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Skin Cancer in Skin of Color

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Abstract

Skin cancers in skin of color often present atypically or with advanced stage in comparison to Caucasian patients. Health care providers must maintain a high index of suspicion when examining skin lesions in skin of color.

Introduction

Skin cancer is the most common malignancy in the United States and represents ~ 35–45% of all neoplasms in Caucasians (Ridky, 2007), 4–5% in Hispanics, 2–4% in Asians, and 1–2% in Blacks (Halder and Bridgeman-Shah, 1995; Gloster and Neal, 2006). The incidence of skin cancer has been increasing among Caucasians (Ridky, 2007), but remains relatively low in people of color. Data have been limited for non-white populations, making accurate determination of incidence and mortality difficult.

The low incidence of skin cancers in darker skinned groups is primarily a result of photo-protection provided by increased epidermal melanin, which filters twice as much ultraviolet (UV) radiation as does that in the epidermis of Caucasians (Montagna and Carlisle, 1991). The larger, more melanized melanosomes of darker skinned groups absorb and scatter more energy than do the smaller, melanosomes of Caucasians (Brenner and Hearing, 2008). Hence, UV radiation, the most important predisposing factor for skin cancer in Caucasians, plays a lesser role in people of color.

When skin cancer occurs in people of color, patients often present with an advanced stage, and thus, worse prognosis in comparison to Caucasian patients (Cormier et al, 2006; Hu et al, 2006). Furthermore, certain types of skin cancer, such as dermatofibrosarcoma protuberans, predominate in people of color (Halder and Bridgeman-Shah, 1995). The anatomic distribution may or may not be different from that seen in Caucasians, depending on the specific type of skin cancer. Treatment is generally the same among all racial groups.

Predictions estimate that by the year 2050, Hispanics, Asians, and Blacks will represent ~50% of the US population (Census Bureau 2000, Gloster and Neal, 2006). Hence, given the often atypical clinical presentation, the difficulty in detecting certain features such as color variegation in dark skin, and pigmentation of some skin cancers that are usually not pigmented in Caucasians, a high degree of suspicion must be maintained by physicians and other health care providers when examining skin lesions in people of color (Halder and Bridgeman-Shah, 1995). In this review, the differences in risk factors, clinical presentation, and mortality associated with skin cancers in Blacks, Asians, and Hispanics as compared to Caucasians will

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be discussed. Forms of skin cancers that can present atypically in people of color will be included and consist of basal cell cancer (BCC), squamous cell cancer (SCC), melanoma, cutaneous T-cell lymphoma (CTCL), Kaposi sarcoma (KS), and dermatofibrosarcoma protuberans (DFSP).

Basal cell cancer

Basal cell cancer (BCC) is the most common type of skin cancer in Caucasians, Hispanics, and Asians (Rubin et al, 2005) **(**Table 1). Hispanics are six times more likely to be diagnosed with BCC than SCC and are more likely to be diagnosed with multiple BCC compared to a solitary SCC (Byrd-Miles et al, 2007). In contrast, BCC represents the second most common skin cancer in Blacks (Halder and Bridgeman-Shah, 1995; Gloster and Neal, 2006). The majority of BCCs in a clinical series at Howard University in Washington, D.C. from 1960–1986 occurred in light-complexioned, as opposed to darker, Blacks (Halder and Bang, 1988). Thus, the frequency of BCC appears to be directly correlated with the degree of pigmentation in the skin, being most common in fair Caucasians and least common in African blacks.

UVR exposure is the most common etiologic factor for BCC in all racial groups (Gallagher et al, 1995) **(**Table 1). Other possible risk factors for BCC include scars (Mora and Burris, 1981), ulcers (Abreo and Sanusi, 1991), chronic infections, immunosuppression (Maloney et al, 2006), previous radiation treatment (Walther et al, 1981), and both physical and thermal trauma (Ewing, 1971; Gloster and Neal, 2006). Genetic disorders such as albinism (Asquo et al, 2007), xeroderma pimentosum (Giannotti et al, 2003), and nevoid basal cell carcinoma syndrome (Kimonis et al, 1997) are also risk factors for BCC.

