MALIGNANT SKIN TUMOURS AS SEEN AT K.N.H. OVER A 10 YEAR PERIOD (1977 - 1986)

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D E C L A R A T I O N

CANDIDATE:

THIS THESIS IS MY ORIGINAL WORK AND HAS NOT
BEEN PRESENTED FOR A DEGREE IN ANY OTHER UNIVERSITY

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C O N T E N T S

INTRODUCTION

Carcinoma of the skin as discussed here refers to superficial malignant skin tumours namely squamous cell carcinoma (SCC), Basal cell carcinoma (BCC) and malignant melanoma (MM). It excludes tumours of skin appendages and connective tissues. It is the most common malignant tumour in man. People living to be 65 years old, 40 - 50% may develop at least one skin cancer during their life time (1). Incidence rates vary from country to country. Occurrence incidence in S.A. Bantu males is given as 0.5/100.000 and U.S.A. (Texas) Caucasian males as 239.7 new cases/100,000 (excluding malignant melanoma) (2). For malignant melanoma) the incidence rate is lower. It ranges from 0.1/100,000 in Japanese males to 16/100,000 in Queensland Australia (2). It is now generally accepted that skin cancers are more common in Caucasians than pigmented races, in whom complete reversal of BCC: SCC ratio and no particular predilection for exposed areas is seen (oluwasanmi et al 1969).

Some of the aetiological factors highlighted in Literature are; exposure to ultra violet (UV) light, effect of ionising radiation on skin, chemical carcinogensis, genetic susceptibility, effect of immunosuppression and development of malignancy on scar tissues, old burns, chronic ulcers and fistulae. Development of skin cancer will therefore depend on multiple factors. That UV light stimulates development of skin cancer is supported by increased tumour occurrence on exposed areas of the skin (2). Pigmented races who gets sun-burn less easily develop tumour less often than Caucasians. Caucasians who work out doors develop more skin tumours and there is inverse relationship between incidence of developing skin cancer and altitude of residence. It is now apparent that sunlight (290 - 320 nm range of U.V. spectrum) is the main causative factor (2).

The amount of U.V. light reaching the Earth surface is dependent on the Earth's ozone layer, which absorbs light and the dust layer which scatters light. Humidity, wind and high surface temperature increase the amount of skin damage caused by U.V. light. The degree of skin protection against these harmful effects is directly proportional to degree of melanogenesis (2). The properties of melanin pigment that make it photoprotective are; its ability to produce a stable radicle, absorb and scatter radiation. There is epidemiological evidence that sunlight is causative in SCC and BCC. BCC develop mainly in sun exposed areas and incidence is directly proportional to degree of exposure to sunlight (1), (3). Sunlight may also play a role in causation of malignant melanoma. Evidence to support this are; the increased incidence as one approaches the Equator; association of increased incidence with out-door recreational activities, predilection for light haired, blue eyed, fair skined individuals and decreased incidence in Blacks; increased incidence in exposed surfaces and predilection for those individuals with defective repair of sun induced Deoxyribonucleic acid (DNA) changes as in Xeroderma Pigmentosa (XP).

There are however contraversial epidemiologic data and no animal model of U.V. light induced malignant melanoma (1). Epstein et;al. suggests that repeated U.V. light injury to the skin may lead to faulty DNA repair and subsequent errors in post ultra-violet light semi-conservative DNA replication and this could lead to increased frequency of malignant change through mutation.

Ionising rediation namely alpha, Beta, gamma and X-rays are associated with causation of skin cancers. Of these, alpha particles penetrate the skin poorly and only produce erythema and pigmentation without sequealae. Beta particles and electrons have been shown to produce acute skin damage with chronic sequalae of atrophy, telangiectasia and lose of skin appendages. Their carcinogenic effect is established in animal experiment in mice, rats and man. (1). Gamma and X-rays are most significant in causing carcinoma. Only low energy X-rays and gamma raysare absorbed by skin to any extent. Point of maximum transfer of energy in high voltage therapy is not at skin surface but at some point below. People at greatest risk of developing skin cancers from exposure to x-rays are those who have long term repeated occupational exposure such as fluoroscopy, deep x-ray therapy with skin absorption, surgicial x-ray therapy for skin effects and accidental exposure to low energy radiation. SCC and BCC develop follwoing radio-dermatitis and the incidence varies from 10 - 29 per-cent (1). Latent period between exposure and development of carcinoma is 4 - 40 years with an average of 7 - 12 years. In man there is no quantitative relationship between developing cancer and amount of roentgen radiation delivered to the skin surface. (1).

Chemical carcinogens were noticed as far back as 1775, when coal tar soot was found to be associated with scrotal carcinoma. Oil or petroleum fraction must have 4 - ring or 5 - ring polycyclic aromatic hydrocarbon to be carcinogenic. Arsenic causes arsenic keratosis and increases the incidence of developing BCC.

Genetic susceptibility to developing skin cancer is shown by the increased incidence and early age of presentation of skin cancer in albinism, an autosomal recessive condition in which the individual lacks enzyme tyrosinase and is therefore unable to elaborate - melanin pigment. Among the blacks, the albinoes have higher incidence of skin cancer than their pigmented counterparts. Xeroderma pigmentosa is a genetic condition where the individual has defective DNA repair following U.V. light injury. The increased incidence of skin cancer in these individuals is higher than in the unaffected population in all races. Malignant melanoma occurs early and is multiple in individuals with positive family history.

There is increased predisposition to developing skin cancers at sites of ulcers, scars and burn scars, chronic cutaneous fistulae. The African is said to show the importance of "mechanical" conditions which cause chronic damage to the skin as discussed above. On the other hand the "whites" demostrate the importance of chronic damage to the skin due to U.V. radiation. However in Nigeria, Oluwasanmi found only 7 per-cent of SCC associated with ulcers. The time interval between burns and development of skin cancer is 7 - 35 years, Lovel et;al. reviewed 500 cases of chronic osteomyelitis and found SCC in 1.8% (1), (3), (4).

Immunosuppression either for renal transplant or for systemic disease may predispose to development of skin cancer. The method of causation being either direct carcinogenisis of the drugs or through their effect in altering the immunological surviellance of the host.

They could therefore potentiate effects of solar irradiation or other carcinogens like oncogenic viruses. A renal transplant patient with resistant Herpes virus infection developed SCC in precisely the same site (1). Westburg et;al reported a case of renal transplant patient who developed 14 SCC; 21 actinic keratoses and 4 keratoacanthomas while receiving azothiaprine and prednisone (5). The incidence of skin cancer in renal transplant patients is seven times more than in controls (1). Mean time of 434 months from transplantation to diagnosis of first cutaneous cancer has been shown (1). These are usually multiple.

Studies of skin cancer done in the tropics among the Black population have tended to suggest that, trauma, chronic ulcers (pyogenic, tropical or varicose) scar tissue and fistulae of chronic nature are among the main predisposing conditions (6), (7), (8). The part played by trauma directly preceding skin cancer is contraversial and needs further research. (2), (9).

The age - sex distribution shows most cancers of skin develop in 30 - 70 years olds. SCC and BCC being more in the older age groups while nodular malignant melanoma is more common in younger people. SCC and BCC are commoner in males, malignant melanoma are common in females with a ratio of 1.5: 1 (1). This higher figure in females is unexplained (2). The disappearance of this difference in mortality figures for malignant melanoma can be attributed to the better prognosis in females.

