

Cutaneous Cancers (Including Melanoma)

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1 Major Determinants of Skin Cancer Carcinogenesis and Their Management in Tropical Areas

Four main factors are associated with the epidemiological profile of skin cancer in the tropics: more intense ultraviolet radiation (UV radiation), a higher proportion of dark-skinned inhabitants, low incomes in association with limited healthcare resources, and high biodiversity including oncogenic microbial pathogens (Fig. 1).

UV radiation is more intense in the tropics because the rays from the sun being perpendicular have a shorter path through the atmosphere and are therefore less absorbed by the atmosphere's layers. In fair-skinned populations, the incidence of three major UV-induced cancers – basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and melanoma (MM) – increases with diminishing latitude.

Skin pigmentation varies substantially across human populations. In the skin, melanin acts as an optical and chemical photoprotective filter which reduces the penetration of UVR into epidermal and subepidermal tissues. Melanin is synthesized in melanosomes, which are distributed to keratinocytes by the dendritic

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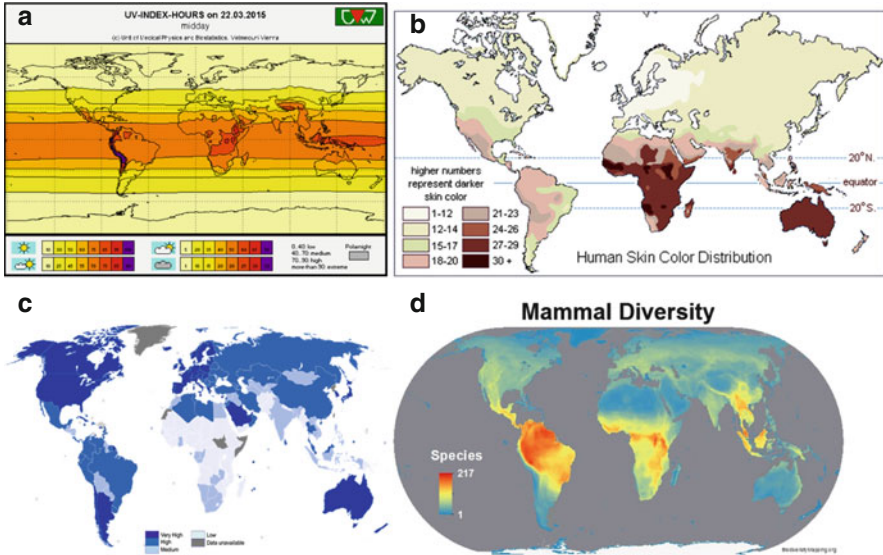


Fig. 1 Four global maps illustrating the four main factors associated with the epidemiological profile of skin cancer in the tropics: (a) more intense ultraviolet radiation (http://www-med-physik.vu-wien.ac.at/uv/uv_online.htm); (b) a higher proportion of dark-skinned inhabitants (Reproduced from <http://anthro.palomar.edu/vary/> by permission of Dennis O'Neil); (c) low incomes in association with limited healthcare resources (Illustrated with a map of the Human Development Index: World map indicating the categories of Human Development Index by country based on 2013 data; <http://hdr.undp.org/en/2014-report>); and (d) high biodiversity including oncogenic microbial pathogens (Illustrated by map of mammal diversity: Jenkins et al. [1]; <http://biodiversitymapping.org/>)

processes of melanocytes. The superior photoprotection of highly melanized skin (dark skin) is due to the high density and distribution of melanosomes within keratinocytes in the epidermis. In 1988, Fitzpatrick describes a scale of 6 phototypes that takes into account both the color of the skin, the susceptibility to sunburn, and the ability to tan after exposure to UVR [2]. Individuals with phototype I (generally with red hair) and II (generally with blond hair and blue eyes) tan little or not at all. The risk of photo-induced skin cancer is very high. People with phototypes III (generally brown hair) and IV (generally dark hair, brown eyes) tan easily. Sunburns are rare. Individuals with skin types V and VI have, respectively, brown or black skin, tan deeply, and rarely or never burn. Relationship between skin pigmentation in indigenous human populations and latitude is traceable to the strong correlation between skin color and UV radiation intensity. The clinal gradation of skin coloration observed among indigenous peoples is correlated with UV radiation levels and represents a compromise solution to the conflicting physiological requirements of photoprotection and vitamin D synthesis [3].