The clinical features of BCC are similar in Blacks, Asians, Hispanics, and Caucasians. Most patients with BCC are elderly and present with asymptomatic, translucent, solitary nodules with central ulceration (Rubin et al, 2005) **(**Table 1). Telangiectasias and a pearly, rolled border in dark skin or in a pigmented tumor may be difficult to discern. Interestingly, when BCC does occur in people of color, pigmentation is present in more than 50% of the tumors (Bigler et al, 1996; Gloster and Neal, 2006) **(**Table 1). In contrast, only 5% of BCCs in Caucasians are pigmented. When pigmented BCC presents in people of color, there are often incorrect diagnoses, such as seborrheic keratoses, malignant melanoma, or nevus sebaceous (Halder and Bridgeman-Shah, 1995). BCCs in Asians have been reported clinically to appear brown to glossy black and have the so-called “black pearly” appearance (Kikuchi et al, 1996). Lesions can occur as nodules, plaques, papules, ulcers, or in more advanced cases, indurated or pedunculated masses.

The anatomic distribution of BCC tends to be similar in Caucasians and people of color **(**Table 1). In a review of BCC’s in Washington, D.C, Halder and Bang showed that 89% of BCC’s in people of color occurred on the head and neck regions (Halder and Bang, 1988). This is also true in Caucasians (Rubin et al, 2005).

Metastatic BCC is rare in all races, with rates ranging from 0.0028 to 0.55 percent (Rubin et al, 2005). However, risk factors for metastasis include a tumor diameter greater than 2cm, location on the central part of the face or ears, longstanding duration, and incomplete excision. The prognosis for metastatic disease is poor, with mean survival ranging from 8 months to 3.6 years (Rubin et al, 2005). Treatment for BCC includes Moh’s micrographic surgery, cryosurgery, and electrodessication and curettage.

Squamous Cell Cancer

Overall, SCC accounts for ~20% of all skin cancers, and excluding melanoma, ~75% of all deaths attributed to skin cancers (Alam and Ratner, 2001). SCC is the most frequently

diagnosed skin cancer in Blacks (Halder and Bridgeman-Shah, 1995) **(**Table 2), and the second most common skin cancer in Caucasians, Asians, and Hispanics.

Predisposing factors for SCC in people of color include scars from thermal and chemical burns (Copcu et al, 2003), chronic leg ulcers (King et al, 2008), and previous sites of radiation. Immunosuppressed patients, such as those with organ transplants or the human papillomavirus, are also at increased risk for SCC (Harwood et al, 2000). Patients with chronic inflammation, such as osteomyelitis, hidradenitis suppurativa, or lupus vulgaris, are also at increased risk for SCC (Halder and Bridgeman-Shah, 1992) **(**Table 2). In Blacks, the most important risk factors for the development of SCC are chronic scarring processes and areas of chronic inflammation. In fact, chronic scarring processes are noted in 20–40% of cases of SCC in Blacks (Gloster and Neal, 2006). Cases of SCC developing in Black and Chinese patients with chronic discoid lupus erythematosus have also been reported (Sherman et al, 1993; Ee et al, 2006).

SCC's are often superficial, discrete, and hard lesions arising from an indurated, rounded, and elevated base (Alam and Ratner, 2001) **(**Table 2). Nonhealing ulcers on the skin of people of color, regardless of original etiology, should be biopsied if present for a significant amount of time (Halder and Bridgeman-Shah, 1992).

People of color develop SCC predominantly in areas infrequently exposed to the sun, such as the legs, in contrast to Caucasians, who develop them in chronically sun-exposed skin (Halder and Bang, 1988) **(**Table 2). For example, in one Howard University series, 15% of SCC occurred in the anus in Blacks (Halder and Bang, 1988) **(**Table 2).

Invasive SCC has the potential to metastasize. The disparity in metastatic rates of SCC between people of color and Caucasians may reflect the tendency for people of color to present with more advanced disease, presumably as a result of delays in diagnosis, or it may be related to the presence of inherently more aggressive tumors (Gloster and Neal, 2006). Unfortunately, SCC that develops within a chronic scarring process, tends to be more aggressive and is associated with a 20–40% risk of metastasis, compared with the 1–4% metastatic rate of sun-induced SCC in Caucasians (Gloster and Neal, 2006). In one series of patients with SCC, the greatest mortality was seen in patients with perianal tumors (Mora and Perniciaro, 1981). SCC that arises from lesions of chronic discoid lupus erythematosus also appear to metastasize at a greater rate than SCC that arises from other preexisting lesions (Halder and Bridgeman-Shah, 1995), so hyperkeratotic or poorly healing lesions in areas of chronic discoid lupus erythematosus in people of color should be biopsied immediately. Most patients with primary SCC have an excellent prognosis. However, if metastatic disease is present, 10-year survival rates are less than 20 percent for patients with regional lymph node involvement and less than 10 percent for patients with distant metastases (Alam and Ratner, 2001). Treatment for SCC includes Moh's micrographic surgery and electrodessication and curettage.

