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A CLINOPATHOLOGICAL EVALUATION AND MANAGEMENT OF CUTANEOUS MALIGNANCIES

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CERTIFICATE

This is to certify that this dissertation entitled **"A CLINOPATHOLOGICAL EVALUATION AND MANAGEMENT OF CUTANEOUS MALIGNANCIES "** is bonafide work done by **Dr. C.K.M Laxmi** under our guidance and supervision in the Department of surgery, Madurai Medical College, Madurai submitted for the M.S.,(General surgery) BRANCH I EXAMINATION, to be held in March 2008, by the Tamilnadu DR.M.G.R. Medical university, Chennai.

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CONTENTS

INTRODUCTION

"What is most difficult of all? It is what appears most simple. To see with yours eyes what lies in front of your eyes" Goths.

Skin, though ubiquitous, is remarkably heterogeneous organ. Because of its complexity, wide varieties of tumours arise from it. Thus there may be tumours from cetodermal origin and mesodermal origin, such as tumours of skin appendages, pigment cells, vessels, muscles and lymphoid tissue. Their diversity combined with body of descriptive data (clinical, histological, and therapeutic) amassed over the past century and deepened in various literature produces confusion.

Interpretation of clinical picture is difficult, for identical manifestations may result from widely difficult causes. However, the great advantage of dealing with cutaneous surgery is that of dealing with an organ that can be seen and felt.

The distinction between normal and pathological structures, benign & malignant neoplasm are more difficult to define when they appear in skin than when found elsewhere. They may also be harbinger of metabolic & visual disturbance.

Malignant skin tumour usually show a disorganized structural pattern of individual cells frequently exhibit structured abnormality such as abnormal size, shape, chromatin pattern and nucleus to cytoplasm ratio. These lesions show infiltrative growth usually rapidly and mitotic figures are not uncommon.

Malignant tumours show infiltrative growth with inversion and destruction of tissue. Metastases are characteristic of malignant tumour.

The potentiality to give rise to metastases is decisive evidence for the malignancy of tumors. For metastases to form, tumour cells must possess a degree of autonomy. This autonomy enables malignant cells to induce foreign tissue to furnish the necessary stroma in which they can multiply. In addition to malignant tumours located largely in situ. Although cytologically malignant, they are biologically still benign.

The criteria for benign and malignancy must be altered somewhat in dealing with skin tumours. For example, one of the rapidly growing tumours of skin, keretoacanthoma, is benign and the most common skin tumour, basal cell carcinoma is slow growing and usually does not metastasize.

While cancers of the skin are more amenable to therapeutic measures than are cancers of other sites, accurate diagnosis presents many problems because of the variations in histological types, of metastasis and different responses to the therapy.

Essentially the principle involved is treatment of skin cancers is early diagnosis, adequate treatment & careful follow up.

Thus, the stud of primary malignant sign tumours is perhaps more intriguing, fascinating & challenging.

AIMS OF THE STUDY

As primary malignant skin tumors comprise of wide variety, it is considered worthwhile to study the following.

1. Common & unusual manifestations of skin tunors seen at GRH, Madurai.
2. Validity of clinical & histopathological diagnosis
3. Management of skin malignancies either palliatively or curatively by following standard procedures in the literature.
4. Education of the patients about harmful effects of etiological factors and early detection of precursor lesion in prevention of skin cancer, so that debilitating surgeries can be avoided later on.

REVIEW OF LITERATURE

Embryology and histology of the skin

The skin has two fold origin; (a) a superficial layer, epidermis which develop from the surface ectoderm, and (b) a deep layer, the dermis, which develops from the underlying mesenchyme.

MORPHOLOGY: Skin is the major organ of the body, forming about 8% of its total mass and having an area of berween 1.2 to 2.2 sq meters. The thickness of skin varies from 1.5mm on the eyelid to up to 4mm on the back. Skin covers the entire surface of the body.

Anatomy of Skin

Skin is the largest organ in the body.

- An outer keratinizing stratified squamous epithelium, which is self regenerating – the epidermis.
- An underlying tough supporting and nourishing layer of fibroblastic tissue – the dermis.
- A variable deep layer, mainly adipose tissue – the hypodermis or sub cutis.

Epidermis: Cells produced by mitosis in the germinal basal layer adjacent to the dermis undergo maturational changes concerned with the production of keratin. The outer keratinized layer is shed continuously and is replaced by the progressive movement and maturation of cells from the germinal layer; thus all of the cells of this lineage are described as keratinocytes. The rate of mitosis in

the germinal layer is generally equal to the rate of desquamation of keratin from the outer surface, in humans, the process of maturation of a cell through to desquamation takes from 25 to 50 days, being rapid in areas exposed to heavy frictional forces.

- Stratum basale
- Stratum spinosum
- Stratum granulosum
- Stratum corneum

Melanocytes: M as seen in micrograph (a) are responsible for the synthesis and release of the brown pigment melanin, which is largely responsible for skin coloration. They are located in the basal layer of the epidermis and appear as round cells with pale – staining cytoplasm scattered infrequently between the low columnar basal cells. From this cell body there are numerous long cytoplasmic processes, which run in the spaces between the keratinocytes of the stratum granulosum. Melanocyte cytoplasm contains secialized membrane- bound oval granules called premelanosomes and melanosomes, which synthesize the pigment melanin.

Skin appendages: Skin has a variety of appendages, principally hairs, sebaceous glands and sweat glands, which are derived embryologically from the surface epithelium (epidermis).

Hairs: Hairs are highly modified keratinized structures produced by hair follicles, which are essentially cylindrical down growths of the surface epithelium, unsheathed by collagenous tissue.

Sebaceous glands: One or more sebaceous glands are associated with each hair follicle; these glands secrete an oily substance called sebum onto the hair surface in the upper part of the follicle. Sebum acts as a waterproofing and moisturizing agent for the hair and skin surface.

Sweat glands: In most areas of the skin, sweat glands are simple, coiled tubular glands, which secrete a watery fluid onto the skin surface by the process of merocrine secretion.

FUNCTION:

Protection: Provides protection against ultraviolet rays, mechanical, chemicals, and thermal insult. Its relatively impermeable surface prevents dehydration; act a s physical barrier to invasion by microorganism.

Sensation: Skin is the largest sensory organ in the body. Contains variety of receptor for touch, pressure, pain and temperature.

Thermo regulation: body is insulated against heat loss by the presence of hair and subcutaneous, adipose tissue. Heat loss is facilitated evaporation of sweat from the skin surface and increased blood flow through the rich vascular network or the dermis.

Metabolic function: Subcutaneous adipose tissues constitute a major store in energy mainly in the form of triglycerides. Vitamin D is synthesized in the epidermis and supplement that derived from dietary source.

Synthesis of melanin: Melanin pigment gives black colour to the skin and hair (Greek word Melanin means black).

1 st step

By Hydroxylation. Tyrosinase (mono oxygenase contains copper)

Tyrosine ⟶ DOPA

2^{nd} step converted to

(dihydroxy Pheny1 alanine) ⟶ Dopaquine ⟶ Indoliquine through a series of reaction involving decorboxylation and oxidation of side chain

Indoliquine is finally polymerized to form melanin.

Step 1 and 2 are enzyme- based reaction

Melanin synthesis is controlled by –

1. MSH

2. ACTH

3. Sex hormone both estrogen and androgen

CLASSIFICATION OF MALIGNANT SURFACE EPIDERMAL TUMOURS. BY WORLD HEALTH ORGANIZATION.[3]

1. Squamous cell carcinoma
    - Spindle cell
    - Acantholytic
    - Verrucous
    - Horn forming
    - Lymphoepithelial

2. Basal cell carcinoma
    - Multifocal superficial (Superficial multicentric)
    - Nodular (Solid, adenoid cystic)
    - Infiltrating
    - Non-Sclerosing
    - Sclerosing (desmoplastic morphemic)

3. Fibroepithelial

4. Basal cell carcinoma with adnexal differentiation
    - Follicular

- Eccrine

5. Basosquamous carcinoma.
6. Keratotic basal cell carcinoma
7. Pigmented basal cell carcinoma
8. Basal cell carcinoma in basal cell nevus syndrome
9. Micronodular basal cell carcinoma
10. Uncommon cutaneous malignaney.

Uncommon cutaneous malignancies

1. Cutaneous angiosarcoma

- Rare, aggressive soft tissue sarcoma derived from blood or lymphatic endothelium.

Presents as flat, painless often pruritic macule or plague with a red, blue or purple color that develops into a mass and ulcerates if left in place.

High incidence of lympha node metastasis (15%).

2. Dermatofibrosarcoma protuberans

* Low grade sarcoma arising from dermal fibroblasts.

Appears as smooth nodule in or immediately beneath the skin.

3. Kaposis sarcoma

Low grade soft tissue malignancy arises from lymphatic or vascular endothelial cells in the skin.

Common in AIDS and other immunosuppressed states.

4. Merkel cell carcinoma

Derived from neuroendocrine cells.

Rapidly growing red-blue nodule most frequently in Head and neck area in older individuals.

11. Adnexal Tumors

Hair follicle

1. Trichoepithelioma
2. Trichofolliculoma

Sebaceous Gland

1. Sabaceous adenoma
2. Sebaceous epithelioma

Apocrine Gland

1. Syringocystadenoma
2. Papilliferous

Ecrine gland

Syringoma

DESCRIPTIVE EPIDEMIOLOGY

Cutaneous malignancies are the malignancies whose incidence is rising faster than any other malignancies. Hence it is aptly called ‘an epidemic of cutaneous malignancies.

The cutaneous malignancies accounts for about 1% of the total malignancy incidence.

All cutaneous malignancies are more common in whites than blacks and occurs less commonly in Asian and black population.

The incidence of Non melanoma cancer ranges from 1,00,000 in Japan to 1000 per 1,00,000 population in Australia. The basal cell carcinoma & squamous cell carcinoma occurs in a ratio of 4:1. Both are more common in males with male to female ratio of 3:1. The incidence of squamous cell carcinoma is 38 per 1,00,000 population per annum. Basal cell carcinoma accounts for 70% of the NMSC.

The incidence of malignant melanoma varies from 30-50 cases per 1,00,000 population per annum with preponderance in males.

Geographical Variations

All cutaneous malignancies are more common in countries in which sun exposure is more, like Australia. The cutaneous malignancies are more common in population of Celtic ancestry.

OCCUPATIONAL SKIN CANCER

Definition: Occupational cancer is a malignancy that results from exposure to carcinogenic forces in an occupational environment.

GENERAL CONSIDERATION ABOUT OCCUPATIONAL CANCER

Predisposing factors

a) Extrinsic factors

1. Nature of carcinogen: There are about 200 polycyclic hydrocarbons, which are carcinogenic apart from arsenic & ionizing radiation. The activity of these carcinogens is affected by presence of Co – carcinogens or anticarcinogens.
2. Physical states of the carcinogens. The effect of carcinogens in the form of dust, aerosol liquid, solid or radiation will vary according to physical state and its capacity to adhere to the skin or penetrate the clothing.
3. Nature of the occupation. The site of lesion is determined by working position, technical demand of job, land environment condition.
4. Intensity of exposure. The effect is directly proportional to it.
5. Duration of exposure. The longer the period of exposure, the more inevitable the out come. This may vary with the individual and carcinogens.

b) Intrinsic factors:

1) Age: The duration of exposure, to carcinogens may amount to many years. Senile atrophic and hypertrophy changes may play a part, but intensity and duration of exposure are of Greater importance than the carcinogens.
2) Sex: Males are more affected than females.
3) Race: Epithelioma is less common in black people because of their more efficient protection against UV rays.
4) Heredity: The influence of heredity is doubtful in occupational cancer though it may play part in personal idiosyncrasy.
5) Nature of the skin: Sebaceous nature of the skin like scrotum, may take up the fat soluble carcinogens.
6) Personal habits: The tendency to reach the nose or ear with oily or tarry hands, general lack of personal cleanliness may determine the site of cancer.
7) Special idiosyncrasy: Many people exposed to carcinogenic material never show any sign of cancer. There may be in certain individuals a metabolic or heredity influence, which operates in direction or the other.

AGENTS PRODUCING SKIN CANCER

A. CHEMICAL AGENTS

1. Arsenic (inorganic or organic).
2. Combustion & distillation products of coal, soot, coal tar, anthralenes.
3. Distillation products of oils, lubrication oil, crude paraffin oils.
4. Processed petroleum products, fuel oil, lubrication oil, crude paraffin oil, and petroleum tars, asphalt carbon black.
5. Combustion of natural gas, types of carbon black.

B. PHYSICAL AGENTS

1. Non ionizing radiations, ultraviolet light.
2. Ionizing radiations; X-rays.

NON-MELANOMA SKIN CANCER

GENO DERMATOSES

Hereditary syndromes enlisted with skin cancers are part of a group of rare disorder characterized by one or more of a large number of abnormities. Their syndromes are known as geno dermatoses.

(i) Nevoid basal cell carcinoma syndrome.(GORLIN SYNDROME)

(ii) Xeroderma pigmentosum

(iii) Epidermodysplasia verruciformis.

(iv) Muir-Torre syndrome.

PRECUSSORS OF EPIDERMAL MALIGNANCIES [12]

1. Actinic keratoses (Solar keratoses) [13]

Actinic keratoses are circumscribed, rough, epidermal lesions that develop on sun exposed skin secondary to chronic solar irradiation. Actinic keratoses (AK) are epidermal neoplasm, consisting of altered epidermal keratinocytes. They are classified as precancerous lesion because at least 20 percent will undergo transformation into invasive squamous cell carcinoma (SCC). Biologically, they are considered to be carcinoma or squamous intraeidermal neoplasm (SIN).

Clinical features: The Actinic keratoses are represented clinically by a rough lesion that is better recognized by palpation than inspection. Most lesions are between 3 & 6 mm in diameter. The AK may be flesh colored, darkly pigmented or erythematous.

Treatment: Chemotherapeutic agents, surgical procedures, irradiation, cold steel or laser procedure. They can also be treated by sharp curettage followed by electrodessication. Cryosurgery with liquid nitrogen is effective therapy for most Actinic keratoses. Topical chemotherapy like 5-FU and topical retinone has been advocated for the reversal of the sun damage to the skin.

Prognosis: The prognosis is good but the lesions may progress to carcinoma. Squamous cell carcinoma that arises from Actinic keratosis rarely metastasizes.

ii) Radiation induced keratoses.

Ionizing radiation used for either diagnostic purposes can induce premalignant keratoses.

1. Treatment of internal malignancies
2. Treatment of cutaneous malignances
3. Treatment of benign skin tumours.
4. Treatment of benign non-tumourous cutaneous conditions.

Prognosis: The lesion is more aggressive than solar keratoses.

Treatment: To remove individual lesion with curettage and electrodessication or excision.

iii) Arsenical keratoses.

It occurs most often on the acral skin, in particular on the volar surfaces. They are punctuate palmar and or plantar lesions. Hundreds of lesions may be present. There is usually symmetrical involvement. Prognosis is determined by the rate of invasive squamous cell carcinoma & its potential for internal malignant disease.

iv) Bowen's disease:

Dr. John Bowen is credited with the first description. It can occur on both sun exposed and non-exposed areas. It is an intraepidermal neoplasia of the skin.