The diagnosis of skin cancer is usually easy and straight forward in late stages of the disease. Early cases can be confused with other skin lesions which are precancerous or benign.

A histological confirmation is therefore always necessary. Actinic keratosis which occurs in areas of chronic sun exposure, presents clinically as a rough scaly surface on an erythematons base. Histologically it shows anaplasia and cellular disarray limited to lower epidermis and basement membrane remains intact. Most actinic keratosis if left untreated for a long time would progress into SCC, however, this transition may require upto 25 years (2). On the other hand SCC is a malignant proliferation arising from the epidermis. It originates from a neoplastic transformation of epidermal keratinocytes. Clinically the tumour often presents initially as a firm erythematous nodule with indistinct margins. The surface of the tumour often develops crusts (plate I). As the tumour enlarges, there is an increase in diameter and elevation and the lesion often ulcerates. SCC has quite a variable course. There may be slow enlargement with only local invasion or rapid growth may occur with wide invassiveness and great risk of metastasis. SCC arising from old burn scars, osteomyelitis sinus, scar tissue or chronic ulcers is more aggressive.

Basal cell carcinoma arises from pluripotential basal cells of the epidermis or hair follicle. The tumour is slow growing and locally invasive. It is only rarely metastasizing BCC has been described in three types:- nodular ulcerative; pigmented; morphea-like and superficial (plate II).

A melanoma is a highly malignant tumour derived from the melanocyte. The three main histological types are:- Lentigo maligna melanoma; superficial spreading melanoma and nodular melanoma.

Some skin nevi are premalignant, especially the compound junctional nevus. (Plate III).

There has been a steady increase in incidence of skin cancer over the years. In U.S.A. the recent National Cancer survey indicates that more than 400,000 new cases of skin cancer are detected each year and a high proportion of these patients live in the sun-belt areas of United States. (1). Age adjusted incidence rates for melanoma derived from cancer registries in Connecticut, New Zealand, Sweden, Norway and Israel show roughly a doubling of the rates during the past 20 - 25 years (2). These increases may be explained to some extent by improved health services and artefacts in reporting, but there is general aggreement that the rise in icnidence is in the main real.

Mortality rates for malignant melanoma have doubled in white population in U.S.A., New Zealand, Norway and United Kingdom and even trippled in Australia over the past 20 years despite significantly improved diagnosis and treatment and increasing survival rates (2). Death rates from all skin cancers, are also increasing. Detailed analysis of mortality and incidence data for malignant melanoma from several countries seem furthermore to indicate a shift in both mortality and morbidity from the older to the younger age groups. The increased incidence in the younger age groups seems to correspond with observed changes in fashion, sportive activities, number of hours spent in out-door work and leisure in the young population (2), (5). Douglas G. et;al. (in cancer of skin Vol 1 pg 418) states that as their urban population have gained more leisure, dressed more scantly and on the whole enjoyed higher standards of of living in areas where the sea, lakes and rivers are available, their exposure to sunlight has increased

Conversely rural occupation has become more mechanised and this may have reduced exposure to the sun. All in all this subject should be approached with an open mind.

Therapy varies depending upon the type, size and location of the tumour. The passage of time has allowed for a careful evaluation of each of the therapeutic modes available today, namely by the approach of dermatologist, chemosurgeon and plastic surgeon. These are mainly cryosurgery; electrodesication; wide excision and immediate skin graft; radiotherapy and chemotherapy.

Skin cancers namely SCC, BCC and malignant melanoma have a low incidence among the blacks but they are not altogether rare. Generally the case fatality is low such that many clinicians, who after all are the ones who have to record and supply much of the necessary data, might consider that further epidemiologic research, which would consume much in the way of staff, time, facilities and money is hardly justified. To sum up, if we are interested in the therapeutic problem of skin cancer only then, further complex investigation are probably not warranted. If however we look at skin cancer as a suitable and unique research tool which might eventually help us to elucidate the general problem of human carcinogenesis then obviously we should press on.

AIM AND OBJECTIVES.

The aims and objectives of this study is to find the incidence, sex and age distribution, presentation, diagnosis and methods of treatment of skin cancers as seen at K.N.H. The results are compared with those of studies in Black and White populations done elsewhere. Reasons for the differences and similarities observed are suggested. Areas of future study are also identified.

METHODOLOGY.

This is a retrospective study of skin cancer as seen at KNH over a ten year period (1977-1986). 109 case files were collected from our Central records and radiotherapy departments. Out of these 22 cases were dismissed from the study because the clinical diagnosis of malignant skin tumour was not confirmed by histological report. That left 87 cases with proven histological diagnosis.

To collect data a proforma was used. This had all the variables; name, age, sex, occupation, residence, site of tumour, lymphnode involvement, method of diagnosis, type of tumour, past medical history, presentation of symptoms, any investigations done, treatment modality, response to treatment, prognosis and follow up. The collected data was then analysed statistically and presented in form of tables and figures. Together with this, all cases of skin cancer entered in our cancer registry from 1977 - 1984 (8 year period) was analysed in terms of tumour type and presentation by site. A total of 1553 cases of skin cancer were recorded out of 9950 case of malignant tumours registered. Pictures were taken of the different types of skin tumours seen at KNH during 1987, and is given here as plates.

RESULTS.

Age and sex distribution.

Of the 87 cases, the youngest patient was 4½ years old female. She was suffering from xeroderma pigmentosa for which she was followed up at the dermatology clinic. At 4½ years the child developed a chronic ulcer on the scalp which on histology was S.C.C. The oldest patients were both 85 years old males. One had malignant melanoma of right (Rt). index finger with metastases. The other had S.C.C. of Rt. leg.

Figure (Fig). I shows the age distribution of the skin cancers. More than half, the patients 29 (59,2%) out of 49 patients with SCC were in their 5 - 6th decade. For malignant melanoma 13 patients (36%) out of 36 presented in their 6th decade. The two patients with BCC one was 38 years old female and the other 56 year old female. The average age of presentation was therefore 55 years for SCC. and 60 years for M.M. for both male and female.

The sex variation is shown in table I. The ratio for SCC was 1:1 male to female. Malignant melanoma was also 1:1 The two patients for BCC were both females. For all tumours together the ratio was 1:1.

Annual distribution:

The annual distribution is shown in fig. II. The average number of patients seen each year was between 2 and 4 for SCC; 4 and 6 for malignant melanoma, BCC. One patient was seen in 1985 and the other 1986. No case of malignant melanoma was seen in 1983.

Presenting symptoms.

This is shown in Table IIa, IIb, IIc, for SCC, malignant melanoma and BCC respectively. The most common presenting symptom overall was swelling, 33 patients (51.6%) out of 70 presented this way. This was followed by ulceration seen in 23 patients (35.9%) out of 70. In SCC ulceratior was most common while swelling was most common presenting symptom in malignent melanoma. The two patients with BCC both had multiple lesions.

Duration of Presenting symptoms.

Fig. III shows the duration of symptoms in years. This was given in 48 patients (55.2%) out of 87. 18 patients (20.7%) out of 87 presented to hospital with history of less than 1 year. 12 patients (13.8%) had had symptoms for 1 year. And 19 patients (20.7%) had symptoms of more than 1 Year. Presentation to hospital was early for malignant melanoma than for SCC. The earliest presentation to hospital was a 58 year old female who had a rapidly growing blackish tumour that bled easily for a duration of three weeks.