Melanoma

Melanoma is the third and most deadly form of skin cancer in all racial groups **(**Table 3). During the 1970s, the incidence rate of cutaneous melanoma increased rapidly by 6% per year, but slowed to 3% per year from 1981 to 2000, and has remained stable since then (SEER Program, 2009). Interestingly, rates of invasive melanoma have increased markedly among Hispanics in California since 1988, with a 1.8% per year increase in incidence of invasive melanomas among Hispanic males between 1988 and 2001, and a 7.3% annual increase in the period between 1996 and 2001 (Cockburn et al, 2006).

Major risk factors for melanoma include intermittent high exposure to sunlight (ie—sunbathing) and chronic cumulative dosages of UVR (ie—outdoor workers) **(**Table 3). Host susceptibility factors include dysplastic nevi, increased number of nevi, freckling, family

history of melanoma, fair complexion, light eyes, and blonde or red hair (Tucker and Goldstein, 2003).

Clinically, melanomas typically present as dark, rapidly spreading patches **(**Table 3). Clues to the diagnosis of subungual (under the nail) melanoma include a pigmented band on the nail with width greater than 3 mm (Hutchinson's sign), variable pigment, rapid increase in size, and the presence of solitary lesions (Gloster and Neal, 2006).

In Caucasians and to a lesser extent, Hispanics, melanomas predominantly occur in sun-exposed skin, whereas in Asians and Blacks, UVR does not appear to be a significant risk factor, and the majority occur in non-sun-exposed skin (ie—subungual, palmar and plantar surfaces, mucus membranes) (Bradford et al, 2008) **(**Table 3). Oral melanomas represent ~7.5% of all melanomas in Asians, and two-thirds of these tumors arise from oral melanosis (Collins, 1984). In non-whites, the plantar portion of the foot is often the most common site, being involved in 30–40% of cases. People of color also have higher percentages of acral lentiginous melanoma (melanoma of the palms, soles, and nailbeds) than Caucasians (Bradford et al, 2008), whereas superficial spreading melanoma is the most frequent subtype in Caucasians and Hispanics (Byrd-Miles et al, 2007).

Multiple studies have demonstrated that 5-year melanoma survival rates of Blacks and Hispanics are consistently lower than those of Caucasians (Reintgen et al, 1982; Rahman and Taylor, 2001; Byrd et al, 2004;). Compared with Caucasians, Hispanics and Blacks tend to present with more advanced, thicker tumors and thus tend to have a poorer prognosis, with higher mortality. In a review of California melanoma cases, tumors thicker than 1.5 mm at presentation increased at 11.6% per year and 8.9% per year among Hispanic males and females, respectively (Cockburn et al, 2006). In a retrospective analysis of case reports to the Florida Cancer Data system, late stage (regional and distant) was more common among Hispanic (26%) and Black patients (52%) compared with caucasians (16%) (Hu et al, 2006). Interestingly, in a review of California melanoma cases, it was shown that even after adjustments for age, sex, histology, stage, anatomic site, treatment, and socioeconomic status, a statistically significant increased risk of death was observed for Blacks compared with Caucasians (Zell et al, 2008). Hence, the poor survival for Black patients with melanoma is not fully explained by differences in treatment or socioeconomic status. All of these results indicated that more primary and secondary prevention efforts are warranted for the control of melanoma in all races/ethnicities, even for those persons who are at a lower risk of developing the disease (Friedman et al, 1994).

Treatment for melanoma includes wide local excision or, sometimes, amputations for melanoma involving the limb, such as acral lentiginous melanoma. Metastatic melanoma is very difficult to treat, but includes isolated limb perfusion with chemotherapy, radiation, IL-2, and experimental cancer vaccines (Markovic et al, 2007).

Cutaneous T-cell Lymphoma

Cutaneous T-cell lymphomas (CTCLs) are the largest group of cutaneous lymphomas, representing ~65% of all cutaneous lymphomas **(**Table 4). There has been an increase in incidence and proportion of CTCL among all lymphomas, and Blacks are twice as likely to be affected as Caucasians (Criscione and Weinstock, 2007). The etiology of CTCL remains unknown and risk factors are poorly documented. The human T-cell lymphotrophic virus type I (HTLV-1), a virus that is endemic in the Caribbean, Japan, sub-Saharan Africa, and South America, has been associated with forms of CTCL (Verdonck et al, 2007). Numerous environmental agents, such as exposure to aromatic halogenated hydrocarbons, have also been reportedly associated with a small proportion of CTCL (Morales-Suarez-Varela MM, et al 2005), but the cause remains largely unknown.