Low- and middle- income countries predominate in the tropics. Limited healthcare resources and limited access to healthcare are very frequent. The main

consequences are diagnostic delays, imprecise diagnoses, and limited therapeutic means. As with other cancers, limited access to pathological examinations, radiotherapy, and chemotherapy complicates the management. However, more than other cancer types, skin cancers can be diagnosed early by clinical examination and can in most cases be treated with surgery under local anesthesia.

Viral infectious agents are responsible for primary cutaneous cancers or cancers with cutaneous focus more often in low latitudes than in high latitudes. The main viruses involved are HHV8, oncogenic HPVs, HIV, and HTLV-1.

2 Epidemiology of Skin Cancer in the Tropics

Despite their frequency, BCC and SCC are not usually recorded by cancer registries due to their high frequency and low to very low case fatality rate. However, it is estimated that the five most common skin cancers in the world are in decreasing order of frequency: BCC, SCC, MM, Kaposi sarcoma (KS), and cutaneous lymphomas. The geographical distribution of these skin cancers shows variations. The first three are most often UV induced, reaching extremely high standardized incidence rates in tropical and subtropical areas where people mostly have fair skin (phototypes I and II). In Australia, a partially tropical country where the population is mainly fair skinned, every year 80 % of new cancers are skin cancers, especially BCC and SCC, respectively, 20 and 10 times more common than MM [4]. Nevertheless, MM is the fourth most common cancer in men (when excluding BCC and SCC) in Australia [4]. Table 1 summarizes and compares the Australian data and shows the influence of environmental ultraviolet radiation (latitude) and constitutional defense ability from ultraviolet radiation in white skin (tan ability) and black skin (indigenous, i.e., Australian Aborigines) [4–7].

In people with dark skin, the incidence of all UV-induced skin cancer is considerably lower. Thus, in the tropics, the incidence of MM is very high among people with fair skin and low in patients with dark skin. The main location of MM is the sole of the foot in these populations from Africa, Asia, and among the Australian Aborigines. This is a variety of MM called acro-lentiginous melanoma (ALM), which is not photo-induced unlike the other forms of cutaneous MM [8].

Kaposi sarcoma (KS) is due to HHV-8, a virus of the herpes virus group, and its incidence increases dramatically when there is a coinfection with HIV. The incidence of KS is very high in countries where these two viral diseases exist simultaneously with high incidence rates. Thus, KS is the most common cancer in several East African countries [9, 10].

In the tropics, the most common lymphoma involving the skin are the adult T-cell leukemia/lymphoma (ATLL) associated with HTLV-1 and mycosis fungoides.

The dermatofibrosarcoma protuberans is rare but could be a bit more frequent in black skin.

Table 1 Age-standardized WHO incidence rates (per 100,000 in habitants/year) for the three principal skin cancers occurring in Australia and associated factors (latitude, skin type categories, ethnic group). Impact of environmental ultraviolet radiation (latitude) and constitutional defense ability from ultraviolet radiation in white skin (tan ability) and black skin (indigenous, i.e., Australian Aborigines)

		BCC	SCC	MM	All cancers ^a except skin cancers
Total by cancer ^b [4, 5]		884	387	40	313
Latitude ^b [4, 6]	High, nontropical ^c	547	232	36	
	Median, partially subtropical ^d	959	432	47	
	Low, partially tropical ^e	1,662	794	65	
Skin type categories ^b [4]	Tans deeply	585	215		
	Tans moderately	722	319		
	Does not tan	1,271	611		
Ethnic group ^f [7]	Indigenous ^g			8	
	Non-Indigenous			32	