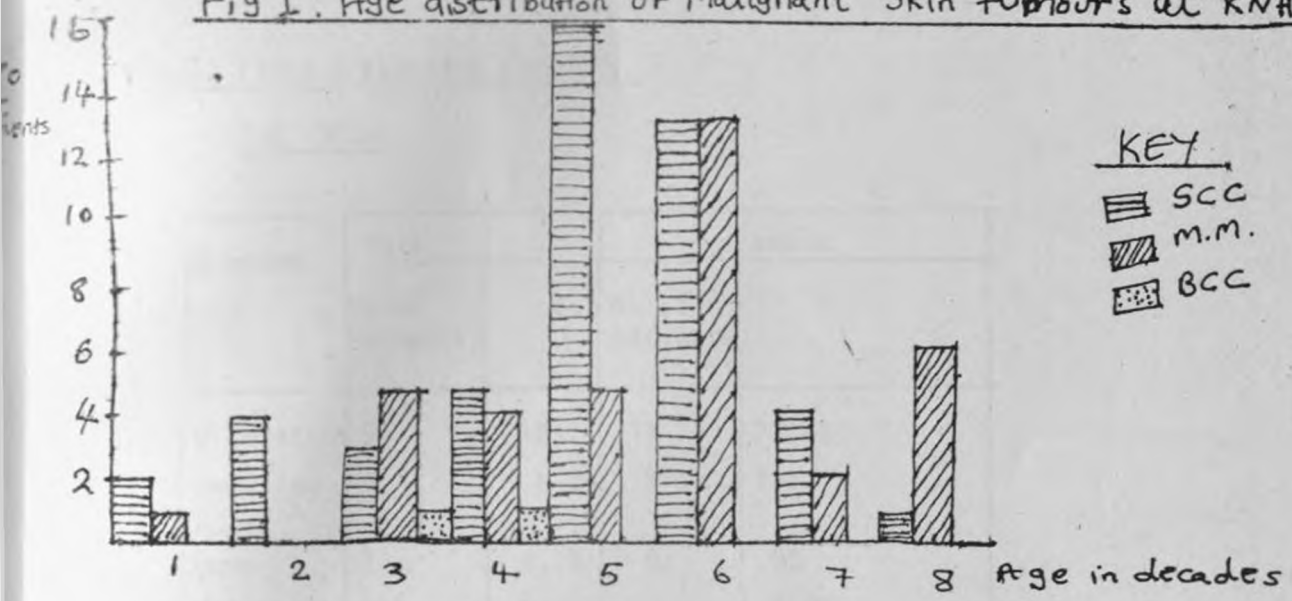
Site.

Site of primary tumour is given in Table IIIa, IIIb, and IIIc for SCC, malignant melanoma and BCC respectively. The majority of SCC were in head and neck 24,patients (48.90%) out of 49 had tumour in head and neck. Out of this 22.4% were females and 26.5% were males. Malignant melanoma was more frequent in lower limbs. 29 patients (80.5%) out of 36 had lesions in the lower limbs. Of these 16 patients (44.4%) were males and 13 (36.1%) were females. BCC presented as lesions on multiple sites in both two patients.

Positive regional lymphnodes. Table IV.

19 patients (38.8%) out of 49 had positive nodes in SCC. of these 7 were females and 12 males. For malignant melanoma 24 patients (66.7%) out of 36 had positive regional lymphnode. Number of male patients Equaled females. There was no mention of involved regional lumphnodes for BCC.

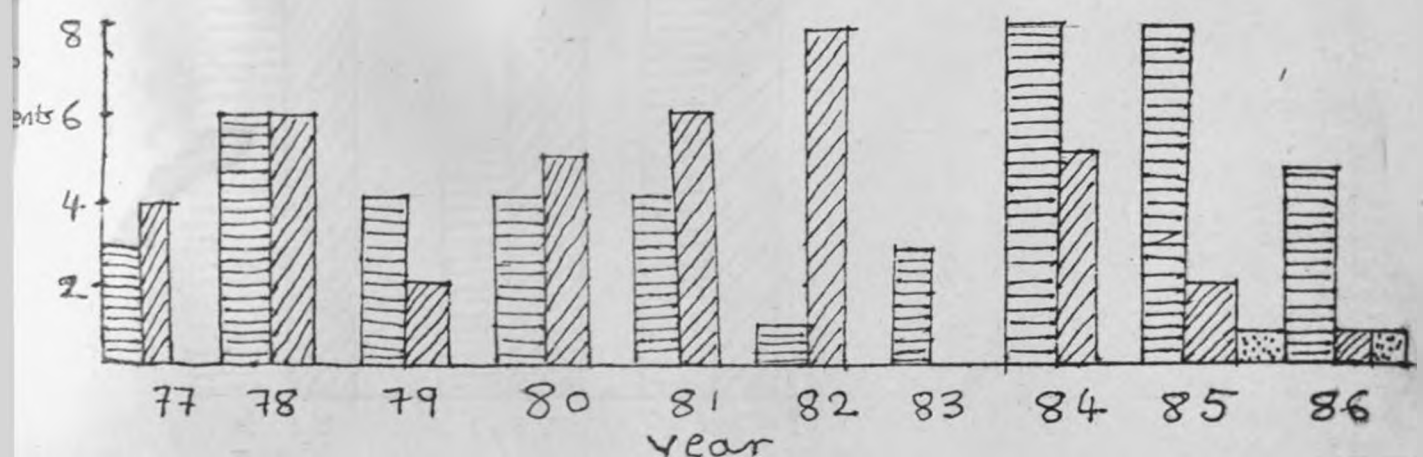
Fig I: Age distribution of Malignant Skin tumours at KNH



Tab. I: Sex incidence.

	SEX.			
TUMOUR TYPE	M	F	TOTAL	M:F RATIO.
SCC	25	24	49	1:1
Malignant Melanoma	18	18	36	1:1
BCC	0	2	2	0:2.
TOTAL	43	44	87	1:1

Fig II: Annual Distribution (1977-86)



Tab II: First presenting symptom.

(a) SCC.