Early CTCL typically presents with patches that are mildly erythematous, slightly scaling, annular, or arcuate and classically involve sun-shielded areas, also known as a “bathing suit distribution” (Smith and Wilson, 2008) **(**Table 4). A hypopigmented form of this disease is seen more frequently in darker skinned people as opposed to Caucasians and is characterized by early onset and good response to therapy (Akaraphanth et al, 2000). Clinically, the lesions present as macules or patches with ill-defined borders and varying degrees of hypopigmentation, although not quite the complete depigmentation seen vitiligo, with no or minimal scaling (Braverman, 1991). Up to 75% of patients with this variant may have a history of prolonged eczematous or psoriasiform dermatitis. These lesions may be confused with those of vitiligo, pityriasis alba, tinea versicolor, sarcoidosis, and postinflammatory hypopigmentation (Halder and Bridgeman-Shah, 1995).

In general, CTCL affects elderly patients and is a chronic disease. Hence, many patients die of other conditions rather than of CTCL. However, if lymph node involvement occurs, the prognosis is guarded and CTCL can be fatal. Palpable adenopathy is associated with a median survival of only 8 years, whereas patients without adenopathy have a survival of 22 years. If a patient has lymphadenopathy, tumors, or cutaneous ulceration, patients survive a median of one year (James et al, 2006).

Treatment involves primarily topical steroids, topical nitrogen mustards or carmustine (BCNU), photodynamic therapy with PUVA or narrow-band UVB (James et al, 2006). In one report on CTCL, it was stated that in the early stages of the disease (before the development or erythroderma and nodal involvement), patients are very responsive to treatment and the disease is potentially curable, whereas individuals in whom the disease is more advanced usually do not respond well to the usual therapeutic modalities (Braverman, 1991).

Kaposi Sarcoma

Kaposi sarcoma is one of the most common cutaneous soft tissue sarcomas **(**Table 5). There are four types of Kaposi Sarcoma: classic KS, which typically occurs in middle aged and elderly men of Mediterranean and Ashkenazi Jew background; endemic KS, which is seen among native residents of equatorial Africa, where it comprises ~10% of reported cancers; immunocompromised KS, which occurs following solid-organ transplantation or in patients receiving immunosuppressive therapy; and epidemic AIDS-related KS, which is the most clinically aggressive form of KS (Schwartz et al, 2008). The human herpes-virus type 8 (HHV-8) virus is a known cause of Kaposi Sarcoma (Dourmishev et al, 2003).

Because KS is one of the common manifestations of AIDS, its incidence and demographic patterns mimic trends seen for AIDS. Approximately 15% of all AIDS patients develop Kaposi sarcoma (Schwartz et al, 2008). In a recent review of cutaneous soft tissue tumors, it was shown that Blacks had a higher incidence rate of KS than Caucasians, 23.5 and 17.5 per 1,000,000 person-years, respectively (Rouhani et al, 2008). Asians had a much lower incidence rate of KS (4.0 per 1,000,000 person-years).

Cutaneous KS is characterized by painless, violaceous macules, nodules, or plaques **(**Table 5). In contrast to Caucasians, the violaceous hue may be difficult to detect in dark-skinned individuals (Halder and Bridgeman-Shah, 1995). KS can be an indolent disease with only skin manifestations commonly found on the lower extremities, or rapidly lead to progressive cutaneous and visceral disease (Schwartz et al, 2008). AIDS-associated KS first manifests as multiple nodules on the upper body and head and neck, swiftly evolving on the skin and in the viscera (Jessop, 2006). Chronic lymphedema may also be found in KS (Schwartz et al, 2008).

Clinical classification of KS may be the best prognostic indicator. Among classic KS patients, prognosis appears to correlate with the degree of immunosuppression and older age. Locally

aggressive KS has an intermediate prognosis, with the old African estimate of a 3-year survival rate of 64% still accurate. Generalized KS, the form seen most commonly in patients with AIDS associated KS, has a 3-year survival rate closer to 0% without therapy (Schwartz et al, 2008).

