Objectives: At the conclusion of this program, the participant will be able to:

- Distinguish the prominent differences between skin of color and Caucasian skin
- Identify characteristics of 3 or more common skin conditions in skin of color

I. Problem
   A. The problem for clinicians when assessing patients with pigmented skin is the lack of guidance and/or evidence. There is a paucity of well-controlled studies for conditions unique to patients with skin of color, so many treatment recommendations are based on case studies or clinical experience alone.\(^6\)
   B. An increase in the awareness and education regarding skin of color for both patients and clinicians is important so that patients’ concerns are addressed at earlier stages and managed more comprehensively.\(^5\)
   C. Understanding racial differences in skin function is essential for skin care, prevention and treatment of skin diseases and injuries.\(^17\)

II. Definition
   A. What is skin of color
      1. People with skin of color are non-Caucasian skin types of various racial and ethnic backgrounds.
      2. Skin of color, or ethnic skin refers to the broad range of skin types and complexions that characterize individuals with darkly pigmented skin, including (but not limited to) persons of African, Asian, Latino, Native American, and Middle Eastern descent.\(^1\)
         b. Latino or Hispanic - Persons of Spanish and indigenous Central/South American descent, including Central Americans, South Americans, and Caribbean-American persons of Spanish descent, including Cuban, Puerto Rican, and Dominican
         c. East Asian - Chinese, Japanese, Korean
         d. Southeast Asian and Pacific islander - Filipino, Vietnamese, Cambodian, Thai, Malaysian, Laotian, Burmese, Hmong descent, Polynesian, Micronesian
         e. Australoid - Australian aborigine, Melanesian descent (new the Republic of Guinea, Papua, Solomon Islands)
         f. Native Americans - More than 560 recognized tribes, including Inuit.
         g. East Indian - Indian, Pakistani, Bangladesh, Sri Lanka
         h. Middle Eastern - Iranian, Iraqi, persons from Saudi Arabia and the Arabian Peninsula (including Kuwait, Bahrain, Oman, Qatar, the United Arab Emirates, Yemen), Lebanese, Afghani, Jordanian, Syrian, Israeli, Turkish, North African (Egypt, Morocco, Algeria, Libya)
      3. Skin color is determined by the amount of melanin in the skin.
         a. Constitutive skin color is the basic melanin content of our skin and is genetically predetermined without the influence of the environment, by ultraviolet (UV) light or hormone exposure. Typically, it’s seen in areas of little or no sun exposure, such as the underside of the upper arm.
         b. Facultative skin color results from influences such as exposure to UV light (sunlight) and hormones, and the way they impact the skin. Tanning, for instance, changes the composition of melanin in the skin and increases the amount and size of melanin produced by melanocytes. Thus, facultative skin is darker than constitutive skin.
4. Melanin
   a. Melanocytes - produce and distribute melanin, the brown pigment of skin, each melanocyte distributes melanin to 30 keratinocytes, as keratinocytes move upward melanin is distributed throughout all layers of the epidermis
   b. The purpose of melanin is to protect your skin by absorbing harmful UV radiation and it darkens when doing so, producing pigmentation.
   c. There is no difference in number of melanocytes between different skin types, but the concentration of epidermal melanin in melanosomes is double in darker skin types compared to lightly pigmented skin types.¹
   d. Racial differences in skin color are attributed to differences in the rate (number, size and groupings) at which melanosomes are produced and melanized in melanocytes and then transferred, distributed and degraded in keratinocytes.⁶

B. Skin Classification Scales
   1. Several numerical classification schemes for categorizing the color of skin have been developed, however they all have limited ability for accurately communicating patient information.
   2. The most widely used classification system used by dermatologists is the Fitzpatrick Scale (also Fitzpatrick skin typing test or Fitzpatrick photo-typing scale). Devised in 1975, this system was designed to classify an individual’s response to ultraviolet radiation (UVR) with respect to burning or tanning ability, however over time; the scale has been used to classify skin color. ⁴