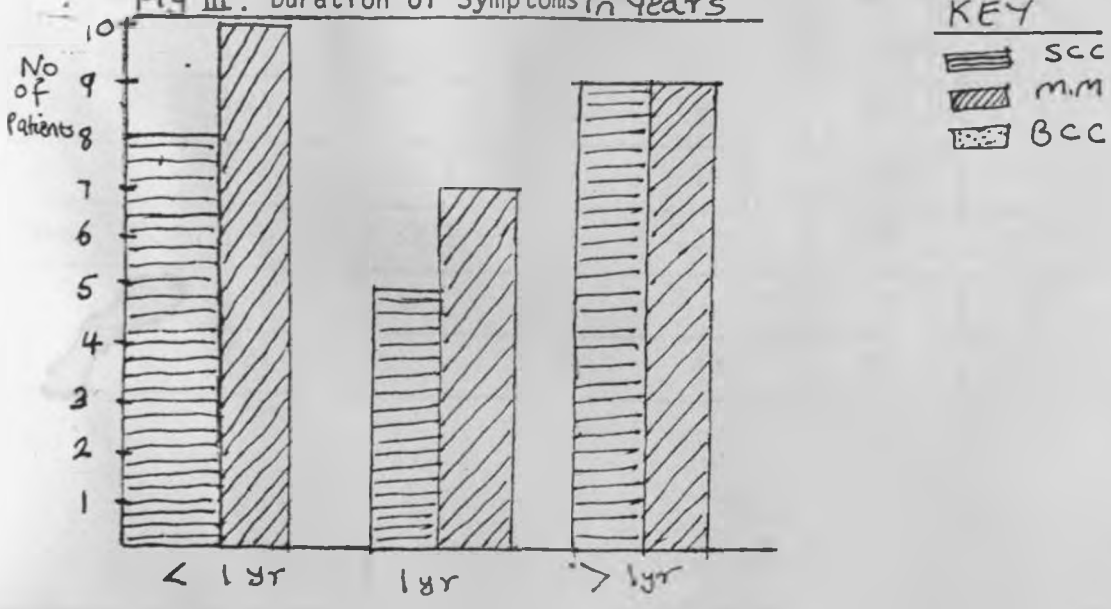
Symptom	Male		Female	
	No of Patients.	%	No of patients.	%
Ulceration	9	18%	11	22.4%
Swelling	4	8.2%	9	18%
Groin Lump	2	4.1%	0	0%
Metastatic	Nil	Nil	1	2.0%
Others	Nil	Nil	Nil	Nil.

b). Malignant melanoma.

Symptom	Male		Female	
	No of patients	%	No of patients	%
Swelling	8	22.2%	12	33.3%
Ulceration	2	5.6%	1	2.8%
Groin lump	Nil	Nil	Nil	Nil
Metastic	3	8.3%	1	2.8%
Pain	1	2.8%	Nil	Nil.

(c) BCC : - all patients presented with multiple lesions.

Fig III: Duration of Symptoms in years



Tab. III: Site of Primary lesion.

(a) SCC.

Site	Male		Female		
	No.	%	No.	%	Total %
Head & Neck	11	22.4	13	26.5	48.9
Lower limbs	10	20.4	9	18.4	38.8
Upper limbs	1	2.0	2	4.0	6.0
Trunk	1	2.0	Nil	Nil	2.0
Penis	2	4.0	Nil	Nil	4.0
Multiple	Nil	Nil	Nil	Nil	Nil.

(b) Malignant melanoma

Site	Male		Female		
	No.	%	No	%	Total%
Lower limbs	16	44.4	13	36.1	80.5
Head & Neck	Nil	0.0	3	8.3	8.3
Upper limbs	2	5.6	1	2.7	8.3
Trunk	Nil	0	Nil	0	0
Penis	Nil	0	Nil	0	0
Multiple	Nil	0	1	2.7	2.7

(c) BCC - all patients had multiple lesions.

Tab. IV: Positive Regional Lymphnodes.

	SEX.					
Tumour	Male		Female		Total	
Type	No.	%	No.	%	No.	%
SCC	12	24.5	7	14.3	19	38.8
Malignant M.	12	33.3	12	33.3	24	66.7
BCC	0	0	0	0	0	0
Total	24		19	43	43	49.4.