All types of KS are radiosensitive. HAART (highly active antiretroviral therapy) has reduced the incidence of KS in HIV infected patients by 10-fold. Effective HAART after 6 months is associated with involution of KS lesions in ~50% of patients. Intralesional chemotherapy, cryosurgery, and radiation have also been used (Schwartz et al, 2008).

Dermatofibrosarcoma protuberans

Dermatofibrosarcoma protuberans (DFSP) is a relatively uncommon tumor with intermediate-to-low grade malignancy (Gloster, 1996) **(**Table 6). Although rare, DFPS accounts for ~10% of all cases of skin cancer in Blacks (Halder and Bang, 1988). In a recent review of cutaneous soft tissue tumors, Blacks (6.4 per 1,000,000 person-years) were found to have higher incidence rates of DFSP than Caucasians and Asians (4.4 and 2.7 per 1,000,000 person-years, respectively) (Rouhani et al, 2008).

The cause of DFSP is unknown. There have been cytogenetic studies showing DFSP tumor cells with chromosomal abnormalities. However, the role of these cytogenetic aberrations play in the pathogenesis of DFSP is unknown (Gloster, 1996).

The clinical features of DFSP are similar in Blacks, Asians, Hispanics, and Caucasians **(**Table 6). DFSP typically presents on the trunk or extremity of adults between 20 and 50 years of age as a violaceous, red-brown, or flesh colored, indurated plaque that with time develops protuberant nodules (Halder and Bridgeman-Shah, 1995). It typically ranges in size from 1 to 5cm and is similar in appearance to keloids. Atypical keloids in people of color, such as those unusual in appearance or occurring in nontraumatized areas of skin or in nontension areas, and keloids with rapid clinical growth should be biopsied (Halder and Bridgeman-Shah, 1995).

DFSP metastasizes in only 5% of cases (Gloster, 1996), and most commonly disseminates hematogenously to the lungs. Although metastasis rarely occurs, DFSP is a locally aggressive tumor with a high recurrence rate. Treatment for DFSP is primarily Moh's micrographic surgery (Gloster, 1996).

Summary

In general, skin cancer is uncommon in people of color when compared to Caucasian. When it does occur, it is often associated with increased morbidity and mortality. Differences in survival rates may be attributed to skin cancers being diagnosed at a more advanced stage, and socioeconomic factors such as lack of adequate insurance coverage and lack of transportation can function as barriers to timely diagnosis and early treatment. In addition to advanced stage at presentation, malignant skin lesions in skin of color often present in an atypical fashion. Because skin cancer prevention and screening practices historically have been lower among Hispanics, Blacks, and Asians and given the changing demographics in the United States, interventions that are tailored to each of these groups will be needed. Public educational campaigns should expand their efforts to educate people of all skin types with emphasis on skin cancers occurring in areas not exposed to the sun (Byrd-Miles et al, 2007), since sunlight is not as important an etiologic factor in the pathogenesis of skin cancer in people of color. Dermatologists and primary care physicians should instruct their darker-skinned patients on how to perform routine skin self-examinations. Physicians should also encourage patients to ask their specialists such as their gynecologist, dentist, and ophthalmologist to look for abnormal pigmentation during routine exams.

To reduce the burden of skin cancer, several prevention methods for all people have been strongly encouraged, including monthly self-examinations, daily use of SPF 30 or greater sunscreen, sunglasses with UV-absorbing lenses, and avoidance of tanning booths (American Cancer Society, 2008) **(**Table 7). In addition, recommendations for clinicians to promote the prevention of skin cancer in skin of color have also been made, including monitoring closely changing pigmented lesions on the palms and soles and hyperkeratotic or poorly healing ulcers in immunosuppressed patients (Halder and Bridgeman-Shah, 1995) **(**Table 7).

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Table 1

Basal cell cancer features in Caucasians and People of Color

	Caucasians	**People of color**
Frequency of disease	Most common skin cancer	Most common skin cancer in Hispanics and Asians
Risk factors	Sun exposure, fair skin, previous radiation therapy, and genetic disorders (albinism, nevoid basal cell carcinoma syndrome, Xeroderma pigmentosum)	Sun exposure, scars, ulcers, previous radiation therapy, and genetic disorders(albinism, nevoid basal cell carcinoma syndrome, Xeroderma pigmentosum)
Typical clinical presentation	Translucent, solitary nodule with central ulceration and telangiectasias	Pigmented BCC, with“black, pearly appearance” common
Anatomic distribution	Head and neck regions	Head and neck regions