BCC basal cell carcinoma, *SCC* squamous cell carcinoma, *MM* malignant melanoma

^aIncluding Kaposi sarcoma, lymphoma, and leukemia

^b2002

^cHigh latitude (>37°S)

^dMedian latitude (29°S–37°S)

^eLow latitude (<29°S)

^f2004–2008

^gAborigines (non-métis-Aborigines have black skin, Phototype VI)

3 Risk Factors and Pathogenesis

UV radiation is strongly associated with the three most frequent skin cancers: BCC, SCC, and MM. Schematically, UVB radiation can directly alter the DNA of epidermal cells, while the oncogenesis due to UVA radiation is more indirect. Spatiotemporal and meteorological factors affect the intensity of radiation: latitude, altitude, environment (sand, snow, water), time of day, period of the year, clouds, air humidity, ozone layer's density, etc. Latitude is an important factor affecting the risk of photo-induced skin cancers; the greatest risk is in the tropics (see Table 1). The temporal profile of the UV radiation dose influences the type of cancer: acute intermittent exposures (with sunburn) are rather involved in BCC and superficial spreading melanoma (SSM), whereas chronic exposure is rather associated with SCC and lentigo maligna (LM). Individual and collective behavioral factors (work, leisure activities and sports, fashion, knowledge of the sunbeam's risks, sunbed room) modulate exposure to UV radiation [11].

The pigmentation of the skin is mainly due to melanin. In the tropics, particularly in Africa, people with dark skin (phototypes V and VI) predominate. This dark pigmentation provides excellent protection against UV-induced cancers. This protection is abolished in melanocytic pathologies: genetic diseases like albinism or acquired diseases like vitiligo [12].

Age is a factor strongly associated with skin cancer, whether it is UV induced (BCC, SCC, MM) or not (classical or endemic KS). For UV-induced cancers, it is difficult to distinguish between the real influence of age and the cumulative dose of UV radiation-related to a longer exposure time. Overall incidence rises sharply from the fourth decade onward. In tropical regions, populations are generally young; with the demographic transition expected in the coming years, we may see an aging of the population which will cause increased incidence of skin cancer.

Sex is an important risk factor in KS (especially for non-HIV-associated forms with a sex ratio of around 10:1) and a relative factor concerning the location of MM (back in men, lower limbs in women). Moreover BCC and SCC are slightly more common among men (probably due to gender differences in exposure to the sun).

Genetic predisposition to skin cancer may exist. Some genetic diseases are accompanied by a specific gene mutation that constitutes a high-risk factor for skin cancer. This applies, for instance, to xeroderma pigmentosum (BCC, SCC, MM), Gorlin syndrome (BCC), or the most common mutations demonstrated in approximately 30 % of familial melanoma (CDKN2A gene mutation) [13]. Other predispositions are not yet clearly identified but likely involve genetic polymorphism predisposing as having a large number of nevi (MM). Skin phototype is also genetically determined.

Some viruses can cause skin cancers: HHV8 and KS especially when there is HIV coinfection, oncogenic HPV types and anogenital cancers (increased risk in case of HIV coinfection), HTLV-1 and adult T-cell leukemia/lymphoma (ATLL), Epstein-Barr virus (EBV) and Epstein-Barr virus-associated B-cell lymphomas, and Merkel cell polyomavirus and Merkel cell carcinoma (MCC). The pathophysiology of viral oncogenesis is described in the relevant chapters of this book. All, except perhaps MCC, are frequently described in the tropics.

Immunosuppression induced by immunosuppressive therapy in organ transplant recipients or caused by prolonged CD4+ lymphopenia in patients HIV+ promotes oncogenesis and skin cancers. In organ transplant recipients, UV-induced skin cancers are increased, especially SCC. In the tropics the risk could be higher because the UV radiation is more intense.

Some medications induce SCC: B-RAF inhibitors (vemurafenib) and voriconazole. These expensive drugs are currently not readily available in the tropics.

