Various classification have been proposed in the past which have required modifications from time to time in the light of most recent ultrastructural,histochemical findings and the reporting of new morphological entities. This study is based on the classification of tumours of skin given in the LEVERS Histopathology of skin 10th edition and Seldon & Helwig, 1974 approved by WHO [4] & this study is confined to the head and neck.

PRIMARY

1.Tumours of the surface epidermis

A.Benign

1. Epidermal nevus
2. Seborrhoeic keratosis
3. Clear cell acanthoma
4. Fibro epithelial polyp
5. Warty dyskeratoma
6. Actinic keratosis
7. Keratoacanthoma
8. Benign lichenoid keratosis

B. Malignant

1. Squamous cell carcinoma 2. Basal cell carcinoma

C.Cysts

Follicular cysts : Infundibular cyst, Trichilemmal cyst, Steatocystoma multiplex, Dermoid cyst, Eruptive vellus hair cyst

Milia

Bronchogenic and thyroglossal cyst

Cutaneous ciliated cyst

Median raphe cyst of the penis.

2. Tumours of the epidermal appendages

Lesions	Follicular differentiation	Sebaceous differentiation	Apocrine differentiation	Eccrine differentiation
Hyperplasia, Hamartomas	Hair follicle Nevus Dilated pore	Nevus Sebaceous Sebaceous Hyperplasia	Apocrine nevus	Eccrine nevus
Benign Neoplasms	Trichofolliculoma Pilar sheath acanthoma Fibrofolliculoma Trichodiscoma Trichoepithelioma Trichoadenoma	Sebaceous adenoma Sebaceoma	Apocrine Hidrocystoma Hidradenoma papilleriferum Apocrine syringocystadenoma Tubular apocrine adenoma Eruptive	Eccrine Hidrocystoma Syringoma Eccrine cylindroma Eccrine poroma Mucinous syringometaplas

	Pilomatricoma Proliferating trichemmal cyst Trichilemmoma Tumour of the follicular infundibulum		adenomatosis of nipple Apocrine Cylindroma	ia Eccrine spiradenoma Nodular hidradenoma Chondroid Syringoma
Malignant neoplasms	Pilomatrix carcinoma Malignant proliferating trichilemmal tumor Trichilemmal carcinoma Trichoblastic carcinoma	Sebaceous carcinoma	Malignant apocrine cylindroma	Porocarcinoma Malignant eccrine spiroadenoma Malignant chrondroid syringoma Eccrine adenocarcinoma Microcystic adnexal carcinoma Adenoid cystic carcinoma Syringoid eccrine carcinoma Malignant eccrine cylindroma

3. Tumors of fibrous tissue

A.Benign

a. Hyperplastic scar b. Keloid

c. Nodular fasciitis d. Dermatofibroma

e. Fibroma f. Angiofibroma

B. Malignant

a. Dermatofibrosarcoma protuberans b. Fibrosarcoma

4. Tumor of blood vessel

A. Benign

a. Granuloma pyogenicum b. Capillary haemangioma

c. Cavernous haemangioma d. Verrucous keratotic haemangioma

e. Glomus tumor group

i. Glomus tumor ii. Glomangioma iii. Angiomyoma

f. Angiokeratoma

i. Mibelli and Fordyce types ii. Fabry type (Angiokeratoma corporis diffusum)

g. Others

B. Malignant

a. Angiosarcoma b. Kaposi's sarcoma

5. Tumours of the fatty, muscular and osseous tissue.

Tumours of the fat

A. Benign

a. Lipoma b. Angiolipoma c. Hibernoma

B. Malignant

a. Liposarcoma

Tumours of the muscle

A. Benign

a. Leiomyoma

B. Malignant

a. Leiomyosarcoma

6. Tumours of the neural tissue

A. Benign

a. Neurofibroma & plexiform neuroma

b. Neurilemmoma (Schwannoma)

B. Malignant

a. Malignant Schwannoma & others

7. Tumors and lesions of the melanogenic system

A. Benign (Naevus)

1. Junctional Naevus 2. Compound Naevus

3. Intradermal Naevus 4. juvenile melanoma

5. Balloon cell naevus 6. Halo naevus

7. Giant pigment naevus 8. Fibrous papule of the nose (involving naevus)

9. Blue naevus 10. Cellular blue naevus

B. Precancerous

1. Precancerous melanosis (Hutchinson's melanotic freckle)

C. Malignant

1. Malignant melanoma

2. Malignant melanoma arising in precancerous melanosis including Hutchinson's melanotic freckle

3. Malignant melanoma arising in a blue naevus

4. Malignant melanoma arising in a giant pigmented naevus

8. Tumours of hematopoeitic & lymphoid tissue:

1.Mycosis fungoides 2.Urticaria pigmentosa

3.Leukemia and lymphoma 4.Reactive lymphoid hyperplasia

5.Benign lmphocytoma cutis 6. Benign lmphocytic infiltrate

7.Langerhans histiocytosis 8.Eosinophilic granuloma

SECONDARY

Metastatic carcinoma of the skin

Skin tumours pertaining to head &neck chosen for this study are as follows

1.Seborrhoeic keratosis	2.Fibroepithelial polyp
3.Epidermal nevus	4.Basal cell epithelioma
5.Squamous cell carcinoma	6.Epidermal cyst
7.Steatocystoma multiplex	8.Trichoepithelioma
9. Nevus Sebaceous	10.Syringoma
11.Keloid	12.Angiofibroma
13.Pyogenic granuloma	14.Neurofibroma

SEBORRHOEIC KEROTOSIS

(Syn. Senile wart, Seborrhoeic wart, Basal cell papilloma, Seborrhoeic verruca, Brown wart)

Yeatman et al[5] on the prevalence of seborrhoeic keratosis in an Australian population reported the frequency of seborrhoeic keratosis in 100 adults in the age group of 15-25, 26-50 and above 75 years. There was an increase in prevalence of seborrhoeic keratosis from 12 % of 15-25 years old to 100 % in the age above 50 years. These are benign tumours composed of epidermal keratinocytes, and is frequently pigmented. It occurs more commonly in the elderly. It may also be seen in the younger age group. Multiple seborrhoeic keratosis may be a familial trait, autosomal dominant mode of inheritance [6] .Its

occasional association in the same patient with fibroepithelioma type of basal cell carcinoma suggest that it could be a nevoid tumour as studied by Pinkus .Het al. [7]

A genetically determined predisposition based on mosaic pattern of aberrant response to epidermal growth factors and inhibitors would explain those cases where a profuse eruption follows an inflammatory dermatosis. It occurs as a manifestation of internal malignancy, usually cancer of gastro intestinal tract. The latter sign is named after Leser-Trelat[8] .Tumour derived circulating growth factors or humoral factors may be involved in the pathogenesis of these lesions.[9] Higher prevalence of seborrhoeic keratosis on the sun exposed areas show that sunlight could be a factor in the etiology of the seborrhoeic keratosis. Because of the verrucous appearance of the lesions HPV has also been suggested as a possible etiology. According to Mackie et al , the males & females are equally affected .[10] Lesions can occur on any part of the body. But they are more common over the face, neck, upper trunk and they can be bilateral, symmetrical or asymmetrical.

Classically it presents as a verrucous plaque with stuck on appearance. The colour is yellowish to light brown. Fully developed lesions are deeply pigmented and covered by greasy scales and are few millimeters to several centimeters in diameter. Lesions over eyelids and flexures are occasionally pedunculated. Usually seborrhoeic keratosis are asymptomatic, rarely itchy. Irritation or infection may cause swelling, bleeding, oozing, crusting, deepening of

the colour due to the inflammation .They do not involute spontaneously. Transient eruptive type may be associated with erythrodermic type of pitryasis rubra pilaris. Clinical variants are stucco keratosis, as described by Willoughby C, Soter [11], a non-pigmented variant of seborrhoeic keratosis, occurring principally on the limbs and, dermatosis papulosa nigra occur commonly in the dark skin individuals, appear early in life and may be multiple in numbers over the face. [12]

The lesions are mostly benign and malignant transformation to basal cell carcinoma, squamous cell carcinoma and bowens disease or anaplastic epithelioma occur rarely as studied by Cascayo CD Eval. [13]

Multiple eruptive seborrhoeic keratosis known as Leser -Trelat sign is associated with multiple internal malignancies [14] like adenocarcinoma of the colon and breast. Malignant acanthosis nigricans present is 35% of patients with Leser- Trelat sign suggesting a similar mechanism of action [15].

Histological types

The histological subtypes of seborrhoeic keratosis are

1. Acanthotic type 2. Melanoacanthoma 3. Hyperkeratotic type

4. Adenoid type 5. Superficial type 6. Irritated type

7. Clonal type representing an intra-epidermal epithelioma of Borst Jadosshon.[16]

Histopathology:

The common features are hyperkeratosis, acanthosis, and papillomatosis. The acanthosis is due to the upward proliferation of the epidermal cells and this is responsible for the stuck on appearance clinically. In the irritated type, squamous eddies are seen numerously. In adenoid type, numerous thin tracts of epidermal cells lined by double row of basaloid cells, extending from epidermis showing branching and interweaving in the dermis. In the acanthotic type, numerous true horn cysts and pseudo- horn cysts are seen. In hyperkeratotic type, numerous digitate upward extensions of lined papilla resembling church spires are seen .In the clonal type, numerous well defined nests of epidermal cells with small, dark stained nuclei are seen. In the melanoacanthotic type, numerous melonocytes are seen scattered throughout the tumour lobules intermingled with basaloid cells.

Treatment:

1. Removal with a curette and the base may be cauterized or electro-coagulated or treated with haemostatic solution such as silver nitrate or ferric sub sulfate (Monsels Solution).

2. Cryotherapy and dermabrasion

3. Topical 5-Flurouracil [17] & Trichloro- acetic acid

4.Surgical excision is not usually indicated except when malignant melanoma is considered as differential diagnosis and when the lesion is large and not responding to other modes of treatment.

5. Laser therapy .

ACROCHORDONS

(Soft fibroma , Acrochordons)

A common benign lesion composed of loose fibrous tissue and occurring mainly on the neck and major flexures as a small soft pedunculated protrusion. A slight female prepondarence was noted with occurence of lesions in pregnancy and menopause. Study of Thappa DM et al with the study group of 35 patients ranged in age from 35 to 73 years, with a mean of 52.03 showed the risk of getting skin tags found to increase with age, but this risk decreased after the fifth decade & the neck was invariably involved, followed by the eyelids, axillae and groin. Of the cases, 62.8% (22 patients) had DM.[18]

Etiology remain unknown, role of growth factor has been suggested. Several reports have suggested an association between the presence of soft fibroma and colonic polyps ,diabetes [19] and acromegaly.[20]

Clinical features:

(a) Typically multiple small, furrowed papules, especially on the neck and in the axillae (1 to 2 mm long) (b) single or multiple filiform, smooth growths in

varying locations, about 2 mm wide and 5 mm long; and (c) solitary bag-like pedunculated growth about 1cm noted on trunk .

Histopathology:

Small furrowed papules usually show hyperkeratosis, regular acanthosis, papillomatosis, and occasionally horn cysts within their acanthotic epidermis. The filiform, smooth growth shows slight to moderate acanthosis and occasionally mild papillomatosis. The connective tissue stalk is composed of loose collagen fibers interspersed with numerous dilated capillaries . Nevus cells are found in as many as 30% of the filiform growths, indicating that some of them represent involuting melanocytic nevi .The bag-like, soft fibromas generally show a flattened epidermis overlying loosely arranged collagen fibers with a varible number of fat cells in the dermis .

Treatment:

1.Chemical cautery 2.Electrofulgration

3. Application of cryotherapy 4. Surgical excision.

VERRUCOUS EPIDERMAL NEVUS

(Syn. Nevus verrucous, Linear verrucous nevus,Linear epidermal nevus.)

The term nevus denotes a circumscribed congenital developmental abnormality resulting in faulty production of mature and nearly mature structures.

Epidermal nevi are hamartomatous lesions arising from embryonic ectoderm. The pluripotent ectodermal cells evolve into a variety of differentiated cell types, including keratinocytes and cells forming the various appendages. Most of the cases occur within the first years of life, rarely reports of elderly, with the oldest being 60 years of life as described by Adams DF et al.[21] These arise due to the genetic mosaicism. Also, verrucous epidermal nevi having the histological features of epidermolytic hyperkeratosis reflects the gene mutation.

Verrucous epidermal nevi occurs at any site but uncommon in face and head, where nevus sebaceous are more common .Lesions may be single or multiple. They follow the lines of Blaschko.[22] On the trunk, they are transverse bands, lesions virtually never cross the midline, but the lesions close to the midline take the course of vertical direction. Mucous membranes may be affected. Linear nevi are seen clinically as hyperpigmented warty growths arranged in linear plaques. Linear nevi may be localized or systematized .The localized type presents at birth with only one linear lesion. It consists of closely set hyperkeratotic papules anywhere in the body. Nevi along the long axis in unilateral fashion is called as nevus unis lateralis. In this form it resembles ILVEN (Inflammatory linear verrucous Epidermal nevus) .But the latter differs from it clinically by the presence of erythema and pruritis and histologically by the presence of parakeratosis and inflammatory changes.

Nevus unis lateralis may be associated with woolly hair [23] and megalopinna.[24] In addition to this, nevi may also be seen in Proteus syndrome, CHILD syndrome, Mc-cune Albright syndrome and in Klippel-Trenaunay syndrome.[25] Occasionally, basal cell epithelioma[26] is observed particularly on the head, in the case of linear epidermal nevi associated with either a nevus sebaceous or a syringocystadenoma papilliferum. Similarly squamous cell carcinoma & bowens disease reported rarely.[27] But in one instance it metastasized to the region lymph node . A study conducted by Vidaurria La et al reported 35 cases of epidermal nevus syndrome among 443 patients with epidermal nevi seen in National Institute of Pediatrics' in Mexico during a 31 year period. [28] It represented 7.9 % of epidermal nevus syndrome were observed in 443 patients with epidermal nevi.

Histopathology:

It can be divided into two types

1. Epidermolytic hyperkeratosis 2. Non-epidermolytic hyperkeratosis.

The common features are hyperkeratosis, acanthosis, papillomatosis, and elongation of rete ridges.

Epidermolytic type or granular degeneration of epidermis shows Compact hyperkeratosis, perinuclear vacuolization of cells in the granular and spinous layer,

peripheral to the vacuolization is indistinct cell margins & an increased number of irregular shaped, large keratohyaline granules.

Some lesions show distinct church spire pattern of acanthosis and hyperkeratosis resembling acrokeratosis verruciformis and Seborrhoeic keratosis. Very rarely it also shows focal acantholytic dyskerotosis. Rarely it may show the features of viral warts, acanthosis nigricans, verrucous phase of incontentia pigmenti as described by Fletches Vs Williams, Lone et al. [29]

Treatment:

It is wise to delay the therapy as the final extent of the process cannot be determined, failure to do so may result in the appearance of new lesions in adjacent site to the treated area. Small linear lesions can be excised. Improvement is achieved by the use of electrodesiccation or Cautery. Cryotherapy or CO2 laser has given inconsistent results. The only effective treatment of this nevi is surgical excision. Topical treatment include podophyllin, retinoic acid, anthralin, calcipitriol are used, but relatively ineffective. Occasionally, using combination therapy will lead to higher efficacy. Systemic retinoids can produce a partial but usually temporary response in some patient.

BASAL CELL EPITHELIOMA

(Syn: Rodent ulcer, Jacobs's ulcer, Basilioma)

Basal cell epithelioma was first described by Jacob in 1827.Krompecher in 1903 found out that the tumour arises from the basal cells of the epidermis. Wallace and Halpert in 1950 described that they were benign tumours of the hair matrix, differentiated to the hair follicle and called them trichoma.[30] According to Lever, they were nevoid tumours or hamartomas arising from the primary germ cells. Pinkus suggested that basal cell carcinoma occurring in later life arises from pluripotent cells that form continuously differentiating into hair, sebaceous & sweat gland.

Predisposing factors are prolonged exposure to the sunlight (light skin color), large doses of Radiation[31] to the face and even to the spine, prolonged intake of inorganic arsenic, thermal injury to the skin[32] , the scars of tuberculosis cutis and small pox vaccination.[33] Mainly they occur over the hair bearing skin. Face is the commonest region where it can involve the inner canthus of the eye, bridge of the nose, along the imaginary line from the tragus of the ear lobe to the angle of the mouth. Oral mucosa may be involved occasionally. It is common in adults and rare in children. There is no sex predilection but males are slightly more affected due to the factors of occupation and sun exposure.

Clinical types of basal cell carcinoma are

1. Nodulo-ulcerative
2. Pigmented
3. Fibrosing / Sclerosing / Morphoea like
4. Superficial

5. Fibroepithelioma

Clinical syndromes

1. Gorlins syndrome [34].

2. Linear unilateral basal cell nevus[35]

3. Bazex syndrome[36].

A study conducted by Christensen LJ et al at Dept of Dermatology, Mayo clinic, Rochester reported that nodular basal cell carcinoma was the most common subtype. [37] Nodulo-ulcerative type-begins as a small waxy nodule that often shows a few small telengiectatic vessels on its surface. It slowly increases in size and undergoes central ulceration and is surrounded by pearly, rolled out borders-classical rodent ulcer. Occasionally they invade deeply, destroying eyes, nose, cartilage and even the dura mater by penetrating the skull[38] .Pigmented type-differs from the above only by the brown pigmentation of the lesion due to the proliferation of melanocytes in the tumour. Fibrosing type- manifests as solitary, flat or slightly depressed indurated yellowish plaque with smooth and shiny surface and ill-defined border, almost on the face. Superficial type -consists of one or several erythematous scaly, slightly infiltrated plaque that slowly increase in size and are surrounded by a fine thread like pearly borders. They show small areas of superficial ulceration and crusting and the center may show atrophic scarring. This type commonly occurs on the trunk. Fibroepithelioma of Pinkus-

consist of one or several raised firm pedunculated nodules covered by erythematous skin and commonly located on the back.

Histopathology:

The characteristic cell is the basalioma cell with a large oval or elongated nucleus and relatively little cytoplasm. The cells do not show any inter-cellular bridges by the light microscope. The nuclei are uniform in size and staining. The connective tissue stroma is arranged in parallel bundles around the tumour masses. The stroma appears mucinous and reacts metachromaticaly. There are retraction clefts around the tumour masses and lacunae are due to the absence of the bullous pemphoid antigen[39] . A mild inflammatory infiltrate may be seen but dense lymphocytic infiltrate is usually seen if the lesion clinically shows ulceration.