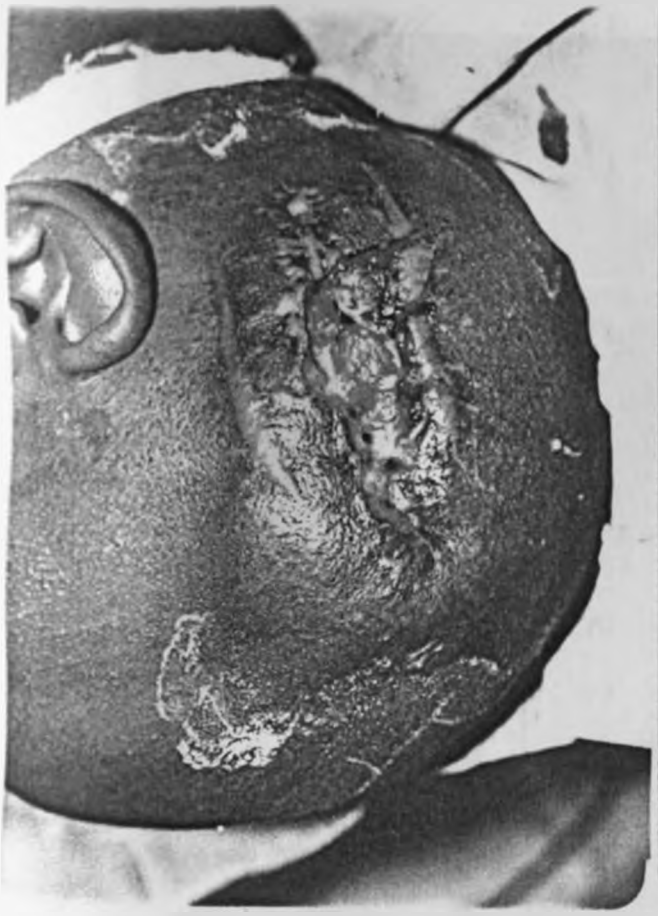


Plate: 2 - Sarcoma of the scalp

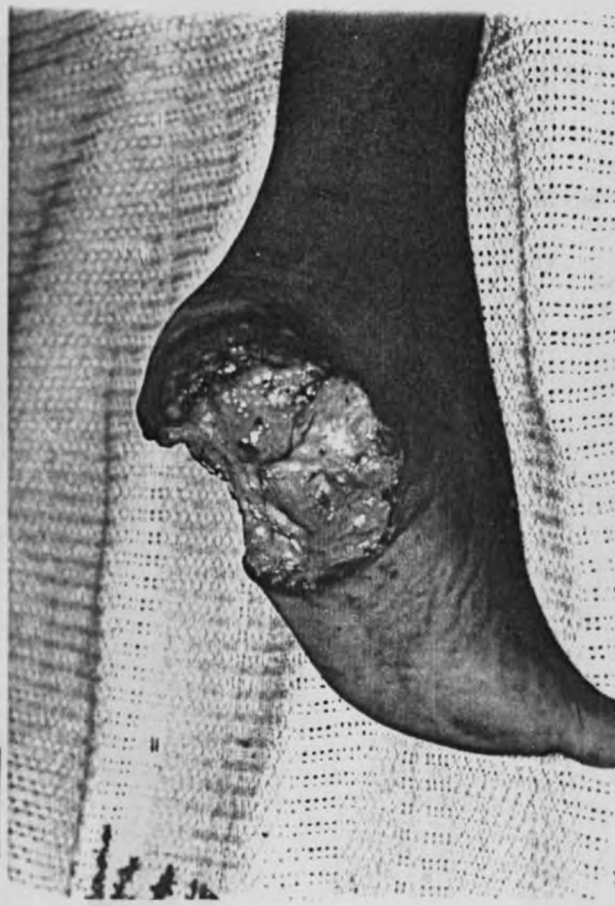


Plate 3: Malignant melanoma in a 20 year old male

Plate 1: Squamous cell carcinoma on the leg of a 65 years old female

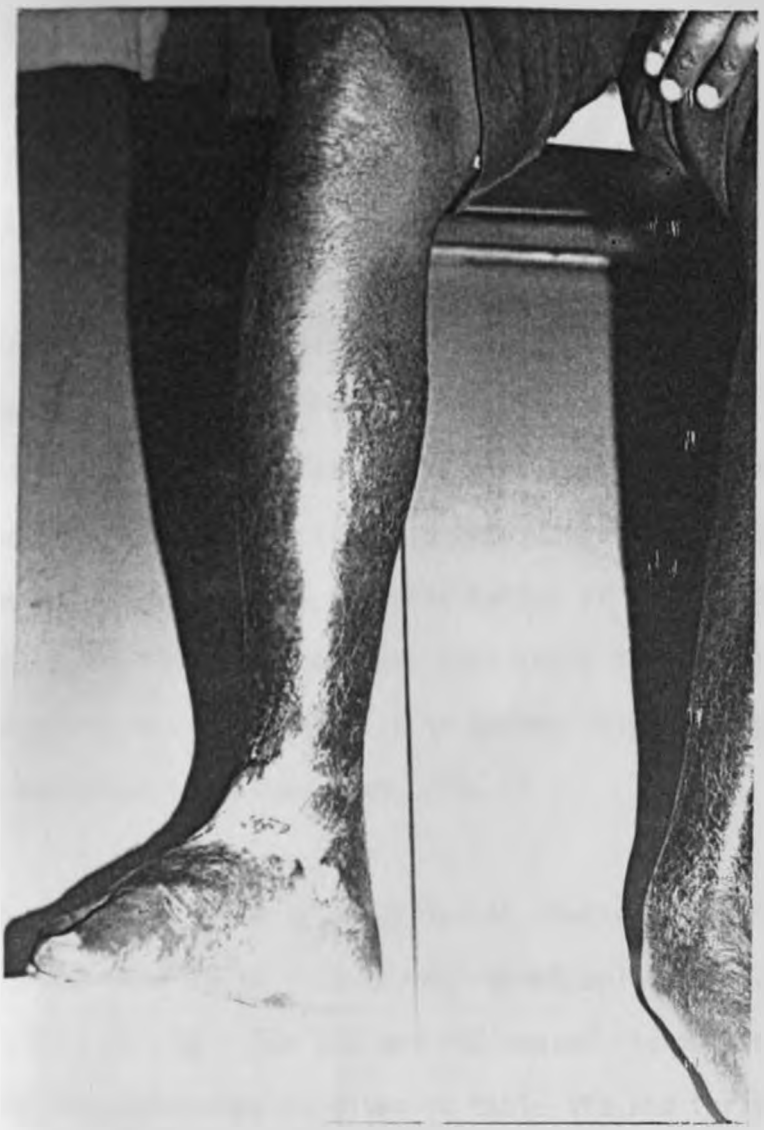


Plate 4: Varicose ulcer in a 55 yr old male Note the varicose veins at the calf medially.

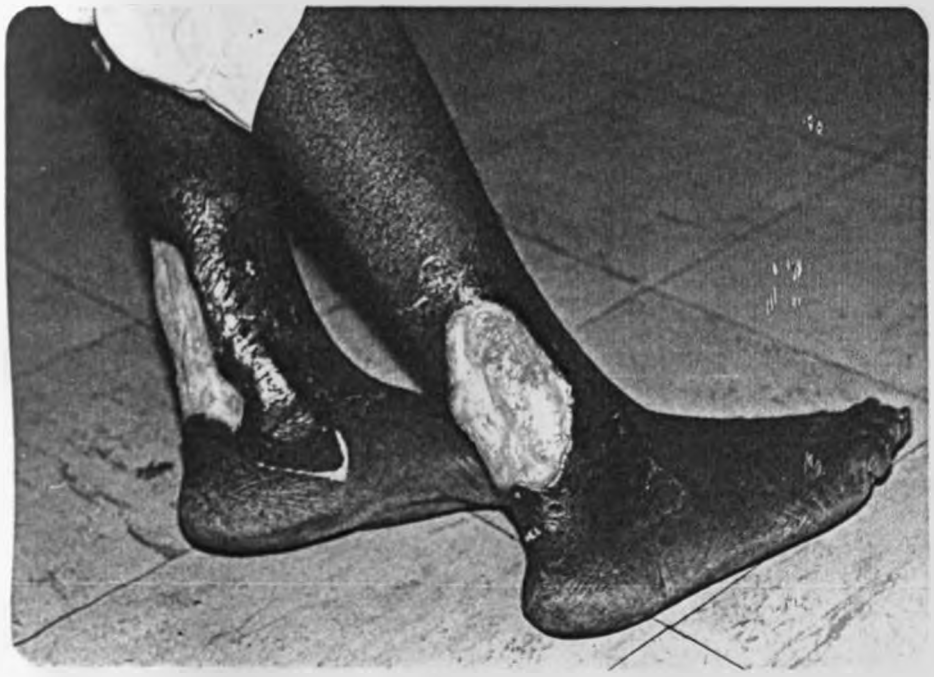


Plate 5: Tropical ulcers on the legs in a 60yr old male

D I S C U S S I O N

Epidemiologic studies have shown that malignant skin tumour are more common in Caucasians than in the pigmented races. The site or the primary tumour in Caucasians is mostly in exposed areas mainly head and and neck followed by trunk and then upper limbs. In pigmented races the most frequent site of presentation is in the lower limbs. The behaviour of skin cancers has repeatedly been shown to be different among the racial groups. It is appreciably more aggressive when encounted in pigmented races (3), (4).

The relative frequency in different countries in Africa varies as follows:- SCC 28.1% - 72.4%; malignant melanoma 15.2% to 28.4%. BCC 0% - 3% (8). For SCC and BCC annual incidences per 100,000 for different countries is given in table VIa and for malignant melanoma in table VIb.

In this study SCC, malignant melanoma, and BCC, will be discussed separately. Attention will be geered to highlighting some of the different behavioural pattern of the tumours among Caucasians and Blacks. Similarities and differences brought up in some of the studies done among different Black population will also be discussed.

SCC

This tumour second only to BCC among Caucasians is number one skin cancer in Blacks. It originates from neoplastic change of epidermal keratinocytes.

TABLE V (a) Examples of incidences of skin cancer (BCC and SCC) annual incidences per 100,000

Country	Male	Female
Uganda (African)	1.7	1.3
Bantus in Johanesburg	1.7	3.0
U.S. Negroes	2.6	3.4
S.W. England	28	15
S.A. Cape Whites	133	72
Texas (Non latin)	168	106
Queensland (whites)	265	156

(Kakande et,al.; E.A. med. J. 1982 Dec)

TABLE V (b) Age - standardised incidence rates for malignant melanoma in various countries

Country	Incidence rate per 100,000		
	Male	Female	Ratio (M:F)
Scotland (Ayrshire)	3.0	3.3	1:1
England (South metropolitan region)	2.1	3.5	1:17
Denmark	3.8	6.1	1:16
Norway	6.8	7.0	1:1
Finland	3.8	3.7	1:1
Sweden	5.3	6.2	1:1.2
New Zealand (non-Maori)	9.4	14.2	1:1.5

(Practioner 1980)

The aetiology is closely linked to the carcinogenic effects of U.V. light on skin. It has been put forward that DNA is the major target for U.V. radiation effect on the skin. Xeroderma pigmentosa patients who have defective repair of DNA following U.V. radiation have an increased incidence of carcinoma of the skin. The youngest patient in this study was 4½ years old female. She had xeroderma pigmentosa for which she was being followed in our dermatology clinic.