Various situations can promote the occurrence of skin cancer: chronic inflammatory conditions (chronic infectious dermatoses, ulcers or old burn scars) and predisposing or precancerous dermatologic lesions (actinic keratosis, lichen sclerosus, discoid lupus). Because mutagenic natural substances exist, plants applied on ulcers in the context of traditional medicine could be at risk, but data are lacking. Carcinogenesis is then of the SCC type.

4 Major Types of Skin Cancer in the Tropics

Connectivity – Internet or mobile phones – being rapidly expanding into many of low- and middle-income countries, a quick access to information and to guidelines could become easier, with updated classifications and updated therapeutic

Table 2 First-line treatment for invasive skin carcinomas, invasive skin melanoma, and Kaposi sarcoma in the context of limited healthcare resources when standard treatment is not available

	Localized tumor	Regional disease treatable by local modalities	More advanced or generalized disease ^a
Melanoma	Surgical excision ^b	Primary tumor excision ^b and regional lymphadenectomy	Surgical resection of isolated metastases Palliative local therapy Other ^c ?
Basal cell carcinoma	Surgical excision ^{d,e} , electrodesiccation-curettage ^f	Surgical excision or radiation therapy	Other ^g ?
Squamous cell carcinoma	Surgical excision ^b	Primary tumor excision ^b and regional lymphadenectomy or radiation therapy	Radiation therapy, other ^h ?
Kaposi sarcoma:			
HIV+	Antiretroviral therapy	Antiretroviral therapy ± radiation therapy or chemotherapy ⁱ	Antiretroviral therapy ± chemotherapy ^j
Non-HIV	Surgical excision or electrodesiccation-curettage or radiation therapy	Surgical excision or electrodesiccation-curettage or radiation therapy or chemotherapy ^j	Chemotherapy ^j

^aMetastases (except regional adenopathy treatable by local modalities)

^bMargins with 1 cm to 3 cm, depending on tumor thickness and location

^cImmunotherapy (+/-) or signal transduction inhibitors (if B-RAF mutation) (+/-) or chemotherapy (dacarbazine)

^dNodular form: margins ranging from 3 to 10 mm, depending on the diameter of the tumor; margin evaluation by pathologist

^eInfiltrating forms or ulcerating tumors: margins ranging from 5 to 10 mm; margin evaluation by pathologist

^fSuperficial forms

^gChemotherapy (cisplatin) or signal transduction inhibitors (Hedgehog pathway inhibitor)

^hMargins ranging: 4 mm if well-differentiated tumors up to 19 mm in diameter, 6–10 mm for larger or less-differentiated tumors or tumors in high-risk locations (e.g., scalp, ears, eyelids, nose, and lips); margin evaluation by pathologist; reexcision may be required if the surgical margin is found to be inadequate

ⁱChemotherapy (cisplatin)

^jBleomycin, liposomal doxorubicin, or liposomal daunorubicin, combination of doxorubicin, bleomycin, and vincristine or bleomycin and vincristine

indications for skin cancers. For example, this information is freely available in English or Spanish languages on the website “<http://www.cancer.gov>” or in French language on the website “<http://www.sfdermato.com>.” The challenges are rather to establish correct diagnosis and to access to treatment options. Here we briefly describe main skin cancers. Table 2 provides guidance on the available treatment options in cases of limited healthcare resources.

4.1 Basal Cell Carcinoma

4.1.1 BCC: General Information

BCC is the most common of all cancers. It is also the least dangerous because it is both slow growing and characterized by an almost total absence of metastatic spread. The most common location is the head, especially the face and nose. Surgical resection is the standard treatment that can sometimes require plastic surgery to optimize the aesthetic and functional aspects. BCC appears on healthy skin, and there is no mucosal involvement. The recurrence risk is higher for lesions in periorificial areas of the face.