Clinical features:

Bowen's disease is most often a slightly scaly, discrete, erythematous plaque with a sharp but with irregular or undulating border. The surface characteristics vary and include hyperkeratosis, fissures, pigmentation, erosions and ulcerations. The lesion usually grows in a slow but progressive manner. Bowen's disease has been linked to at least four potential etiologic agents, actinic damage, arsenic ingestion, radiation therapy and viral agents.

Treatment: It includes surgical excision, curettage, and cryotherapy with liquid nitrogen, lacal irradiation. Topical chemotherapy with 5 – FU, laser surgery and microscopically controlled surgery.

Tar Keratoses:

Keratoses and cancer of the skin following chronic exposure to tar, pitch, coal, soot and mineral oil. The lesions are karyolyic or waxy and occur on skin.

SQUAMOUS CELL CARCINOMA

DEFINITION:

Primary cutaneous Squamous cell carcinoma a malignant neoplasm of the keratinizing cells of epidermis.

Epidemiology

Non-melanoma skin cancer is the most common form of human cancer, with an estimated incidence of 1 million cases per year. Basal cell carcinoma (BCC) accounts for 80% of these cases. SCC accounts for 20% non-melanoma skin cancers.

Risk factors and pathogenesis

1. Radiation: 80% of UV light induced SCC of the skin develops on the arms, head and neck. Ultraviolet light is biologically significant in UV B (280 - 320 nm wavelength) range. This mechanism of UV light carcinogens is attributed to chromosomal changes, induction of DNA mutations, altered DNA methylation and activation of oncogenes. Radiation is known to be immuno suppressive and may impede the usual tumour surveillance mechanism.

SCC more often found in areas of sun-exposed skin. A higher incidence of SCC is seen in lower latitudes. The incidence of SCC in proportionately related to chronic cumulative sun exposure.

2. Immunosupression: Renal transplant patients have an inroad risk of SCC of the skin. Some studies have noted cyclosporine increases incidence of skin cancers.

3. Human papilloma virus (HPV): The infection with HPV has been implicated in the pathogenesis of SCC. The HPV subtype 16 and 18 found to have the closest association with transformation to SCC.

4. Hereditary: Xeroderma pigmentosa is a rare autosomal redessive disease. These patients develop SCC, BCC and malignant melanoma. Treatment is absolute sun avoidance.

5. Oncogenes: Over expression of the p53 protein has been in up to 40% of SCC of the skin. No P53 reactivity was observed is normal skin.

6. Chromosome abnormalities: Frequent loss of heterozygosity of markers from 3p (23%), 9p (41%), and 17p (33%) in seen. This finding suggests that there are certain genes important in the development of SCC of the skin.

7. Chemicals: Arsenic, polycyclic hydrocarbons, Doxorubicin, Alkylating agents, mechloroethamine has also been implicated.

8. Lymphopoliferative disease: Patients with CLL and Non Hodgkins lymphoma may develop aggressive SCC. Patients with cutaneous T-cell lymphoma are at increased risk for SCC (Sezary Syndrome).

9. Scar carcinoma (MARJOLIN'S ULCER): Marjolin, in 1828, first reported the occurrence of SCC in long standing burn wounds. This lesion is most

apparent in those wounds that are clinically unhealed or unstable. Changes include increase in size of the ulcer, change in the appearance, particularly with appearance of rolled and everted edges with surrounding induration. Lymphnode metastases are higher in this group UV induced SCC reaching up to 20%.

10. Sinus tracts: Squamous cell carcinoma can develop in chronic sinus tracts e.g.; osteomyelites. The frequent site of involvement is the tibia. The pressure of ulceration or a mass in the region of the sinus is an indication for biopsy. SCC of the skin metastasizes to regional lymph nodes at the rate of 30%.

HISTOPATHOLOGY

SCC's grow at a rate of 1.85 mm per month. It behaves more aggressively and has a highest incidence of metastases because of its stromal independence. At the periphery of some SCC's dermosomes may be absent, which allows tumor cells to appear to float free in the stroma. This allows tumors cells to be implanted in to lymph nodes, blood vessels, or adjacent normal tissue during a surgical procedure. Microfilaments are important for cell mobility and are most numerous in tendon cells at the advancing edge of a cancer.

Microscopy

- Full – thickness epithelial dysplasia or anaplasia (in situ).
- Dysplasia or anaplasia in lower most epidermal layer with single cell on nested transgression across basement membrane invasive.

- Hyperkeratosis with formation of atypical parakeratoses.
- Individual cell zonal necrosis.
- Keratin pearl formation.

MODES OF LOCAL SPREAD.

SCC follow the path of least resistance as they spread through tissue along peridchondrium, periosteum, or tarsal plate, embroyonal fusion planes, vestiges of embryo genesis, appear to offer little resistance to penetration and spread of this tumors. SCC does not readily invade muscle, when it does, the spread in often parallel to the muscle fibres.

Perineural spread is more common in SCC than in BCC. Tumors affecting the nerves may spread in a centripetal and centrifugal direction and may travel a considerable distance.

PATTERY OF METASTASES

SCC usually first metastases to the regional lymph nodes and distal spread in the absence of node spread in rare. Regional metastases usually occur within 3 years from the time of treatment of the primary tumors. Unusual & atypical metastases also occur. Intransit and satellite lesions may also be seen. The most frequent sites for metastases are the lung, skin, bones and parotid gland. The five-year survival rate of metastases in SCC in 34%.

HISTOLOGIC AND CLINICAL VARIANTS OF SCC

1. SCC in situ (Bowen's disease, Querat's erythroplasia)

Two different forms of SCC in site exists. The first is Bowen's (BD), and the second is non-invasive SCC arising within an AK.

Non- invasive SCC

In Grade I, the SCC has not penetrated below the level of sweat glands. In some areas basal layer may be intact.,

In Grade II, the invading cell mass is poorly demarcated from the stroma. Many of the nuclei are atypical, and only a few incompletely keraitinised horn pearls are present.

In Grade III, keratinisation is absent in many areas and the horn pearls are not present. Malignant dyskeratoses is observed.

In Grade IV, keratinisation is almost non-existent; almost all of the nuclei are atypical.

2. Adenoid, Acantholytic or pseudoglandular SCC.

This variant of SCC in almost exclusively found in the sun exposed areas of elderly patents, especially the face and ears. Microscopically tumour appears nodular or cup shaped.

3. Spindle cell SCC.

A poorly differentiated variant, the tumour cells are elongated and spindle shaped. They are often pleomorphic & have atypical nuclei. Mitoses are common.

4. Clear cell carcinoma of the skin.

It is a rare variant of SCC, has significant incidence of perineural involvement & metastatic spread. Numerous clear cells are intermingled with areas of typical keratinizing SCC.

5. Signet ring SCC

It is unusual variant. These tumours are Polydifferentiated invade deeply and frequently and recurs multiple times. Signet ring cells are formed by monofilaments arrangement around the nucleus in concentric rings.

6. Verrucous carcinoma.

Ackerman first coined the term verrucous carcinoma in1948. In 1954, Arid et al, were the first in the English literature of report cutaneous lesions termed epithelioma cuniculatum because the tumour resembled a rabbit warren.

Etiology: Trauma & chronic irritiation have been suggested as contributory to development of verrucous carcinoma, especially because of its prosperity to develop in burn scar and on the plantar surface.

Clinical manifestations:

The lesion is a warty surfaced, exophytic tumor, often with many sinuses that may exudates greasy, malodorous material upon pressure. It can grow to be a boggy fungating mass. Cutaneous lesions most often occur on the plantar surface of the foot but have been seen on the face, back, leg and hand. Duration of the tumour is variable, from 2 months to 44 years.

Differential diagnosis: Plantar wart, corn, condyloma acuminatum, pyogenic granuloma, amelanotic melanoma, and eccrine poroma.

The course of verrucous carcinoma is usually indolent, but it can grow aggrsively locally. Distant metastatic spread has not been described. The prognosis is excellent.

Pathology: It is low – grade differentiated SCC that is usually both exophytic & endophytic. Superficial papillomatosis, hyperkeratosis are classical features.

Treatment: Surgical excision is the treatment of choice. Recurrence rates following excision range from 5% to 6%. If the margins are positive immediate reexcision is advised.

TREATMENT OF SQUAMOUS CELL CARCINOMA

1. Conventional surgical excision

This is regarded by many as the treatment of choice for primary SCC. Excision with primary closure. Local rotational or advancement flaps or full thickness graft to repair the defect when carried out by a trained operator, usually produces a good cosmetic result and provides the pathologist with a specimen that allows confirmation of the completeness of excision and identification of histological factors associated with increased risk of local and systemic recurrence. Although under treatment because of lateral or deep tumour

extension may be identified in conventionally processed surgical specimens, there is potential to miss cases of imcomplete excision as only a small proportion of the tumour edge is examined using conventional histopathological techniques. Usually a margin of 5 to 10 mm is given during excision.

2. Micrographic Surgical technique

Frederick E Mohs, in 1936 introduced the technique of systematically excision and mapping the excised tumours until obtaining normal tissue.

This technique has been used most widely for the treatments of recurrent tumors. Previously irradiated tumors, and anatomic sites with a high rate of recurrence.

Advantages: Tissue preservation, lower recurrence rate, and a tumor free site for recurrence.

Disadvantages: They include requirement of special staining, time consumption, inconvenience for the patient and expense.

3. Field destructive therapies.

Their therapies rely on a physical insult that is lethal to the lesion to be treated. Healing of the wounds is by secondary intention.

a. Cryosurgery: Uses liquid nitrogen via an application probe as spray. It is reserved for premalignant lesions and for the lesions located on the eyslids, face, ears, neck & trunk. Caution should be taken when treating the nasolabial fold, inner canthus of the eyes, and preauricular areas.

b. Electro desiccation & curettage: A dull curette is used to remove the tumor. Although not sure, the curette will remove friable tissue & usually will spare normal issue. Local anesthesia is used. Electro desiccation then performed to give a 1 to 2 mm zone of tissue destruction in and about the wound. It heals by secondary intention.

c. Radiotherapy: Usually treatment is 400 cGY delivered in 10 to 16 fraction for tumor less than 5cm & 4500 to 4500 cGY in 15 to 30 fraction for larger tampon. The field of irradiation includes the lesion & 3mm margin. The irradiated area develops acute radiation dermatitis that resolves in 4 to 6 weeks. The 5 years cure rate for SCC in reported to be 90%.

Disadvantages:

1. Inconvenience of daily treatments
2. Radiation alters tumor host interaction.
3. Recurrence is difficult to treat because of persistent damage to the site of treatment.
4. Potential risk of radiation induced tumors.

   This modality is reserved for patients over 65 years, debilitated patients & those with high surgical risk.

d. The CO2 laser: A cutting instrument is used for patients with bleeding disorders on anticoagulants, or those patients with contraindication to the use of electro cautery.

e. The Nd: Yag laser: Is used for the treatment of SCC, as the light is absorbed deep into the skin, producing more extensive thermal necrosis at a deeper level.

f. Photodynamic therapy: This uses photosensitivity compounds that here the ability to bind specifically to tumor cells. E.g. Hematoporphyrin derivatives. This produces free radicals that damage the tumor on exposure to light to 630 nm.

Disadvantages: Photosensitivity for a period of weeks, with mandatory avoidance of all sunlight. It is applicable to multiple, difficult, metastatic or end stage lesions.

g. Topical – 5- florourocil (5-FU): It is applied topically in the form of 5% cream or a 2% of 5% solution twice a day. Redness, soreness & swelling occurs within 3 weeks on the face and with in 6 weeks on the legs. Healing can take 3-6 weeks, and usually retains the form of hypo-pigmented lesion. This treatment modality may have its place in those patients with multiple superficial lesions or in those patients for whom cosmetic results may be very important.

h. Retinoids: Proved useful in the treatment of SCC. Oral iso -1-retinoate and e-retinoate have been used.

Disadvantage: Patient intolerance and non- compliance secondary to nausea & anorexia with a concomitant increase in liver function tests.

i. Immunomodualtion: Interferon – alpha 2b, alpha 2c & gamma have been used both as intralesional injections and as systemic modalities. Intralesional depositions of interferon – alpha 2b enhances local T cell mediated immune response to act on the tumour cells.

Follow up: SCC that recurs or metastasizes usually does so with in 3 years the risk of metachronous lesions however remains throughout patient's life.

BASAL CELL CARCINOMA

INTRODUCTION:

Basal cell carcinoma (Basal cell epithelioma) is a malignant epithelial neoplasm of the skin, which is derived from the basal cells of epidermis and adnexal structures. Most commonly arising in areas of chronic sun exposure. It is a slow growing tumour that rarely metastasizes. However, it is capable of causing extensive local tissue destruction and slow death if left untreated or inadequately treated.

INCIDENCE & EPIDEMIOLOGY:

Basal cell carcinoma (BCC) is the most common cancer, which occurs in humans. BCC occurs most commonly in men & manifests between 40-50 year of age.

Histiogenesis: It arises from

1) Basal cells of the epidermis
2) Infundibulum and outer root sheath of the hair follicle.
3) Primordial epithelial germ cells
4) Cells of the pilosebacious unit.

I. Ultraviolet Exposure

The most common factor involved in the pathogenesis of skin cancer is UV Light (UVL). Fair skinned individuals who burn easily & tan poorly are of greatest risk for developing pre cancerous & cancerous lesions of the skin. The UVL rays most important in cancer carcinogenesis are 290 to 320 nm.(UVB)

When combined UVB and UVA are much core carcinogenic than when either in administered alone. Cumulative exposure to sunlight over a prolonged period (20 to 30 years) is required for tumour development. Epidermal DNA damage is thought to be the primary mechanism by which carcinogenesis is mediated.

II. Scars & tranma:

They also play a role in the development of BCC. It arises in superficial scars & burns; most of these tumors arise on the head & neck & not the extremities.

Besides burns, other form of trauma such cuts have also been associated with the acute onset of carcinoma.

III. Radiation exposure:

X-ray irradiation may give rise to BCC's. The minimal amount of exposure necessary for carcinoma formation is a dose of 1000 rads.

IV. Arsenic:

Arsenic plays a role in cutaneous & visceral carcinogenesis Sources include well water, insecticide, medication, mining , smelting & sheep dipping. Superficial muticentric & nodular BCC's have been observed in patients exposed to arsenic.

V. Nitrogen mustard.

It is used topically for the treatment of Mycosis fungoides. It is a carcinogen & enhances photo carcinogenesis.

VI. Chicken pox scars are more commonly affected.

VII. Immunosuppression:

An increased incidence of malignant neoplasms is reported in immunosuppressed individuals. Immunodeficiency enhances photo oncogenesis.

GROWTH AND MODES & SPREAD:

The stroma appears to be important not only in the induction of BCC but also in its survival. This marked stromal dependence explains why the BCC so rarely metastasize. An adequate blood supply is necessary for the survival BCC's. Regression may occur centrally or peripherally. During growth the stroma surrounding the tumour is thin & there is minmal inflammation. Metastases with BCC are rare. Spread commonly occurs via the lymphatic to regional nodes & via the blood stream to the long bones & lungs. Other sites, including the skin, may be affected. Implantation in the lungs may also occur by aspiration of fragments of the tumours in head & neck region.