The amount of hours of sun exposure appears to count among the caucasians. In these racial group the male: female ratio for skin cançer is high. The men do more out-door work than female and so have more hours of exposure to the sun. (3). In this study male: female ratio was 1:1. In Uganda Africans it is 1.7: 1.3 (6). Oluwasanmi in Nigeria gives it at 1:1. Among the pigmented races most SCC arise from chronic ulcers and old scar tissue (3). Sun light is thought not to be the main promoting factor in development of SCC in the Black population. In a country like Kenya, the society is mainly agricultural. Most of the farm work is done by women. As such one would expect to find more SCC developing in females than males. Females would be expected to have more ulcers on the legs secondary to cuts, pricks and bites sustained in the course of farm work. Schrek in 1944 stated that 18% of all SCC of the scalp, trunk, legs and arms develop in preexisting scars (6). In this study 20 patients (40.08%) out of 49 had chronic ulcers preceding to development of SCC. In 2 patients (4.08%) out of 49 SCC developed on old burn scars. One patient tumour developed on chronic osteomyelitis sinus. In Nigeria 15(7%) out of 219 patients SCC developed on chronic ulcers. (7). In only 26 (12%) patients were other antecedent lesions like hypopigmentation, trauma, warts, fistula, schistosomias, or albimsm and non-specific dermatosis observed (7).

Davis et;al, in 1968 saw that nearly all Uganda Africans with SCC had antecedent chronic ulcers; 477 out of 663 patients (71.6%)

In this study, 24 (48.9%) out of 49 patients, SCC presented on the head and neck. The breakdown being forehead one; scalp five; occiput two; cheek five; temporal area two; nose one and eyelids two. Oluwasanmi in Nigeria (1969) had 27.1% of his patients presenting with SCC in the head and neck. In Uganda (Davies et;al. 1968) 8% of patients had head and neck SCC. In South Africa Bantus 40%, U.S. Negros 57.9% and U.S. Whites 70.3% (7). The observed difference of SCC presenting more frequently (70.3%) in head and neck in whites compared to Blacks (57.9%) could be of aetiological significance. The incidence is not altogether low in Blacks in the tropics. As such it is suggested that sun exposure is more probably the main carcinogenic factor in both Black and Whites as apposed to the popular theory of chronic ulcers predisposing to SCC development among the Blacks.

That SCC can arise from old burn scar was first noticed by Celsus in the 1st century. Later in the classical article by Treves and Pack 1930, it was shown that age of the burn scar was more important than age of the patient. It takes approximately 36 years from the time of burns to when one develops SCC on the scar. The frequency of SCC arising from an old burn scar is given as 2% (4). However there is no report exisitng to show the incidence of burn scars that undergo malignant degeneration. In this tudy one patient had SCC arising from old burn scar.

A previous study by Kakande I 1980 showed that 5 patients had SCC arising from old burn scars. Could this mean that at KNH more burns are getting skin grafted and hence the decrease in number of patients developing SCC on old burn scars? Lovel et;al. reviewed 500 cases of chronic osteomyelitis and found SCC in 1.8% (1). In other series the percentage varies from 0.23 - 0.5% (4). In this study 1 patient (2%) out of 49 developed SCC on chronic osteomyelitis sinus. Chronic osteomyelitis sinus therefore does not seem to be a major predisposing condition to developing SCC.

Trauma has been suggested as promoting factor in developing skin cancer by many Writers. They postulate that SCC develop more frequently in the lower limbs in Africans who walk bare-footed, because repeated trauma promotes development of SCC from skin that is chronically irritated. But has the incidence of SCC developing in the lower extremities decreased with the urbanisation of many African countries? In Kenya from 1974 to 1983 there has been a steady decline in the number of SCC entered in the Kenya Cancer Registry every year, from 185/year in 1974 to 116/year in 1983. This also raises the question of whether the figure of 48.9% SCC developing in head and neck in this study compared to 8 -12% stated in a previous study by Kakande in 1982 is actually significant. If so then the change in way of life from rural to urban over the years has made head and neck more predisposed to developing SCC than lower limbs by way of change in clothing and occupation.

SCC is hardly seen before puberty. This study had 4 patients before puberty. The youngest was 4½ years old female with XP who developed SCC on the scalp.

Second was 13 years old male albino with BCC exised from left (L+) forehead 1 year before he developed SCC on L+ temporal area. Third was 16 year old male who developed SCC on old burn scar. Lastly there was a 14 year old female with SCC developing on the skin over the parotid. Out of the 4, three patients (75%) had predisposing conditions to developing SCC before puberty.

The pathology of tumour any where, requires that it is well defined in terms of size, site and duration as well as its morphological and behavioural pattern. This in part affects the tendency to metastasize. SCC arising from chronic ulcers or old scar tissue is more aggressive and metastisize early (4). Scar tissue SCC of the lower extremities metastasize earlier than that of the scalp. In this study 19 (38.8%) out of 49 patients had positive regional lymphnode involvement. The question as to whether this is due to delay in reporting to hospital or because these tumours were more aggressive is not clear. Most patients, 13 (26.5%) out of 49 presented within a year or less, from the on set of symptoms.

Diagnosis of SCC can be clinical but should be confirmed by histology. In this study all the 49 cases had confirmatory histology report. One case of a 38 year old female with lesion on the Rt scapular clinically diagnosed as SCC was found on histology to be dermatofibroma protuberance. Methods of obtaining tissue for histology was by incisional or excisional, biopsy. Other methods of biopsy currently in use in other centres are punch, shave and incisional, if the lesion is more than 1 cm and involves vital structures like the nose, eyes or lips. Excisional biopsyis recommended for lesions of 1 cm diameter or less. Frozen section and needle biopsy may also be used. Needle biopsy is not encouraged because the sample error is high

Treatment should be decided upon inventory analysis of human factors and tumour factors. For the patient factors age, resulting scar, multiplicity of lesions, whether lesion is a recurrence and if the patient wants to be hospitalised or not should be considered. For the tumour factors one should consider invassiveness and presence of lymph-nodes and whether they are mobile or fixed. The mode of treatment is then decided on. Most centres advocate electrodesication for lesion of less than 1 cm diameter. Presumably in an older patient who does not wish to be hospitalised. It is simple and can be done on out patient basis. It is useful for management of recurrent tumour as well. Excision and closure is suitable for lessions less than 1 cm. It is also done on out patient basis. In the early 1950s doctors would wait for six months to one year to see if there were any recurrences before undertaking reconstructions. Currently taking a larger margin of excision when performing major reconstruction is advocated. This margin rather than being 0.5 - 1 cm may be as much as 2 - 3 cm with possible frozen section before resurfacing of the resulting defect. In this study 25 (51%) out of 49 patients had deep x-ray therapy (DXT); 6(12.2%) had excision followed by DXT and 5(10.2%) had excision and skin graft, immediate or delayed. Its fair to point out that majority of patients here had very extensive lesions not possible to excise completely. There was also quite a high proportion with head and neck lesions for which DXT was found to be the best mode of treatment.