There are three main types of BCC, from least to most aggressive: superficial (erythematous plaque), nodular (pearly appearance with telangiectasia), and infiltrating (scar plate). BCC usually progresses slowly in the form of a superficial spreading and sometimes evolves into ulceration (Fig. 2a). A spreading toward profound tissue layers such as muscles and bones by contiguity may appear over time with inability to achieve a complete surgical resection.

The standard treatment consists of a surgical excision of the entire lesion with pathological control including section edges to ensure that the removal is complete [14, 15]. Recommended margins depend on several clinicopathological factors. The margins vary from 3–4 mm to over 10 mm and are based on the risk of local recurrence. Surgical resection complemented by pathological examination of frozen sections or Mohs surgery can reduce these margins especially for infiltrating type. Nonsurgical treatments are possible: radiotherapy, cryotherapy, electrodesiccation-curettage, electrocautery, topical imiquimod, and phototherapy; these three techniques are to be reserved for superficial forms of BCC. In case of advanced forms, therapeutic possibilities involve chemotherapy, targeted therapy (in development), or radiotherapy.

4.1.2 BCC in the Tropics

Among the long-established populations in the tropics, there is an important melanin pigmentation that protects the skin from UV radiation resulting in a relatively low incidence of BCC. BCC is usually pigmented or “tattooed” and rather affects skin phototype IV to V. The darkest phototypes (VI) are virtually unaffected by this type of cancer. Among people with very fair skin in the tropics, carcinoma incidence rates are extremely high as previously described (see Table 1). The risk of Caucasian Australians developing a BCC before age 70 is greater than 50 % [4].

4.2 Squamous Cell Carcinoma

4.2.1 SCC: General Information

SCC has metastatic potential, which depends on: 1) clinical criteria: type, size (very low if less than 1–2 cm in diameter), location, existence and nature of previous precancerous lesion, recurrence, and neurological symptoms of invasion; 2)



Fig. 2 Pictures of different types of skin cancer: (a) recurrent nodular-ulcerative BCC partially pigmented on the face with diagnostic delay; (b) hypochromic mycosis fungoides on black skin – early lesions; (c) SCC developing on burn scars (left arm); (d) multipapular type of ATLL in a HTLV-1+ woman (back of the left hand); (e) KS strictly localized to the right lower limb with lymphedema and nodules in a patient coinfecting with HHV8 and HIV – absence of the characteristic purple color of KS on dark skin; and (f) MM-type ALM located on the sole of the foot in a patient of African origin

histopathologic signs: depth of dermal invasion, degree of differentiation, and histological type; 3) association with immunosuppression.

The cancer progression is initially local (ulceration, sometimes deep) or locoregional (lymph node metastasis in the first lymph node) and then metastatic. SCCs in situ may have specific clinical aspects: Bowen's disease or Bowenoid papulosis. SCC can grow on the mucous membranes (labial, buccal, anogenital) with possible

involvement of oncogenic HPV. SCC frequently appears on precancerous lesions like actinic keratosis and is then mostly UV induced. Sometimes SCC develops from chronic cutaneous inflammation, sites of prior burns (Fig. 2c), or long-standing skin ulcers.

The standard treatment for localized forms or forms with locoregional lymph node involvement is surgical excision with 5–10 mm margins for the primary tumor, depending on the criteria mentioned above [14, 16].

Other therapeutic methods can be used such as radiotherapy, cryotherapy, electrodesiccation-curettage, and electrocautery. Chemotherapy is rarely effective on metastasized forms; nevertheless, it can reduce the size of locally very advanced lesions which then allows surgery or radiotherapy. Targeted therapies are currently under development. For SCC in situ, nonsurgical treatment can be discussed as, for instance, cryotherapy, topical 5FU, or photodynamic therapy.