BCC can demonstrate aggressive local growth, spread, and destruction. BCC always follows the path of least resistance. It is for this reason that invasion of bone, cartilage & muscle is not common and occur last in the disease. Once in a plane there can be extensive spread of tumour & destruction of normal tissue.

CLINICAL VARIANTS OF BCC

I. Nodular BCC (Nodulo ulcerative, Rodent ulcer).

This is the most common variant of BCC. Typically lesion is a small pink or red, well-defined nodule with a transplant appearance and overlying telangectasias, ulceration may also occur. Melanin pigment may be present in visible amount. Although slow growing, these tumors may reach a large size and extend deeply. Thus the term rodent ulcer.

II. Superficial multicentric BCC.

It is commonly found on the trunk and extremities. Areas of spontaneous regression characterized by scarring & hypo pigmentation may be present. Multiple lesions may also be present.

III. Morpheic BCC

It resembles a plaque of morphea (localized scleroderma). Typically, the lesion in is indurated, ivory in colour, with overlying telangectasia. The morphemic BCC in noted for its sub clinical spread and high recurrence rate after treatment.

IV. Cystic BCC

Cystic degeneration in a BCC in not often clinically obvious. They may have clear or blur gray cystic appearance and exude a clear fluid if punctured or cut.

V. Basosquomous Variety (Basal cell carcinoma with squamous metaplasia or metatypical carcinoma)

This variant of BCC is much more aggressive & destructive in its behavior & more likely to metastasize & recur after treatment. The more squamous cells in typical BCC give greatest potential for metastases. The incidence of metastases with this variant is 9.7%

VI. BCC with on aggressive (Infiltrative or micronodular) Growth pattern.

These lesions are flat or only slightly elevated plaques they are ill defined in contrast to the purely nodular BCC.

VII. Premalignant fibroepithelioma of pinkus

This is rare variant of BCC. Lesions situated on the lower back. The typical lesion is a smooth, slightly red, moderately firm nodule that may be pedunculated.

HISTOLOGY

- Nests and cords of atypical basaloid cells with peripheral palisade.
- Individual cell necrosis and mitotic activity.
- Variably mucinous stroma
- Stromal epithelial separation artifact
- Adnexal differentiation common.

TREATMENT

SURGERY

Surgical excision is the most commonly used method of tumour treatment by surgeon. The minimal margin for resection of a basal cell cancer is 2 to 5 mm.

A reasonable surgical strategy in the plan for resection of the lesion at one sitting with delay in the final closure of the surgical defect. The surgical site is dressed with an occlusive, moist dressing and second surgical procedure is scheduled 5 to 7 days later when complex reconstructive options can be planned. In the case of positive surgical margins, immediate re-excision on early reoperation of a suspected recurrence is advised.

MOHS MICROGRAPHIC TECHIQUE

Moh's technique is based on two fundamental principles. First, that all basal cell cancers spread by contiguous growth and second, that all tumour cells must be excised to achieve a cure.

Procedure

There are two techniques:

1. Fixed tissue technique
2. Fresh tissue technique.

1. **Fixed tissue technique**

This technique involves the application of a fixation, zinc chloride paste to a 1.0 to 2.0 cm zone surrounding the clinically evident tumour. In excising the fixed tissue, another, wider margin (up to 2.0 cm) is excised around the areas. This procedure is not used robustly because the fresh tissue technique allows more rapid completion of the procedure without the pain of zinc chloride fixation, and primary repair of the defect.

2. **Fresh tissue technique**

The clinically apparent border of the tumours is outlined with gentian violet and used to generate the surgical map with reference to regional anatomy. The affected area is painted & draped. The area in anesthetized by regional or local anesthetized by regional or local anesthesia

The tumour is debulked by simple curettage. This process may help to reduce the number of excisional layers necessary to eradicate the tumours. The first micrographic specimen is obtained though excision of the base of the lesion in a saucer like manner with a border of 2 to 3 mm of skin from the edge of the defect made by the curette. After the skin is superficially scored in a circle to indicate the area to be excised, a second set of score marks is made at the superior and inferior edges to preserve orientation of the specimen. These superficial incisions are made before the specimen is removed.

The piece of tissue is excised with the blade beveled slightly inward. The angle of the blade become more severely beveled as it cuts deep to the surface until the plane of the incision is horizontal to the skin surface. This thin wafer of excised tissue has a thickness of 2-3 mm & is excised without any holes made in the specimen. The specimen is placed with proper orientation on an anatomically marked transfer card.

The specimen is subsequently prepares for frozen section. To ensure proper orientation of the specimen, a schematic drawing of the surgical defect is

made with reference to the regional anatomic landmarks. Aligning incisions are marked on the diagram. The excised tissue is cut into pieces, each 2 sq cms in size, which fit into the freezing chuck of the cryostat. Each of these blocks of tissue is given number, which is known as section number. Indelible marking dyes are applied to the opposing cut surfaces of the specimen.

Frozen sections are cut sequentially from each section. The technician begins with the first section & continues with each consecutive section. Optimal cutting temperature medium is placed on the cryostat chuck & the specimen is inverted & placed horizontally on the chuck so that deep margin faces up. The specimen molded so that skin margin is elevated until it is in the same plane as the rest of the specimen. The whole specimen in flattened with the heat extractor. The tissue is sectioned at a thickness of 4 to 8 mm. the first sections processed represent the deepest & most lateral margins & are most important. The section are dried on a slide warmer and stained with methylene blueor hemotoxylin-eosin. After the sections are stained, a cozen slip is applied & each slide is carefully examined microscopically. If the residual tumour is present, the patient returns to the procedure room for removal of areas with persistent tumour. In this second stage of the surgical procedure, the excisions is performed in a manner similar to the first stage.

3. **Curettage & electrodessication.**

The tumour is debulked using curette down to the normal tissue, which is indicated by the sound on curettage in contrast the tumour is soft & friable. Electrodessication is used to destroy any residual tumour cells & produce hemostasis. Used in superficial BCC, nodular BCC, selected cases of Bowen's disease, keratoacanthomas and hypertropic actinic keratoses.

Disadvantages & Contraindications.

- Aggressive histology like Morpheic BCC, recurrent BCC & SCC.
- Larger tumour more than 1 cm especially on face, recurrent.
- Longer duration of disease as they have tendency to become more infiltrative and break down into smaller more invasive tumour with fibrosis.
- Tumours in high –risk areas like skin cancers along the embryonal fusion planes, which have the potential for deep invasion and higher rate of recurrence. Those areas include mid face under the eyes, preauricular & post auricular areas and the Para nasal, nasolabial & inner canthal areas.

4. **Cryosurgery (Liquid Nitrogen)**

the advantages of the technique are that it requires no local anesthesia; there is no bleeding & scarring in very acceptable. Disadvantages include the lack of a specimen of a specimen for pathological evaluation and healing period

that may take weeks. Morpheform, infiltrative and recurrent lesions have a lower rate of cure with cryosurgery methods.

5. **The carbon dioxide laser**

It is used on a cutting & cauterizing tool for the surgical removal of skin cancer. Nd YAG laser may be useful for field destruction because its light is absorbed deeply in the skin.

6. **Topical Chemotherapy:**

5 FU, function as a local autotoxin agent and promotes on enhanced immunologic response at the local level.

7. **Topical & oral retinoids**

These treatments have been shown to produce tumor regression but not complete cure.

8. **Intralesional interferon**

Lesions are injected with 1.5 million 1U of interferon depot – alpha 2b, there times per week for 3 weeks. Cosmetic results are excellent.

9. Photodynamic therapy

Hematoporphyrin or delta-amino levulinic acid makes tumour cells photosensitive to light of 625-630 mm. A disadvantage is patient remains photosensitive for weeks, requiring avoidance of sunlight.

10. **Systemic Chemotherapy**

Cisplatin appears to be more effective. Temporary regression & palliation are goals of treatment.

11. **Radiation therapy**

effective treatment generally involves 200 rad fractions administered 5 days per week for a total dose of 5000 rads. Reduction is then performed with an additional 1000 rads delivered to the region at greatest risk for recurrence.

Indication for adjuvant radiation therapy

- Extra capsular spread of tumour beyond lymph node capsule
- Nerve invasion
- Vascular invasion
- Lymphatic invasion
- Close margin (<1mm)

Disadvantages: They include capability or histological assessment, poor margin control in ill – defined tumours & radiation dermatitis.

T N M Staging of Cutaneous Cancer AJCC Primary tumor staging, criteria for confirmer of the skin.

Criteria.	**Criteria.**
Tx	Not assessable
To	No evidence of primary tumor
Tis	Carcinoma in situ

T1	Tumor 2 cm in greatest dimension
T2	Tumor>2 cm & <5 cm in greatest dimension
T3	Tumor >5 cm is greatest dimension
T4	Tumor invades deeper structure (Cartilage, bone, muscle)

AJCC Staging criteria for regional nodes of the head & neck.

Stage	Criteria
Nx	Not assessable
No	No evidence of lymphatic metastases
N1	Mitosis in single ipsilateral lymph node <3cm in greatest direction
N2a	Metastasis in single ipsilateral lymph node >3cm but <6cm
N2b	Metastases is multiple ipsilateral lymph nodes <6cm
N2c	Metastases is bilateral or contralateral lymph nodes <6cm.
N3	Metastases to any lymph node > 6cm.

Stage Groupings for carcinoma of the skin.

Stage	Tumour	Node	Metastases
O	Tis	No	Mo
I	T1	No	Mo
II	T3	No	Mo

III	T4	No	Mo
	Any Y	N1	Mo
IV	Any T	N1	M1

RECOMMENDED TREATMENT FOR SUBTYPES OF BCC

1. Nodules > 1cm (Not in high risk area): Cryosurgery (may need to combine with curettage, are only for lesion <2cm). Excision radiation, Mohs surgery (If>2cm).
2. Superficial Multicentric: Shave excision with curettage, currttage and electrodessication, 5-FU (may need to use with curettage or occlusion), Cryosurgery, excision (poor choice if multiple lesion on large lesion, and especially if on trunk), Radiation (extremely superficial X ray required; not a usual and preferred method of treatment), Mohs surgery (if recurrent).
3. Morpheiforn: Mohs surgery, Excision (only if mohs surgery is not available), Radiation.
4. Aggressive growth pattern (Any location): Mohs surgery, Excision, Radiation.
5. Field fire: Mohs surgery (allow wound to on its own if possible), Excision, & cryosurgery are poor alternatives especially if possibly dealing with recurrent BCC.

6. Recurrent: Mohs surgery, Excision (only if mohs surgery not available)
7. Incompletely cured: Re excision in conventional manner or by mohs surgery.
8. Unresectable and advanced disease: Cisplatin + Doxorubicin + Radiation,
9. Systemic Metastases: Cisplatin + Doxorubicin; may use with radiation necessary.

MELANOMA SKIN CANCER

DEFINITION:

Melanomas are a product of malignant transformation of melanocytes, which are neural crest cells that produce pigment melanin. It most commonly occurs in keratnised, sun-exposed skin, but may also occur in unusual locations such as the retina of the eye and in mucous membrane of the nose, mouth, anus & uvula.

Epidemiology

The worldwide increase in melanoma has been evident for the past several decades.

Armstrong and English found that rates varied from as low as 0.2 per 1,00,000 in parts of Japan to 40 per 1,00,000 is Queensland, Australia. Asian populations such as those in Hong Kong, Singapore, China, India & Japan experienced the lowest rates. Melanoma incidence has been found to be higher in populations residing closer to the equator, an indication of a positive correlation with sun exposure.

Risk factors:

1. Family history: Between 5% & 10% of melanoma patients have a family history of the disease.
2. Personal Characteristics: Persons with blue eyes, fair & red hair and pale complexion are at higher risk for melanoma. Hair color is easier to evaluate than skin color.
3. Skin reaction to sunlight: People who get sunburned easily and tan poorly are at a increased risk for melanoma. A recent case control study suggests that childhood sun exposure is protective only among those able to tan.
4. Freckling: Freckling whether in childhood or adulthood is a characteristic pigmentation pattern relation to poor sun tolerance and to an increased risk of melanoma.
5. Benign melanocytic nevi: The number of nevi rather than their size is related to higher melanoma risk.

6. Anthropometric indices: Height, weight and body surface area have revealed a significantly increased risk for melanoma with increasing height in both sexes. The tallest persons were at increased relative risk than the shorter men.
7. Immunosuppressive states: Renal transplant recipients may have an increased risk of developing melanoma.

SUN EXPOSURE:

Exposure to sunlight is found to be the most important risk factor for melanoma. Conclusions relating sunlight exposure to melanoma include 1) correlation of melanoma with latitude & measured ultraviolet (UVB) radiation, increases as the latitude decreases, and those living in coastal regions have higher incidence. 2) The apparent protection of racial pigmentation & 3) the changing incidence related to migration.

Recent studies have indicated a decrease of ozone layer by 3% to 7% since 1969. Each percentage decrease in the amount of ozone causes melanoma incidence to increase by 1%.

Fluorescent lights: At short lengths <295 nm, the absorbed dose of UV light from the florescent light may be greater than that from the sun. Chemicals, alone or in combination with UV radiation, can induce melanoma.

Ionizing radiation: People are exposed to large variety of environmental xenobiotics through industrial processes such as food, drugs, cosmetics, air &

water, especially the chlorinated swimming pool water & open swimming water polluted by halogenated compounds.

Occupation: Occupational groups such as chemicals, chemical workers and chemical engineers have been found to have increase risk of melanoma. There is strong correlation between melanoma & high socioeconomic status.

Diet: High intake of fat especially polyunsaturated fat is associated with increased risk. Krik patric et al., examined intake of vitamin A, victory antioxidants, and other dietary components for the risk of melanoma.

PATHOLOGY

BENIGN SIMULATORS OF MELANOMA.

A) Dvsplastic Nevus: In 1820 Dr. William Norris described dysphasic Nevus syndrome. The dysphasic nevus is a histopathologically intermediate step between the junctional Nevus & insitu malignant melanoma. It is also considered a marker for heritable malignant melanoma. A dysplastic Nevus may arise de novo or within pre-existing dermal nevic components. Clinically dysplastic nevi resemble small melanomas. They are macular with an irregular shape or at most, slightly raised with a finely pebbled surface. The most striking feature is heterogenecity.

Size	:	often > 7mm
Number	:	Many (>50-100)

Symmetry	:	Symmetric
Erosion \ulceration	:	None.
Location	:	usually trunk > limbs >head
Symptoms	:	None
Surrounding skin	:	Normal.

B) Atypical Melanocytic hyperplasia

It consists of proliferation of atypical cells. Clinically, these lesions appear as irregularly shaped, irregularly pigmented tan / brown macules.

C) Spitz nevus

The compound nevus of spitz was first described by Sophie spitz. It is a benign Melanocytic nevus. The characteristic lesion is dome shaped, caused by a proliferation of nevomelanocytes in a variably hyperplastic epidermis. The characteristic architecture of spitz nevus consists of large, superficial nests, which become smaller nests in the deeper portions. Clinically, the compound nevus of spitz usually presents as pink tan, rapidly growing papule no greater than 0.5 to 1.0 cm in size. One helpful sign is discoscopy.

D) Spindle cell Nevus of Reed

A variant of spits nevus described by Dr. Richard Reed. The pigmented spindle cell nevus occurs on the dorsal surface predominantly in young women. It is a uniformly pigmented dark papule with a flare of pigment.