<table>
<thead>
<tr>
<th>Fitzpatrick Skin Type</th>
<th>Skin Color</th>
<th>Common hereditary backgrounds</th>
<th>Visual Reaction to Sun</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Pale White</td>
<td>Nordic, Scandinavia (Swedish, Danish)</td>
<td>Always burns; never tans</td>
</tr>
<tr>
<td>II</td>
<td>White</td>
<td>Irish, English, Welsh</td>
<td>Usually burns</td>
</tr>
<tr>
<td>III</td>
<td>Light Brown (naturally tan)</td>
<td>Asian, Mediterranean (Italian, Greek)</td>
<td>Mildly burns; tans relatively well</td>
</tr>
<tr>
<td>IV</td>
<td>Moderate brown</td>
<td>Hispanic, Middle Eastern, African-American, Native American</td>
<td>Rarely burns; tans well</td>
</tr>
<tr>
<td>V</td>
<td>Dark brown</td>
<td>Hispanic, Middle Eastern, African-American, Native American, Southeast Asian</td>
<td>Very rarely burns; tans easily</td>
</tr>
<tr>
<td>VI</td>
<td>Black</td>
<td>African-American, Southeast Asian</td>
<td>Least likely to burn; tans very darkly</td>
</tr>
</tbody>
</table>

³The Fitzpatrick scale isn't particularly helpful in nursing/wound care assessment because of its focus on the effects of sun exposure and because dark-skinned people fall into primarily one category. ⁵For clinical skin-color assessment, visual inspection and asking patients about their normal skin color are the best methods. ⁵

III. Distinguishing Characteristics in Ethnic Skin Color
   A. Asian and black skin has a thicker, more compact dermis than white skin with the thickness being proportional to the degree of pigmentation. ¹⁰
      1. Dark skin has extremely compact bundles of collagen and in greater numbers resting just below the epidermis, and “the dermal-epidermal junction length in dark skin is about threefold that in light skin”. ⁹
B. Ethnic skin is very prone to either a loss or gain of color adjacent to other areas of the body, hypopigmentation or hyperpigmentation also known as dyschromia, which is often the result of cutaneous inflammation, injury, or dermatologic treatment.
   1. Most cases of post-inflammatory hypopigmentation improve spontaneously within weeks or months if the primary cause is removed; however, it can be permanent if there is complete destruction of melanocytes.5,6
   2. The exact cause of how hyperpigmentation occurs after an injury is unknown but might result as an influence of inflammatory mediators and reactive oxygen species.7
   3. Pigmentary disorders are one of the most common skin concerns among women and persons of color, can cause psychological and emotional distress and can pose a significant negative impact on a person’s health-related quality of life.7

C. Fibroblasts are reportedly larger and more numerous in dark skin than those found in light skin, suggesting heightened activity (or reactivity), which may influence keloid and hypertrophic scar formation complications particularly common in individuals of African and Asian ancestry.8,10
   1. Likelihood of scarring among ethnic groups is 15 times higher than in Whites, and should be discussed with a patient of color that is facing surgery or treatment of the skin that involves some level of trauma.3

D. Transepidermal water loss (TEWL) is defined as the total amount of water vapor lost through the skin and appendages, under non-sweating conditions.
   1. Black and Asian skin have higher transepidermal water loss (TEWL) when compared to white skin.3,13,14 Which leads to the conclusion that Black and Asian subjects have a more compromised epidermal barrier function and would most likely be more susceptible to irritants.13
   2. Additionally, because transepidermal water loss is greater in Blacks than Whites, it may account for greater increases in xerosis, though studies are inconclusive and ongoing.3

E. Black skin has a lower pH compared to Whites with some variations according to body sites and depth of skin layers.13,14
   1. Typically skin has a pH in the acidic range varying from 5.5 to 6.5. A pH of less than 7 is acidic and a pH of greater than 7 is alkaline.

F. Stratum corneum in black skin is more compact with a greater cohesion between keratinocytes that results in vesicles and bulla that remain intact longer than similar lesions on white skin.3 In addition; stratum corneum contains melanosomes, whereas light skin has no melanosomes in stratum corneum.27

G. Individuals with darker skin tones are naturally more susceptible to vitamin D insufficiency and deficiency. Darker skin, which contains more melanin than relatively lighter skin, offers natural protection from UV radiation. Melanin, the principal skin pigment, reduces but does not block cholecalciferol [vitamin D3] synthesis.12 Thus, longer periods of sun exposure are required for equivalent vitamin D synthesis in people with dark skin.12
   1. Optimal vitamin D status - at least 100 nanomoles per litre (nmol/L) of 25(OH)D. Vitamin D deficiency is defined by some as 25(OH)D levels below 50 nmol/L.
   2. To achieve 25(OH)D ≥75 nmol/L, recommended that European ancestry individuals with high sun exposure need 1300 IU/d vitamin D intake in the winter and African ancestry individuals with low sun exposure need 2100–3100 IU/d year-round.12