4.2.2 SCC in the Tropics

In patients with white skin living in low latitudes, the SCC incidence is very high and situated between BCC and MM [4] (Table 1). SCC usually occurs in a context of actinic skin with multiple actinic keratoses, solar lentiginos, or epidermal atrophy. In people with darker skin, SCC is much less common but more common than BCC and MM [17]. On all skin types, some chronic tropical infectious, chronic ulcers, or chronic inflammatory dermatoses can become cancerous, usually after a long-standing evolution of 10 or more years [17, 18]. This applies to plantar ulcers in leprosy, phagedenic ulcers, chromomycosis, lobomycosis, Buruli ulcers, burn scars, chronic cutaneous lupus, etc. [19–21]. The application of plants on wounds, during traditional treatment, can question the carcinogenic potential of natural substances. A frequent problem is the difficulty for pathologists to differentiate between pseudoepitheliomatous hyperplasia (frequent in biopsies of ulceration edges) and SCC. In case of doubt, it is necessary to repeat biopsies in the middle of the ulcerated area.

4.3 Melanoma

4.3.1 MM: General Information

MM is less common than BCC and SCC in high latitudes; however, its incidence has increased significantly in people with very fair skin over the last 30 years. MM's metastatic potential depends on tumor thickness measured during pathological examination according to Breslow. Breslow thickness below 1 mm has a good prognosis; beyond 1–1.5 mm, prognosis is worse, especially if the lesion is ulcerated. A relatively small tumor mass can spread quickly, resulting in a melanoma case fatality rate of more than 10–20 % of all cases.

The extension is sometimes locoregional (lymph node metastasis). Lymph node dissection of the sentinel node does not improve survival. Visceral metastases occur either simultaneously or later. Primitive mucosal lesions (mouth, vagina) or other localizations like intraocular lesions are rare. MM most often appears on healthy skin (about 80 % of all cases), more rarely from a nevus. Patients with a giant congenital nevus have a high risk of developing MM.

The risk factors of MM are fair skin phototype (type I and II mainly) and ethnicity; UV radiation intensity and cumulative dose depending on latitude, altitude of residence, the type of sun exposure (intermittent/occasional, resulting in sunburn or not), and the sun-exposed body regions; age (incidence increases sharply after the age of 40; melanoma is very rare in children); personal and family history of melanoma; high number of nevi (>50 or >100) and/or atypical nevi; and certain genetic diseases (xeroderma pigmentosum) or predisposing mutations (10 % of all patients) [13].

The main types of melanoma are superficial (SSM), most common in white skin; nodular (NM), with a poorer prognosis because usually diagnosed at a later stage; acral (ALM), most common in dark or black skin; and lentiginous (LM), a form of melanoma in situ that occurs on the sun-exposed skin of elderly people, mainly on the face. Photoinduced melanomas are either induced by intermittent sun exposure (including sunburn) like in SSM and NM or by a high cumulative UVR dose in case of chronic exposure as in LM. The latter are very rare on black skin (phototype VI). On the contrary, plantar, palmar, and subungual (mainly in ALM) MMs do not seem to be induced by sun radiation [8]. In the USA, ALM incidence, although low, is similar in white and black populations [8].

The surgical margins of the primary lesion vary from 5 to 30 mm according to the Breslow depth [14, 22, 23]. In case of metastases, there is no curative treatment except for very rare cases with a single resectable metastasis. Chemotherapies are ineffective. The recent development of targeted therapies and immunotherapies in metastatic melanoma allows prolonged remissions, but these treatments are currently very expensive. Radiation therapy can sometimes be useful.

4.3.2 MM in the Tropics

ALM is the most common MM in Africa, among Aborigines in Australia, and among Chinese and Indians in Asia (Fig. 2f). Diagnostic delays are very frequent in Africa, so the prognosis is bad [24]. A differential diagnosis problem arises in clinical examination of the hands and feet in Africans because of the high frequency of palmoplantar pigmentations and pigmented nail strips [25]. Furthermore, on physical examination, achromic melanoma can be confused with KS or pyogenic granuloma.

A study in tropical area in Australia shows that sunscreen use in adults is beneficial in preventing MM [26]. These should be a general recommendation for fair-skinned populations living in the tropics.