RADIAL GROWTH PHASE MELANOMA.

Definition: Radial Growth phase melanoma describes the predominantly intrapidermal proliferative phase of malignant melanoma. The cells comprising the intraepidermal proliferation are similar in morphology to the dermal cells. Mitosis is common in the intraepidermal component.

The radial growth phase of cutaneous malignant melanoma of all forms consists of a gradually expanding macular to slightly maculopapualar lesions in skin that is associated with micro invasion by single cells or small nests of cells. These lesions are not associated with risk for metastases & are virtually 100% curable by surgery.

TYPES OF RADIAL GROWTH PHASE MELANOMA:

1. SUPERFICIAL SPREADING
2. LENTIGO MALIGNA
3. ACRAL – LENTIGINOUS / MUCOSAL

MELANOMA SITU:

Melanoma in situ refers to a purely intraepidermal process of malignant melanocytic proliferation. The clinical lesions of melanoma in situ usually resemble the lesions of the radial growth phase of either superficial spreading melanoma or acral lentiginous melanoma, but without areas that are pigmented and palpable.

VERTICAL GROWTH PHASE:

Vertical growth phase describes the onset of a process in which cells originating in the epidermis have the capacity to form an expansile nodule in the papillary dermis and / or infiltrate the reticular dermis and subcutaneous fat. The vertical growth phase can arise de nova. In which case the lesion is designed Nodular Melanoma.

VERIANTS OF VERTICAL GROWTH PHASE MELNOMA:

MINIMAL DEVIATION MELANOMA: It is a histological variant of malignant melanoma and is thought to have intermediate prognostic implications. It may contain an intraepidermal or radial growth phase compinent.

If a radial growth phase component can be diagnosed as superficial spreading melanoma, lentigo maligna melanoma, or acral lentiginous melanoma and contains an expansive nodule with minimal deviation features in the dermis, the lesions designed Malignant Melanoma.

NEVOID MELANOMA: Histologically appears benign but behaves with aggressiveness. Two predominant architectural patterns are observed (1) a dome-shaped pattern (spitzoid) and (2) a verrucoid pattern.

DESMOPLASTIC MALIGNANT MELANOMA (DMM): It is another variant of vertical growth phase malignant melanoma. First described by Conley et al in 1971. DMM frequently presents clinically as a pink to tan dermal or subcutaneous nodule in the head and neck region of elderly persons.

The hallmark histologic feature is the thick fibrotic collagenous stroma in the dermis, within which malignant spindle cells are arrayed.

HISTOLOGIC PROGNOSTIC PARAMETERS:

1) DEPTH OF INVASION OF THE TUMOUR:

Anatomic Levels of invasion (Clark): The anatomic levels of invasion describe five levels based on anatomic landmarks. Level I is intraepidermal proliferation of melanoma cells without any invasive component – melanoma in situ. Level II is single – cell infiltration of the papillary dermis or infiltration by small nests of cells of the same size as the intraepidermal nests. Level III is a widening of the papillary dermis by an expansile nodule of tumour cells that compress the reticular dermis. Level IV is a broad infiltration of single cells into the reticular dermis. Level V is infiltration of tumour cells into the subcutaneous fat.

CLARK'S MICROSTAGING CRITERIA

Level	Anatomic Depth of invasion
I	In situ melanoma confined to epidermis
II	Invasion into papillary dermis
III	Invasion to but not into reticular dermis
IV	Invasion into reticular dermis
V	Invasion into sub dermal fat

2. LINEAR DEPTH OF INVASION: The linear depth of invasion according to Dr. Alexander Breslow is the measurement in millimeters from the top of granular cell layer to the deepest tumour cell. In an ulcerated lesion the measured depth of invasion is taken from the depth of the ulcer base rather than from the granular cell layer adjacent to the ulcer. A mucosal melanoma is measured from the most superficial keratinocyte to the deepest tumour cell. Traditionally lesions less that 0.75 mm are considered to be at low risk for metastases and virtually curable by surgery. Lesions in the range of 0.75 to 1.5 (0.86 -1.69) mm are considered to be in the intermediate to low risk range and are associated with onset of the early vertical growth phase. Lesions in the range of 1.5 to 4.0 mm are intermediate to high risk, and lesions greater than 4.0 mm are definitely high risk.

3. PHASE OF TUMOR PROGRESSION: Horizontal or radial growth phase lesions are curable by surgical excision, and lesions in the vertical growth phase have a risk for metastases that sequentially increases with the progressive measured depth of invasion.

4. HISTOGENIC TYPE: Lentigo maligna melanoma was considered to have a better prognosis than other types of melanoma, nodular melanoma has a worse prognosis.

5. HOST RESPONSE: The presence of an inflammatory infiltrate composed predominantly of lymphocytes is a favorable prognostic indicator, Clark et al.

6. REGRESSION: In the vertical growth phase, regression is recognized as areas of dense fibrosis containing macrophages within a given tumour nodule. Regression was found by Clark et al , to have a favorable prognostic significance when defined as the absence of tumors in both the epidermis and fibrotic dermis.

7. ULCERATION: Thicker tumours more commonly ulcerate. Ulceration, greater than 6 mm in depth was shown to have a striking difference in survival at 5 years according to Day and co-workers.

8. MITOTIC RATE: The mitotic rate of the vertical growth phase is a significant parameter. If the mitotic rate was greater than 6/ ^{mm2} the survival rate was 40%, whereas those with less than 6/ mm2 had an 80% survival rate, Kopf and associates.

9. MICROSCOPIC SATELLITES: Day et al found that isolated nests of tumour and measuring 0.05 mm in diameter or greater has a significant effect on survival. The presence of these nests, called microscopic satellites.

TNM STAGING OF MALIGNANT MELANOMA

Classification	Thickness (mm)	Ulceration status
T1	<1.0	a. Without ulceration and level II /III b. With ulceration or level IV /V
T2	1.01 -2.0	a. Without ulceration b. With ulceration
T3	2.01 – 4.0	a. Without ulceration b. With ulceration
T4	>4.0	a. Without ulceration b. With ulceration

Classification	No.Of Metastatic Nodes	Nodal Metastatic Mass
N1	1 node	a. Micro metastasis

		b. Macrometestasis
N2	2 or 3 nod5es	a. Micro metastasis b. Macrometestasis c. In transit metastasis (es) / satellite (s) without metastatic nodes.
N3	4 or more metastatic nodes, or matted nodes, or in transit metastasis (es) / satellite (s) with metastatic node(s)	

M. Classification	Site	Serum LDH Level
M1a	Distant skin, subcutaneous or nodel metastasis	Normal
M1b	Lung metastasis	Normal
M1c	All other visceral metastases	Normal
	Any distant metastasis	Elevated

Clinical Staging				Pathologic Staging			
0	Tis	NO	MO	0	Tis	NO	MO
IA	TIa	NO	MO	IA	T1a	NO	MO
IB	T1b	NO	MO		T2a	NO	MO
	T2a	NO	MO		T2a	NO	MO
IIA	T2b	NO	MO	IIA	2b	NO	MO
	T3A	NO	MO		T3a	NO	MO
IIIB	T3b	NO	MO	IIB	T3b	NO	MO
	T4a	NO	MO		T4a	NO	MO
IIC	T4b	NO	MO	IIC	T4b	NO	MO
III	Any T	Any N	MO	IIIA	T1-T4a	N2a	MO
					T1-T4a	N2a	MO
				IIIB	T1-T4b	N2a	MO
					T1-T4b	N2a	MO
					T1-T4a	N1b	MO
					T1-T4a	N2b	MO

					T1-T4a,b	N2c	MO
				IIIC	T1-T4b	N1b	MO
					T1-T4b	N2b	MO
					Any T	N3	MO
IV	Any T	Any N	Any M	IV	Any T	Any N	Any M

RECOMMENDED SKIN MARGINS FOR WIDE LOCAL EXCISION FOR MELANOMA

Primary site	**Thickness**	**Recommended Margin**
Extremities	< 1mm 1 – 4mm > 4 mm	1 cm 2 cm 2-3 cm
Head and neck	All	1-2 cm
Scalp	All	2-3 cm

ELECTIVE NODE DESSECTION

The rationale for ELND is based on the concept that melanoma metastasizes sequentially from lymph nodes to distant sites

Malignant melanoma can disseminate though both the lymphatic and hematogenous routes. For intermediate thickness lesions, the proportion of lymphogenous spread is higher than for thicker lesions. ELND be applied to patients younger than 60 and those with primary lesions 1 to 2 mm thick or combination of these two parameters, ELND has also been used for staging purposes.

ELND should not be performed on

1) Patients whose primary malignant melanoma are in situ or have a maximal thickness of less than 1mm. the incidence of regional lymph node dissection is not justified.
2) Patients whose primary malignant melanomas are in the midline of the head and neck or the trunk. Bilateral nodal dissections in these two regions of the body in the absence of a clearly demonstrable therapeutic advantage are not justified. Whether radioisotopic studies will greater the definition to this group remains to be seen.
3) Elderly patients with serious intercurrent disease. They should not undergo ELND unless primary is very thick and lies directly over this nodal group.
4) Patients with systemic metastases.

SENTINEL NODE BIOPSY:

Morton et al, developed the method of intraoperative lymphatic mapping and sentinel node biopsy. The sentinel node identified and subjected to frozen section. When the node is negative the procedure is terminated. When the sentinel node is positive, about 37% probability exists that additional node(s) are positive and therefore a complete node dissection is required.

THERAPEUTIC NODE DISSECTIONS:

1) Groin Dissection
2) Axillary node dissection
3) Neck dissection
4) Popliteal node dissection

ADJUVANT TREATMENT FOR PATIENTS WITH POSITIVE REGIONAL NODES:

Adjuvant treatment with interferon -2b increases significantly, the 5 years disease free survival of patients from 26% to 37%

INTRANSIT METASTASES:

Local recurrence is considered as that occurring with in 2cm of the surgical scar of the primary site. It should be treated with wide resection as a primary lesion, supplemented ideally by regional perfusion or infusion. Intransit metastases are those occurring beyond 2 cm from the primary site and are usually

muttiple, they may occur at any place between the primary site and the regional lymph nodes as skin or subcutaneous nodule.

The standard treatment is hyperthermic perfusion. Residual lesion is resected. Intralesional bacillie Calmette – Guerin some has been used in the past with some success among immunocompetent patients. Intransit lesions of the trunk are not amenable to regional chemotherapy.

RESECITON OF DESTANT METASTASIS:

Malignant melanoma disseminates widely through the hematogenous route. It can involve any organ or tissue. Patients likely to benefit from resection of distant metastases are those with a relatively small number of lesions. Surgery for distant metastatic disease is also indicated for the palliation of symptoms.

ISOLATED HYPERTHERMIA AND CHEMOTHERAPY PERFUSION:

The goal was to deliver the maximally tolerated chemotherapeutic does to a regionally confined tumour area while limiting systemic toxicity. Isolation was further enhanced by appropriately placed proximal tourniquets.

DRUGS AND DOSAGES:

Melphalan (Alkeran), known as pheylalanine mustard, has been the drug of choice. Malphalan is usually given in 150 to 200 – mg aliquots into the arterial line at 3 –min intervals. Melphalan perfusion is continued for 1 hour. The M.D Anderson staging system is used because it fits the clinical situation for limb melanoma.

M.D ANDERSON STAGING SYSTEM

Stage	Definition
I	Primary diseases
II	Local recurrences or satellites within 3cm of the original tumour
III A	Intransit diseases more than 3cm from primary tumour
IIIB	Regional node involvements
IIIAB	Intransit diseases and positive nodes
IV	Distant diseases metastases (includes positive iliac of supraclavicular nodes)

CHEMO IMMUNO AND RADIO THERAPIES IN THE TREATIMENT OF MELANOMA:

I. CHEMOTHERAPY:

The best –studied single agents for the treatment of melanoma are dacarbazine (DTIC) nitrosoureas, interleukin – 2, and interferon – alpha

COMBINATION CHEMOTHERAPY:

Three –drug combination have been developed that incorporate a vinca alkaloid such as vincristine into DTIC based combinations.

Two drug combinations of DTIC and cisplatin, DTIC and vindesine, vindesine plus eisplatin, have been evaluated.

Two combination regimens – BHD, which consists of carmustine (BCNU) hydroxyurea, and DTIC and BOLD, which includes bleomycin, vincristine. Lomustine (CCNU) and DTIC, induce response in 30% to 40% of treated paints.

Combination regiment reported to have a responses rate of 50% to 55% contains DTIC, CISPLATIN, BCNU AND tamoxifen (Darthmouth regimen).

BIOLOGIC THERPY:

The combination of chemotherapy plus cytokines is called biochemotherapy or chemoimmunotherapy. Its underlying rationale is the combining of active agents with potentially different and complementary mechanism of action.

II IMMUNOTHERAPY: In 1908, Paul Erlich proposed the possibility of using the immune system to eradicate tumour. Therapeutic manipulation of the immune system can be broadly designated immunotherapy.

III RADIATION THERAPY:

Sufficient biologic and clinical evidence now exists to refute that melanomas are uniformly radiation resistant.

Radiation therapy has been established as a simple and cost effective treatment modality for palliation of patients with symptomatic metstatic spread.

PREVENTION OF SKIN CANCER:

There is broad consensus in the scientific community supporting the etiologic importance of sun exposure for both melanoma and nonmelanoma skin cancer.

STAGE BASED INTERVENTIONS

There is an emerging consensus among behavioral scientists that matching intervention to the stages of change can enhance the efficacy and generalizability of treatment programs. These include:

1) Sun Sensitivity Assessment and Feedback
2) Sun scanner
3) Sun damage instant photography

BASIC FACTORS IN EARLY DIAGNOSIS:

To make a diagnosis, the physician must have a high index of suspicion and through knowledge of :

1. The clinical characteristics of early malignant melanomas.
2. The clinical features of the common types of pigmented lesion.
3. The characteristics of precussor lesions-e.g.Dysplastic nevi and certain congenital melanocytic nevi

4. Other factors that increase the risk of developing a malignant melanoma –e.g. Familial history of malignant melanoma, many melanocytic nevi, excessive sun exposure, light coimplexion, history of sunburns and susceptible age.

Detection includes ABCDs (Asymmetry, Border irregularity, Color variation, Diameter enlargement).

DANGER SIGNS OF MALIGNANT MELANOMA:

Charge in Color, Change in Size, Change in Shape, Changes in Elevation. Change in Surface, Change in Surrounding Skin, Change in Sensation, Change in Consistency.

A complete annual examination of the skin by a physician is recommended for everyone, supplemented by monthly self – examinations by the patient.

SELF-EXAMINATION OF THE SKIN:

Routine self-examination of the skin is inexpensive, non invasive, and free of danger. The patient takes part of the responsibility for identifying early malignant melanomas of the skin at time when such lesions curable most early malignant melanomas are macular and grow in diameter for some time before they become elevated. Flat lesions are almost always curable, whereas lesions that develop plaques, papules or nodules have a greater risk for metastases. The goal is to recognize early malignant melanomas when they are flat and curable.

Malignant melanomas " write the message in the skin with its own ink and therefore all the easy to see. Some see do not comprehend" Dr. Neville Davis.