Table 2

Squamous cell cancer features in Caucasians and People of Color

	Caucasians	**People of color**
Frequency of disease	2nd most common skin cancer	Most common skin cancer in Blacks; 2nd most common in Hispanics, Asians
Risk factors	Sun exposure, fair skin, immunosuppression, human papillomavirus infections, scarring	Chronic scarring/inflammation from burns, leg ulcers, radiation, lupus, immunosuppression, human papillomavirus
Typical clinical presentation	Superficial lesion arising from indurated, rounded, elevated base	Sore that will not heal(bleeding or developing a crust)
Anatomic distribution	Sun exposed areas (head and neck, hands)	Skin infrequently exposed to the sun (legs)

Table 3

Melanoma features in Caucasians and People of Color

	Caucasians	**People of color**
Frequency of disease	3rd most common skin cancer; incidence increasing	3rd most common skin cancer; incidence increasing in Hispanics
Risk factors	Sun exposure, fair skin, family history, increased number of nevi, dysplastic nevi	Sun exposure; unknown for acral melanoma
Clinical presentation	Dark, rapidly spreading macules or patches arising from pigmented nevi	Dark, rapidly spreading macules or patches arising from pigmented nevi; Rapidly changing pigmented band on the nail(Hutchinson's sign)
Anatomic distribution	Often on trunk or lower legs	Often on palmar, plantar and subungual areas (acral melanoma)

Table 4

Cutaneous T-cell lymphoma features in Caucasians and People of Color

	Caucasians	**People of color**
Patterns of disease	Most common cutaneous lymphoma	Most common cutaneous lymphoma; Blacks twice as likely to be affected
Risk factors	Largely unknown	HTLV-1 endemic in Caribbean, Japan, sub- Saharan Africa, and South America
Clinical presentation	Erythematous, slightly scaling, annular or arcuate patches	Hypopigmented lesions presenting as macules or patches with ill-defined borders and no or minimal scaling
Anatomic distribution	Involves sun-shielded trunk“bathing suit distribution”	Involves sun-shielded trunk“bathing suit distribution”

Table 5

Kaposi sarcoma features in Caucasians and People of Color

	Caucasians	**People of color**
Patterns of disease	One of the most common cutaneous soft tissue sarcomas	Blacks have higher incidence rates of KS than Caucasians. Asians have a much lower incidence rate.
Risk factors	Mediterranean and Ashkenazi Jew background; immunosuppression (organ transplants); AIDS; HHV-8	Endemic KS in Africa; immunosuppression (organ transplants); AIDS, HHV-8
Clinical presentation	Painless, violaceous macules, nodules or plaques	Painless, violaceous macules, nodules or plaques; violaceous hue may be difficult to detect in darker skinned populations.
Anatomic distribution	Commonly on lower extremities.	Commonly on lower extremities.

Table 6

Dermatofibrosarcoma protuberans features in Caucasians and People of Color

	Caucasians	**People of color**
Patterns of disease	Rare	~10% of all skin cancers in Blacks; Blacks have higher incidence than other groups
Risk factors	Unknown	Unknown
Clinical presentation	Violaceous, red-brown, or flesh colored indurated plaque that with time develops protuberant nodules.	Violaceous, red-brown, or flesh colored indurated plaque that with time develops protuberant nodules.
Anatomic distribution	Trunk or extremities	Trunk or extremities

Table 7

Recommendations for Patients and Clinicians for the Prevention of Skin Cancer in Skin of Color

Recommendations for Patients
Perform routine skin examinations and report any new and/or changes of existing skin lesions to health care providers.
Sunscreen (sun protection factor 30 or greater) should be applied to skin, regardless of complexion, during periods of prolonged exposure, especially during peak hours of sunlight (10 am–2 pm).
Wear sunglasses that have UV-absorbing lenses.
Avoid tanning booths.
Recommendations for Clinicians
Pigmented lesions in people of color, particularly those of mucosal, palmar, plantar, and subungual surfaces, should be monitored closely and biopsied if any enlargement or ulceration is noted.
Hyperkeratotic or poorly healing lesions in people of color with chronic discoid lupus erythematosus should be biopsied.
Nonhealing ulcers on skin of color, regardless of original etiology, of significant duration should be biopsied.
Atypical appearing keloids and those appearing in atypical nontraumatized locations on skin of color or in nontension areas should be evaluated.
CTCL should be included in the differential diagnosis of hypopigmentation disorders of people of color.