From the histological point of view basal cell carcinoma divided into 3 groups: Undifferentiated - circumscribed, infiltrative

Differentiated – keratotic , cystic , adenoid

Mixed

There are 4 uncommon histological variants of basal cell carcinoma

1. Adamantinoid type-resembles dental adamantinoma
2. Granular type-resembles granular cell tumour
3. Clear cell type-contains glycogen vacuoles in cytoplasm
4. Matricial type -shows shadow cells as in pilomatricoma

Treatment:

According to Pillsbury statement, the cure rate is 100% when the lesion is recognized and intervened rapidly. The choice of therapy depends on the site the size and the number of lesions. Therapy is not satisfactory when the basal cell carcinoma involves the orbit, nose or ear.

1. Surgery

If the lesion is large-wide excision followed by either full thickness graft or Curettage and cauterization must be done by a competent plastic surgeon.

2. Mohs Micrographic Surgery

This method is used following curettage & desiccation to determine the clear zone in cases of invasive and infiltrative tumour in difficult sites.

3. Cryosurgery with liquid nitrogen

4. Curettage and cauterization

5. Interferon therapy and photodynamic therapy are useful.

7. Radiotherapy

This is best for elderly and for extensive tumour lesions especially involving inaccessible sites (eyelids). Radiotherapy is contraindicated for recurrent lesions and Morphoeic type of basal cell carcinoma which is radio resistant.

7. Combination therapy : Surgery + Irradiation of the lesion.

8. Local cytotoxic therapy

a. Topical 20% 5-FU ointment b. Topical 20% Podophyllin resin.

SQUAMOUS CELL CARCINOMA (SCC)

It is a malignant tumour arising from the kerartinocytes of the epidermis. Pott was the first to describe the malignant nature of the squamous cell carcinoma in 1775 .Squamous cell carcinoma is strongly associated with advanced age with a sharp increase in incidence after the age 40. It is twice common in men than women. Melanocortin-1 receptor, is a major determinant of skin pigmentation and hair color. Several variant of MCIR alleles are associated with increase of squamous cell carcinoma that was independent of skin type and hair color. MCIR gene is highly polymorphic with more than 20 variants [(40)].

Nuzhat Yauman et al studied 75 cases of malignant skin tumours of which squamous cell carcinoma were 30 cases, basal cell carcinoma were 36 cases, malignant melanoma 5 cases, bowens disease 1 case and malignant trichilemmoma 1case . [(41)]

Predisposing factors are UV exposure, Ionizing radiation ,Environmental carcinogens, Immunosuppression, Scars, Burns or chronic heat exposure, Inflammatory dermatosis, Precursor skin lesions (angiokeratoma, bowens disease), Genodermatosis (Xeroderma Pigmentosum, Porokeratosis). Human papilloma virus infection.

Squamous cell carcinoma presents as a firm, flesh colored or erythematous keratotic papules or plaque, but squamous cell carcinoma also sometimes will be pigmented. Other presentations of squamous cell carcinoma are ulcer, nodule and as cutaneous horn . Margins may be distinct, firm, elevated. Progressive tumour invasion ultimately results in fixation to the underlying structures. Lymph node involvement may be present and is due to the metastasis.

Oral squamous cell carcinoma presents in patients with long history of smoking, tobacco chewing and alcohol abuse. Squamous cell carcinomas of the oral cavity are common in males, palate, and tongue are the most common sites[(42)]. Oral squamous cell carcinoma most commonly evolve from lesions of erythroplakia and it is usually asymptomatic and presents as persistent, rough, patch or plaque that ultimately becomes firm and nodular. Lower lip Squamous cell carcinoma begins as a papule of actinic chelitis or scaly leukoplakia with slow progression to a tumour nodule..

Histopathology

The hallmarks of invasive squamous cell carcinoma are the extension of atypical keratinocytes beyond the basement membrane and into the dermis, the absence of connection between tumour cells and the epidermis . Tumours appear as single mass or small group of nests of cells. The lower border may broadly impose on the dermis or be represented by individual foci of micro invasions.

Invasive tumour is confined to dermis, subcutaneous involvement is unusual. There are typically varying proportions of normal appearing and atypical squamous cells with increased mitosis, aberrant mitotic figures, nuclear hyperchromasia and loss of intercellular bridges. Squamous differentiation is seen as a foci of keratinisation, concentric rings of squamous cells called horn pearls. Loss of differentiation is associated with decreased keratin production.

Histological subtypes:

1. Adenoid type-tubular microscopic pattern and keratotic acantholysis
2. Clear cell type-keratinocytes appears clear as a result of hydrophic cytoplasmic swellings and accumulation of lipid vacuoles
3. Spindle cell type-spindle shaped atypical cells
4. Signet ring type-rare with concentric rings composed of keratinocytes and large vacuoles corresponding to dilated endoplasmic reticulum
5. Basaloid type
6. Verrucous type-Acanthosis and papillomatosis are more
7. Mucinous type.

Metastatic rate of Squamous cell carcinoma is 0.5-6% [(43).] It is common in tumours that are large, recurrent and if it is involving the deeper structures. [(44)]

High risk Squamous cell carcinoma are considered if diameter >2cms ,depth >4mm ,tumour involving the bone & muscle , location –ears, lips ,tumour arising from the scars , immuno suppression & absence of inflammation.

Treatment:

1. Surgical excision-wide excision with a clear margin of 4mm, if the lesion is <2mm and 6mm if the lesion >1cm
2. Mohs microsurgery surgery-for high risk cases
3. Radiation- for superficial, small lesions.

EPIDERMAL CYST

(SYN: Epidermoid cyst, epithelial cyst, keratin cyst, sebaceous cyst, infundibular cyst, epidermal inclusion cyst)

It is the most common of all the cysts .They result from the proliferation of the surface epidermal cells lying within the dermis. Production of keratin and lack of communication with the surface are responsible for the cyst formation. Most epidermal cysts arise from the occluded pilosebaceous follicles as described by Mc.Gaven and Binnington in 1966.

Epidermal cyst may occur during puberty or in adult life and affect both sexes. They are frequently seen over the face, scalp neck, shoulder, and trunk. [45] It may be solitary or multiple. Epidermal cysts are slow growing cysts, elevated, round, firm, intradermal or subcutaneous tumours. They are dome shaped

protuberances that are mobile over the deeper structures. They are tethered to the overlying epidermis and occasionally they may show central keratin filled blackish or bluish punctum. A clinico pathological study conducted by ChandrasekaranV et al reported that 40% had punctum in 34 patients of epidermal cysts.[46] On rupture, a cheesy, odoriferous material may be expressed.

Rarely malignant transformation may occur [47] as described by Delaretz J. These may go for complications like secondary infection, predominantly by anaerobes [48] and dystrophic calcification.

Histopathology

The cyst wall is composed of all the layers of epidermis in the dermis and the lumen is filled with laminated keratin. In sectioning with haematoxylin and eosin, melonocytes and melanin pigment of keratinocytes. Rupture of the cyst into the dermis elicits a foreign body granulomatous reaction-keratin granulomas containing numerous multinucleated giant cells. Malignant degeneration of the epidermal cyst is interpretated either as pseudo carcinomatous hyperplasia in a ruptured epidermoid cyst [49] or as a proliferating trichilemmal tumour [50].

Treatment:

Complete surgical excision is required if the cyst becomes symptomatic. An inflamed cyst is better incised & drained and phenolised. An inflamed,non-infected cyst can be treated by intralesional triamcinolone 5mg per ml.

STEATOCYSTOMA MULTIPLEX

(Syn: Sebocystomatosis, Epidermal polycystic disease)

Steatocystoma multiplex was first described by Pringle in 1899[51] .Several cases have been inherited as autosomal dominant trait [52] .Occasionally if it occurs as a solitary, non inherited, in adults it is called as simplex type[53] . A clinicopathological study of 64 cases of steatocystoma multiplex by Cha .S. et al, department of dermatology ,Seoul, Korea reported most of the cases were sporadic, average age being 26 years and the distribution over the chest arms and axilla . [54] It occurs due to the mutation in keratin 17 gene.

Men are affected more in the previous studies but now both sexes are equally affected .The lesions may present from birth or develop at puberty or shortly after that . The lesions are usually located over the upper trunk, especially over the sternum, axilla, arms ,face and sometimes over the scrotum. The lesions are numerous, small, round, soft to firm cystic nodules adherent to the overlying skin and measure 1-3 mms in diameter. The color varies from the flesh color to yellowish .On puncture, it discharges an oily fluid in some instances. They are asymptomatic but secondary inflammatory changes may occur with suppuration and scarring. Associated conditions are acrokeratosis veruciformis ,hidradenitis, hypohidrosis ,hypothyrodism, hypotrichosis, lchthyosis,koilonychia, pachyonchia congenita[55] and lichen planus. It is also associated with natal teeth.

Histopathlogy:

The cyst wall is intricately folded consisting of several layers of epithelial cells and the central portion consists of homogenous horny layer that protrudes irregularly in to the lumen. Characteristic feature is the presence of flattened sebaceous glands lobules either within or close to the cyst wall. Occasionally cysts contain lanugo hair .

Treatment:

It is usually asymptomatic. For extensive cases, dermabrasion may be useful. Isotretinoin can be useful in suppurated cases [56] .The best treatment for these lesions in the face is inserting a needle and extripating the contents without removing the cyst wall.

TRICHOEPITHELIOMA

(Syn: Brooke's tumour, Milia with telengiectasia, Epithelioma adenoids Cysticum, Benign cystic epithelioma)

A benign neoplasm with differentiation directed towards hair structures, particularly follicular germs. Trichoepithelioma is classified as –solitary and multiple.

Solitary-Classical, Giant , Desmoplastic.

Multiple trichoepithelioma is due to the mutation in the gene CYLD [57].

Ziprkowski et al conducted a study in trichoepithelioma in which 50 % of the patients had family history of trichoepithelioma. Multiple trichoepithelioma is transmitted as autosomal dominant trait.[58] Initially the lesions appear in the childhood and gradually increase in number. The lesions are rounded, skin coloured ,firm papules and nodules of varying sizes, located mainly in the nasolabial folds, nose, forehead ,cheeks, upper lip and occasionally in the neck scalp and upper trunk. Rarely ulceration and malignant transformation to basal cell carcinoma may occur. Trichoepithelioma may be associated with Brookes-Spiegle and Rombo syndrome[59] systemic lupus erythematosus and myasthenia gravis.

Solitary trichoepithelioma of classical type is more common than multiple variety. It is not inherited and consists of a firm, elevated, flesh colored nodule usually less than 2cms in diameter. Its onset is in childhood or early adult life commonly seen over the face. Giant form of this tumour is a rare type that occurs in later life mainly in the thigh and perianal region. Desmoplastic form occurs mostly on the face and is markedly indurated[60], clinically it presents with a raised annular border and a depressed centre resembling granuloma annulare. It occurs in adolescence & young adult females.

Histopathology:

It consists of mainly two components in the dermis namely the horn cysts and basaloid cells. The former consist of fully keratinized center surrounded by

basaloid cells similar to the basal cell carcinoma , but lacking nuclear atypia and frequent mitosis. The keratinisation formed is abrupt and complete and is called as Trichilemmal keratinisation. The tumour islands are composed of basaloid cells arranged in a lace like or adenoid or solid aggregates. They show peripheral pallisading of the cells surrounded by fibrous tissue like stroma .The fibroblasts surrounding the basaloid cells lack the retraction artifact typical of basal cell carcinoma. Sometimes the horn cysts may rupture producing a foreign body granulomatous reaction and calcium deposition may also occur. The desmoplastic form shows narrow strands of basaloid cells, multiple horn cysts and a dense desmoplastic stroma..

Histogeneis

It is assumed that horn cysts represent alteration in hair shaft formation and the basaloid cells surrounding horn cysts are analogous to the hair matrix cells.

Treatment:

For Solitary lesions, complete and adequate surgical excision is the best method, followed by the primary closure grafting if necessary .For multiple lesions, plastic repair with cosmetic care is the best mode of therapy. Electro cautery, Cryotherapy and Dermabrasion have also been tried. CO2 laser is also useful.

NEVUS SEBACEOUS

(Syn: Organoid nevus, Nevus Sebaceous of Jadosshon)

Jadosshon definition of the organoid nevi is stable localized malformation of the skin through excess or deficiency of one or more of the normal mature constituents such as hair, glands , epidermis, or connective tissue and it excludes adenomas and often, less mature tumours. The evolution of nevus sebaceous includes 3 stages,

1. Early stage in infancy and childhood often characterized by under development of the hair and sebaceous glands, usually located on the scalp or face & it consists of circumscribed slightly raised, hairless plaque, often linear in configuration
2. An intermediate stage, usually begins with puberty and leads to the development of the glands and maturation with papillomatous hyperplasia , lesion becomes verrucous and nodular.
3. Late stage is due to the complications; development of the benign and malignant tumours.

Chang,Yi et al done a clinico pathological study of nevus sebaceous of 104 cases.[61] Among them, 48 were males and 56 were females. The age ranged from 3-60 years, but the mean age was 23.5 years. Most of the nevus sebaceous developed before 10 years (83 %).Mostcommon site was scalp (70 %). This nevus is seen at birth or may appear in early life as a solitary lesion usually located

on the scalp or face. Some patients may show associated features of epilepsy, mental retardation, neurological defects, skeletal defects called as nevus comedonicus syndrome. Nevus may appear sporadically. Familial nevus has been described but is exceedingly rare [62] .

Histopathology

In the first few months of life, the sebaceous glands are well developed. Thereafter in childhood they regressed and reduce in size and number. The presence of incompletely differentiated hair structures is typical of this nevus. Some hair follicles consists of dilated, keratin filled infundibula showing multiple buds of undifferentiated cells. At puberty ,large number of mature sebaceous glands with papillomatosis and hyperplasia of the epidermis are seen which is the characteristic feature. Mature apocrine glands are also present.

In adults various types of appendage tumours develop secondarily within the lesions of nevus commonly syringocystadenoma papilliferum and less commonly nodular hidradenoma, syringoma, chondroid syringoma, sebaceous epithelioma, trichilemoma and proliferating trichemmal cyst. Rarely, basal cell carcinoma, squamous cell carcinoma, apocrine carcinoma and malignant eccrine poroma have also been reported .Mutations in the PATCH gene is reported for basal cell carcinoma in the sebaceous nevus [63]

Therapy

Surgical excision with primary closure can be done. Lesions can be removed by tissue expansion technique. Dermabrasion and CO2 laser are also effective but recurrence is possible.

SYRINGOMA

(Syn: Syringocystoma, Hidradenoma eruptives)

Patrizi et al reviewed 29 cases of syringoma of which only one patient complaints of itching, two cases had solitary lesions, six patients had only eye involvement and eighteen patients were eruptive syringoma. [64] Syringoma are benign adenomas arising from the intra-epidermal portion of the eccrine sweat duct. Syringoma occurs predominantly in women at adolescent age or later in life. The lesions are multiple but solitary type is also reported .They are small skin colored, soft elevated, flat topped papules of size 1-5mm in diameter with angular outline .They are commonly seen in bilateral symmetrical pattern below the lower eyelids but may also occur in cheeks, neck, axilla, trunk, abdomen and rarely vulva [65]. It has also been reported in patients with Downs syndrome .[66]

Classification:

1. Multiple 2. Solitary 3. Eruptive
4. Unilateral 5. Occult 6. Inapparent
7. Annular 8. Acral 9. Chondroid

Unilateral type may occur rarely in the face or upper chest. Occult type occurs in scalp in association with diffuse thinning of hair or cicatricial alopecia. Inapparent type have also been described as incidental finding in close approximation with basal cell carcinoma. Annular type is the one in which the papules are arranged in the annular fashion in the trunk. Acral type are of multiple lesions appear as symmetrical grouped erythematous papules over the dorsa of fingers and hands in young men. Chondroid syringoma, a special type-a benign tumour of eccrine sweat gland with stromal participant. Clinically, the tumours are firm, intradermal or subcutaneous nodules occurring most commonly on the head and neck and of size 0.5 to 3 cms in diameter.

Histopathology:

It shows numerous small cystic ductul structures lined by two rows of flattened epithelial cells, where the inner row of cells are vacuolated. These cystic ducts are embedded in dermal fibrous tissue stroma. The lumina contains amorphous debris. Some of the ducts have tail like projections of epithelial strands into the fibrous stroma giving a typical Tadpole or Comma like appearance .In addition ,solid strands of basophilic epithelial cells are also seen independent of ducts. There is also a distinctive clear cell variant with an epithelial lining consisting of cells with large cytoplasm and glycogen. This type is associated with diabetes mellitus [(67)]

Histogenesis

Enzymes, histochemical and electron microscopic studies have established syringoma as a tumour with differentiation directed towards intraepidermal eccrine duct. Histochemical examination of syringoma tumour cells shows strongly positive reaction for eccrine enzymes namely succinyl dehydrogenase, phosporylase, and leucine aminopeptidase.

Treatment:

The treatment is required mainly for cosmetic reasons. ElectroCautery, chemocautery, and dermabrasion are found to be useful. [68] Surgical excision can be done for solitary large lesion.

KELOID:

Albert in 1825 described keloids as true when arising spontaneously and false when arising at sites of trauma**.**[69] A keloid (cheloid,meaning 'crab claw') is a benign, well-demarcated area of fibrous tissue overgrowth that extends beyond the original defect.

Precipitating factors are surgery, lacerations, tattoos, burns, injection sites, bites, vaccination, piercing and blunt trauma. Another risk factor is presence of foreign material either exogenous (e.g. suture material) or endogenous (e.g. embedded hair). Persons with blood group A are also have an increased tendency

to develop keloid . Keloids quite commonly follow ear piercing, especially in black people with a predisposition.

Keloids occur in all age groups although mainly in the third decade of life. Both sexes are equally affected[70,71] . It is more common in blacks as compared to whites[72]. According to Bayat et al study , 211 cases of keloid scarring with 137 (65%) females and 74 (35%) males, the formation of keloid scars in multiple anatomical sites was more common in younger age groups & more than 50% (111) of all keloid cases had a positive family history of keloid scarring, and family history was strongly associated with the formation of keloid scars in multiple sites as opposed to a single anatomical site. [73]

Keloids are variable in size from 2 to 3 mm papules to large pendulous tumours. The lesion appears as a firm, mildly tender or pruritic, bosselated, well-demarcated pink or red plaque with regular margins to irregular claw-like projections occurring more frequently on shoulders, chest, neck, upper arms, earlobes and cheeks. Keloids tend to regress after several years. Lesions on the beard area sometimes undergo central suppurative necrosis. Malignant degeneration has been reported .

There is a genetic association with other fibromatoses such as Dupuytren's contracture[74] with Dubowitz's syndrome[75] ,Ehlers Danlos

Syndrome, pachydermoperiostosis and Rubinstein Taybi syndrome.[76] Keloids form readily in acromegalics, and after thyroidectomy in young patients.