MATERIALS AND METHODS

All the 40 patients admitted in surgical wards of GRH Madurai as cases of skin malignancies during the period of November 2005 to October 2007 are included in our study as time bound study.

Inclusion criteria:

1. All patients admitted to surgical wards as cases or primary cutaneous malignancies.
2. patients willing for surgery.

Exclusion criteria:

1. Patients of secondary cutaneous malignancies
2. Patients of cutaneous sarcoma
3. Patients treated on OPD basis.
4. Patients not willing for surgery.

Methods:

All the 40 patients admitted in the surgical wards as cases of cutaneous malignancies during our study period were included in the study. Method of study consists of

1. Detailed history and physical examination
2. Relevant systematic examination
3. Histopathological diagnosis
4. Study of various modes of treatment.

Histopathological diagnosis:

The diagnosis of the cases was confirmed by histopathology. Edge biopsy was performed in large ulceroproliferative growths and subjected to histopathology. The enlarged regional lymph nodes were either subjected to the FNAC of the lymph nodes or excisional biopsy.

Routine Investigations:

Hb%, urine routine, blood urea and serum creatinine were the basic investigation performed in all cases.

Radiological Investigations:

Investigations like Chest X-ray, ultrasound abdomen were performed when indicated.

OBSERVATION AND RESULTS

TABLE NO.1 Age and Sex distribution of primary skin malignancies

Age	Sex		Total	Percentage
	Male	Female		
30-39	5	1	36	15%
40-49	6	2	48	20%
50-59	8	4	12	30%
60-69	3	1	4	10%
70-80	8	2	10	25%
Total	30	10	40	100%

Above table shows age & sex distribution of skin cancer in our study. There is male preponderance with 80% of male being offense with compound to 20% females M.F. ratio being 4:1.

TABLE 2: OCCUPATION

Age	Sex		Total	Percentage
	Male	Female		
Agriculture	20	2	22	55%
Coolie	10	-	10	25%
House wife	-	6	6	15%
Railway Employee	2	-	2	5%
Total	32	8	40	100%

This table shows agriculture field worker (55%) suffers more skin cancer than any other form of occupation. This is due to increased rate of sun exposure and thus ultraviolet radiation on working outside in the field for long hours.

Chart 2: Skin cancer distribution in different occupation

TABLE -3: TYPE LESION

Symptom	Total	Percentage
Ulcerative	8	20%
Ulceroproliferative	22	55%
Nodule	6	15%
Swelling with ulcer	4	10%

The above table shows type of the lesion, which predominates in my study. Of the four type ulceroproliferative (55%) is most common. Rest of the type has got almost equal presentation.

Chart 3: Type of Lesion

TABLE 4: SITE

SITE	SCC	BCC	MM
Face & Neck	6	8	--
Back & Gluteal ragion	10	-	--
Leg	2	2	--
Foot	8	-	4

This table shows sites of distribution of skin cancer. In general lesions of lower extremity 40% predominate followed by face (35%). The highest density of nin melanoma skin cancer & melanoma in both sexes was on the usually exposed parts of the head and neck & the lowest density on rarely exposed sites.

TABLE NO.5: PREDISPOSING FACTORS

Factors	SCC	BCC
Chronic ulcer	9	2
Chronic Sinus, osteomyelitis	1	--
Albinism	-	1
Chronic sun exposure	16	7

In our study chronic sun exposure (65%) was the most common predisposing factor found. We had one patient with albinism with basal cell carcinoma over face. Here all the patients are agricultural field worker and coolies and are considered as chronic sun exposed patients.

TABLE 6: Lymph node

Lymph node	SCC	BCC	MM
Insignificant	8	0	
Significant	6	0	2

In our study totally 40% cancer had lymphadenopathy in that 8 cases (20%) were significant and 8 cases were having insignificant lymphadenopathy.

SIGNIFICANT LYMPHADENOPATHY

SCC can metastasize to regional lymph nodes at the rate of 30%. But in our study it was found to be 24.4% of SCC. Because of the less no of cancer studied & for only one year duration, lymphatic spread can't be compared to the standard.

TABLE 7: Incidence of primary malignant tumor of the skin from NOV 2004 to OCT 2007.

Malignant tumor	Total	Percentage
Squamours cell carcinoma	26	65%
Basal cell carcinoma	10	25%
Malignant melanoma	4	10%
Total	40	100%

Of the malignant tumor 26 were SCC and 10 were BCC & 4 were malignant melanoma. In our study the results are not correlated with the

European & American population statistics in which basal all carcinoma is the commonest.

OBSERVATION OF SQUAMOUS CELL CARCINOMA

TABLE 8: SQUAMOUS CELL CARCINOMA AGE INCIDENCE

Age group	No.Cancer	Percentage
30-39	2	7.7%
40-49	2	7.7%
50-59	10	38.4%
60-69	4	15.4%
70-80	8	30.8%

Regarding the age wise incidence in our study, it is more concentrated in the age group between 50-80 years. The youngest patient suffering from SCC was 30 yrs old and the oldest was 80 yrs old. The highest incidence was in 50-60 age groups.

TABLE 9: SQUAMOUS CELL CERCINOMA SEX INCIDENCE

The number of male cases affected is 22 & 4. SCC is 5 times more prevalent in males compared to females. The increased ratio can be explained by increased out door activity.

SCC	No of cases	Percentage
Male	22	84.6%
Female	4	15.4%

TABLE 10: Anatomic site

Site	SCC	Percentage
Face & Neck	6	23.1%
Back	-	-
Thorax & upper extremities	-	-
Gluteal region	10	38.4%
Lower extremities	5	38.4%

In our study SCC is most commonly seen in lower limbs followed by face & gluteal region.

OBSERVATION OF TREATMENT OF SQUAMOUS CELL CARCINOMA

Squamous cell carcinoma comprises lesions all over the body with maximam occurrence at the lower extremities followed by face. In our study all the cancers were treated either curatively or palliatively.

The operability of the cancer were identified mainly by site & size of the primary tumors, number of lesions, tumour border, & involvement ofunderlying structure like bone & muscle. The palpable lymphnodes were subjected to FNAC. And those, which were negative for malignancy, were treated with antibiotics.

In our study majority of primary tumors were treated with wide local excision with split skin Grafting (WLE & SSG). There were two cases of varrucous carcinoma, which were treated by WLE & SSG.

Two cases were presented with lesion on the fore foot and they were treated with transmetataral amputation.

Two patients with lesion over the leg were treated with WLE with reconstruction with calf flap. The flap was well taken.

One patient with lesion over right leg, which was very large, was treated with Above Knee amputation.

Two patients with lesion over left side of the nose was treated with WLE and forehead flap. This cancer was positive for tumour margin.

The patient was given two cycles of cisplatin and 5fU followed by radiotherapy. Case was followed for 3 mths with no local recurrence.

Two patient were presented with significant inguinal lymphadenopathy. One patient was treated with radiotherapy to inguinal nodes & another with chemotherapy using CIsplatin and 5 – FU

In our study, low incidence of the amputation shows acceptability of the other forms of treatment over debilitating surgery.

OBSERVATION AND DISCUSSION OF BASAL CELL CARCINOMA

TABLE 11: Basal Cell Carcinoma Age Incidence

Age Group	No.Of cancer	Percentage
30-39	4	40%
40-49	4	40%
50-59	-	-
60-69	-	-
70-80	2	20%

Basal cell carcinoma presents a great diversity of appearance & is known as great imitator. It is the most common skin malignancy noted in western countries. The table shows the distribution of BCC age wise.

TABLE 12: Anatomic Site

Site	No. Of cancer	Percentage
Nose	2	20%
Angle of eye	2	20%
Forehead	2	20%
Neck	2	20%
Lower limb	2	20%

The above table shows the site of BCC. All the lesions are above the line joining the lobule o fear to ala of the nose. There findings are similar to those published by Gold berg H.

TABLE 13: SEX INCIDENCE OF BASAL CELL CARCINOMA

BCC	No. Of Cases	Percentage
Male	6	60%
Female	4	40%

In the study Reizner GT & Chakong T-et al. showed basal cell carcinoma is more prevalent in females than males contrary to squamous cell carcinoma distribution.

Our study does not go in favour of the above study.

TABLE 14: Occupational Incidence of Basal Cell Carcinoma

Occupation	No. of cases	Percentage
Agricultural labour	4	40%
Coolie	4	40%
House wife	2	20%
Total	10	100%

No particular occupation has predilection for BCC even though agricultural labour (60%) are most common in our study.

Presenting complaint

In our study 6 patients presented with ulcer & 4 patients with nodules. Lesions of BCC can present as papule, ulcer & pedunculated mass. In our study no cases had significant regional lynphadenopathy or metastasis. Metastatic BCC is very rare, incidence reported being 0.0028%.

OBSERVATION OF TREATMETN OF BASAL CELL CARCINOMA

In our study we had ten cases of basal cell carcinoma, six males and four females. One patient who was an albino, presented with ulcerative type of BCC over the neck. He was treated with wide local excision and primary closure. Rest nine patients were treated with wide local excision and spilt skin grafting.

A minimum of 5mm margin was given in all patients Chemotherapy & radiation were not used in any of the patients.

OBSERVATION OF MALIGNANT MELANOMA

The lowest incidence rate of 0.1 -0.2 per 1,00,000 person per year were found in India. Incidence of melanoma normally increases steeply until 50 years of age. After that the rate slow down & some studies show melanoma incidence is slightly higher in females. It has increases incidence in people who work outside. All the above statement about the melanoma could not be studied in our series as we got only four cases out of 20 cases.

DISCUSSION OF THE CASE

1. Kannan 48 yr male, presented with ulcerative growth over right great toe for 2 months. Patient gave history of thorn prick to the same site 1 years back.

On examination there was a 10x12 irregular dark brown to black ulcer with everted edge. Surrounding skin was hyper pigmented and had 2 palpable inguinal nodes. Edge biopsy of the ulcer showed malignant melanoma & FNAC of lymph node came as metastatic deposits fo malignant melanoma.

He was treated with below knee amputation withright inguinal block dissection and post operatively given one cycle of Dacarbazine. Patient lost to follow up.

2. Yamuna, 52 Old female, presented with ulcerative growth over left sole for 1 ½ yr.

On examination there was an ulcerative growth 3x2 cm with everted edges. The ulcer was blackish brown in color. She had 3 palpable inguinal nodes. Biopsy of ulcer preved melanoma & FNAC of the lymphnode came as metastatic deposits of melanoma.

She was treated with wide local excision and inguinal block dissection. Postoperatively he was given 3 cycles of dacarbazine chemotherapy. Patient was followed for 3 mths without recurrence.

DISCUSSION

Cutaneous malignancies have always posed a challenge to surgeous, who over the decade have attempted to tackle problem. There has been recent increase in the in the incidence of cutanieous malignancies woeldwide, which is aptly termed as ‘an epidemic of skin makignancies.’ Various modalities of treatment have evolved over time with major advances in recent times.

In our present study 40 patients were admitted to the surgical wards which were evaluated and treatment with various modalities.

In our study the mean age of presentation was 53 years with the commonest age group being 50-60 years. A study by T. Milan et al showed that there was increase in incidence with the increasing age. The high incidence of cutaneous malignancies with the increasing age is due to cumulated risk of sun exposure.

Table 15. Comparision of the age of presentation

Age (Years)	Present study No of cases	Milan et al No of cases
15-29	0	2
30-44	12	10
45-59	14	13
60-74	12	25
75+	2	18

In our study there were 28 male patients of non melanoma skin cancer (77.78%) and female patients (22.22%). According to study by Chuang T Y, PopencuNA et al. Non melanoma skin cancer occurs predominantly in males when compared to females in the ratio of 3:1. Our study goes in favour of that study.

Table 16 comparision of sex incidence

Sex	**Chuang TY, Popencu NA et al**	**Present study**
Male	75%	77.78%
Female	25%	22.22%

In the present study the squamous cell carcinoma predominantly present in the lower llimbs 76.9%. in a study by Bernstein et al the squamous cell carcinoma most commonly presented over head and neck and dorsum of hands

which are sun exposed areas. In the present study basal cell carcinoma 80 percent of cases were preset over the head and neck. This goes in accordance with the study by McCormack C et al, where the most lesions were situated above the line joining the tragus and ala of the nose.

In the present study most common mode of presentation was ulceroproliferative growth (55%). We could not find any other study in support of our finding.

In their study, Kennedy C and Bajdik C D reported that basal cell carcinoma was most common among the non melanoma skin cancer. In the present study squamous cell carcinoma was the predominant (72.22%) which doesn't correlate with the literature. Since the present study involved only a small number of cases and it is a time bound study. The results could not be compared with the other studies.

In their study, Johnson T M et al reported that exposure to UV radiation as the most common and important predisposing factor in the causation of Non melanoma skin cancer. In our study we found that of all the patients of non melanoma skin cancer 22 patients were agricultural workers (55%) and 10 patients were manual labours (25%). Since all these patients were involved in outdoor work, they were considered to be the individuals at risk of skin cancer because of sun exposure. In our study we had 9 patients of squamous cell carcinoma with history of chronic ulcer and 1 patient of history of chronic sinus,

which were considered to be one of the redisposing factors for the causation of squamous cell carcinoma.

In our study all the patients who were clinically suspected as cases of cutaneous malignancies were subjected to edge biopsy of the lesion. The clinical diagnosis was confirmed by the Histopathological diagnosis. The enlarged lympth nodes were subjected to FNAC for the confirmation of tumour metastases. In our study only 4 cases of squamous cell carcinoma were positive for the metastatses (15.4%). According to the literature squamous cell carcinoma metastasizes at the rate of 30%.

In our study conventional surgical excision of wide local excision was the main modality of the treatment for the primary tumour. Because of lack of facility other modalities like Moh's technique, electrodessication and curettage were not used. Moh's technique was the ideal method of treatment for all the lesions, especially those situated over the head and neck. Other field destructive methods like radiotherapy, cryosurgery which are effective in the treatment of small lesions specially those over the face, head and neck which help in preservation of skin. However these techniques will not assure negative margins. A minimum margin of 5mm to 10mm was given for excision of squamous cell carcinoma and minimum of 5mm margin was given for the excision of the basal cells carcinoma. Bradland DG, Zitelli JA, in their study reported that a minimum

of 6mm margin is sufficient in the excision of squamous cell carcinoma and 2mm margin for excision of basal cell carcinoma.

In their study Rowe DE et al showed the comparable efficacy of conventional surgical excision to that of Moh's technique and electrodessication. In their study they reported the recurrent rate of 1-3.1% for Moh's technique, 3.7% for electrodessication and 8.1-10% for conventional surgical excision in the management of non melanoma skin cancer. In our study we did not have any recurrences in our follow up period.

In all the cases the defect after the wide local excision was closed by primary closure or split skin graft. Two patients with lesion over the leg were treated by calf flap for reconstruction. Two patients with lesions over the forefoot were treated by trans metatarsal amputation Two patient with a very large lesion over the right leg was treated by above knee amputation. In our study low rate of amputation showed the efficacy of wide local excision with adequate margin in the management of non melanoma skin cancer.