4.4 *Kaposi Sarcoma*

4.4.1 **KS: General Information**

KS is usually considered as a cancer, but this is subject to controversy. Pathophysiology is probably based on oligoclonal proliferation of spindle-shaped cells of lymphatic endothelial origin, which form vascular channels followed by an extravasation of red blood cells.

The viral origin has been proven and involves HHV-8, a virus of the herpes group. Men are much more affected than women especially in forms without associated immunosuppression (sex ratio=10:1). The global distribution of HHV-8 in human populations shows a north-south gradient, particularly in Africa. Some forms of immunosuppression, especially a coinfection with HIV, greatly increase the risk of KS and in particular of disseminated forms. KS mostly develops on skin especially of the lower limbs, but all organs may be affected except the nervous system.

The classification is epidemiological and clinical: classic or Mediterranean form mostly in the elderly; endemic (or African) form in Africa, which is more diffuse and occurs at an earlier age; associated form related to iatrogenic immunosuppression in organ transplant recipients; and AIDS-related epidemic forms with a high frequency of organ involvement.

The clinical appearance of skin lesions depends on the patient's pigmentation. On fair skin and mucous membrane (mouth, digestive tract, bronchi), lesions adopt their characteristic red maroon or purple color. On dark skin, lesions are pigmented and less typical. The lesions may be macular, papular, plaque like, nodular, or tumoral. The existence of lymphedema was first reported in African patients.

It is important to confirm the diagnosis by pathology. Whenever a KS is suspected, serology or a quick HIV capillary blood test must be carried out because the initial therapeutic management of KS in HIV+ patients is specific.

The main complication is related to the development of visceral lesions including pulmonary and digestive extensions. Pulmonary extensions are more severe (respiratory failure). Digestive forms can result in bleeding. Lymph node damage can cause dramatic lymphedema of the limbs or genitals.

There are various treatment options: surgery, radiotherapy, cryotherapy, chemotherapy, treatment of immunosuppression, and immunotherapy [14]. It should be noted that bleomycin is a relative inexpensive and effective drug in many cases of KS. Its advantage resides in the absence of induce cytopenias. Nevertheless, the total dose cannot exceed a certain maximum because of the risk of pulmonary fibrosis. The main indications for treatment are: (1) KS associated with HIV: treatment always starts with HIV antiretroviral treatment which alone can make the lesions disappear; however, immune reconstitution inflammatory syndromes are possible; (2) KS with visceral involvement: indication of chemotherapy (+/- antiretroviral treatment of HIV +); (3) cutaneous Kaposi with few lesions in the elderly: a local treatment may be sufficient.

4.4.2 KS in the Tropics

The epidemiology of KS in Africa has its particularities. It is the most common of all cancers diagnosed in Central and East Africa including Uganda due to extreme endemicity of HHV-8 in this region (prevalence > 50 %) combined with a not yet controlled HIV epidemic [9, 10]. Furthermore symptomatology of KS skin lesions is different, the purple-red appearance of the lesions being masked by the melanin pigmentation of dark skin (Fig. 2e). Finally in Africa, pediatric forms have been described which then concern lymph nodes first.

4.5 Other Types of Skin Cancer in the Tropics

Mycosis fungoides: a cutaneous epidermotropic T-cell lymphoma. It is rare, but incidence rate is highest among black in the USA. Mycosis fungoides has the particularity on black skin to begin with hypochromic lesions that can make the diagnosis difficult (Fig. 2b).

Adult T-cell leukemia/lymphoma (ATLL): the disease is due to a virus named HTLV-1. Skin lesions are frequent and polymorphic (Fig. 2d). The type of skin lesion is an independent prognostic factor in some studies [27].

Dermatofibrosarcoma protuberans: rare but slightly more common on black skin [28].

Dermatoses in paraneoplastic syndromes: among paraneoplastic dermatoses, one is frequently evoked in patients of African origin, acanthosis nigricans, not because it is more common in black skin but because it can be confused with pseudoacanthosis nigricans affecting mostly black women who are overweight.

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