H. Signs of Aging
   1. Because skin of color is less susceptible to sun-induced photodamage, clinical manifestations of aging are less severe and typically occur 10 to 20 years later than those of age-matched white counterparts.15
2. However the same photoprotection afforded by differences in melanosome and melanin characteristics also causes frequent hyperpigmentation in skin of color, and may be responsible for divergent responses observed in burn injuries.\textsuperscript{16}

I. The classic signs of skin damage, injury, and response may present differently in Caucasian and dark pigmented skin due to varying amounts of melanin. When there is an extremely high melanin content the color of the skin can be so dark that it is difficult to assess any changes in color.\textsuperscript{32}

IV. Assessment
A. Some skin conditions/ diseases present differently or ‘atypically’ in a patient with dark skin, so it is important that clinicians are aware of these differences when they assess patients.\textsuperscript{18,19}
B. Holistic Assessment\textsuperscript{18}
1. Detailed history of the skin condition - should include information on its duration, occurrence, and any variations.
2. General assessment - state of mind, physical abilities, cultural needs, and physiological status.
3. Assessment of the patient’s knowledge.
4. Specific physical assessment of the skin
   a. Use all senses. Look, listen (to the patient/family), touch and smell.
   b. Careful inspection and palpation of the skin should be based on a head-to-toe assessment with focus on areas at high risk for breakdown.
   c. Evaluate skin temperature, skin dryness, itching, bruising, and changes in texture of the skin and nail composition. Observe for color and uniform appearance, skin thickness, skin symmetry, lesions, trauma, or skin breakdown.
C. Tips for Assessment with Skin of Color
1. Use natural or halogen light, not fluorescent lighting. Fluorescent light imparts a bluish tone to dark skin and makes it harder to see skin changes.\textsuperscript{5}
2. Use an additional light source such as a penlight to illuminate hard to see skin areas such as the heels or sacrum.\textsuperscript{20}
3. Avoid wearing tinted lenses when assessing skin color.
4. If using photography with assessment, it is recommended to use a flash with the camera, as the flash makes demarcation between normal skin tones and those that are injured easier to see.\textsuperscript{28}
5. May need to remove gloves to perform skin assessment as it is critical to feel skin temperature changes when assessing dark skin.\textsuperscript{5}
   a. Most clinicians use the back rather than the palm of their hand to assess the temperature of a patient’s skin.\textsuperscript{20}
   b. Remember that increased skin temperature can be a sign of fever or impending skin problems such as a Stage I pressure ulcer or a diabetic foot about to ulcerate.
   c. Touch the skin to evaluate if it is warm or cool.
   d. Compare symmetrical body parts for differences in skin temperature.
6. Know the person’s normal skin tone so that you can evaluate changes.\textsuperscript{20}
   a. Assess non-injured areas first. Ask the patient/family to point out an area of normal skin color, temperature, and texture.\textsuperscript{5}
   b. Exposed areas may vary in color with unexposed areas.
7. Check skin areas with the least amount of pigment – palms, soles, tongue, palate, oral mucosa, conjunctive, sclera, and nail beds.\textsuperscript{5}
8. Note that macerated skin from too much moisture may also appear lighter or feel soft or boggy.\textsuperscript{20}
9. Light skin usually darkens as it heals, dark skin may lighten as it heals before returning to its normal color.
   a. Repigmentation of scars depends on the availability of melanocytes and this will depend on the mechanism of injury; in partial thickness injuries the wound bed will contain residual
adnexal elements which will be able to provide melanocytes and epithelial cells to the neo-
epithelium. However, in deep injuries in which all adnexal elements have been removed or
destroyed by the wounding process, the only available source for melanocytes will be
the wound edges. These wounds will take longer to heal and will heal with a
hypopigmented center in contrast to the surrounding unwounded skin.