In our study Four patients of squamous cell carcinoma had metastases to the inguinal lymph nodes. One patient was treated with chemotherapy with cisplatin and 5-FU and the other patient with radiotherapy. One patient with positive surgical margin was treated with chemotherapy with ifosfamide and methotrexate with radiotherapy. There was no local recurrence in that patient during the follow up period of 3 months.

IN our study wide local excision was the main modality of the treatment. By following the minimal surgical margins prescribed by the literature we did not have the single local recurrence during the period of followup.

CONCLUSION

The ubiquitousness, the protean manifestation and clinical importance makes cutaneopus malignancy ideal subject for study. However because of very nature of the disease its variability in sites of presentation, patient seeks consultation in diverse departments. Hence the study compiled in one department, as is the case study, and any statistical conclusions drawn may not fully represent the magnitude of the problem. However the study does serve of gain a prespective of the problem & to gain insight into the subject.

Even though the cutaneous malignancy has not reached the epidemic proportions as in westen countries, the magnitude of problem is large in this part of Tamilnadu. It is highly relevant study for surgeon because of the variety of clinical presentations and treatment possibilities available.

Among the primary malignant tumor of skin the incidence of squamous cell carcinoma was highest at 65% followed by BBC at 25%.

Males (80%) were more affected than females (20%). A high incidence was found in those people who work outdoors.

Lesions on the lower extremity (40%) predominate closely followed by lesion on head & neck (35%).

The incidence of SCC carcinoma was highest in lower limb. This may be related to repeated trauma and infection of that part. The cases in our study are from low socioesconomic status in whom the usage of foot wear is practically non existent, which may lead to repeated trauma.

The BBC occurrence most common in the region above the line joining the ear lobe & ala of nose which is in relation to increased sun exposure.

The squamous cell carcinoma although ubiquitous in distribution, occurs more commonly in lower limb. Trauma leading to a non healing ulcer or healing after months to year with scar formation shows malignant characteristics. The malignancy appears long often the original injury.

The edge biopsy was used as the main stay in the diagnosis. The anatomical site, number of lesion, tumous border, and size of lesion, age of the patient were considered while planning wide local excision. A margin of 0.5 cm to 1 cm was given in all cases. No recurrences were reported in our study.

Four melanoma cases were presented in our study. All the cases had lesions over lower limb with metastases in inguinal lymphnodes. Edge biopsy confirmed the diagnosis in all. Wide local excision with a margin of 2 cm and below knee amputation was performed. In two cases inguinal block dissection was done followed by chemotherapy with Decarbazine.

The future of skin cancer treatment belongs to immunotherapy. Either specific active immunization with appropriate vaccines or adoptive immuno therapy must be based on the principle of immune reaction.

Skin cancer can be prevented by adequate education of the public about the harmful effects of over exposure to sunlight & usage of foot wear which prevents repeated trauma. Early detection of premalignant, suspicious lesions by skin self examination must also be carried out.

SUMMARY

This study was conducted in the surgincal wards of Govt. Rajaji Hospital, Madurai November 2005 to October 2007 to evaluate various clinical manifestations, histopathological diagnosis and various treatment modalities used to treat primary cutaneous malignancies.

40 patients in the age groups of 30 to 80 years were included in our study, detailed history was taken and based on clinical diagnosis of primary cutaneous malignancy, biopsy of the lesions were performed. FNAC or biopsy of regional lymph nodes was performed when they were enlarged.

After confirmation of the diagnosis, must of the patients were treated by wide local excision followed by primary closure or split skin grafting. When the regional lymph nodes enlarged which were positive for tumour, patients were

treated with inguinal block dissection with chemoradiation or chemoradiation alone.

Mean age of presentation was 53%. Males (80%) were most commonly involved compared to females (20%) in the ratio 4:1. Agricultural field worker (55%) was the most common occupational group involved. Ulceroproliferative growth (55%) predominate followed by lesions over face (35%). IN the present study chronic sun exposure (65%) was the most common predisposing factor found. Squamous cell carcinoma (65%) was the most common histological type in our study. In our study most patients (75%) were managed by wide local excision as the sole mode or the part of treatment.