Pathology :

There is a vascular proliferation along with fibroblasts enlarges and transforms into thickened nodular mass of varying sizes containing greatly thickened, compact, glassy, eosinophilic collagen bundles and proteoglycan. This persistent transformation of swirl-like fibroblast clusters into hyalinized collagen bundles appears essential for keloid growth.[77]

Histogenesis:

Keloid fibroblasts, unlike those from hypertrophic scar tissue, are hyperresponsive to both TGF which is abundant in healing wounds[78] and PDGF [79]. It has been suggested that decreased apoptosis may play a role, allowing the keloidal fibroblasts to proliferate and produce more collagen [80,81].

Treatment:

Non-essential surgery should be avoided in the sites of predilection. If surgery is necessary, simple excision can be done. Radiotherapy, including superficial X-rays, electron beam therapy or implantation with iridium-192 wires, may prevent recurrence following surgery.

Small keloids can respond to silicone gel (e.g. Silastic) held in place with adhesive tape[82].

Prior freezing with liquid nitrogen before the injection causes oedema, which allows the triamcinolone to be injected more readily.

Intralesional 5-fluorouracil and 585-nm flashlamp-pumped pulsed dye laser produced comparable improvement in one study [83].

ANGIOFIBROMA:

(Adenoma sebaceum of Pringle)

Tuberous sclerosis, a dominantly inherited disorder, is characterized by the triad of mental deficiency, epilepsy, and angiofibromas of the face. In the past, the symmetrically distributed, small, red angiofibromas of the face were mistakenly called adenoma sebaceum. However, the sebaceous glands are generally atrophic.

The angiofibromas consist of numerous small, red, smooth dome shaped papules 1-4 mm in size occurs in a symmetrical distribution in the nose, nasolabial folds, on the cheeks, and over the chin most commonly appear in early childhood. Other organs are frequently involved.[84] Additional cutaneous manifestations may include asymmetrically arranged, large, raised, soft, brown fibromas on the face and the scalp, subungual and periungual fibromas, and so-called shagreen patches, usually found in the lumbosacral region and consisting of slightly raised and thickened areas of the skin. Their diagnostic significance lies in

the fact that they are present at birth or appear very early in life, and thus are the earliest cutaneous sign of tuberous sclerosis [85].

Genetic linkage studies show linkage to chromosome 9q34 (TSC 1) or to chromosome 16p31 (TSC 2) [86,87] in families with tuberous sclerosis. Two-thirds of cases are sporadic and are assumed to result from new mutations many of which are in TSC 2[88].

Multiple tumors are commonly found in the brain (gliomas, often calcified), retina (gliomas), heart (rhabdomyomas), and kidneys (angiomyolipomas)

Histopathology:

It is characterized by the dermal proliferation of spindle, stellate or multinucleate giant cells and dilatation of capillaries. Occasionally, multinucleate giant cells are also present. sometimes, there are vascular proliferation and perivascular proliferation of fibroblasts in addition to vascular dilatation. In old lesions, there may be perifollicular proliferation of collagen, leading to the compression of atrophic hair follicles by concentric layers of collagen with the absence of elastic tissue.

Treatment:

No specific treatment are available. Cosmetic surgery, diathermy , dermabrasion ,Pulsed Dye laser may be tried.

PYOGENIC GRANULOMA:

(Lobular capillary hemangiomas ,proud flesh. Bloody wart)

In 1904, pyogenic granuloma was proposed by Haitzell [89]. A vascular nodule that develops rapidly, often at the site of a recent injury, and which is composed of a lobular proliferation of capillaries in a loose stroma.

It is considered to be part of a reactive or infective process following trauma, drugs like retinoid & indinavir therapy , insect bite , burns ,cryotherapy , hormonal factor , underlying arteriovenous malformation. Many authors believe it to be a primarily a vascular disturbance.

A study conducted by Hessa Al Wayli et al , reported among 45 cases of pyogenic granuloma ,the most commonly affected site was the gingiva (87.09%), followed by the lip (9.67%) and buccal mucosa (3.22%) and the medical status were as follows: healthy(80%), pregnant(10%), diabetic(6.66%) and kidney transplanted (3.33%). & most of the cases (40%) were seen in the 31-40 years age group. [90] Usually occurs in children and young adults but may occur at any age. Common sites include face, finger, mucous membrane of oral cavity (gingiva, lips, tongue, buccal mucosa, and palate. Other less common sites are trunk, arms, leg, external genitalia, conjunctiva, cornea, larynx. Clinically, these are polypoid or pedunculated, reddish brown nodules usually less than 2cm in diameter. Often the surface of the lesions are ulcerated and bleed easily. Solitary lesions most commonly located on head & neck region[91].

Variants:

1. Epulis gravidarum is a variant of pyogenic granuloma that presents in the oral cavity during pregnancy
2. Satellite
3. Disseminated / eruptive
4. Intravenous
5. Subcutaneous

Histopathology:

It is characterized by polypoid mass of angiomatous tissue protruding above the surrounding skin & often constricted at its base by a collarette of acanthotic epidermis. An intact flattened epidermis may cover the entire lesion but surface erosions are common. In ulcerated lesions , a superficial inflammatory cell reaction with mild inflammatory reaction in the deeper dermis can give rise to an appearance suggestive of granulation tissue, but inflammation does not appear to be an intrinsic feature .The angiomatous tissue tends to occur in discrete masses or lobules, resembling a capillary hemangioma by Mills et al. Normal mitotic figures may be present. Extramedullary hematopoiesis has been reported in the stroma.

Treatment:

Curettage with cauterization or diathermy coagulation of the base if pedunculated lesions.

Shave, punch, or scalpel excision may be curative if the lesion is completely removed. Chemical cauterization with silver nitrate & ligation of the base.

Topical imiquimod cream and alitretinoin gel[92]

Laser & cryotherapy.[93]

NEUROFIBROMATOSIS

(VON RECKLINGHAUSON'S DISEASE):

VonRecklinghausen – coined the term neurofibroma in 1882. A neurofibroma is a tumour composed of neuromesechymal tissue (Schwann cells, perineural cells, fibrolasts, & mast cells). Solitary forms of cutaneous neurofibromas are relatively common in adults & equally present in both sexes. VonRecklinghausens disease (NF1), most common form of neurofibromatosis, characterised by Café-au-lait macules, freckles, multiple NF, and lisch nodules with autosomal dominant inheritance. Incidence of NF1 is 1:3,500. No gender or racial predisposition.

A diagnosis of NF1, according to the National Institutes of Health Consensus Development Conference Statement , is based on two or more of the following criteria:

1. Six or more café-au-lait macules of over 5 mm in greatest diameter in prepubertal individuals and over 15 mm in greatest diameter in postpubertal individuals;

2. Two or more neurofibromas of any type or one plexiform neurofibroma;

3. Freckling in the axillary or inguinal regions;

4. Optic glioma;

5. Two or more Lisch nodules;

6. A distinctive osseous lesion such as sphenoid dysplasia or thinning of the long bone cortex with or without pseudoarthrosis;

7. A first-degree relative (parent, sibling, offspring) with NF1 by the above criteria.

Riccardi classification:

TYPE 1: classical

TYPE 2: central or acoustic NF

TYPE 3:mixed of type1 & type2

TYPE 4:variant of NF2 with more cutaneous involvement.

TYPE 5: segmental due to post zygomatic mutation

TYPE 6: only CALM but in two successive generations.

TYPE 7: late onset

TYPE 8: definite NF but do not fit into other categories

Clinical features:

CALM: 1st feature sharply defined, light brown patches, borders like coast of California can be present anywhere except scalp and eyerows.

Freckling: It consists of small tan brown , macules that are 1-3 mm in diameter, diagnostic only when they are present in axillae or groin.

Neurofibroma:

Classical circumscribed- sessile & pedunculated

Subcutaneous

plexiform – diffuse [elephantiasis & cimcumscribed

Lisch nodules (pigmented iris hamartomas) appear as dome-shaped lesions found superficially

around the iris on slit-lamp examination. They occur in over 90% of patients and increase with age. They are asymptomatic but help to confirm the diagnosis

Other cutaneous featuers are hyper and hypopigmentation , blue macules, hypertrichosis, Juvenile xanthogranuloma, Cutaneous angioma, macroglossia, Cutis verticis gyrata.

Histopathology :

Well cimcumscribed non- encapsulated tumour of dermis composed of thin spindle shaped cells with elongated wavy nuclei, regularly spaced among thin, faintly eosinophilic collagenous strands. Cells and collagen are spaced either in a

loose or homogenous pattern. Blood vessel are seen. Nerve fibres seen with Bodian stain & Mast cells by Giemsa stain. Rarely mucoid degeneration of collagen are also seen.

Treatment:

Reassurance & Genetic counselling.

Psychotherapy, Speech therapy

Cutaneous Neurofibroma with CO2 laser.

Antiepileptics,& antihistamines:Ketotifen- 1-2 mg/kg

Farnesyl transferase inhibitor .

AIMS AND OBJECTIVES OF THE STUDY

To find out the

1. Overall frequency of skin tumours of head & neck reported in the out-patient department of dermatology, chengalpattu medical college , chengalpattu.
2. Age & sex predominance of the various tumours encountered.
3. Different clinical presentation such as morphology, site and association with other skin & systemic conditions.
4. Histopathological features of the various tumours encountered.

MATERIALS AND METHODS

100 patients presenting with different forms of cutaneous tumours of head & neck as their main complaints were selected for the study from the skin department of chengalpattu Government Hospital, chengalpattu during the one year period from October 2008 to September 2009 at random. The provisional diagnosis were mainly made by clinical presentations..

The age and sex of all the hundred cases along with their occupation were recorded .The duration of the skin lesions in all the patients was also noted. Specific and relevant histories were taken from certain cases with skin tumours and they included history of prolonged intake of any internal medication like inorganic arsenic containing preparation which may lead to basal cell epithelioma. Family history regarding the presence of tumours was also elicited and it was relevant in trichoepithelioma and steatocystoma multiplex. Menstrual, marital, parturition histories were taken in the female patients. History of medical and surgical intervention for the above complaints if any was also noted in all the 100 patients.

Thorough clinical examination of the skin lesions was carried out in all the cases with special reference to the site, number, size, shape, color, surface, borders, consistency, tenderness and compressibility of the lesions. Whether the lesions were grouped or discrete, sessile, or pedunculated or whether there were

any attachment to the underlying structures or the overlying skin were also observed.

Careful general and systemic examinations were carried out. Investigations like complete haemogram ,blood sugar, renal function test , VDRL ,HIV status and skin biopsy in the form of both excision and incision biopsy were carried out. The sections for histopathological examination were stained with haematoxylin and eosin and studied in both low and high power magnifications.

In selected cases, X-ray skull, Barium meal study, Barium enema, Upper GI –Endoscopy, ultrasonogram were carried out.

Most of the patients were treated surgically in the form of complete excision of the lesions. Few cases were treated with electrocautery & cryotherapy.

OBSERVATONS

Of 100 patients studied, 46 were males (46.66 %) and 54 were females (53.33 %). The distribution is given in Fig 2.

The age and sex distribution is depicted in Table – 1.

INCIDENCE OF VARIOUS TYPES OF SKIN TUMOURS

TABLE- 1

S.NO	Classification	NO. of cases	%
1	Tumours of surface epidermis	62	62
2	Tumours of epidermal appendages	21	21
3	Tumours of the fibrous tissues	5	5
4	Tumours of the vascular tissues	4	4
5	Tumours of the fatty, muscular and osseous tissue	-	-
6	Tumours of the neural tissue	8	8
7	Disorders of nevus cells and melonocytes	-	-
	TOTAL	**100**	**100%**

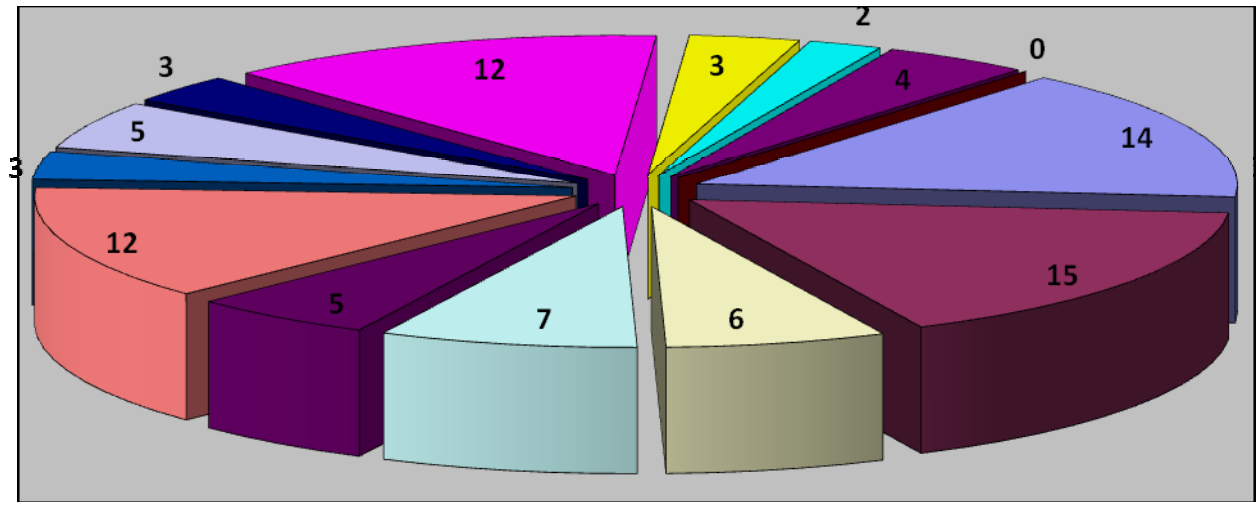
AGE AND SEX DISTRIBUTION OF 100 SKIN TUMOURS

TABLE-2

Age group (yrs)	**No. of cases**		**Total**	Percentage
	M	**F**		
Birth-1 year	-	-	-	
2-10	3	2	5	5
11-20	7	5	12	12
21-30	12	11	23	13
31-40	7	12	19	19
41-50	10	11	21	21
51-60	7	13	20	20
	46	54	100	100

DISTRBUTION OF TUMOURS AS IN 100 PATIENTS

FIGURE -1



Seborrhoeic keratosis = 14

Acrochordons =15

Epidermal nevus = 6

Basal cell carcinoma = 7

Squamous cell carcinoma = 5

Epidermal cysts =12

Trichoepithelioma =5

Nevus sebaceous =3

Syringoma = 12

Keloid =3

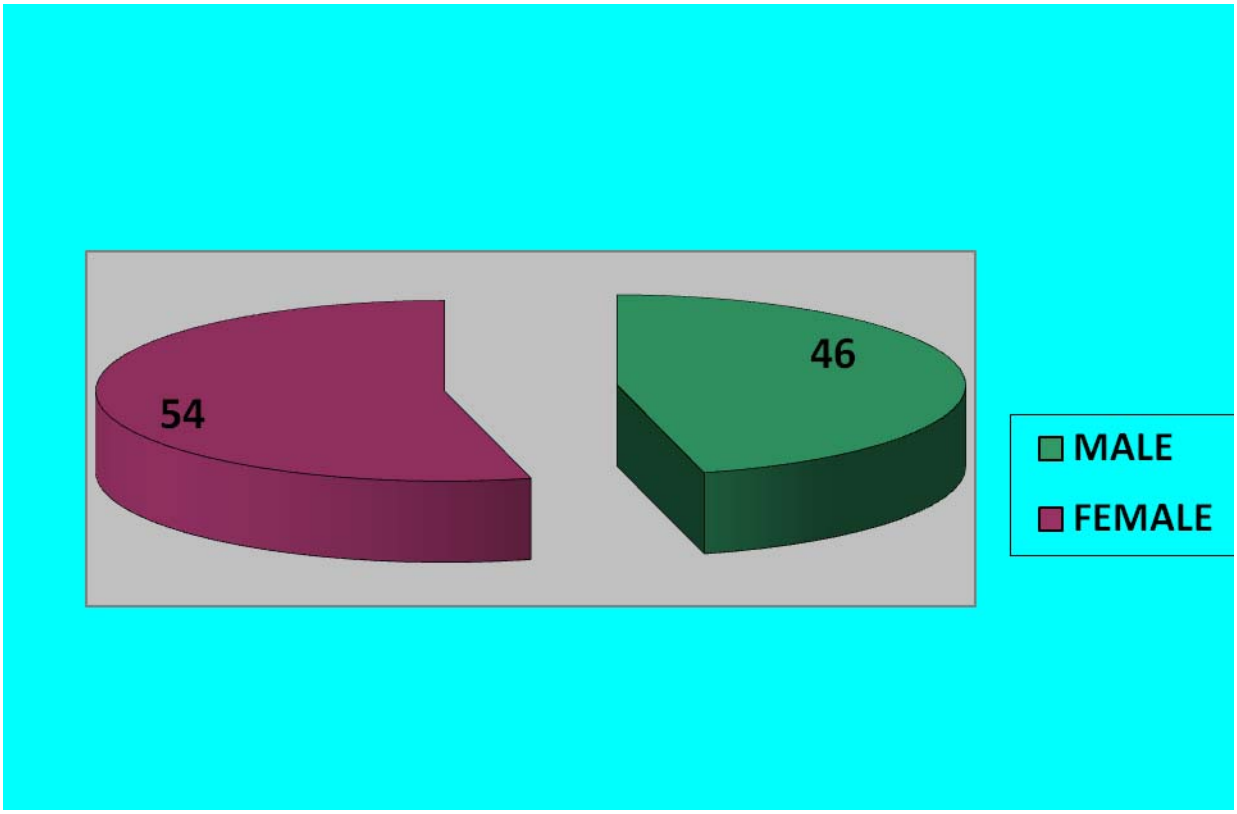
Angiofibroma =2

Pyogenic granuloma =4

Steatocystoma multiplex =3 **Neurofibroma =8**

MALE – FEMALE CASE DISTRIBUTION

FIGURE-2



1. SEBORRHOEIC KERATOSIS.

Among fourteen cases encountered ,8 were male patients (57%) and 6 female patients (43%).The age and sex distribution in these cases is given in Table- 3. Majority of the patients were in their sixth decade.