10. Assessment of tissue circulatory status in dark skin
   a. Color changes will appear more subtle than light skin
   b. Assess patient from a neutral position and then with the area elevated approximately 15°
and dependent for about 5 minutes, and compare.
   c. Assessment of capillary refill time for darkly pigmented skin should be attempted at the
tips of the second or third fingers. In addition, examine nail beds by applying pressure
to the second or third finger, if the skin under the nail blanches, it will provide color
comparison for assessing pallor or cyanosis. The slower the return of color the more
diminished the vasomotor function.
   d. Pulse oximetry may be less reliable for darkly pigmented skin. Darker pigments are thought
to cause errors in SpO2 readings. Among patients with hypoxia (low SaO2) who have darker
skin pigments, errors in SpO2 are increased in up to 10% of cases.

D. Color Assessment in Light and Dark Skin
   1. In general, lesions that appear red or brown on light skin often present as black or purple on
dark skin.
   2. Pallor – loss of color in skin due to absence of oxygenated hemoglobin
      a. Light Skin – White skin loses its rosy tones; Skin with natural yellow tones appears more
         yellow
      b. Dark Skin – Black skin loses its red undertones and appears ash-gray. Brown skin becomes
         yellow-tinged; skin looks dull; Conjunctive and oral mucosa appears gray, white, or ashen.
   3. Absence of Color – Congenital or acquired loss of melanin pigment
      a. Light Skin – Albinism appears as white skin, white or pale blond hair, and pink irises. Vitiligo
         appears as patchy milky-white areas, especially around the mouth.
      b. Dark Skin - Albinism appears as white skin, white or pale blond hair, and pink irises. Vitiligo
         is very noticeable as patchy milk-white areas.
   4. Cyanosis – Mottled blue color in skin due to inadequate tissue perfusion with oxygenated
      blood.
      a. Light Skin – Skin, lips, and mucous membranes look blue-tinged. The conjunctivae and nail
         beds are blue.
      b. Dark Skin – Skin may appear a shade darker. Cyanosis may be undetectable except for the
         lips, tongue, and oral mucous membranes, nail beds, and conjunctivae, which appear pale
         or blue tinged. Grayish white color around the lips or tongue, or maroon tinge to the nail
         beds.
   5. Erythema – Redness of the skin due to increased visibility of normal oxyhemoglobin.
      a. Light Skin – Readily identifiable over entire body or in localized areas. Local inflammation
         and redness are accompanied by higher temperature at the site.
      b. Dark Skin - Difficult to detect. Localized areas of inflammation appear purple, purple-blue,
         violet, eggplant color or darker than surrounding skin. May be accompanied by higher
         temperature, hardness, swelling.
   6. Jaundice – yellow undertone due to increased bilirubin in the blood.
      a. Light Skin – Yellow first in sclerae, hard palate, and mucous membranes; then generalized
         yellow tint over all skin.
b. Dark Skin – Yellow tint visible in the sclerae, oral mucosa, junction of hard and soft palate, palms of the hands, and soles of the feet. Note: If patient wearing yellow clothing, remove before the assessment.

7. Carotenemia – yellow orange tint caused by increased levels of carotene in the blood and skin.
   a. Light Skin – Yellow-orange seen in forehead, palms, soles. No yellowing of sclerae or mucous membranes.
   b. Dark Skin – Yellow-orange tint most visible in palms of the hands and soles of feet. No yellowing of sclerae or mucous membranes.

8. Hemosiderin Staining - When blood leaves a ruptured blood vessel, the red blood cell dies, and the hemoglobin of the cell is released into the extracellular space leaving dark pigment in the surrounding tissue.
   a. Light Skin - Skin becomes reddish, rusty, brown or gray; may appear as a ring around pressure ulcers or as discoloration of the skin of the lower leg of patients with venous disease
   b. Dark Skin - Discoloration or darker than surrounding skin.
   c. Assessment Tip: Hemosiderin Staining usually occurs close to the wound edges, whereas injury-related color changes usually extend out a considerable distance and are accompanied by the other signs of inflammation.  

E. Assessment of Stage I pressure ulcer injury in Dark Skin

1. National Pressure Ulcer Advisory Panel definition of Category/Stage I: Non-blanchable erythema
   a. Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.  
   b. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue. Category I may be difficult to detect in individuals with dark skin tones. May indicate “at risk” persons.

2. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Visual assessment cannot be trusted in patient with dark skin, rely on assessment of skin temperature, change in tissue consistency and pain to identify sDTI.
   a. Hyperpigmentation “increased browning”, or darkening of tissue compared to surroundings.
   b. Palpation: SHOULD BE USED on ALL Dark Skinned Pts on high risk areas.

3. As with light-colored skin remove pressure and allow the area to be exposed to ambient room temperature for 5-10 minutes before examination.

4. Perform a head-to-toe inspection of the skin focused on high-risk areas such as bony prominences, skin folds, and under medical devices.

5. Assess localized skin color changes. Any of the following may appear:
   a. The color of intact dark skin remains unchanged (does not blanch) when pressure is applied over a bony prominence.
   b. Color changes that differ from patient’s usual skin color occur at site of pressure ulcer.
   c. If patient previously had a pressure ulcer, that area of skin may be lighter than original color.
   d. Localized area of skin may be purple/blue or violet instead of red.

6. Assess tissue consistency
   a. Edema and induration indicate abnormal fluid accumulation and occur when pressure causes the skin layers to separate, allowing interstitial fluid to accumulate, which may suggest further tissue damage, abscess or a wound infection.
b. Edema (non-pitting swelling) may occur with induration of more than 15mm in diameter and may appear taut and shiny.  

7. Skin Temperature Assessment  
   a. Localized heat (inflammation) is detected by making comparisons to surrounding skin.  
   b. Localized area of warmth eventually will be replaced by area of coolness, which is skin of tissue death. 

8. Assess for pain, recently relieved pain and or discomfort in high risk areas. 

F. Normal Pigmentary Variations In Skin Of Color  

1. Knowledge of normal variations in skin is crucial in evaluating and treating patients with skin of color. 

2. Pigmentary Demarcation Lines  
   a. Normal boundaries of the skin that represent a transition between darker and lighter melanin pigment distribution. In all skin types, the dorsal skin surfaces are relatively hyperpigmented compared to the ventral surfaces. In individuals with darkly pigmented skin, visible lines of demarcation between dorsal and ventral surfaces are more conspicuous. 
   b. Incidence - 79% of black female adults and 75% of black men have PDL of at least one type and 15% of white females have at one PDL. 
   c. Types of Demarcation Lines  
      1) Type A – Location: Anterolateral upper arms, pectoral area; Hyperpigmented; Also known as Voigt’s or Futcher’s lines 
      2) Type B – Location: Posteromedial aspect of lower legs; Hyperpigmented; often arise during pregnancy 
      3) Type C – Location: Vertical line in presternal area; Hypopigmented 
      4) Type D – Location: Posteromedial area of spine; Hyperpigmented 
      5) Type E – Location: Chest from mid-third of clavicle to periareolar skin; Hypopigmented 
      6) Three facial patterns (F-H) have been commonly described in the skin of Indians. Their appearance is distinctive enough to be recognized as a fairly common variant of normal pigmentation in the Indian sub-population. 
         - Type F – These are dark patches that appear 'V' shaped or as an inverted cone on the side of the face in the region between the cheek and the temple. They are invariably similar and of almost equal intensity on both the sides. 
         - Type G – These are also dark patches in the same location but have two inverted cones lying in close proximity, looking like the letter ‘W’, with a faint strip of normal color in between. Both patterns F and G show evenly diffuse pigmentation with a well-defined margin which is better appreciated from a little distance. 
         - Type H – These are two symmetrical linear bands of darker color extending from just below the angle of the mouth to the sides of the chin. In many women there is often an additional band running just below and parallel to the lower lip. 
   