Currently it is advocated that treatment requires wide excision with immediate repair. Post operative radiation can be given if there is concern about recurrence.With the advent of microvascular surgery and musculocutaneous flaps, there are practically no major defects that cannot be reconstructed immediately.

Prosthesis are useful in nose ear and orbit defects. Elderly patients, those with heart disease, pulmonary disease, nervous disease or diabetes should not be subjected to multiple staged reconstruction. Chemotherapy may be used for large and extensive head and neck lesions which are inoperable. Drugs like methotrexate and vitamins A and C have been tried. In this study most common chemotherapentic agent used was methotrexate given parenterally. Lymphnodes that are enlarged should be given 4 - 6 weeks after excision of tumour, patient is put on antibiotics meanwhile. If they do not regress then lymphnode dissection should be carried out. In this study only one patient underwent lymphnode dissection. One patient had a below knee amputation because of bony involvement.

Considering the fact that skin cancer is known to be more aggressive and to metastisize early in the Blacks, it is suggested that as part of management of SCC regional lymphnodes should be dissected more often than is shown in this study.

BCC

This skin cancer arises from the basal cell layer of the skin at the junction of epidermis and dermis. The basal cell has the potential for outward growth and replication, as such neoplastic change can easily arise from it. It is the most common skin cancer in caucasians. It is only rarely seen in Blacks. Europeans who have migrated into the U.S.A., South Africa and Australia have the highest incidence rates. It is given as 265/100,000 in males in Queensland Australia and 28/100,000 in South West England (1). Among the Blacks and oriental races it occurs in association with pre-existing conditions like XP, Basal cell naevus syndrome or albinism. Albinism is an autosamal recessive condition while basal cell naevus syndrome is autosomal dominant. XP is incomplete sex-linked recessive. The aetiology is thought to be due to reduction of DNA synthesis and altered mitotic activity following exposure to U.V. light of wavelength 230 - 320 nm. The tumour is locally invassive but death is rare. Only 109 cases have been reported in world literature upto 1977 that showed metastasis (1). (Phillip Lasson M.D. pg. 304). Lymphnodes when involved are characteristically stony hard and rough feeling on palpation. In this study 2 patients (2.3%) out of 87 had BCC. Both had occulo-cutaneous albinism, their lesions were in multiple sites, no sign of metastasis was noted.

The treatment in both cases was by radiotherapy alone. Both patients at the time of the study were still on follow up. In the caucasians treatment is mainly by curretage and electrodesication. This is done mostly by dermatologists who undoubtedly see the vast majority of BCC patients. Cure rate by this method is given as 95% (1). It is recommended for lesions which are superficial and less than 2 cm in diameter. Cure rate for radiotherapy is 96% (1). Cryotherapy and application of 5 - fluorouracil can also be used. Systemic chemotherapy has not been found useful in metastatic disease both in BCC and SCC.

Malignant Melanoma

Melanoma is a malignancy the incidence of which is increasing in frequency. The incidence of melanoma has increased almost 50% in the past decade (1). The most highest incidence is found in fair skinned whites and lowest percentage in Blacks. The figure is given as 20 times more frequent in white than Black Americans (10). In Blacks the lesion is more common in the sole of feet, more deeply invassive and more advanced stage at first presentation than in whites. In a study done in America the five year survival rate for Black Americans was 23% (10). Blacks had a significantly worse prognosis. This emphasizes the aggressiveness of this rumour in Blacks. 36% (41.4%) out of 87 patients in this study had malignant mel noma. The male female ratio was 1:1.

Sunlight is the key factor in the aetiology of malignant melanoma. In addition other sun-related factors of importance have been determined. Skin colour is one of them. In 1896 Rudolph Matas, observed that it was rare to find skin cancer in Blacks. A number of years later, Thomas Paul wrote "the pigment of the skin stands as a sentinel guarding the underlying skin from the baneful effects of sunlight". But this malignancy, is not altogether rare in Blacks. The angle of sun exposure is important. The more direct the exposure (the closer to the equator), the higher the incidence. The incidence in southern cities of United States such as Atlanta is twice that in northern cities such as Detroit (1). The study in Queensland of more than 1000 patients gives a well documented distribution of the tumour. The peak age in the study was 45 years. This study, peak age was 60 years. The mean age in Nigeria is 50 years, while in Uganda itis given as 50 - 59 years (7). In the American Blacks it is frequently in 60-69 years old. It would appear that this tumour has its peak at a younger age among the Blacks in Africa, compared to those in America.

But in the Blacks in general the tumour develops later than in Europeans in Queensland, Australia. This points to a genetic factor at play in the aetiology of malignant melanoma. In this study the only case of malignant melanoma in the under 20 yrs old was in a female aged 15 yrs. She presented with a lesion on the dorsum of her foot and progressed in one month to metastasize in the chest extending to the hilum, followed by unconsciousness and death. No treatment could be instituted. Oluwasanmi in Nigeria had 4 patients who developed malignant melanoma before age 20 yrs. The youngest was 8 yrs old. Kakande in an earlier series at KNH noted malignant melanoma in a 16 yrs old. This tumour is generally more aggressive and progresses rapidly in the under 20 yrs.

Trauma has been implicated in the aetiology of malignant melanoma. The tumour is more common in the lower extremities in the Blacks. Lewis in 1967 postulated that the foundamental reason why malignant melanoma is common in the sole of feet among Uganda Africans was because of the high incidence of potentially unstable collection of melanocytes seen in this area. And that it is genetically determined. Similar studies have not been carried out in any other Black population for comparison. The actual role played by trauma is debatable. Lewis was of the opinion that trauma may well play a part in transformation of areas of pigmentation into junctional and then malignant change. Other factors known to have causative role in development of malignant melanoma are xeroderma pigmentosa, which combines development of SCC, malignant melanoma and fibrosarcoma, at an early age. Some studies have shown a female preponderance (11). Oestrogen and progesterone- oestrogen combination are thought to have a stimulatory action on melanocyte activity. In New Zealand the female excess is most marked during the reproductive age.

In this study male: female ratio was 1:1, no patient presented with lesions during pregnancy, No patient complained of preexisting lesion enlarging during pregnacy. No history was available whether any of the female patients was on oral contraceptives.

In Queensland most lesions developed in the sun exposed areas of the body. The back, chest and upper extremities were the commonest sites in males, whereas in female patients the back, lower leg and upper extremities predominated (1). In this study 16 male patients (44.4%) and 13 female patients (36.1%) out of 36 had tumour in the lower limbs. The distribution here was left leg more than Rt leg. Most lesions were on the foot, and specifically heel and sole. This contributed to (86.2%) 26 out of 29 patients with lesion of the lower limbs. Next common area was upper limbs where 4 (11.1%) out of 36 patients had lesions in this area. This compares well with figures given in Nigeria 67%; U.S. Negroes 79.2%; Uganda Africans 87.8% compared to U.S. whites where only 30.4% of tumor occurs in lower limbs (7). This study had 3 patients with lesions on the head and neck. Of these, in one case lesion was on the nostril, another on the buccal cavity and last one on the upper eye lid. 4 patients had lesions on the upper limb namely index finger, arm and thumb.