TABLE-3

AGE AND SEX DISRIBUTION OF 14 CASES OF SEBORRHOEIC KERATOSIS

Age group (yrs)	No. cases		Total	Percentage
	M	F		
10-20	-	-	-	-
21-30	-	-	-	-
31-40	-	-	-	-
41-50	4	2	6	43
51-60	4	4	8	57
	8	6	14	100

All the cases were asymptomatic except 3 males who complained of pruritis over the lesions. The lesions appeared as multiple verrucous pigmented papules and plaques of varying sizes covered by scales, stuck on to the skin surface, distributed mainly over the cheeks, temple region and fore head (Fig:3). All the three males who had pruritis had similar lesions over the neck, chest, back & upper limbs. They were submitted for investigations to exclude any internal malignancy and that included barium meal, upper GI –endoscopy, ultrasonography and occult blood which proved to be negative.

Excision biopsy of a single lesion was done and histopathology with haematoxylin & eosin stain showed the classical features of marked hyperkeratosis, irregular acanthosis and papillomatosis (fig:4) with the presence of pseudo and true horn cysts and basaloid cells.

2. ACROCHORDONS

Among fifteen cases encountered ,8 were male patients (53%) and 7 female patients (47%).The age and sex distribution in these cases is given in Table-4. Majority of the patients were in their fifth decade.

TABLE-4

AGE AND SEX DISRIBUTION OF 14 CASES OF ACROCHORDONS

Age group (yrs)	No. cases		Total	Percentage
	M	F		
10-20	-	-	-	-
21-30	1	-	1	7
31-40	3	1	4	27
41-50	4	4	8	53
51-60	-	2	2	13
	8	7	15	100

All the cases were asymptomatic.The lesions appeared as multiple,small, skin coloured soft to firm papules of varying sizes with few pedunculated surface, distributed mainly over the face, neck, axilla.(Fig:5). All were submitted for investigations to look for diabetes mellitus and intestinal polyposis .

Excision biopsy was done & histopathology with haematoxylin & eosin stain showed the classical features of flattened epidermis overlying loosely arranged collagen fibers with in the dermis (Fig:6).

3. EPIDERMAL NEVUS.

Among 6 cases of epidermal nevus with one case of linear epidermal nevus were observed during in this study ,2 were male patients(33 %) and 4 were female patients (77 %). Among them the lesions were present since birth in 2 female as in the Table- 5.

TABLE-5

AGE AND SEX DISTRIBUTION OF 6 CASES OF EPIDERMAL NEVUS

Age group (yrs)	No. cases		Total	Percentage
	M	F		
birth-10	1	1	2	33
11-20	1	2	3	50
21-30	-	1	1	17
31-40	-	-	-	-
41-50	-	-	-	-
	2	4	6	100

Clinically, the lesions were seen as closely set, keratotic, pigmented verrucous papules in a linear fashion of size 2x1 cms distributed unilaterally over the left side neck (fig.7).Incision biopsy was done and histopathology with haematoxylin & eosin stain showed marked hyperkeratosis, irregular acanthosis with elongation of the rete ridges and papillomatosis. (fig:8)

4. BASAL CELL EPITHELIOMA (BCE)

Seven cases were encountered during the period of study, of which 2 were males(29%) and 5 were females (71%). The age and sex distribution in these patients is depicted in the Table-6 .Of these 7 patients ,4 had nodulo-ulcerative and 3 had pigmented type (fig.9).All the patients had the lesions of basal cell carcinoma in the face mainly over the cheeks, nose, pre-auricular region &ears.

TABLE-6

AGE AND SEX DISTRIBUTION OF 7 CASES OF BASAL CELL CARCINOMA

Age group (yrs)	No. cases		Total	Percentage
	M	F		
10-20	-	-	-	-
21-30	-	-	-	-
31-40	-	-	-	-
41-50	1	2	3	43
51-60	1	3	4	57
	2	5	7	100

Biopsy was done and histopathology with haematoxylin & eosin stain showed tumour masses of basaloid cells and horn cysts with peripheral palisading and clefts around the islands of basalioma cells with peritumoural lacuna(fig.10).

All the patients were advised surgical intervention with complete excision followed by cosmetic reconstruction in the plastic surgery department. The patients were followed up regularly.

5. SQUAMOUS CELL CARCINOMA

Two male and three female cases were reported during the study. Sites involved were lips, oral mucosa,around the eye and one female case was associated with xeroderma pigmentosum (fig.11 &12). Incision biopsy of the lesion was done and histopathology with haematoxylin & eosin stain showed massive hyperplasia and horn pearls (fig.13 and 14). .

6. EPIDERMAL CYST

Epidermal cysts were encountered in 8 male patients (66 %) and 4 female patients (34%), as in Table-7

TABLE-7

AGE AND SEX DISTRIBUTION OF 12 CASES OF EPIDERMAL CYSTS

Age group (yrs)	No. cases		Total	Percentage
	M	F		
10-20	3	0	3	25
21-30	5	0	5	42
31-40	-	3	3	25
41-50	0	1	1	8
51-60	-	-	-	-
	8	4	12	100

The lesions were asymptomatic in all the cases. These lesions appeared as elevated, rounded, firm, intradermal cystic nodules of 1 to 2 cms in diameter with stretched and shiny surface, found mainly over the scalp and neck with punctum in 6 cases (fig:15) & the number of cysts ranged from 1 to 3 and were not attached to the underlying structures.

Excision biopsy was done and histopathology with haematoxylin & eosin stain showed the cyst in the dermis, with cyst wall showing all the layers of true epidermis with lamellated keratin inside the cavity (fig.16).

7. STEATOCYSTOMA MULTIPLEX

TABLE-8

AGE AND SEX DISTRIBUTION OF 5 CASES OF STEATOCYSTOMA MULTIPLEX

Age group (yrs)	No. cases		Total	Percentage
	M	F		
10-20	-	2	2	40
21-30	1	-	1	20
31-40	1	-	1	20
41-50	1	-	1	20
51-60	-	-	-	-
	3	2	5	100

3 males(60%) & 2 female patients (40%) patients with steatocystoma multiplex were seen during the study period as in the table -8. Clinically, the lesions appeared as numerous, small, smooth, round, soft to firm, yellowish cystic nodules of size varying from 1 to 3 cms in diameter, adherent to the overlying skin and were found predominantly over the chest and neck (fig:17). The lesions, to start with were single and in course of few months, new lesions appeared. On puncturing the cysts, oily fluid was extruded in all the three cases. There were no other associated skin lesions in both the patient.

Excision biopsy of single lesion was done and histopathology with hematoxylin & eosin stain showed intricately folded cyst (fig:18) in the dermis

and numerous sebaceous gland lobules attached to both sides of the cyst wall & contents of the cyst were eosinophilic.

8. TRICHOEPITHELIOMA

Five cases of trichoepithelioma were encountered. All were adults. Of whom 2 were male(40%) and 3 female(60%). The lesions were seen as raised, rounded, skin colored, firm papules and nodules of size varying from 2 to 8 mms in diameter, with few showing telengiectasia over the surface. They were located on the face especially over the paranasal areas,forehead and cheeks (fig:19).One of these female patients underwent plastic surgical repair with good cosmetically results. All the patients reported only for cosmetic disability.

Excision biopsy of a lesion was done and histopathology with haematoxylin & eosin stain showed multiple horn cysts and islands of basoloid or basalioma cells as in the (Fig:20), which lack high grade atypia and mitotic activity.Cosmetic surgical repair is the best mode of therapy.

9. NEVUS SEBACEOUS OF JADASSOHN

Four adult patients reported with these lesions of whom, 3 were males and 1 female. The lesions in all the 4 patients were present since birth but as smaller and flat ones. The patients also gave history of increase in the size of the lesions with verrucous excrescences since adolescence. Clinically they were seen as circumscribed, hyperkeratotic, thickened, verrucous, plaque of size varying from

2 to 4 cms in diameter and were distributed over the parietal and fronto-pareital region of the scalp (fig:21).No other associated eye, skeletal and nervous system involvement.

Excision biopsy was done and histopathology with haematoxylin& eosin staining showed marked hyperkeratosis, irregular acanthosis, papillomatosis, numerous mature sebaceous glands seen in the upper dermis(fig:22).

The lesions in all the cases were excised with good cosmetic results.

10. SYRINGOMA

TABLE-9

AGE AND SEX DISTRIBUTION OF 12 CASES OF SYRINGOMA

Age group (yrs)	No. cases		Total	Percentage
	M	F		
10-20	-	1	1	7
21-30	-	3	3	20
31-40	2	4	6	60
41-50	-	1	1	7
51-60	-	1	1	7
	2	10	12	100

12 cases of syringoma were observed. All were adults and among them only two were male (17 %). The age and sex distribution of syringoma is shown in Table – 9 The syringoma lesions were skin colored, firm, elevated, flat-topped papules of size ranging from 1 to 5 mms in diameter with angular outline and were

distributed mainly over the face (fig.23) especially below the lower eyelids. In one adult female, the lesions were also seen in the neck and the vulvar region, in addition to face. All the patients sought medical help only for cosmetic disability.

Excision biopsy of a single lesion was done and histopathology with haematoxylin & eosin stain showed numerous small cystic ductal structures lined by 2 rows of flattened epithelial cells, embedded in dermal fibrous stroma. Some of the ducts also had tail like projections of epithelial stands into the stroma giving a tadpole appearance (Fig :24)

11. KELOID

Three cases of keloid were encountered. All were adults. Of whom 1 was a male(33%) and 2 females(67%). The lesions appeared as firm, mildly tender, pruritic, bosselated, well-demarcated tumours seen on earlobes and the back of the neck(Fig:25).One of these female patients underwent plastic surgical repair with good cosmetic results. But the lesion recurred after 6 months.

Excision biopsy of a lesion was done and histopathology with haematoxylin & eosin stain showed nodular mass of vascular proliferation along with fibroblasts containing greatly thickened, compact, glassy, eosinophilic collagen bundles (Fig:26). Cosmetic surgical repair is the best mode of therapy.

12. ANGIOFIBROMA

Only one male case and one female case of adolescent age group with Tuberous sclerosis were reported in our study. The lesions appeared as numerous small, red, smooth dome shaped papules 1-4 mm in size ,occurs in a symmetric distribution in the nose, nasolabial folds, on the cheeks, and on the chin(Fig:27) Both of them had angiofibroma with the age of onset around 10 years of age along with the history of seizures. Excision biopsy of a lesion was done and histopathology with haematoxylin & eosin stain showed the dermal fibrosis, vascular proliferation with the perivascular proliferation of fibroblasts(Fig:28).

13. PYOGENIC GRANULOMA

One male and three female cases were reported during the study. All were young adults & they had single lesion. Sites involved were lower lips and forehead (fig.29). All were screened for HIV. Excision biopsy of the lesion was done and histopathology with haematoxylin & eosin stain showed the polypoid mass of angiomatous tissue protruding above the surrounding skin & often constricted at its base by a collarette of acanthotic epidermis (fig:30)

All the lesions were cauterised with adequate hemostasis.

13. NEUROFIBROMA

Neurofibroma were encountered in 4 male patients (50%) and 4 female patients (50%), as in Table-10 with the age of onset around the first decade.

TABLE-10

AGE AND SEX DISTRIBUTION OF 8 CASE OF NEUROFIBROMA

Age group (yrs)	No. cases		Total	Percentage
	M	F		
Birth - 10	-	1	1	13
10-20	1	0	1	13
21-30	3	2	5	63
31-40	-	-	-	-
41-50	-	-	-	-
51-60	-	1	1	13
	4	4	8	100

The lesions were asymptomatic in all the cases except in 2 cases who had pruritus. These lesions appeared as multiple soft to firm cutaneous papules and nodules (Fig: 31) with numerous CALM, axillary freckling ,lisch nodules .

Excision biopsy was done and histopathology with haematoxylin & eosin stain showed well cimcumscribed non- encapsulated tumour of dermis composed of thin spindle shaped cells with elongated wavy nuclei, regularly spaced among thin, faintly eosinophilic collagenous strands. (Fig :32).

Genetic counseling were given to all the patients .

DISCUSSION

SEBORRHOEIC KERATOSIS

Seborrhoeic wart was the second common tumour (14 %) in our study.In this study, all the cases reported were above forty years of age as in the study of Yeatman et al . [5] According to Mackie et al, the males & females are equally affected. [10] In this study, male outnumbered the female. Cosmetic disfigurement was the main complaint in all the cases. The lesions of seborrhoeic wart were classical in their morphology, distribution and histopathology in all the cases. Electrocautery was considered as the choice of therapy.

ACROCHORDONS

It is the most common tumour (15%) in our study . Study of Thappa DM et al with the study group of 35 patients , 62.8% (22 patients) had DM. [18] In our study, among 15 patients, 6 (40%) had diabetes mellitus & none of them had intestinal polyposis.

EPIDERMAL NEVUS

6 % of the study cases (100) had epidermal nevus. The age of occurrence was at birth or little later as seen in these patients. These lesions were classical in their morphology, distribution and histology in all the patients. A study conducted by Vidaurida la Cruz H et al, reported 7.9 % of epidermal nevus

syndrome in 443 patients with epidermal nevi. [28] .But in this study no case of epidermal nevus syndrome was reported out of 6 cases of epidermal nevus.

No patient showed systemic associations like seizures and visual disturbances. The adult patients were encouraged to undergo surgical excision, which is the best mode of therapy.

BASAL CELL EPITHELIOMA (BCE)

Seven cases of basal cell carcinoma among 100 patients were reported in our study . Among them 2 had severe pain & others had come for their cosmetic disability. Prolonged exposure to sunlight could be the cause for the tumour in 2 of the patients who were manual labourers, with prolonged sun exposure.

Nodulo-ulcerative basal cell carcinoma was the common type seen in all the cases in this study as the study conducted by Christensen LJ et al. [37] The adult onset, clinical features and histopathology coincided with the literature reports. None of the other types of basal cell carcinoma like superficial, fibrosing, pigmented, fibroepithelioma types were encountered.

A good response was observed in all these patients who were motivated to undergo surgical excision considering the local invasive nature of the tumour.

SQUAMOUS CELL CARCINOMA

In a study conducted by Nuzhat Yasmmen et al, listed 30 squamous cell carcinoma among the 75 cases studied (40 %). [41]But in our study,among 100

cases, squamous cell carcinoma were in seventh order (5 %). One of case was associated with xeroderma pigmentosum as shown in the (Fig:12). Clinically all the cases presented as ulceroproliferative growth and histopathology were consistent with squamous cell carcinoma.

EPIDERMAL CYST

Epidermal cysts were 12 % in our study cases (100). In this study punctum was seen in 50% of the cases which correlates well with ChandrasekaranV et al study .[46] All these patients were asymptomatic excepting the cosmetic disability. The lesions were classical in their morphology and histology. Cosmetically a good response was observed in all the cases following complete excision of the cysts.

STEATOCYSTOMA MULTIPLEX

The incidence of steatocystoma multiplex was 3% in our study cases(100).Cha .S. et al, reported most of the cases were sporadic.[54] But in our study, 2 patients had family history. The lesions were classical in their morphology, distribution, and histopathology in all the cases. Puncturing the lesions extruded oily fluid which helped to diagnose the condition clinically. There were no other associated skin lesions in these cases.[10]

TRICHOEPITHELIOMA

Five cases (5 %) were reported in our study (100 cases). Cosmetically trichoepithelioma were more disfiguring than syringoma. Ziprkowski et al [58] reported that 50 % of the patients with trichoepithelioma had family history but no familial occurrences have been reported in our study. The lesions in all the cases were classical in their morphology, distribution and histopathology. Neither the special types such as giant or desmoplastic nor ulcerative and malignant transformation were encountered .

NEVUS SEBACEOUS

The incidence of nevus sebaceous was 4 % in our study (100cases). Chang Geng et al, reported 83% of the cases among 104 cases developed before 10 years in contrast to all the cases presented at birth in this study.[61] The lesions were classical in their morphology, distribution and histology in all the cases. None of the patients had any systemic associations like skeletal or neurological defects as a part of neurocutaneous syndrome or malignant transformation in the lesions was noted.

Cosmetically acceptable surgical removal was carried out in all the cases. In general, all the patients with these lesions are better encouraged to undergo wide excision of these lesions, even at the early age to prevent the rapid progression of lesions at puberty and possible malignant transformation.

SYRINGOMA

Incidence of the cases (12 %) in the study (60 cases) were having syringoma. Two of these patients(17%) had familial predisposition & most of the patients in our study were in the adult age group and were females (83 %). All were asymptomatic & no other special types such as eruptive or chondroid syringoma were encountered in our study which is in contrast to Patrizi et al study.[64] The lesions were classical in their morphology, distribution and histology in all the 12 cases.

KELOID

The incidence of keloid was 3% in our study cases(100). Similar to Bayat et al study[73], females outnumbered the males in our study and one female case had positive family history . Two of the cases developed keloid following six months after ear piercing & one patient underwent surgical excision but recurred after six months. The lesions were classical in their morphology, distribution and histology in all the 3 cases.

ANGIOFIBROMA

2 % of the study cases (100) had angiofibroma with shagreen patches and periungual fibromas. Both of them had history of seizures. Histopathological features were characteristic and consistent with the literature reports.

PYOGENIC GRANULOMA

4 % of the study cases (100) had pyogenic granuloma . In our study , all the cases were healthy and no pregnancy was reported. The age of occurrence were in third and fourth decade in all the cases which is similar to Hessa Al Wayli et al study. [90] These lesions were classical in their morphology, distribution and histology in all the patients.

NEUROFIBROMA

8 % of the study cases (100) had neurofibroma with equal male & female ratio. All the patients were asymptomatic except the two patients who had pruritus. One of the male patient had scoliois. None of the patients had systemic involvement. These lesions were classical in their morphology, distribution and histology in all the patients.

CONCLUSIONS

A study of clinicopathological features of tumours of head & neck was done on 100 patients at random over a period of one year from October 2008 to September 2009 attending the outpatient department of skin & STD, Chengalpattu Medical College, Chengalpattu.

1. The commonest skin tumours recorded were surface tumors of epidermis(62%) followed by tumours of epidermal appendages (21%), tumours of neural tissue(8%), other tumours constitutes(9%).

2. Majority of the patients reported with the skin tumours of head and Neck were adults . The tumours such as epidermal nevus, nevus sebaceous, neurofibroma occur in childhood (23 %) and these need awareness among the parents to bring the children at an early age.

3. The highest age incidence observed in this study was in the fifth decade (21%) followed by sixth decade (20%).

4. The reporting of the skin tumours was more by the females (54 %) compared to males (46 %) probably because of their cosmetic awareness.

5. The commonest tumour encountered in this study was Acrochordons (15%) with classical histopathology.

6. The second common tumour was seborrheoic keratosis (14%). All the patients had their lesions in the sun exposed areas like face & neck except one male & one female who had multiple lesions all over the body and having classical histopathology.

7. Epidermal cyst and syringoma(12 %) were the third most common tumour encountered in this study and with classical histopathology.