3. Infant Hyperpigmentation 
   a. Black infants have localized areas of hyperpigmentation. 
   b. The usual areas of involvement are the helix of the ears, lips, fingernail and toenail matrix areas, penis, scrotum, vulva, nipples, umbilicus, axillae, and anal orifice. 
   c. Between 60% and 85% of all black infants have these localized areas of darkness. 
   d. Finger, toe, nail matrix, nipple, penis, scrotum, and vulvar hyperpigmentation seems to persist for the duration of one’s life, whereas earlobe and axillary hyperpigmentation seems to disappear during the first year of life.
4. Nail pigmentation (longitudinal melanonychia/melanonychia striate)
   a. Longitudinal bands of brown pigment starting from the matrix and extending to the tip of
      the nail plate; occurs on fingernails more than toenails
   b. Often occur as an acquired condition in pigmented skin, and there is often a history of
      trauma.
   c. Incidence varies according to skin pigmentation with percentages that may be as high as
      77% in African-Americans over 20 years of age and up to 90% in those over 50 years of age
      but varying with depth of skin color, 10% to 20% in Asians, and about 1% in Caucasians.
   d. Differential Diagnosis – There seems to be no association of melanonychia with any
      systemic diseases; however, melanoma must be ruled out.
      1) A grayish background and thin, regular gray lines are common characteristics in ethnic
         type nail pigmentation.
      2) Longitudinal black lines of subungual melanoma tend to be involvement of one nail
         with a width of 6 mm or more, Brown-black band, irregular in color, spacing, thickness,
         and parallelism, with over 75% of subungual melanomas involving either the great toe
         or the thumb.
      3) If unsure, recommend dermatologist consult.

5. Palmer and Plantar Hyperpigmentation
   a. Hyperpigmented macules on the palms and soles area normal pigmentary variant in people
      with skin of color. These lesions vary in size and shape and may be sharp or ill-defined
      with a reticulated appearance.
   b. Creases on the palms often present with hyperpigmentation and may contain
      hyperkeratotic papules or pits. Most often occurring in the creases of the palms and
      soles, punctate keratoses are 1- to 5-mm depressed comedo-like keratinous plugs,
      Punctate keratoses are a benign normal variant seen most often in black patients.
   c. Study result showed: Hyperpigmented macules and patches of the palms were present in
      35% of black adults examined and in more than 50% of those over age 50 but absent in
      black infants.
   d. Since hyperpigmentation of neither the palms nor the soles is present at birth, trauma
      leading to postinflammatory changes may be the precipitating factor.

6. Idiopathic guttate hypomelanosis
   a. A benign and asymptomatic skin manifestation characterized as diffuse hypopigmented
      macules, or white spots.
   b. Most often found on the anterior shins and sun-exposed parts of the forearms.
   c. Affects all races, is more frequent in women, and tends to increase in incidence with age.
   d. The sizes of the lesions vary from 1-10mm, but are most commonly 1-3 mm in diameter.
   e. Individuals with very dark skin, the initial lesions are often yellow-brown in color.
   f. It is present in more than 90% of black senior citizens, with the legs, thighs, abdomen,
      arms, and back involved. Rare in children and young adults.
   g. The cause is not known, but it appears to be related to the effect of the sun on
      melanocytes, which makes them weak/ineffective.
   h. Patients may worry that these macules are vitiligo.

G. Ashy Skin
   1. “Ashy skin” or “Ashiness” is a lay term describing a condition characterized by any xerotic
      process with loss of natural skin shine, in other words, very dry skin.
   2. Common physiological skin condition that may develop in people with dark skin complexion.
   3. Associated with a whitish or grayish coloring (much like ashes left after something has been
      burned, hence the term) that appears on brown and darkly pigmented skin. All skin tones can
experience “ashiness.” Caucasian and pale Asian skin can look chalky from dead cells on its surface; it’s just that the dead skin cells are more noticeable on darker skin.  

4. Ashy skin is commonly found on arms, elbows, lower legs, knees and heels.

5. The spontaneous desquamation rate (sloughing/exfoliation) of the stratum corneum in African-Americans is 2.5 times greater than that seen in Caucasians and Asians, which may account for the increased frequency of xerosis seen clinically in African-Americans.

6. Dry skin can be caused by a variety of issues, however the main cause is that one’s skin does not have the necessary moisture on the top layer of their skin.

7. Treatment
   a. Agent to maintain skin moisture.
   b. Emollient lotions or creams are one of the most effective treatments
   c. Avoid products that contain alcohol because they evaporate and their drying action compounds the original problem.
   d. Petroleum based products seal the skin surface and prevents what little lubrication is made from evaporating, but they do not penetrate the surface of the skin and do not replace skin moisture.
   e. Alpha hydroxy acids are frequently used to treat xerosis. Alpha hydroxy acids include glycolic, citric, lactic, mandelic and tartaric acid. Through a chemical process, these acids accelerate the softening of the skin, dissolving or peeling the outer layer of the skin to help maintain the skin’s capability to hold moisture. Lactic acid in concentrations of 2.5 percent to 12 percent is the most common alpha hydroxy acid used for moderate to severe xerosis.
   f. Examples: Atrac-Tain® Cream, Eucerin® 10 % Urea Lotion, Lac-Hydrin 12%.