CHART 1 AGE AND SEX DISTRIBUTION SKIN CANCER

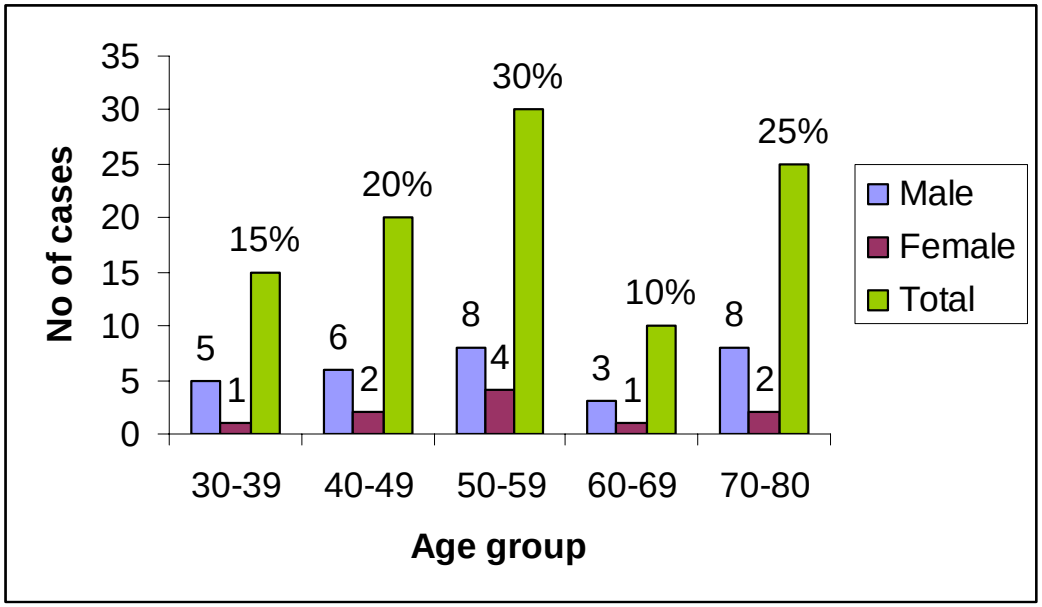


TABLE 2 SKIN CANCER DISTRIBUTION IN DIFFERENT OCCUPATION

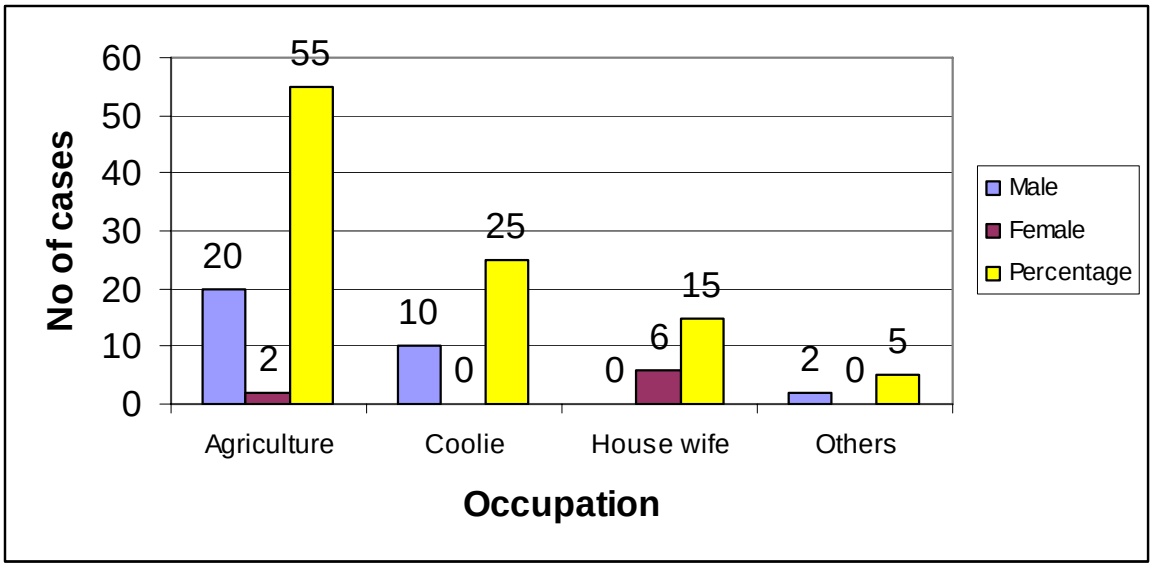


TABLE 3 TYPE OF LESION

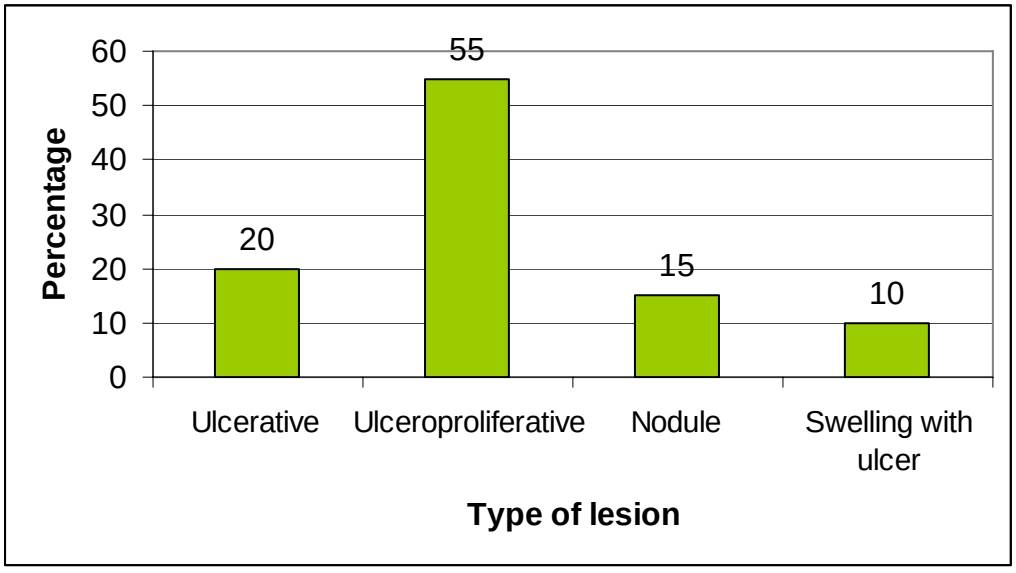


TABLE 4 DIFFERENT SITES IN SKIN CANCER

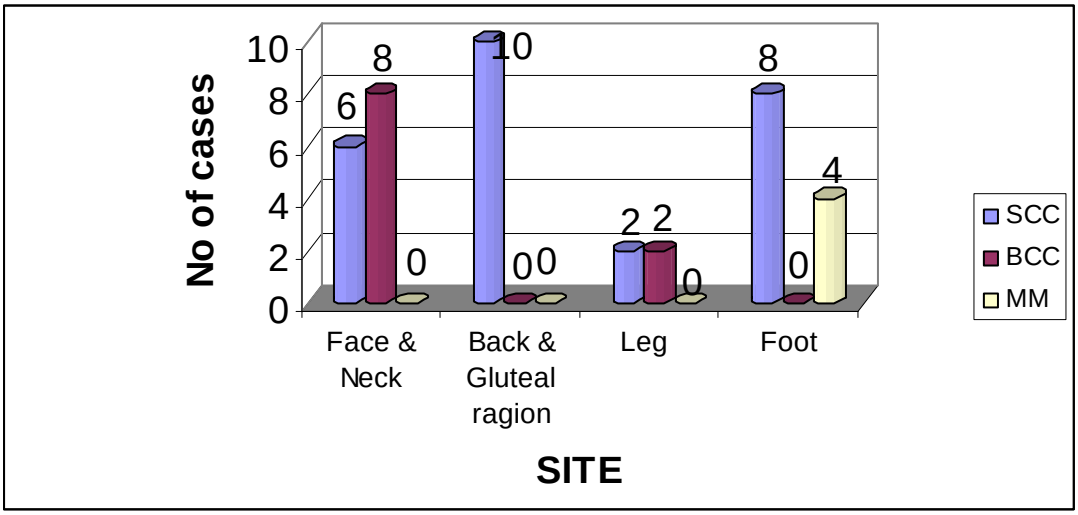


TABLE 5 Incidence of primary malignant tumor of the skin

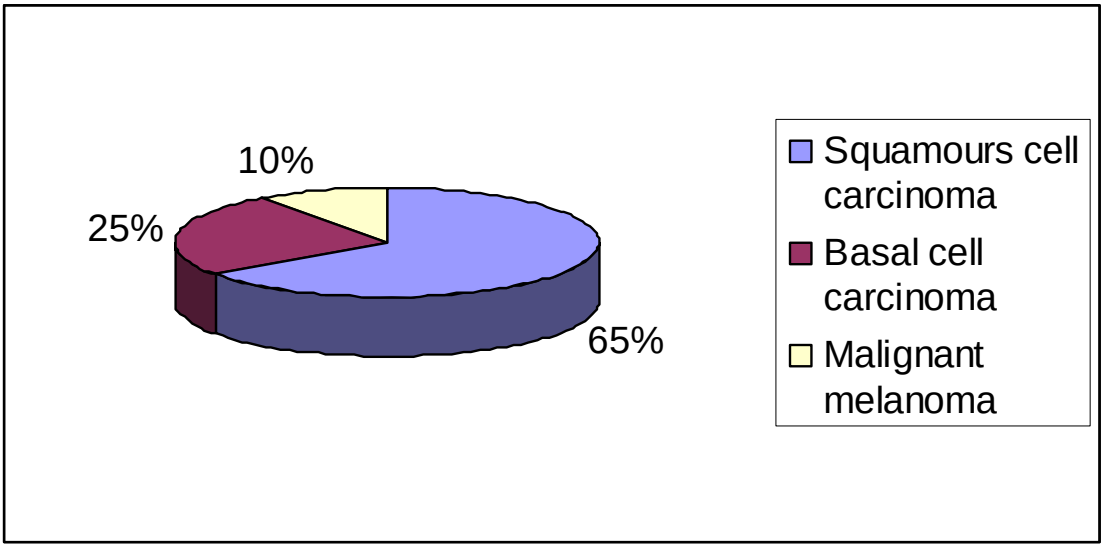


TABLE 6 SQUAMOUS CELL CARCINOMA AGE INCIDENCE

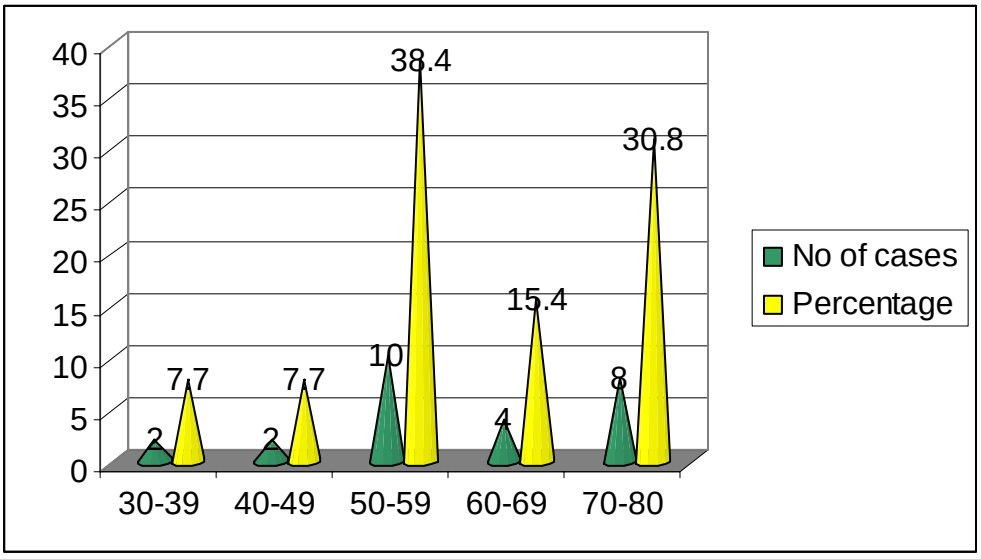


CHART 7 SQUAMOUS CELL CERCINOMA SEX INCIDENCE

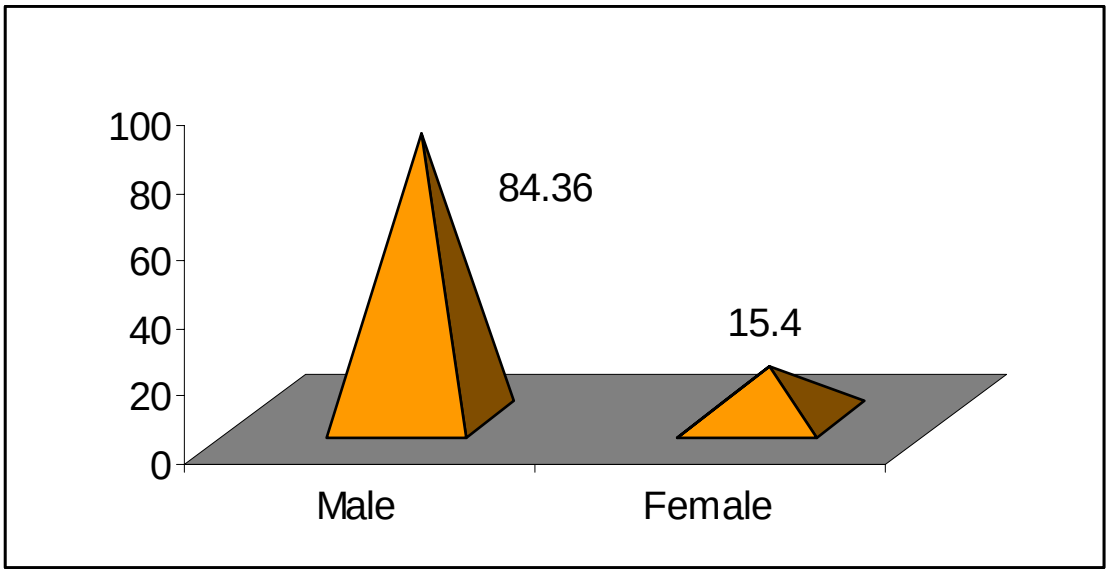
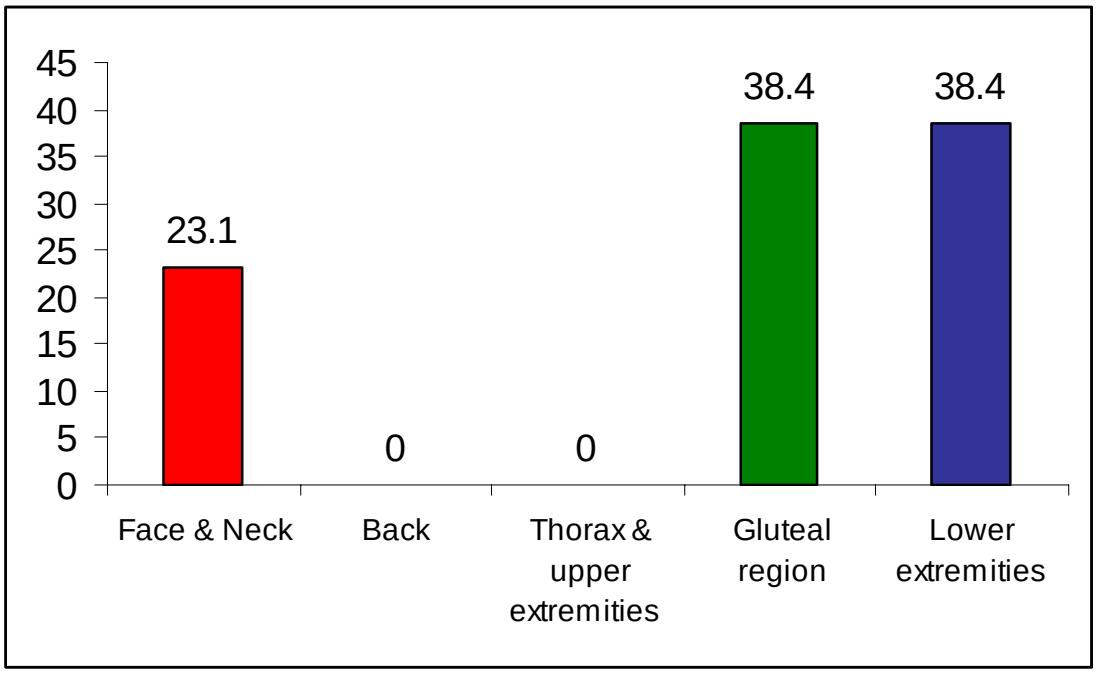
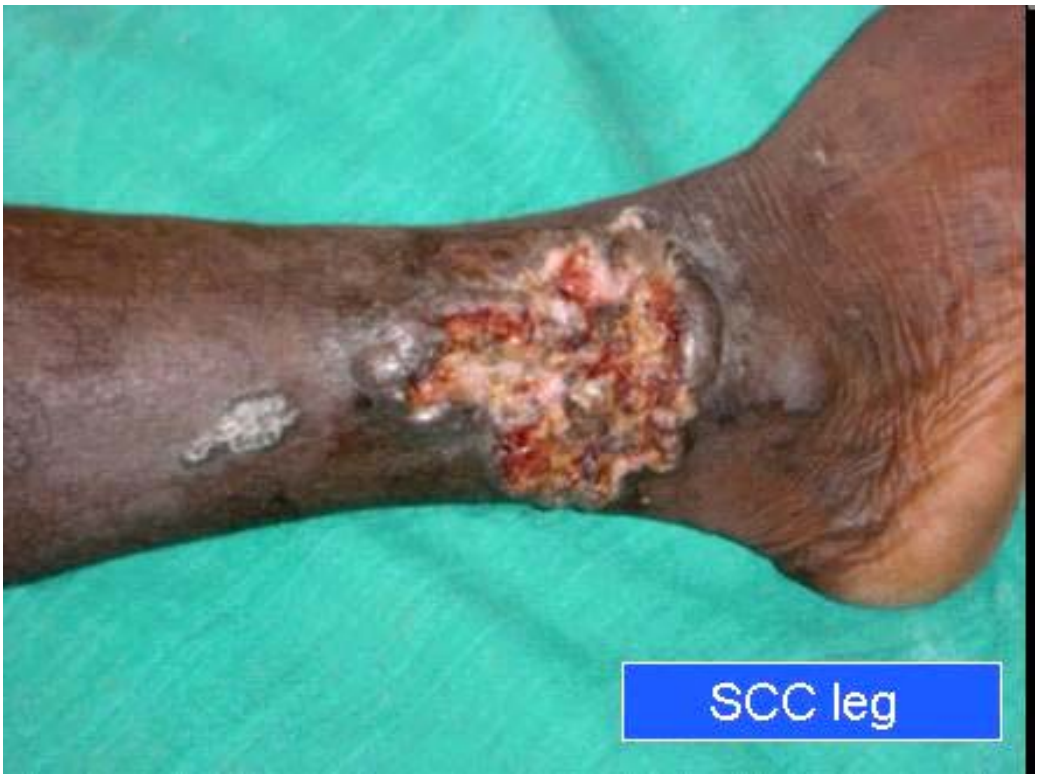
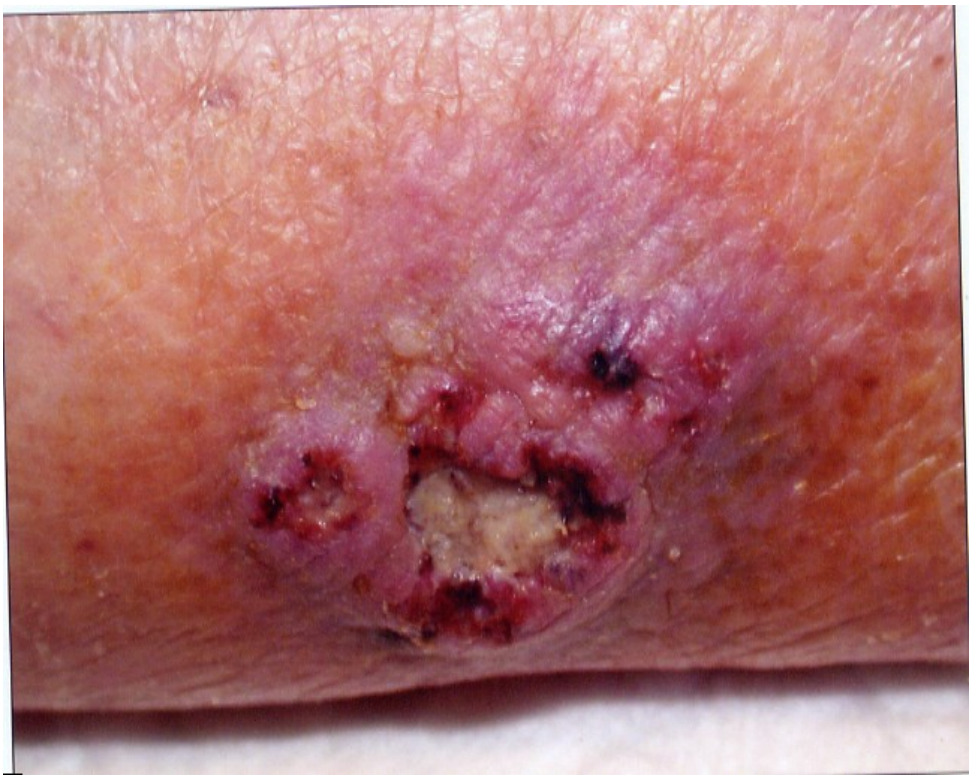


CHART 8 ANATOMIC SITE


45
40
35
30
25
20
15
10
5
0
23.1
0
0
38.4
38.4
Face & Neck
Back
Thorax & upper extremities
Gluteal region
Lower extremities