8. Neurofibroma (8%) , Basal cell carcinoma (7%) , Epidermal nevus (6%), squamous cell carcinoma(5%), trichoepithelioma (5 %), pyogenic granuloma(4%), nevus sebaceous (3%) , steatocystoma multiplex(3%), keloid(3%), angiofibroma(2%) were also encountered in our study with classical clinical and histopathological findings.

9. Rare case like xeroderma pigmentosum with squamous cell carcinoma was encountered in a female child.

10. In this study, among 15 patients with acrochordons , 6 (40%) had diabetes mellitus

11. Familial occurrence were noted in 2 cases of syringoma, 2 cases of steatocystoma multiplex and one case of keloid.

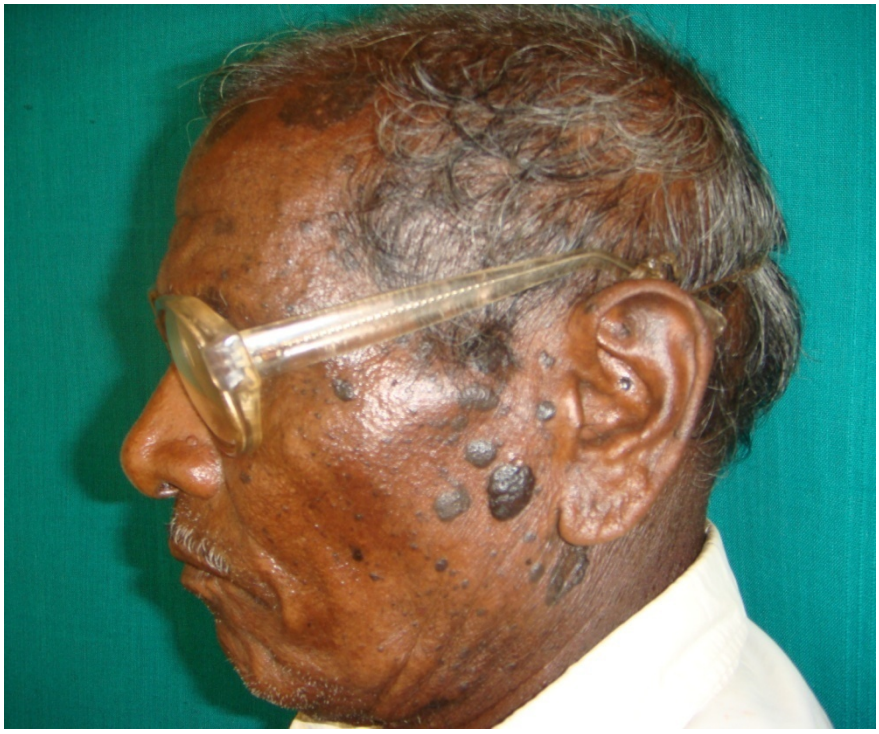


Fig:3 – Seborrhoeic keratosis over the face

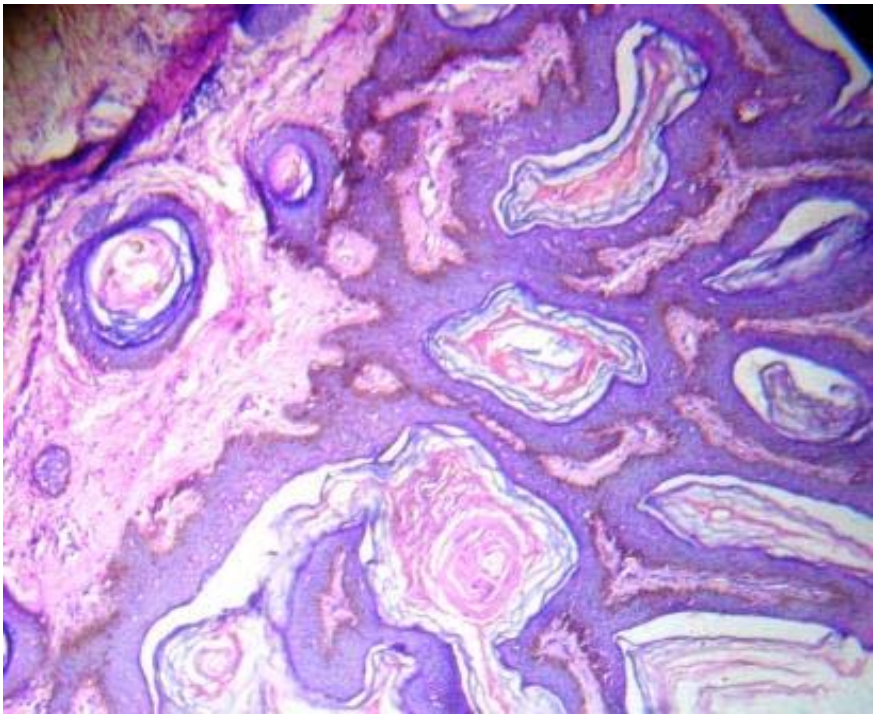


Fig: 4 – Histopathology of seborrhoeic keratosis showing horn cysts.

Fig:5 –Multiple skin tags (both sessile & pedunclated) over the neck.

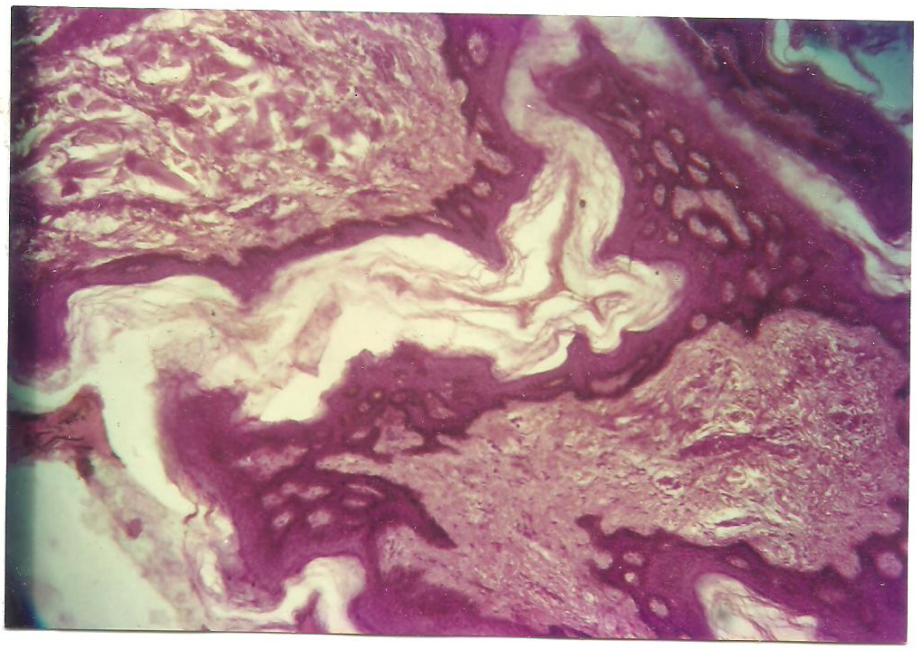


Fig:6 -Histopathology of Acrochordons with proliferation of fibrocollagenous tissue.

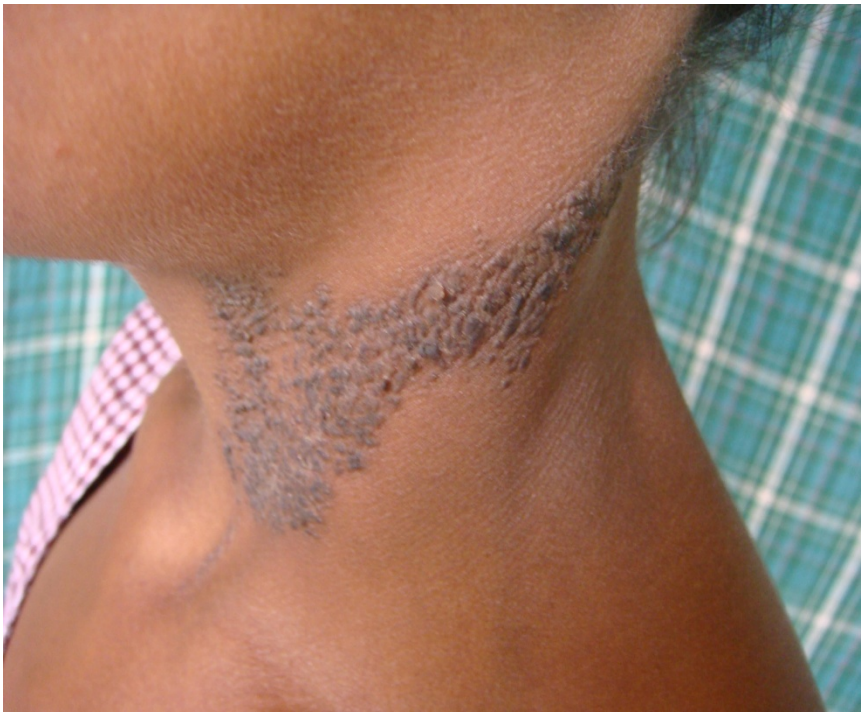


Fig: 7 –Epidermal nevus over the side of neck

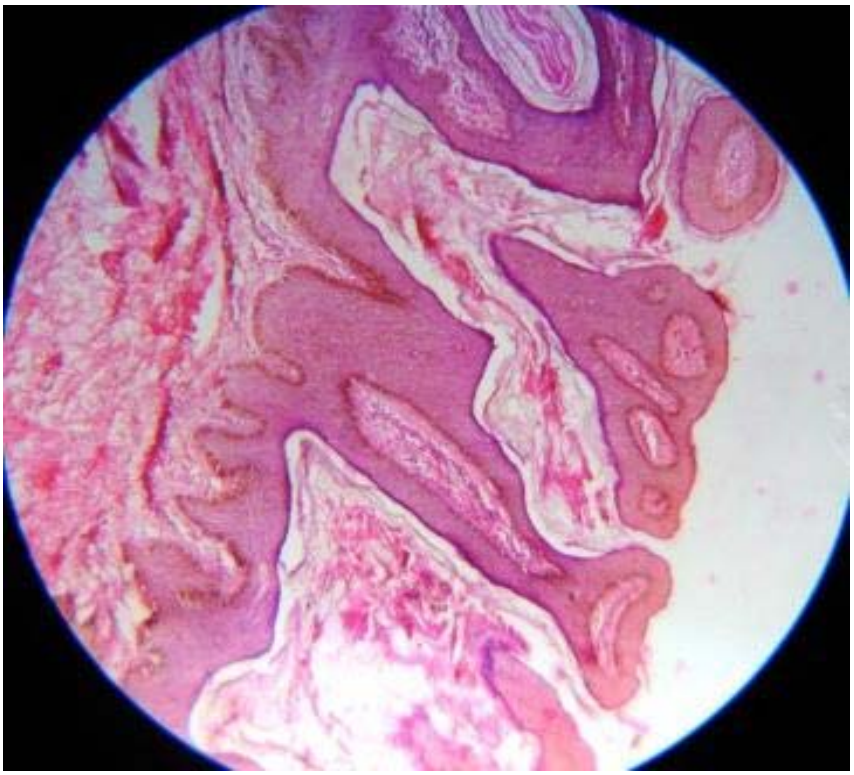


Fig:8- Histopathology of epidermal nevus showing hyperkeratosis, acanthosis and papillomatosis.

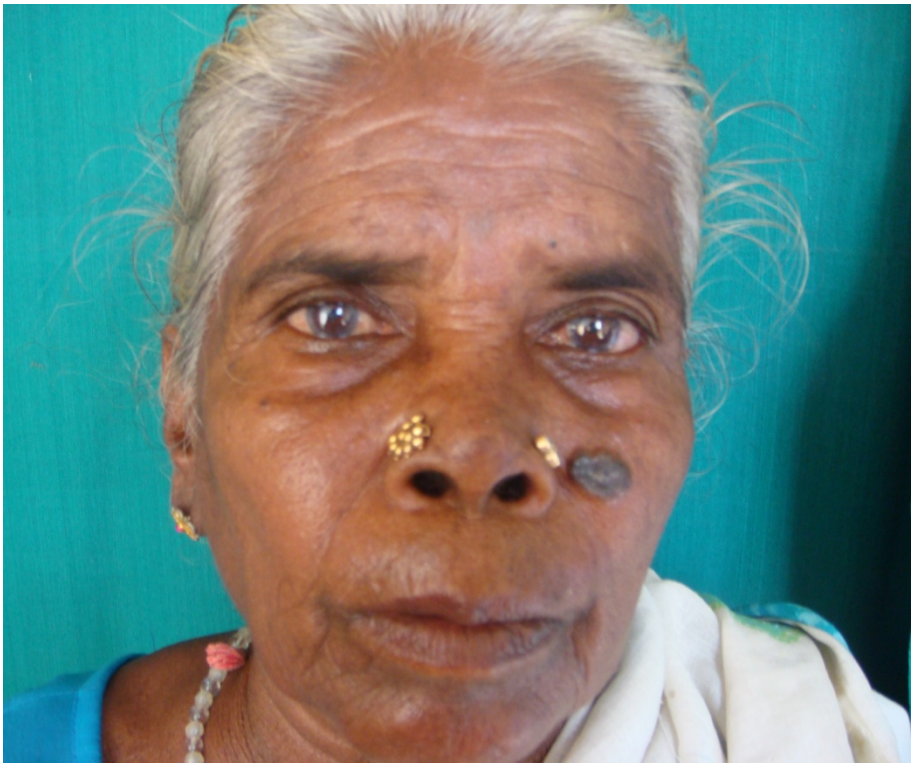


Fig:9 – Noduloulcerative basal cell carcinoma over the face.

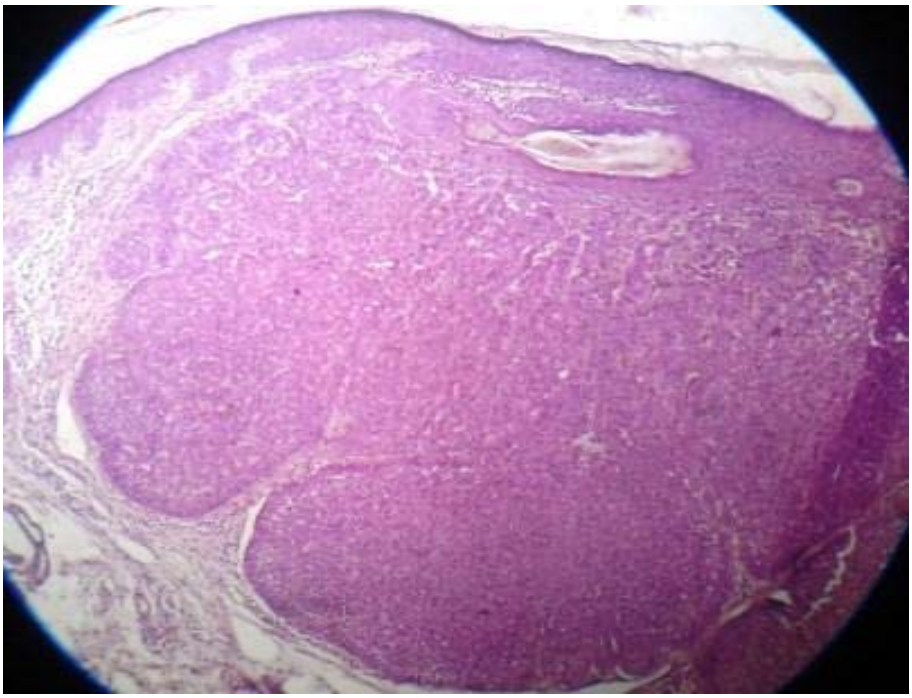


Fig:10 – Histopathology of basal cell carcinoma showing islands of tumour cells with peritumoural lacuna.

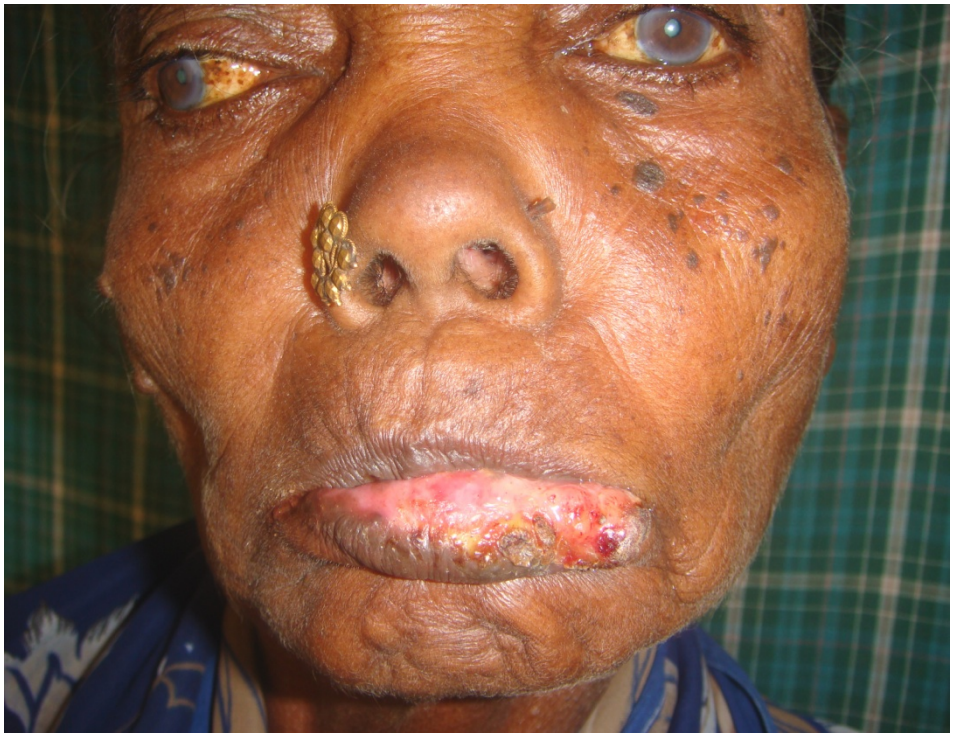


Fig:11- Squamous cell carcinoma in the lower lip.