V. Pigmentation Disorders
   A. Pigmentary disorders can affect all skin types, individuals with darker skin, including Asians, blacks, Latinos, and American Indians, are more susceptible. Pigmentary lesions may be more visible in skin of color and are often resistant to treatment.
   B. Pigmentary disorders can cause psychological and emotional distress and can pose a significant negative impact on a person’s health-related quality of life.
      1. In a prospective cohort study on the prevalence of pigmentary disorders and their impact on quality of life:
         a. 47.3% of patients admitted being self-conscious about their skin to some degree
         b. 32.7% reported feeling unattractive because of their skin
         c. 32.7% put effort into hiding pigment changes
         d. 23.6% thought their skin affected their activities
         e. 21.8% thought others focused on their skin
   C. Hyperpigmentation
      1. Melasma
         a. Melasma is an acquired symmetric hypermelanosis characterized by hyperpigmented patches on sun-exposed areas of the face, neck, back and forearms. 
         b. Clinical Presentation
            1) Patches appear as irregular light-brown to gray-brown patches
            2) Most common is symmetrical irregularly shaped areas of hyperpigmentation on both cheeks
         c. Although the exact cause of melasma is unknown, it is strongly associated with three factors: hormonal influences, ultraviolet (UV) radiation, and genetic predisposition.
         d. Incidence
            1) It predominantly affects women, with men comprising only 10% of all cases.
2) Affects all racial groups but is most prevalent in darker-skinned individuals (Fitzpatrick’s skin types IV–VI), such as Hispanics, East Asians, Southeast Asians, and blacks who live in areas of intense ultraviolet (UV) radiation. 

3) Occurs commonly during pregnancy, with data suggesting an incidence of 50–70% in pregnant women. 

e. Chronic, relapsing disorder that can be managed effectively but not cured. Effective treatment of melasma often has a prolonged course, and the patient must be aware of the “long-term commitment” 

f. Treatment

1) Minimizing UV exposure
2) Minimizing contributing hormonal influences
3) Hydroquinone Topical
   - Primary topical ingredient for inhibiting melanin production
   - “Gold standard” of melasma treatment either as monotherapy or combined with other agents 

4) Chemical peels, microdermabrasion, Fraxel, Laser treatments

2. Postinflammatory hyperpigmentation (PIH)

a. Discoloration that is left on the skin after an underlying skin disease has healed such as areas of prior inflammatory disease, infection, allergic contact or irritant reactions, injury from prior procedures or trauma, sites of papulosquamous or vesiculobullous disease, and medication reactions.

1) Most common causes are injuries such as scratches, burns, cuts, rashes or bruises. 
2) Ordinary conditions such as acne or pimples are a very common cause of PIH in individuals with brown skin. 
3) PIH can also be caused by injury to the skin resulting from sunburns, surgery or cosmetic procedures such as chemical peels, dermabrasion, lasers and cryotherapy 

b. Incidence

1) Although postinflammatory hyperpigmentation occurs in whites, but more frequently affects skin-of-color patients, including African Americans, Hispanics/Latinos, Asians, Native Americans, Pacific Islanders, and those of Middle Eastern descent. In the Asian population it is more prevalent among Asians with darker skin, such as Malays and Indians, than those with lighter skin, such as the Chinese. 
2) Occurs with equal incidence in males and females 

c. Clinical Presentation

1) Manifests as macules or patches in the same distribution as the initial inflammatory process, borders are often hazy.
2) In the epidermis, the lesions tend to be brown, but hyperpigmentation in the dermis causes lesions to have a dark gray or gray-blue hue.

d. Treatment

1) Epidermal pigment may take 6–12 months to fade, whereas dermal pigment may be present for years. 
2) Treatment includes topical skin-lightening agents and physical modalities such as chemical peels, microdermabrasion, and laser treatments; however these treatments may induce hyperpigmentation due irritation and inflammation of the skin. 
3) Hydroquinone (HQ), a chemical lightening agent that is applied directly to the skin is considered the “gold standard of topical treatment. 
   - Hydroquinone products can be purchased over the counter at 1% to 2% concentrations, or by prescription at 3% to 4% concentrations.
- Hydroquinone should be carefully applied to the dark marks—avoiding normal-appearing skin—once or twice a day for up to 6 months.45