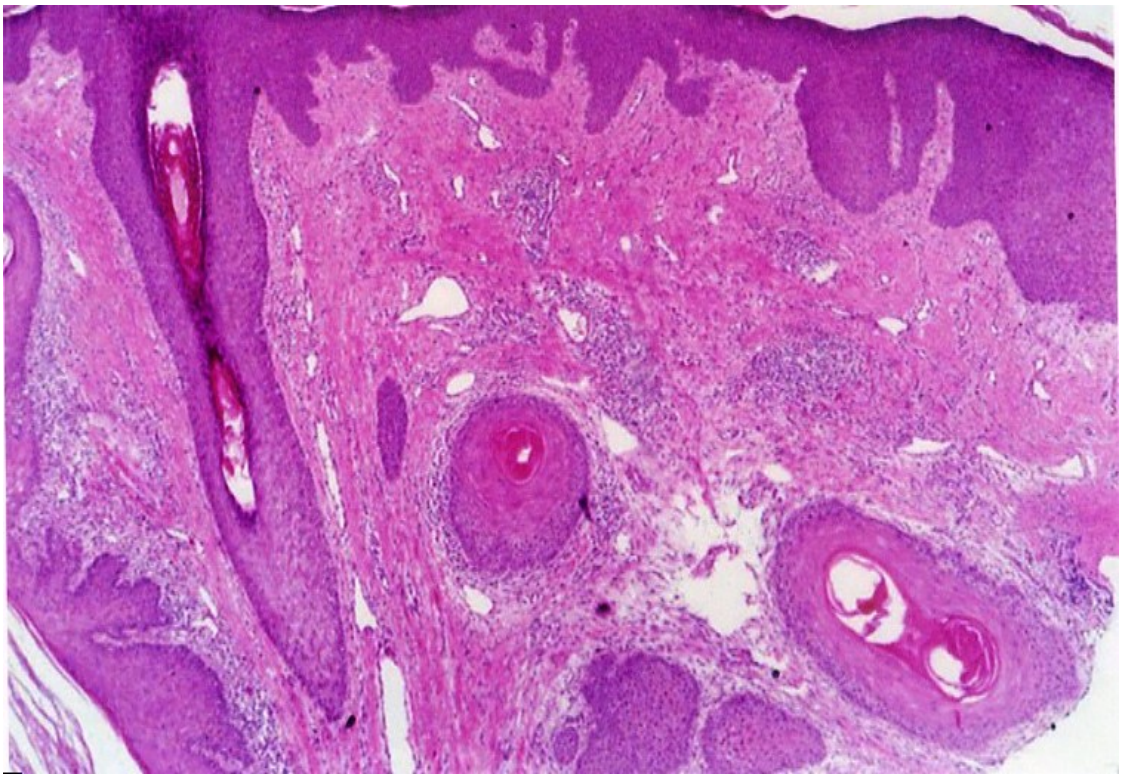


SCC OF LEG

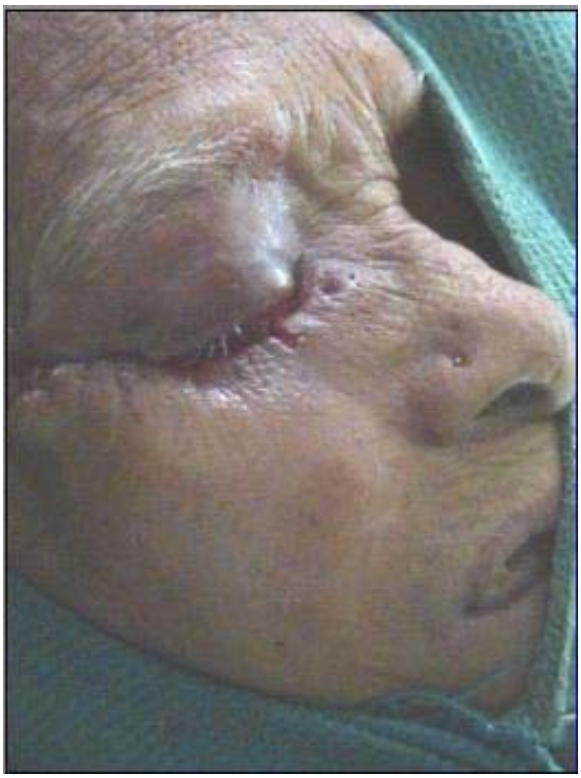



Wide Excision with cortex of Fibula

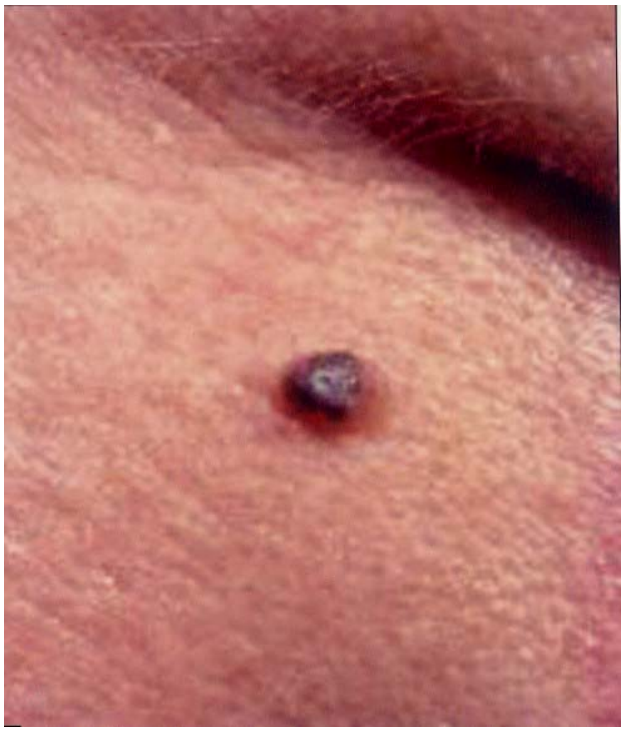
HISTOPATHOLOGY OF SCC



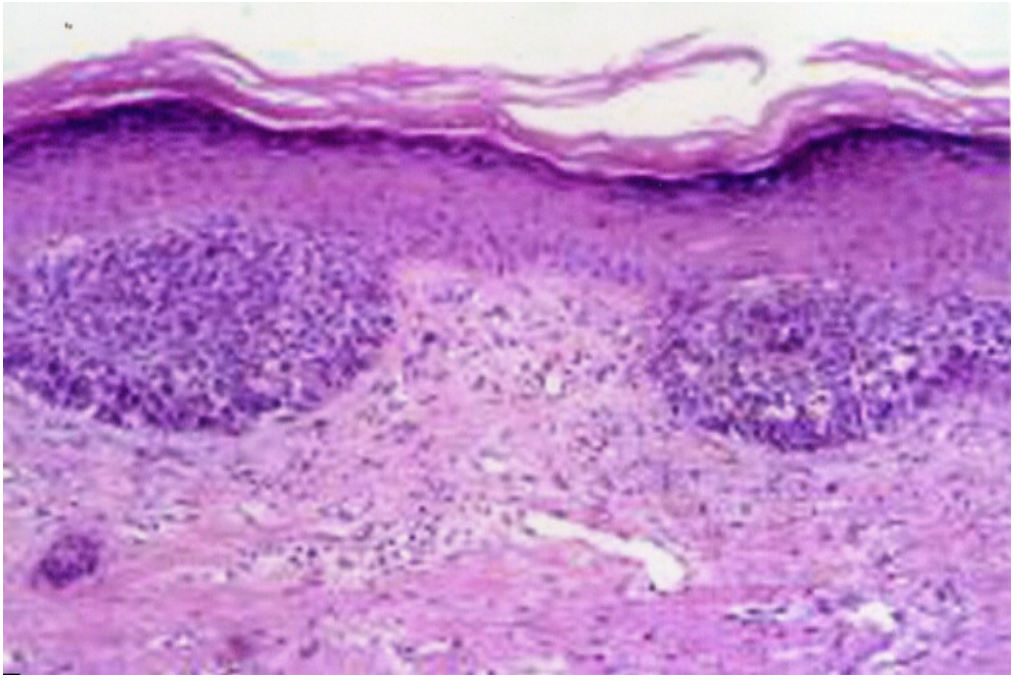
BCC OF INNER CANTHUS



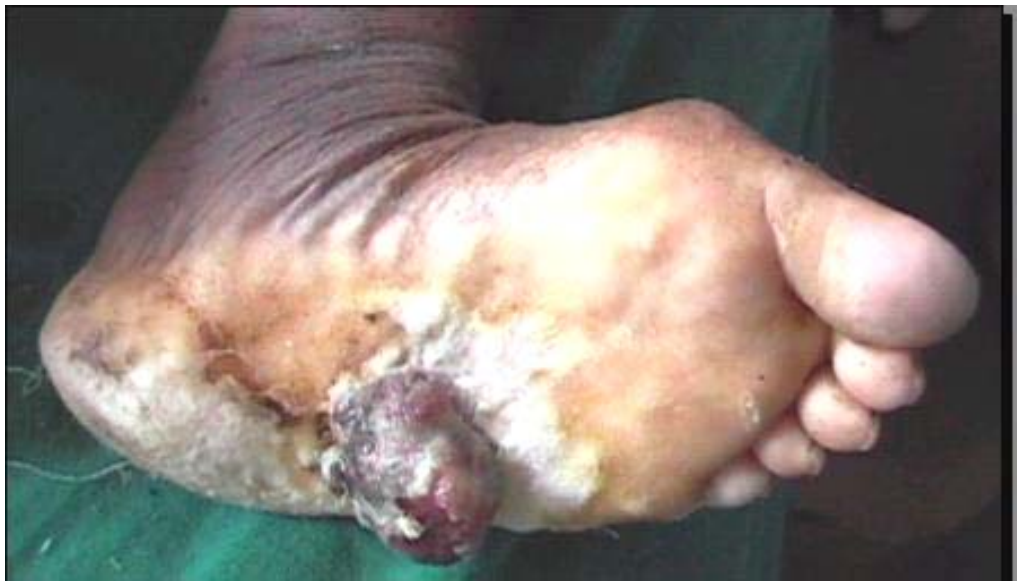
BCC OF FACE

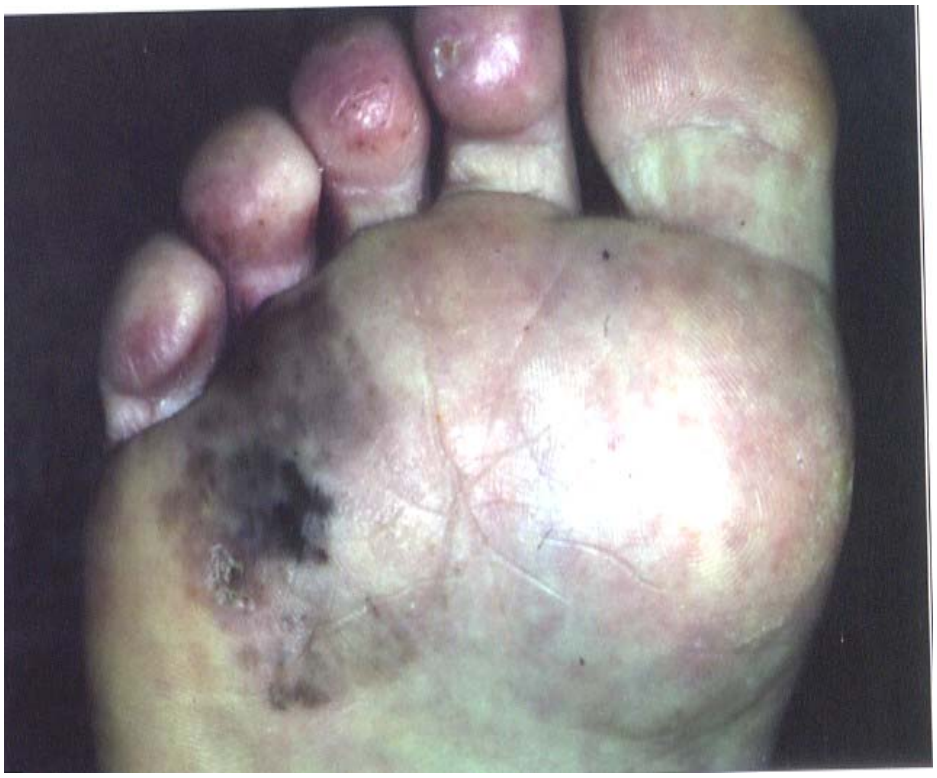


HISTOPATHOLOGY OF BCC

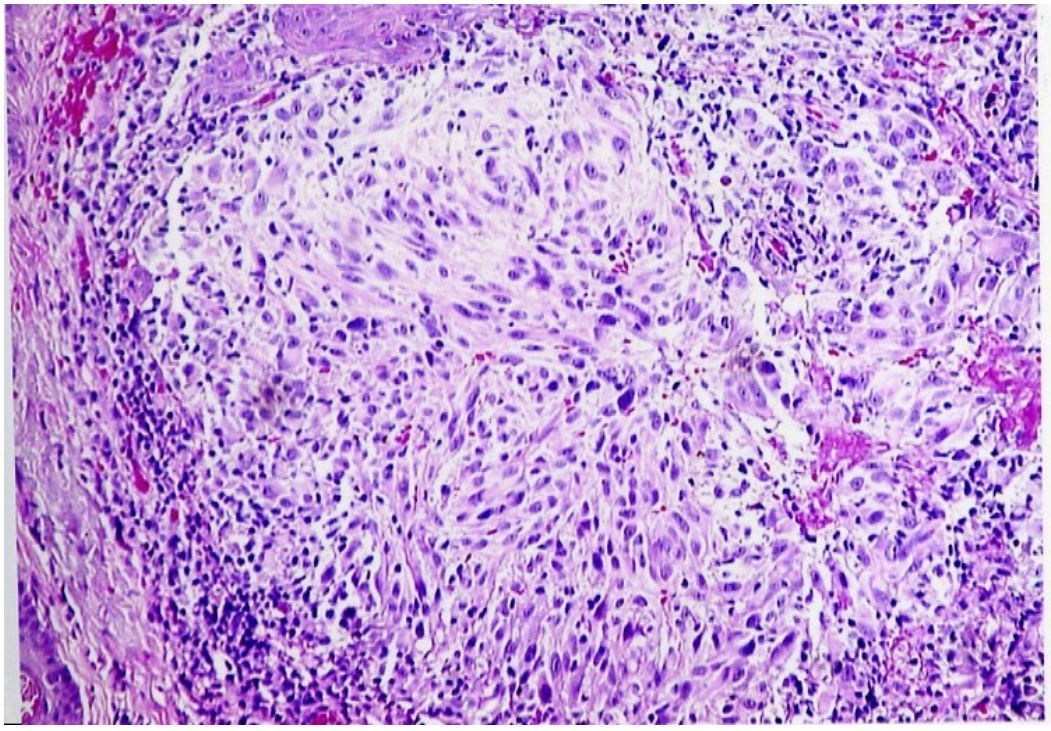


MALIGNANT MELANOMA OF FOOT





HISTOPATHOLOGY OF MALIGNANT MELANOMA



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ANNEXURE

PROFORMA

Name: Age: Sex:

IP No.: DOA: DOD:

Chief Complaints: a. Ulcerative Growth

Nodule / swelling

Discolored patches

H/o Presenting illness:

Ulcerative Growth / Nodule / Swelling / Discolored patches

Duration:

Number:

Mode of onset – Sudden / insidious / previous mole

Site:

Size:

Shape:

Colour:

Progress- Increasing / Remains same

Secondary changes:

Associated with pain:

Associated with fever:

Discharge:

Others:

Past History: H/o similar swelling / ulcer patch

H/o Skin cancer

H/o Radiotherapy

H/o chronic exposure to chemicals

H/o chronic exposure to sun

Family History: H/o similar complaints in the family member

Personal History: Occupation

Habits: Smoking / Alcohol / Tobacco

Sleep: Disturbed / Good

Appetite: Good / poor

Socioeconomic status:

General Physical Examination:

Built: Nourishment: Pulse

BP: Temp: Skin Complexion: Fair / Mod / Black

Icterus: Clubbing: Pedal edema:

Lymphadenopathy: Yes / No

If yes Group: Number: Size:

Consistency: Tenderness: Fixity:

Systemic Examination

Per Abdomen:

Respiratory System:

Cardiovascular System:

Central Nervous system:

Local examination

Ulcerative Growth / Nodule / Swelling / Discolored Patch

Inspection:

Number:

Site:

Size:

Shape:

Colour:

Surface:

Border:

Floor:

Margin:

Edge:

Surrounding skin:

Skin over the swelling:

Discharge:

Satellite nodules:

Palpation: Size:

Shape:

Tenderness: Non tender / tender

Temperature : Reduced / Normal

Surface:

Extent:

Plane of the swelling:

Borders:

Edge:

Base:

Bleeding / friability:

Adjacent structure / skin:

Sensation:

Regional lymphadenopathy: Yes / No

If yes

Group: Number: Size:

Consistency: Tenderness: Fixity:

Clinical diagnosis

Investigation:

Hb% TC: DC: ESR:

Blood group: Alb: Urine Micro: Sugar:

Blood Urea: LFT: S. Bilurbin:

Serum creatinine: T. Protein

FBS: S. Albumin Globulin

Chart X-ray: SGOT:

Ultrasound abdomen:

CT Scan: Chest / Abdomen / brain ETC.

Primary

FNAC:

Secondary LN:

Biopsy:

Post-investigational diagnosis:

Pre operative preparation

Operative procedure:

Histopathological report

Final Diagnosis:

Follow up:

KEY TO MASTER CHART

SCC - Squamous Cell Carcinoma

BCC - Basal Cell Carcinoma

MM - Malignant Melanoma

Agri - Agriculture

AKA – Above Knee amputation

BKA - Below knee amputation

FF - Forehead flap

CF - Calf flap

HW - Housewife

Insig Ing Ln – Insignificant Inguinal lymph node

Ing BD – Inguinal block dissection

PC – Primary closure

R. Leg – Right Leg

R FF - Right fore foot

RE – Railway employee

Reg - Regional

RGT – Right great toe

R glu - Right gluteal region

Sig Ing N – Significant inguinal lymph node

SSG – Split Skin graft

SW - Swelling

UG - Ulcerative Growth

UL - Ulcer

UGP - Ulceroproliferative growth

WLE - Wide local excision

ABSTRACT

AIMS AND OBJECTIVES

To study the various modes of presentation of cutaneous malignancies, clinical correlation with Histopathological diagnosis and the management by following standard procedures in the literature.

MATERIALS AND METHODS:

All the patients admitted in the surgical wards at GRH Madurai as cases of skin malignancies during the period of November 2005 to October 2007 are included in the study. Patients who were not willing for surgery, patients of cutaneous sarcoma, cases of secondary cutaneous malignancies and patient treated on O P D basis were excluded from the study.

RESULTS AND CONCLUSION:

Total of 40 patients (30 males and 10 females) were admitted in surgical wards as cases of skin malignancies. 65% of them were squamous cell carcinoma, 25% were basal call carcinoma and 10% were malignant melanoma. Most common mode of presentation was ulceroproliferative growth and the lesions were most commonly found on the lower limbs (40%) followed by lesions on the head and neck (35%). Most common age group affected was between 50-60 yrs. A high incidence was found in those working outdoors. Early detection of lesion treatment with wide local excision gave satisfactory results.

KEY WORDS: Squamous cell carcinoma, Basal cell carcinoma, Malignant Melanoma, Moh's technique.

List of abbreviations

AKA-Above knee amputation

AK-Actinic keratoses

BCC-Basal cell carcinoma

BKA-Below knee amputation

MM-Malignant melanoma

SCC-Squamous cell carcinoma

SSG-Split skin grafting

Sw-Swelling

Ul-Ulcerative

Ul prol-Ulceroproliferative

WLE-Wide local excision