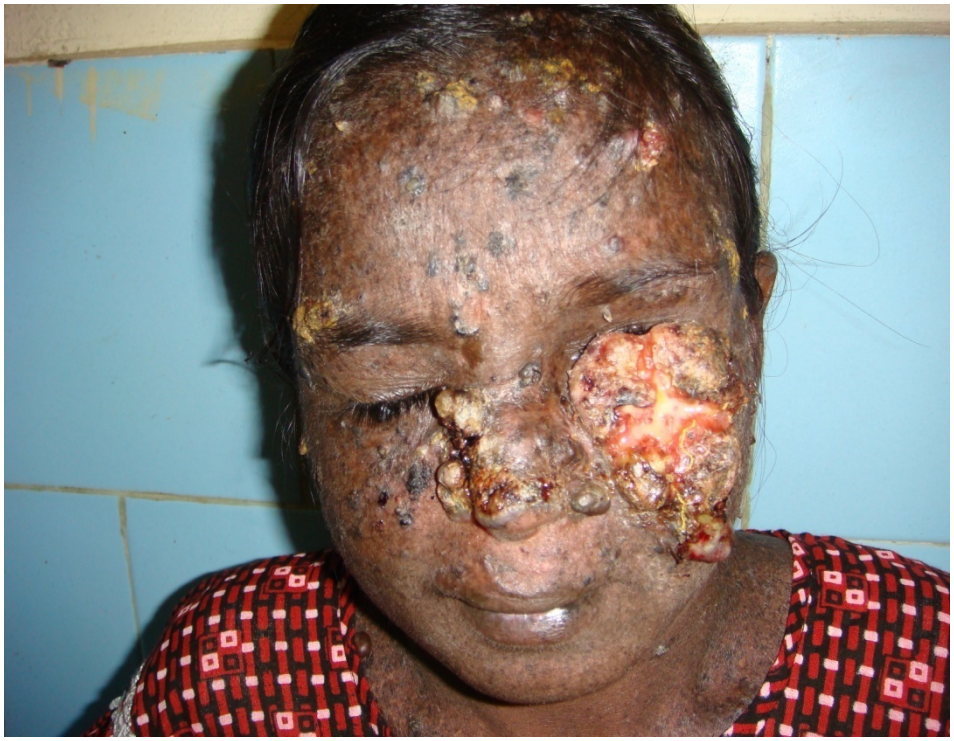


Fig:12– Xeroderma pigmentosum with squamous cell carcinoma over the face

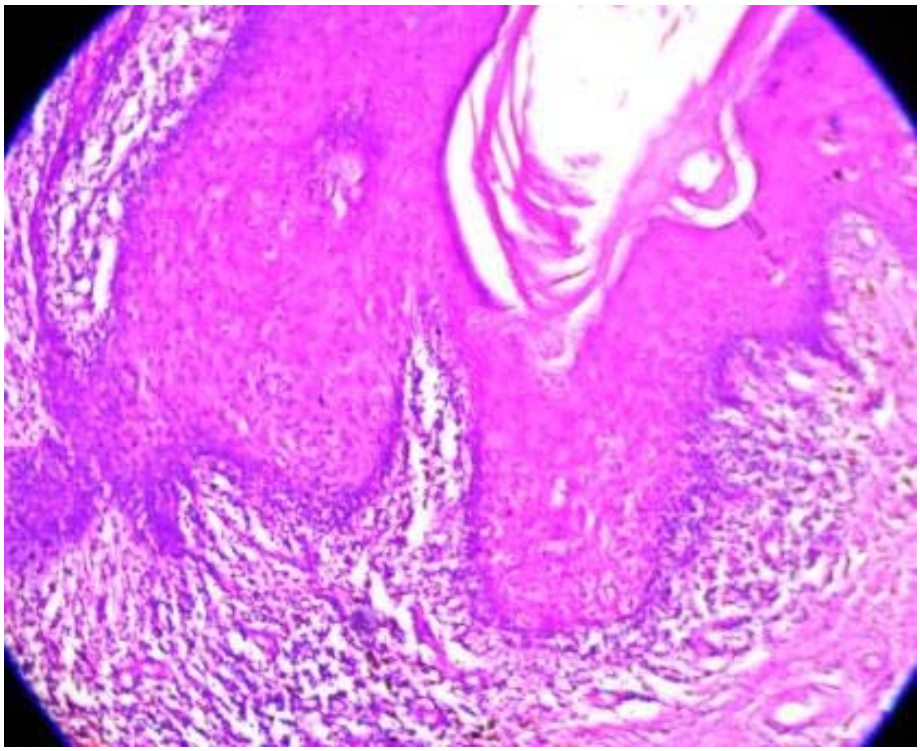


Fig:13 –Histopathology of squamous cell carcinoma shows massive hyperplasia.

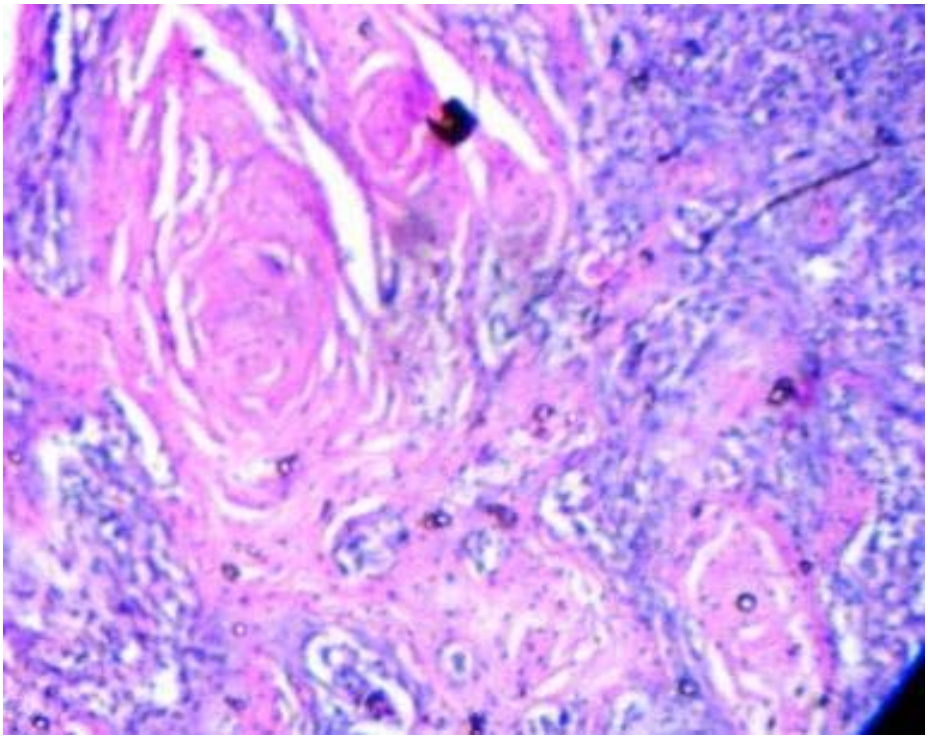


Fig:14–Histopathology of squamous cell carcinoma shows horn pearls.

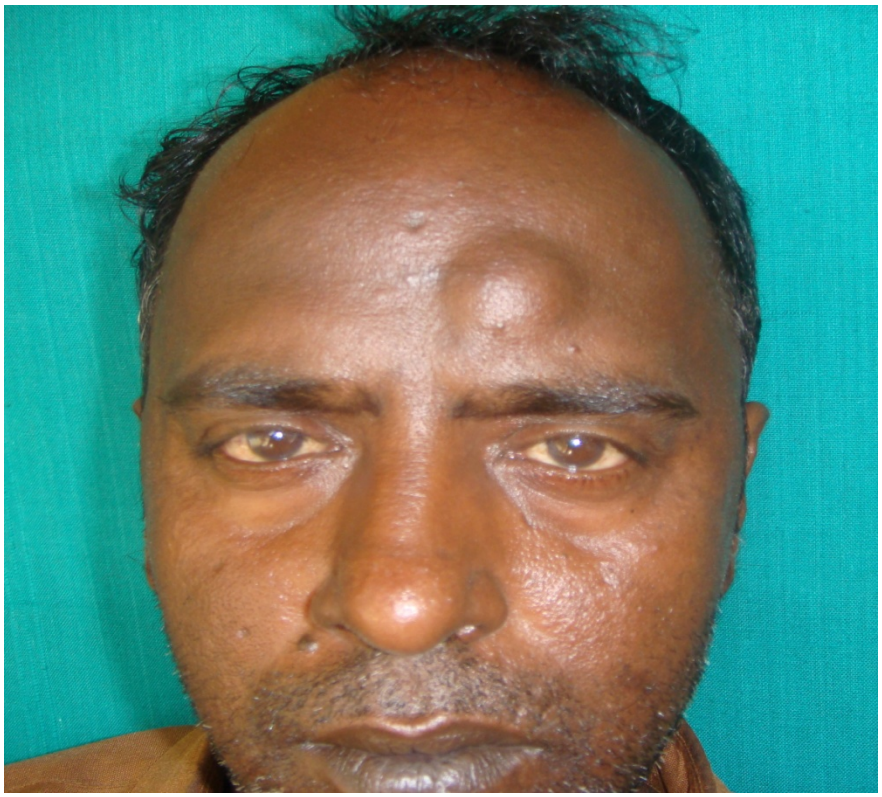


Fig:15 – Epidermal cyst over the scalp.

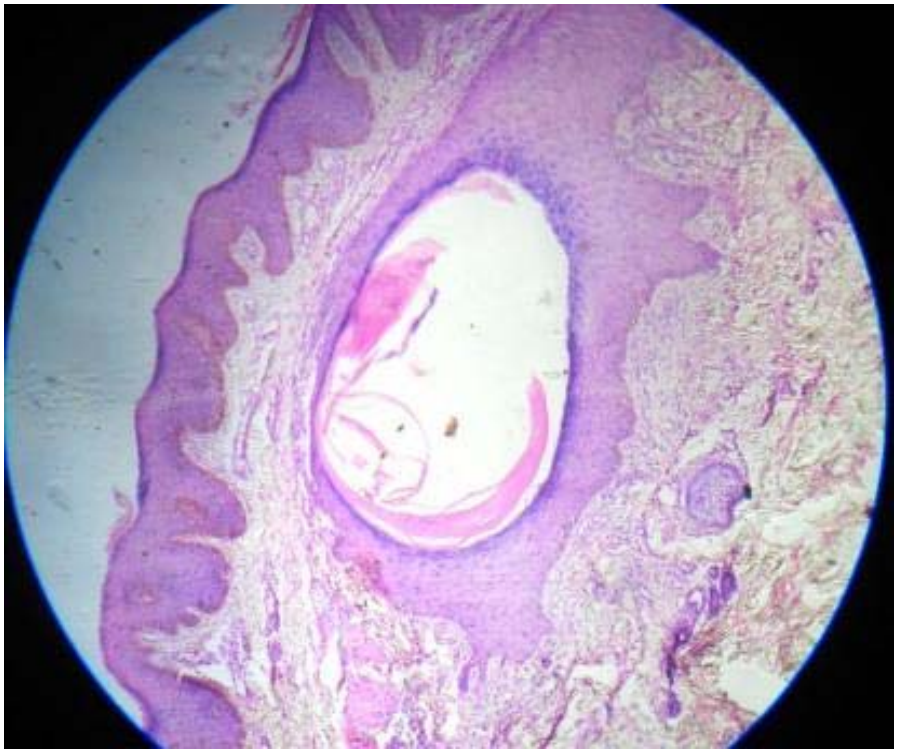


Fig: 16- Histopathology of epidermal cyst with keratin in the cavity.

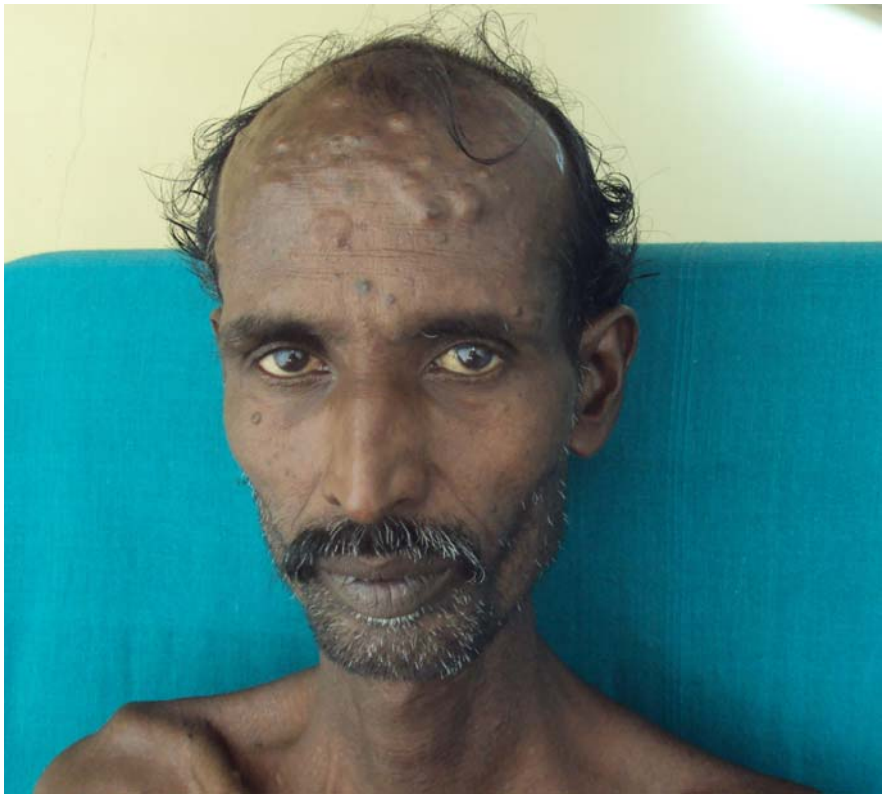


Fig:17– Steatocystoma multiplex over the neck and chest.

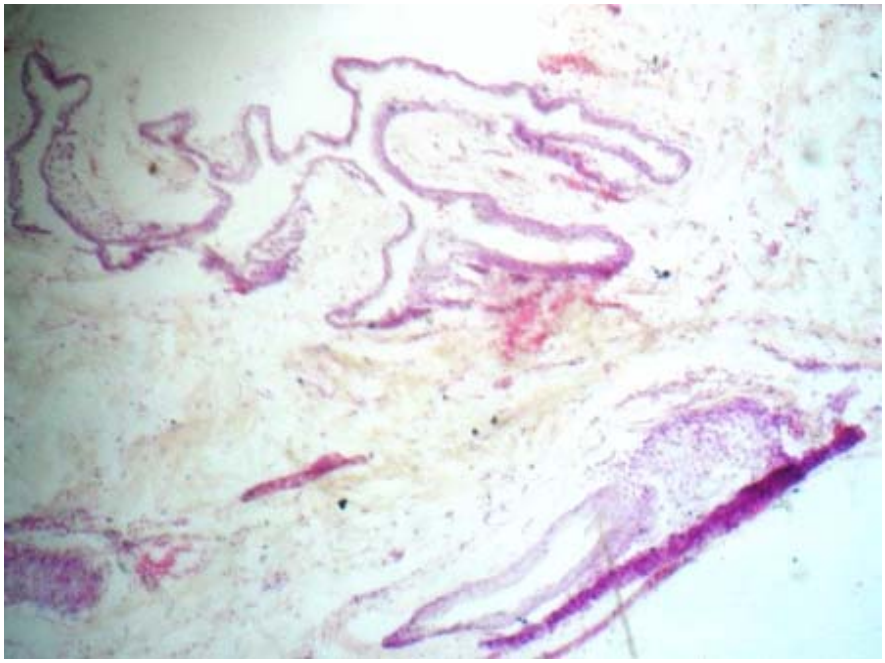


Fig:18 – Histopathology of the steatocystoma multiplex showing intricately folded cyst

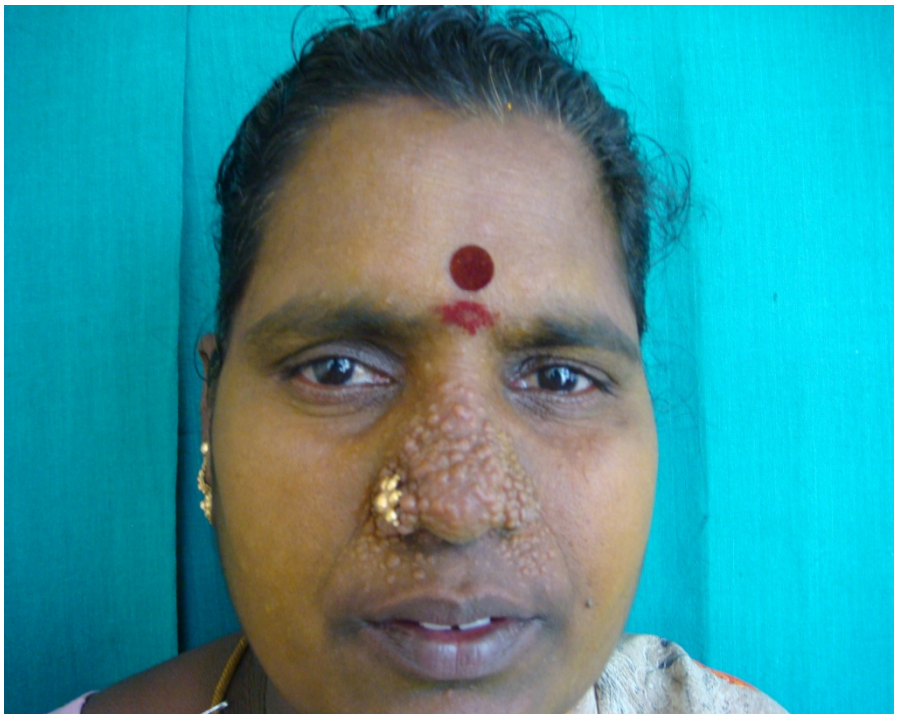


Fig:19 – Multiple trichoepithelioma of the face.

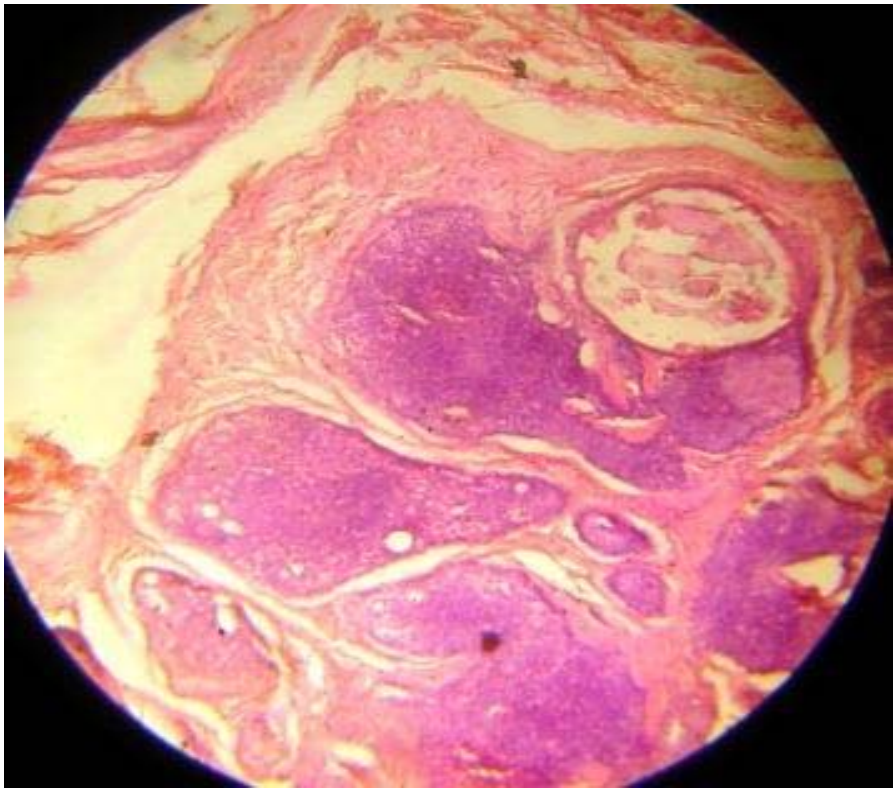


Fig:20 -Histopathology of trichoepithelioma shows basaloid cells and horn cysts.