Presenting symptoms of this tumour in caucasians is usually a complaint by the patient of change in the characteristics of a pre-existing lesion. Among the Blacks, the most common presentation is by an ulcerated tunour with positive regional lymphnodes. In this study 20 (55.6%) out of 36 patients presented with history of a swelling, while only 3 (8.3%) out of 36 patients presented with ulcerated tumour mass. Lymphnode invovement was recorded in 24 (66.7%) out of 36 patients. This is in keeping with figures given by Kakande I (10). In Australia the commonest presenting symptom was increase in size (47.7%) and a lump (swelling) in 3.1%) (10).

The average duration of symtoms before patients presented to hospital was, in 17 (47.2%) out of 36 patients in a year or less and 9 (25%) out of 36 patients with symptoms of more than a year's duration. Kakande I in an earlier study at KNH found average duration of symptoms to be 13.7 months in males and 16.6 months in females. He suggested that the poor prognosis in males could therefore not be due to late presentation to hospital. The better pregnonsis in females has been reported in other studies (2) · (10)· (11).

The different histological types of malignant melanoma are; lentigo maligna commonly seen in the elderly; superficial spreading and nodular melanoma commonly in the young. In this study 3 cases had amelanotic melanoma; 3 had pleomorphic melanoma and 3 had nodular melanoma. The rest 27 (75%) out of 36 patients were reported just as malignant melanoma. In this study no mention was made of the stage of the disease either by clinical, Clark level or Breslow classification. It would appear that most cases having presented with positive regional lymphnodes, the condition was regarded as advanced and staging thought not to significantly alter the mode of treatment offered. But in order to gauge prognosis staging should have been given.

In this study most common modality of treatment used was wide excision and skin grafting in 19 (52.8%) out of 36 patients. Next was excision without skin graft. In 8 (22.2%) out of 36 patients only radiotherapy was offered. In Nigeria in the absence of radiotherapy, radical or local excision, amputation and gland dissection was the mode of treatment (7). In an earlier study at KNH over half the patients had excision with or without skin grafting. No significant response was noted in the use of radiotherapy, chemotherapy or BCG vaccination. (11).

In this present study only one patient was offered lymphnode dissection, he was then lost to follow up after 2 years. The only patient amputated at below knee level, later presented with metastatic disease causing collapse of 3rd lumber spine. A study in America showed that 5 years survival was at 22% for Blacks (10). The pgrnosis was worse in males than females, with median survival of 26.3 months and 40 months in males and females respectively. The white American had a highly significant survival rate than Black American. Multiple regression analysis or survival among Black patients showed that; ulceration, number of mitosis and stage of the disease were suggestive prognostic factors. In the white patients, Clark level, ulceration and Breslow depth of invassion was shown to be significant prognostic factors (10).

In Nigeria follow up of 50 patients showed that 33% were known to be alive between 1 and 3 years. In the present study, 2 patients with very advanced disease were offered no treatment. Two died before treatment could be instituted. Follow up was generally poor and over 50% of patient were lost to follow up within a year. Treatment of malignant melanoma has changed drastically in the past decade. The empirical approach has been modified with the advent of qualitative and quantitative methods of gauging tumour depth and a better understanding of the kinetics of tumour growth. Emergence of a rational and statistically valid approach to prognosis and therapy, has allowed an enhanced patient survival with diminished morbidity. Classification of malignant melanoma into various groups and its differentiation from other benign and malignant pigmented lesions have been clarified. Surgery remains the mainstay therapy less radical procedures are now advocated for Clark low level malignancies.

The role of nodal dissection remains important in selected patients. Chemotherapy is useful in prolonging the disease-free interval in some patients with metastatic disease, although survival in these patients is still poor. The potential role of chemotherapy is in the eradication of micrometastatic disease especially stage I patients. The prospect of immunotherapy is exciting and it is hoped that in the future it will be possible to safely manipulate a human hosts' immune system to eliminate micro-metastasis in malignancies.

SUMMARY

During the period of this study (1977 - 1984) 9886 malignant tumours were recorded in the Kenya cancer registry. Of these, carcinoma of cervix was the number one tumour. It formed 15% of all tumours. Carcinoma of the skin (including SCC and BCC) was number two forming 10.2% of all tumours. Malignant melanoma formed 3.9%. The least frequent tumour was Carcinoma of rectum at 1.3%. In Nigeria a similar study showed that a total of 6133 malignant tumours were recorded in their Cancer Registry in a period of 8 years and the relative frequencies of the different tumours is given in Table VI.

There is conflicting evidence about the incidence of skin cancer in pigmented races, including Africans. Shapiro and Colleagues in 1953 found 50 (8.4%) cases of skin cancer out of 590 cases of malignant disease in South African Bantus, over a period of 3 years. He concluded that it was a rare condition. Stainer in 1954 found only one American Negro with carcinoma of scrotum in Los Angeles out of 135 patients. Schrek in 1944 found that skin cancer accounted for only 3% of tumours in American Negroes. Davies and his colleagues in 1968 found that superficial cancers of skin constituted up to 15% of all cancers diagnosed in Uganda Africans. A survey carried out by National Cancer Institute of U.S.A. in 1947 - 48, the crude incidence rate of skin cancer in non-whites was about one-sixth to one-twentieth that of whites. Low ratio frequencies have also been obtained in other pigmented races in India (Khanolkar 1950), Indonesia (Kouwenaa and Sutomo 1957), North Africans (Mussini and Montpellier 1951).

In developing countries record keeping and demographic data are either not accurate or not available. Most of the quoted studies have been done mainly in the nineteen fifties and sixties. These studies do need updating. Data obtained from these studies may have been affected by standard of primary health care, diagnostic facilities and criteria and socio-economic status of the population studied. As such comparison should not be parallel, all the same these results are increasingly useful with a view to identify factors suspected of carcinogensity in these tumours. One point that is clear in all these studies is the relatively low incidence of skin cancer in Blacks compared to caucasians. In the Caucasians the incidence is again higher in Europeans who have migrated to the tropics compared to those in temperate climates. The aetiological factors in development of skin cancers in Blacks is not alltogether clear. The only factor that has stood the test over the years is that skin pigment seems to protect the Blacks from the baneful effects of sunlight. Malignant melanoma has been shown in this study to be more aggressive in the Black population. (10). The aetiological role of trauma in development of malignant melonoma is shown but it does appear to be a procarcinogen and not carcinogenic in its own right.

It is not clear what makes skin cancers to be more aggressive with poor prognosis in the Blacks. Late presentation alone does not seem to be the only reason for this. A critical look at the immune system especially in the Blacks may probably give an answer to most of these unanswered questions.

TABLE OF % OF COMMON MALIGNANT TUMOURS

TABLE VI

Type of Tumour	Kenya (1977 - 83)		Nigeria (1960 - 67)	
	No.	%	No.	%
Cervix	1496	15.0	618	10.7
Squamous Cell Carcinoma	1006	10.2		
Malignant melanoma	383	3.9		
Basal Cell Carcinoma		0.6		
Lymphoma	876	8.8		
Breast	715	7.2	303	4.9
Liver	592	5.9	438	7.1
PNS	608	6.1		
Oesophagus	653	6.6		
Lungs	-	-	58	0.94
Stomach	434	4.4		
Prostate	590	5.9	127	2.07
Rectum	132	1.3		
Kaposis	251	2.5		
TOTAL	9886		6133	

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