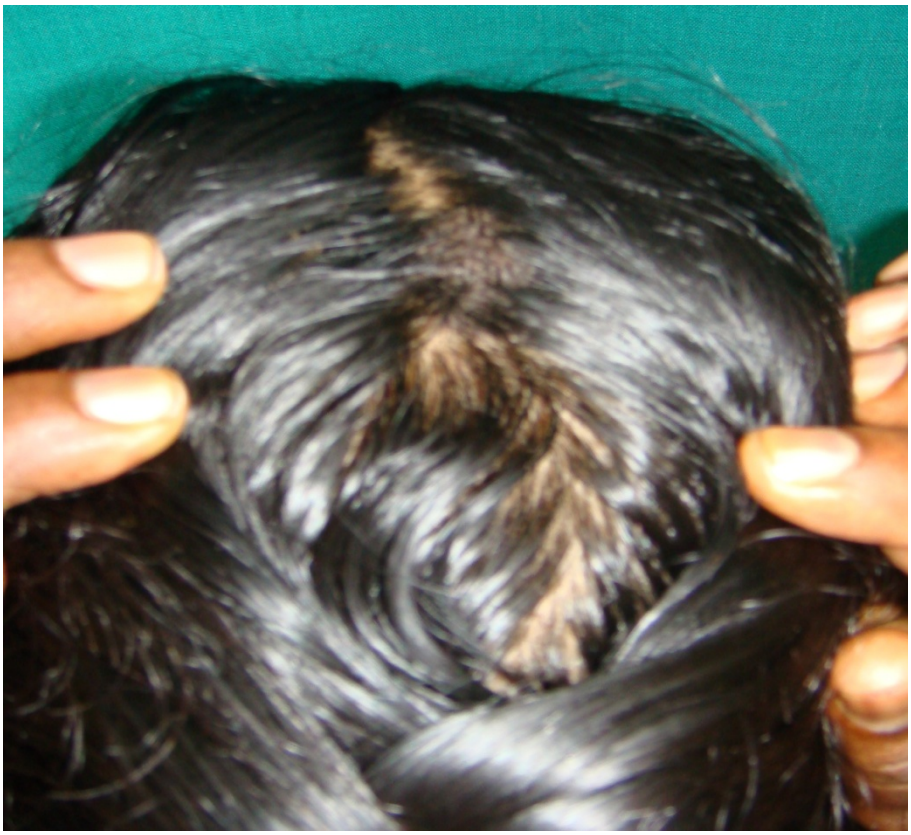


Fig:21– Nevus sebaceous of the scalp.

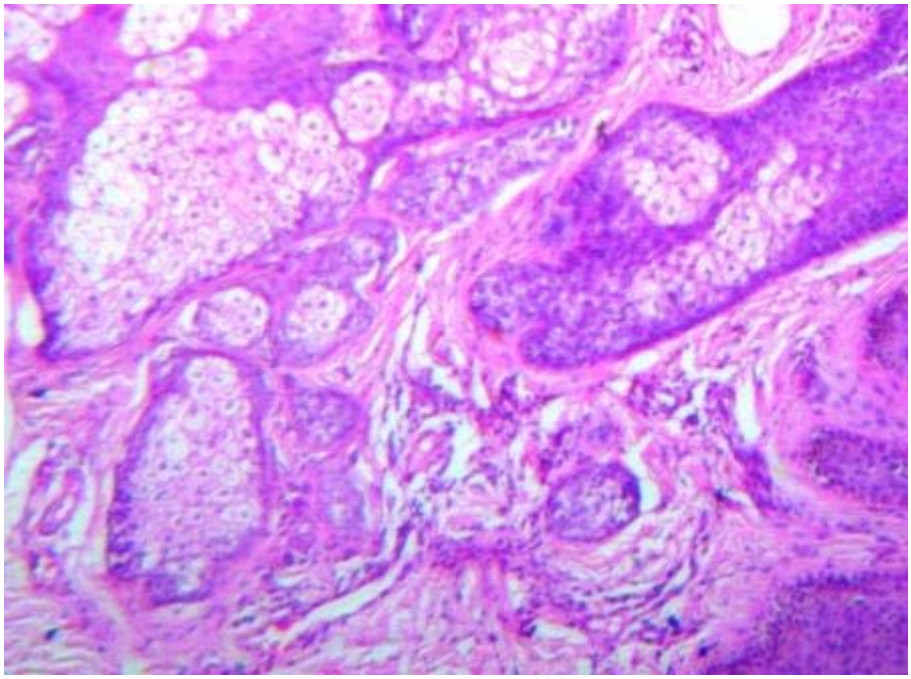


Fig:22 –Histopathology of nevus sebaceous shows proliferation of mature sebaceous glands.

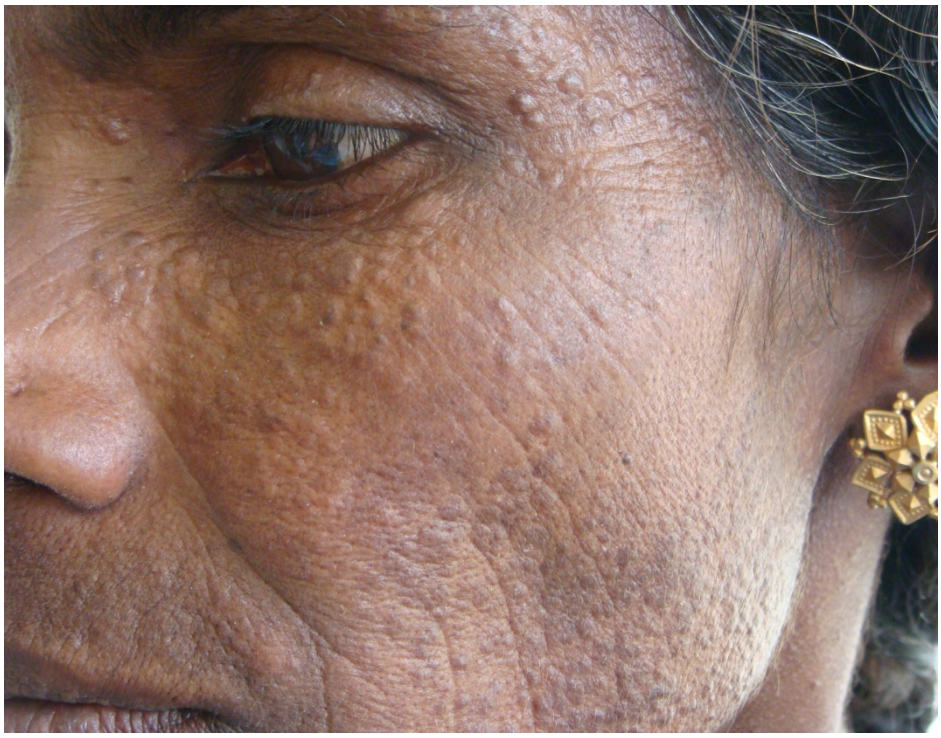


Fig:23 – Syringoma of the infra orbital region.

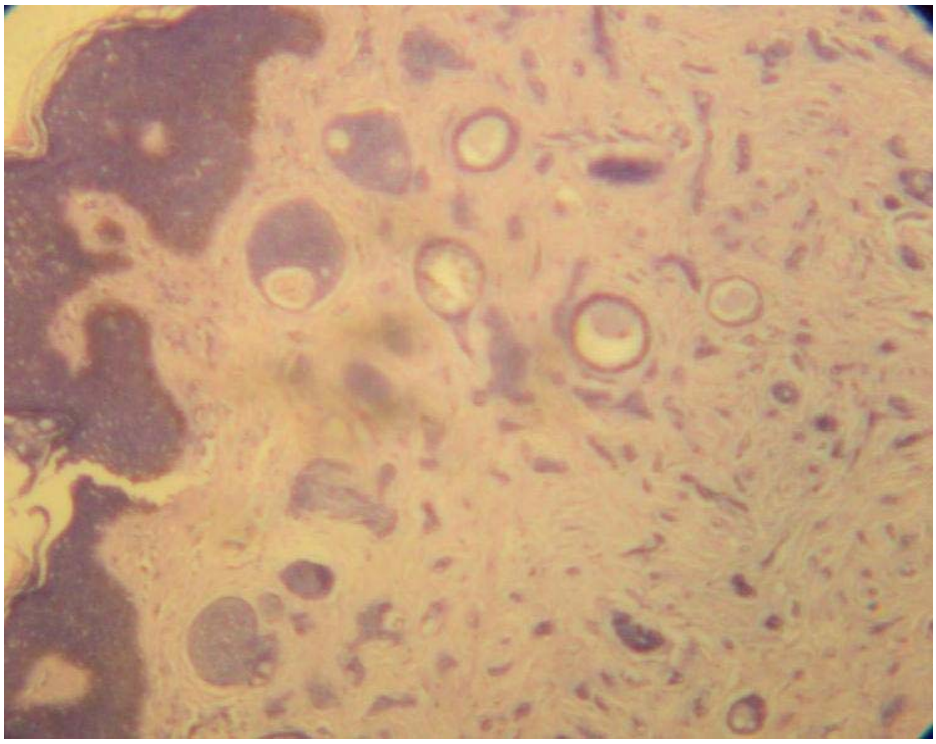


Fig:24 – Histopathology of syringoma shows numerous cystic ductal structures in the stroma. Some of the ducts show tadpole appearance.

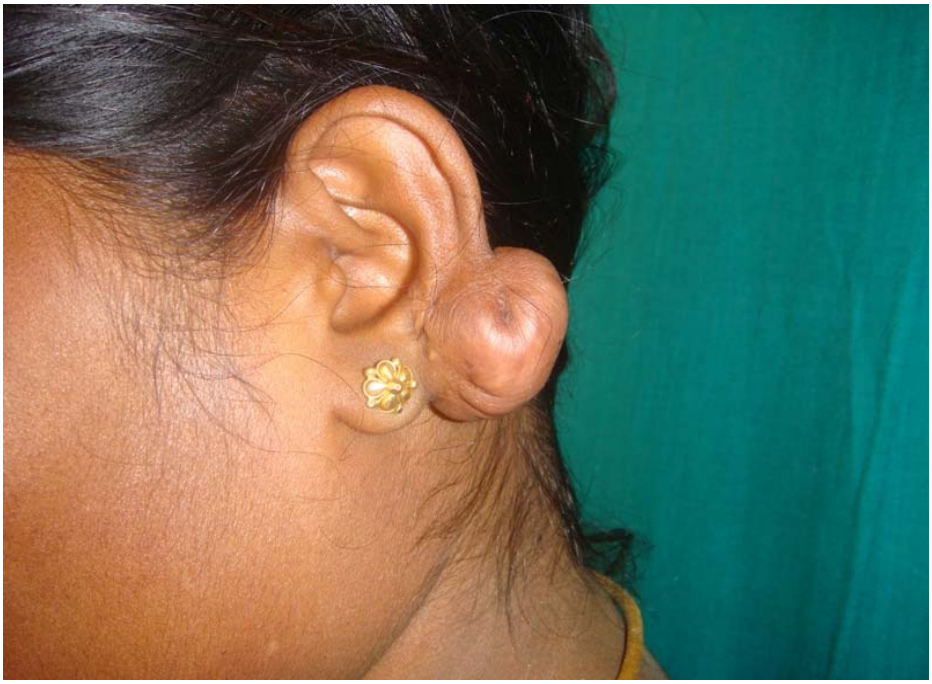


Fig:25-keloid over the left ear

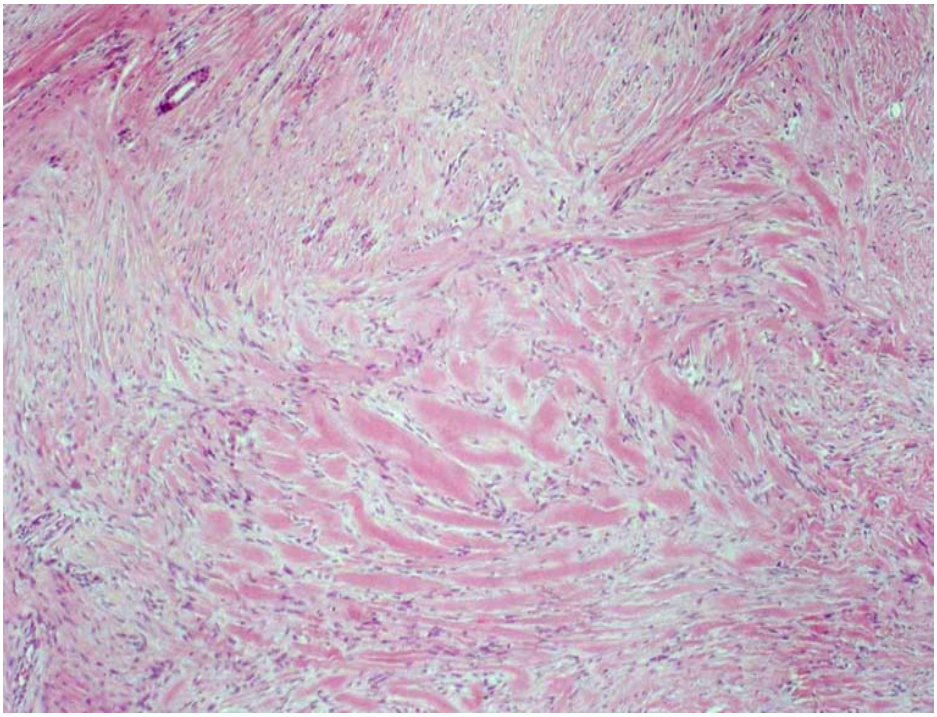


Fig: 26-Histopathology of keloid demonstrating thick hyalinized collagen bundles

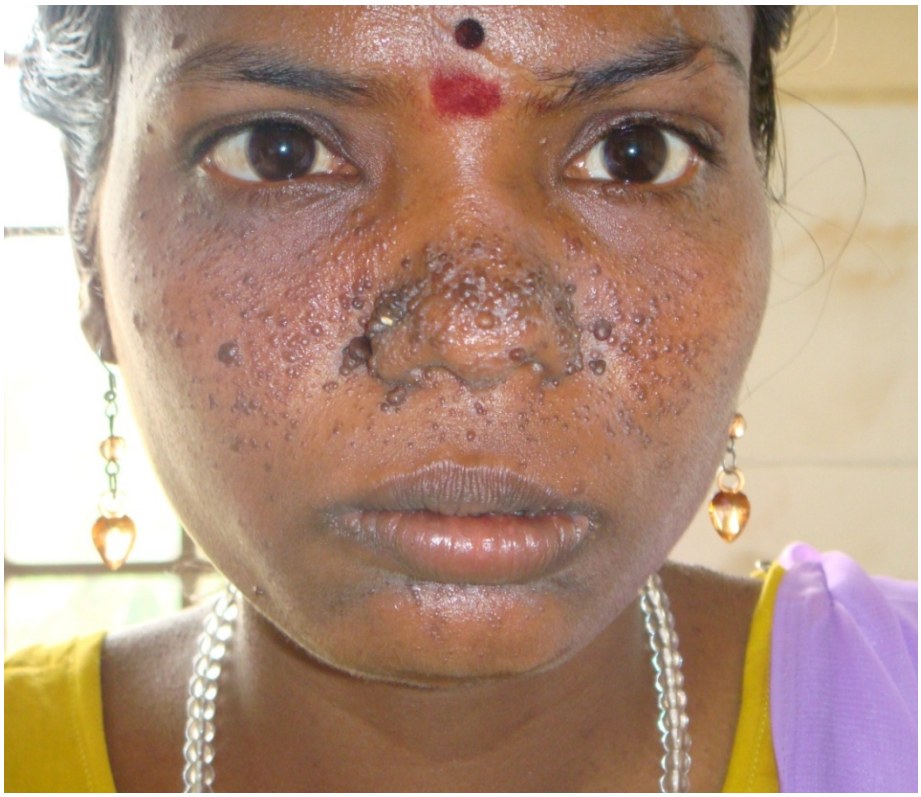


Fig:27 -Facial Angiofibroma in a patient of Tuberous sclerosis.

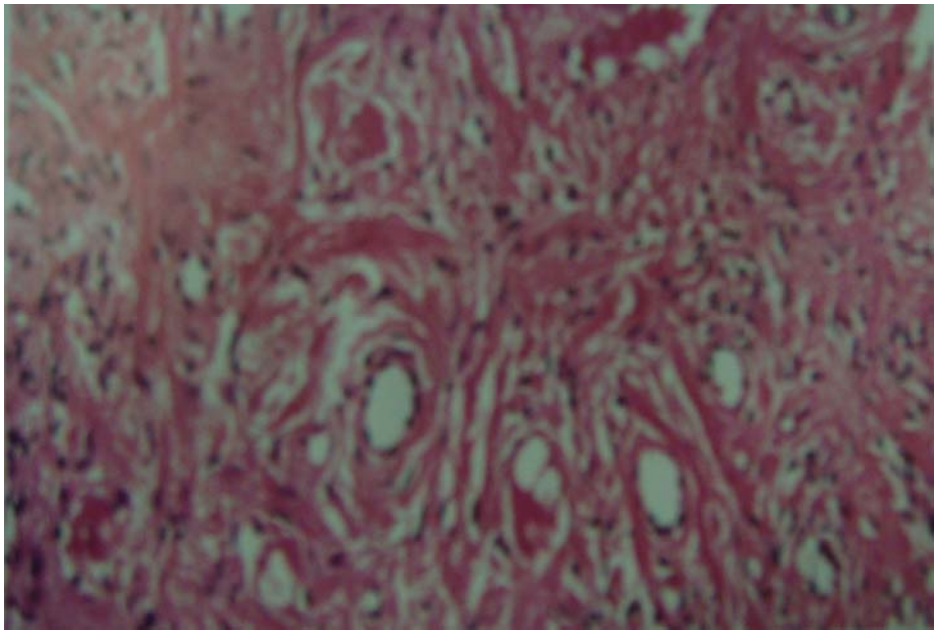


Fig:28 – Histopathology of Angiofibroma showing sclerotic, hyalinized collagenous stroma surrounding the telangiectatic blood vessels.

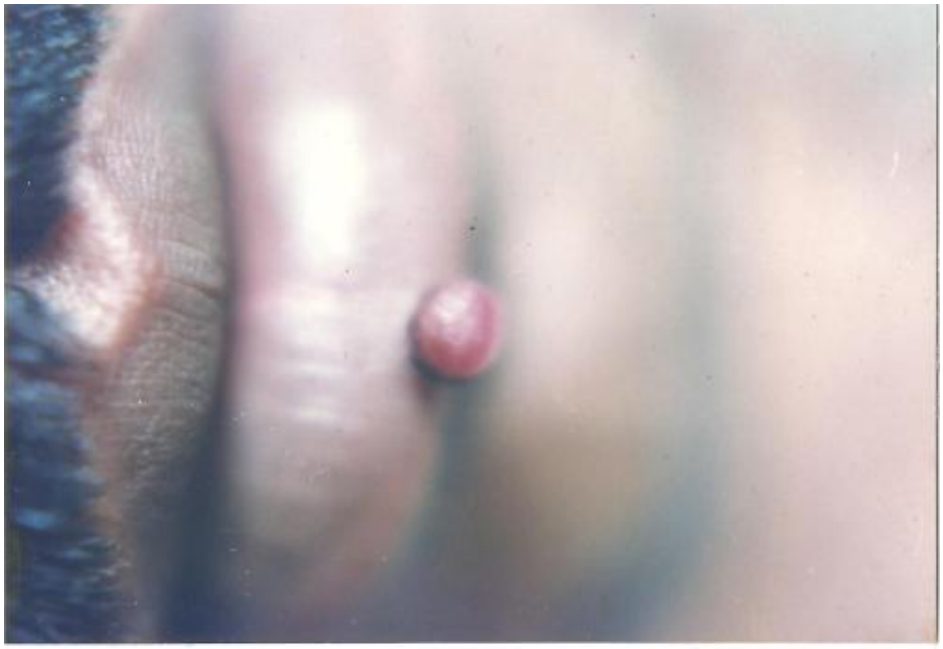


Fig : 29- Granuloma pyogenicum over the lower lip

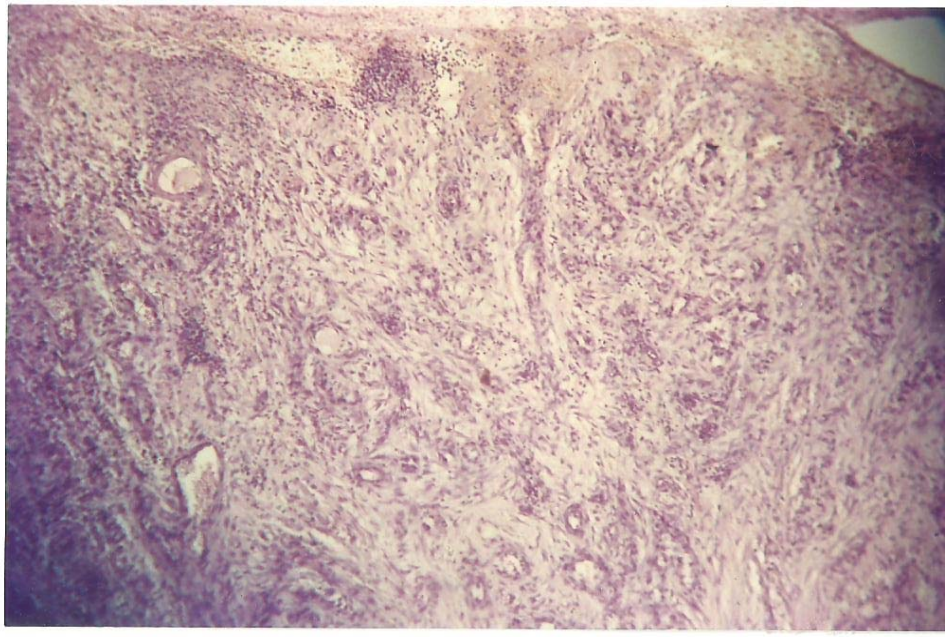


Fig:30-Histopathology of Granuloma pyogenicum showing the lobules of proliferated capillaries.

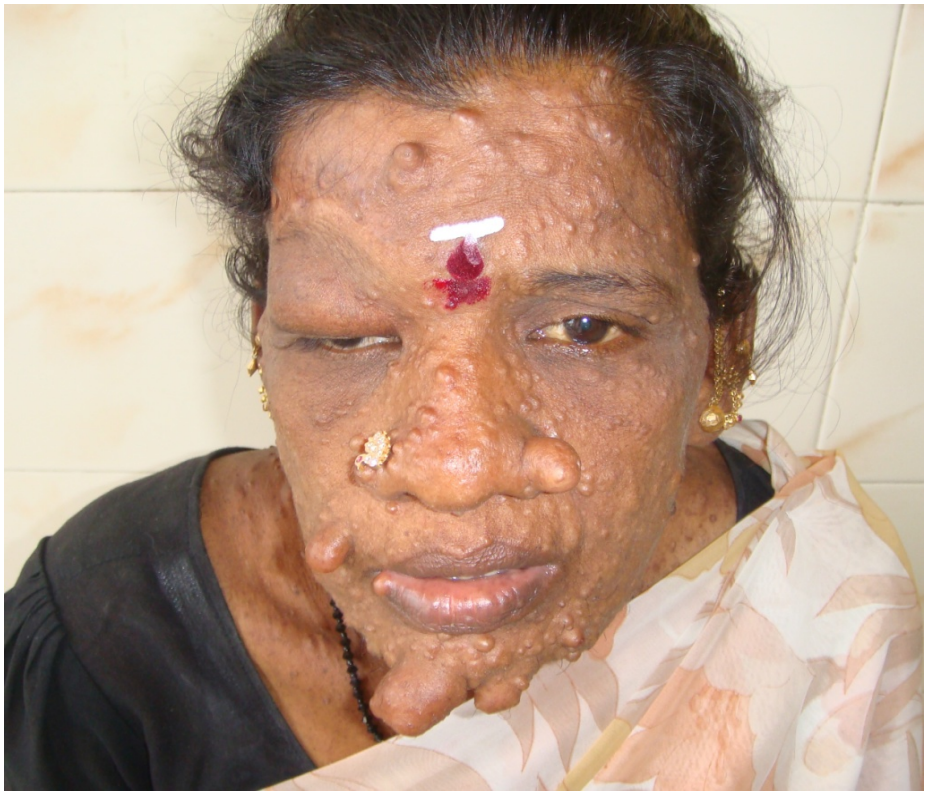


Fig:31 - Multiple Neurofibromas over the face. Note the Café -au lait macules

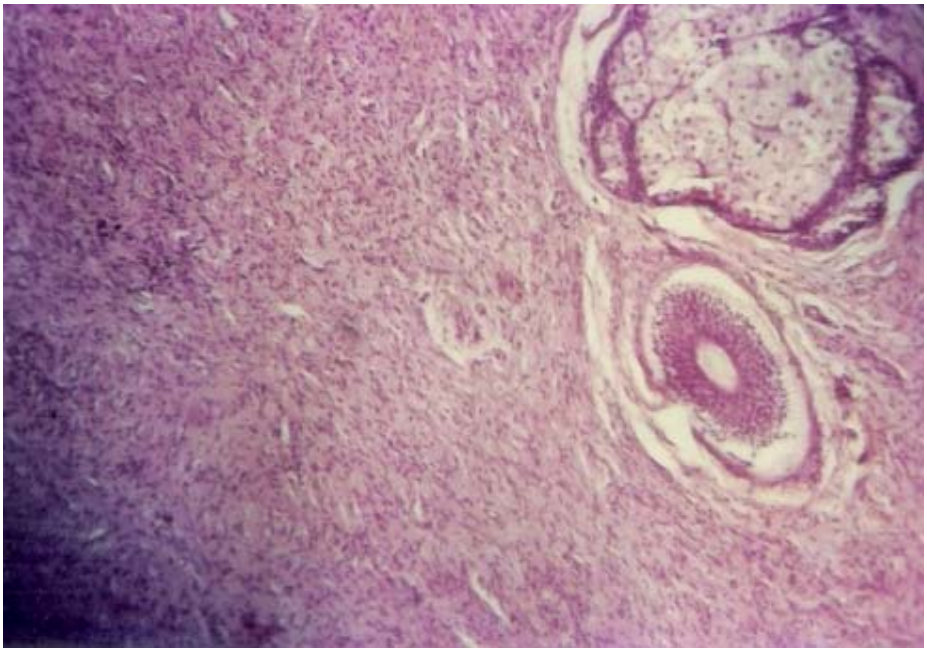


Fig:32 - Histopathology of Neurofibroma showing thin wavy nerve fibres.

APPENDICES

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PROFORMA

Name : **Sex : M / F** **Age :**

Case no : **Hospital no :**

Address : **Occupation** :

Presenting complaints :

Skin lesions

Site

Duration of complaints

Others

Symptoms : pain / burning sensation / itching / cosmetic

Fear of malignancy / discharge / others

Previous treatment : medical / surgical / radiotherapy / others

Consanguineous / Non-Consanguinous

Family history : yes / no

Excessive sun exposure : yes / no

General examination :

Systemic examination :

Dermatological examination :

Primary lesion : macule /papule /patch /nodule /others

Site of the lesion :

Number :single /2-10 /11-20 / >20

Size :

Shape :

Discrete :

Extension :

Pigmentation :

Erythema :

Linearity :

Surface : **flat topped** **elevated** **raised**

Papillomatosis **smooth** **shiny**

Verrucous **fungoid** **indurated**

Scaling **crusted** **ulcerated**

Nodular **telengiectasia**

Sessile / pedunculated :

Border / margin :

Warmth :

Tenderness :

Bleeds on touch :

Consistency :

Attachment to the underlying structures :

Regional lymph node enlargement :

Any similar lesions :

Other associated findings :

Hair :

Nail :

Mucous membrane :

INVESTIGATIONS

1. Complete blood count

2. Blood group

3. Mantoux

4. VDRL

5. ELISA for HIV

6. Random blood sugar, blood Urea. Serum creatinine

6. SKIN BIOPSY

7. Ultrasound/ CT Scan

MASTER CHART

S.NO	AGE (yr) /SEX	DIAGNOSIS	SITE	AGE (yr) OF ONSET	ASSOCIATED CONDITIONS
1	54/M	Seborrhoeic keratosis	Cheeks, temples	46	-
2	52/M	Seborrhoeic keratosis	Face, forearms	41	-
3	60/M	Seborrhoeic keratosis	Cheeks, neck	51	-
4	46/F	Seborrhoeic keratosis	Temple.neck	40	-
5	56/M	Seborrhoeic keratosis	Forehead	49	-
6	42/F	Seborrhoeic keratosis	Nose, cheeks	40	-
7	41/M	Seborrhoeic keratosis	Cheeks	40	-
8	51/F	Seborrhoeic keratosis	Behind the ear	46	-
9	41/M	Seborrhoeic keratosis	Cheeks	41	-
10	57/F	Seborrhoeic keratosis	Cheeks, neck	44	-
11	44/M	Seborrhoeic keratosis	Cheeks ,Forehead	43	-
12	42/M	Seborrhoeic keratosis	Cheeks	40	-
13	56/F	Seborrhoeic keratosis	Forehead, cheeks	49	-
14	52/F	Seborrhoeic keratosis	Nose, cheeks	43	-
15	36/M	Acrochordons	Face, neck	35	Diabetes mellitus
16	39/M	Acrochordons	Neck	37	-
17	42/M	Acrochordons	Neck, axillae	42	Diabetes mellitus
18	43/F	Acrochordons	Face,Neck, axillae	43	-
19	53/F	Acrochordon	Face,Neck,	45	Diabetes mellitus
20	28/M	Acrochordon	Face,Neck,axillae	28	-

S.NO	AGE (yr) /SEX	DIAGNOSIS	SITE	AGE (yr) OF ONSET	ASSOCIATED CONDITIONS
21	35/F	Acrochordon	Cheeks.Neck	35	-
22	35/M	Acrochordon	Face,Neck	35	Diabetes mellitus
23	45/M	Acrochordon	Face,Neck	41	-
24	54/F	Acrochordon	Face,Neck	45	Diabetes mellitus
25	43/F	Acrochordon	Face,Neck	43	-
26	44/M	Acrochordon	Face,Neck	41	-
27	43/F	Acrochordon	Face,Neck	42	-
28	49/M	Acrochordon	Face,Neck	40	Diabetes mellitus
29	50/F	Acrochordon	Face,Neck	35	-
30	14/F	Epidermal nevus	Left cheek	2	-
31	9/F	Epidermal nevus	Side of the Neck	Birth	-
32	5/M	Epidermal nevus	Temple	3	-
33	16/F	Epidermal nevus	Right cheek	Birth	-
34	12/M	Epidermal nevus	Face-cheek	1	-
35	24/F	Epidermal nevus	Neck	12	-
36	41/M	Basal cell carcinoma	Left cheek	40	-
37	56/F	Basal cell carcinoma	Left ear	51	-
38	45/F	Basal cell carcinoma	Right cheek	42	-
39	57/M	Basal cell carcinoma	Nose	50	-
40	60/F	Basal cell carcinoma	Left cheek	55	-
41	49/F	Basal cell carcinoma	Outer canthi of righteye	47	-
42	60/F	Basal cell carcinoma	Right cheek	54	-
43	60/F	Squamous cell carcinoma	Lower lip	57	-
44	13/F	Squamous cell carcinoma	Lower lip, around the eye	11	Xeroderma pigmentosum
45	60/M	Squamous cell carcinoma	Upper lip,	58	-

S.NO	AGE (yr) /SEX	DIAGNOSIS	SITE	AGE (yr) OF ONSET	ASSOCIATED CONDITIONS
46	59/F	Squamous cell carcinoma	Right cheek	55	-
47	56/M	Squamous cell carcinoma	Lower lip	55	-
48	26/M	Epidermal cyst	Side of the neck	21	-
49	32/F	Epidermal cyst	Nape of the neck	25	-
50	30/M	Epidermal cyst	Forehead	20	-
51	14/M	Epidermal cyst	scalp	31	-
52	42/F	Epidermal cyst	Forehead	40	-
53	29/M	Epidermal cyst	Face	22	-
54	35/F	Epidermal cyst	Neck	31	-
55	20/M	Epidermal cyst	Side of the neck	15	-
56	21/M	Epidermal cyst	Vertex	17	-
57	26/M	Epidermal cyst	Side of the neck	21	-
58	32/F	Epidermal cyst	Nape of the neck	25	-
59	15/M	Epidermal cyst	Forehead	10	-
60	42/M	Steatocystoma multiplex	Face,Neck	35	-
61	13/F	Steatocystoma multiplex	Neck	11	-
62	34/M	Steatocystoma multiplex	Face,Neck	28	-
63	34/F	Trichoepithelioma	Central face,nasolabial folds	28	-
64	36/F	Trichoepithelioma	Central face	31	-
65	27/M	Trichoepithelioma	Central face	16	-
66	36/F	Trichoepithelioma	Central face, Para nasal folds	31	-
67	26/M	Trichoepithelioma	Central face	18	-
68	14/M	Nevus sebaceouS	Scalp	Birth	-
69	8/M	Nevus sebaceous	Scalp	Birth	-

S.NO	AGE (yr) /SEX	DIAGNOSIS	SITE	AGE (yr) OF ONSET	ASSOCIATED CONDITIONS
70	30/F	Nevus sebaceous	Scalp,	Birth	-
71	9/M	Nevus sebaceous	Scalp	Birth	-
72	33/F	Syringoma	Face-infraorbital region	20	-
73	21 /F	Syringoma	Face-infraorbital region, nose	19	-
74	40 /F	Syringoma	Face-supraorbital region	36	-
75	54 /F	Syringoma	Face-infraorbital region ,neck	40	-
76	39/M	Syringoma	Face-infraorbital region	31	-
77	36/F	Syringoma	Face-infraorbital region	25	-
78	25/F	Syringoma	Face-infraorbital region	19	-
79	19/F	Syringoma	Face-infraorbital region	18	-
80	31/M	Syringoma	Face-infraorbital region	21	-
81	31/F	Syringoma	Face-infraorbital region	29	-
82	26/F	Syringoma	Face-infraorbital region	22	-
83	42/F	Syringoma	Face-infraorbital region	28	-
84	30/F	Keloid	Earlobules	28	-
85	33/F	Keloid	Earlobules	25	-
86	25/M	Keloid	Earlobules,back of the neck	20	-
87	20/F	Angiofibroma	Nose,malar area, forehead	8	seizures
88	19/M	Angiofibroma	Nose,malar area,	10	seizures
89	20/F	Pyogenic granuloma	Scalp	20	-
90	25/F	Pyogenic granuloma	Forehead	24	-
91	35/M	Pyogenic granuloma	Lower lip	35	-
92	30/F	Pyogenic granuloma	Lower lip	29	-
93	25/M	Neurofibroma	Generalised cutaneous papules & nodules,numerous CALM macules, axillary freckling ,multiple B /L lisch nodules,	5	Scoliosis,
94	30/M	Neurofibroma	Generalised cutaneous papules & nodules,numerous CALM macules,no lisch nodules	6	pruritis

S.NO	AGE (yr) /SEX	DIAGNOSIS	SITE	AGE (yr) OF ONSET	ASSOCIATED CONDITIONS
95	8/F	Neurofibroma	8-10 cutaneous papules & nodules involving face trunk, 2 CALM macules	4	-
96	15/M	Neurofibroma	Skin lesions present over the scalp 2 in number,No CALM , lisch nodules absent	4	-
97	26/M	Neurofibroma	Generalised cutaneous papules & nodules,numerous CALM macules, present , B /L lisch nodules,	8	-
98	60/F	Neurofibroma	Generalised cutaneous papules & nodules,one plexiform NF numerous over the left leg,CALM macules,multiple B /L lisch nodules, axillary freckling ,	9	Intense pruritis
99	23/F	Neurofibroma	2 nodules over the face . 2 CALM macules ,no lisch nodules	15	-
100	30/F	Neurofibroma	Generalised cutaneous papules & nodules,numerous CALM macules,no lisch nodules, axillary freckling	14	-