D. Hypopigmentation

1. Tinea versicolor
   a. Superficial fungal infection, characterized by changes in skin pigment due to colonization of the stratum corneum by a dimorphic lipophilic fungus of the normal flora of the skin.50,51
   Not considered contagious.
   b. Clinical Presentation
      1) Multiple macules and/or patches of variable appearance hypopigmented, hyperpigmented, ivory to tan, dark brown or erythematous surrounded by normal skin.50,51
      2) Predominantly affects the sebaceous areas of back, chest, abdomen, neck, upper limbs and face.50,51
      3) Lesions are usually asymptomatic but may be mildly pruritic. The pruritus is more intense when the patient is excessively warm.
      4) Involved skin lesions fail to tan in the summer.
   c. Incidence
      1) Seen in light and dark skin equally, however often more apparent in persons of color because of greater contrast.50
      2) More common in teenagers and young adults than in older people.
      3) Occurs more frequently in areas with higher temperatures and higher relative humidities
   d. Treatment
      1) The condition does not leave any permanent scar or pigmentary changes, and any skin color alterations resolve within 1-2 months after treatment has been initiated.50,51
      2) Recurrence is common, and prophylactic therapy may help reduce the high rate of recurrence.
      3) Treatment is topical antifungal shampoos, creams, or lotions, including selenium sulfide, terbinafine, or imidazoles, or pulsed systemic antifungal therapy with oral ketoconazole, fluconazole, or itraconazole.50,51
         - Selenium sulfide lotion is liberally applied to affected areas of the skin daily for 2 weeks; each application is allowed to remain on the skin for at least 10 minutes prior to being washed off.50,51
         - Topical azole antifungals can be applied every night for 2 weeks.50,51

2. Vitiligo
   1) Vitiligo is an acquired pigmentary disorder of the skin and mucous membranes, and it is characterized by circumscribed de-pigmented macules and patches. Vitiligo is a progressive disorder in which some or all of the melanocytes in the affected skin are selectively destroyed.52
   2) Cause
      a. The predominant finding in the de-pigmented areas of vitiligo is an absence of epidermal melanocytes.52,53
      b. The precise cause of the loss of these epidermal melanocytes is unknown.
      c. Theories regarding loss of melanocytes include autoimmune mechanisms, cytotoxic mechanisms, and an intrinsic defect of melanocytes, oxidant-antioxidant mechanisms, and neural mechanisms.52,53,54
   3) Clinical Presentation
      a. White or hypopigmented macules or patches. The lesions are usually well demarcated, and they are round, oval, or linear in shape and vary in size.
b. Lesions enlarge centrifugally over time at an unpredictable rate.
c. Initial lesions occur most frequently on the hands, forearms, feet, and face, favoring a perioral and periocular distribution.

4) Incidence
   a. Vitiligo can occur at any age, but it usually occurs before 20 years in nearly 50% of patients and affects nearly 1–2% of the world population.
   b. Vitiligo has an equal incidence in all racial and ethnic groups.
   c. Patients with vitiligo experience low self-esteem, isolation, job discrimination, stigmatization, depression, and embarrassment in social and sexual relationships.55

5) Treatment
   a. No single therapy for vitiligo produces predictably good results in all patients; the response to therapy is highly variable.
   b. Therapeutic objectives for vitiligo should include both stabilization of the disease and re-pigmentation of vitiliginous skin lesions.52
   c. Commonly used medical therapies for vitiligo include topical and systemic steroids, narrow-band ultraviolet phototherapy, psoralen with ultraviolet A (PUVA), targeted phototherapy, nutritional vitamin supplementation, immunomodulators, and calcipotriol.52,54

6) Cosmetics
   a. Cosmetics are helpful to disguise the vitiligo (cosmetic camouflage).
   b. Dyes, stains, and make-ups can be applied and with specialist help the results can be very satisfactory.
   c. Dihydroxyacetone-containing "tan without sun" products; take care not to apply to the normally tanned skin because this will also look darker.
   d. Water-resistant concealing make-up.
   e. Resources for makeup:
      - Coloration Makeup http://www.colortration.com/index.htm
      - Giorgio Armani at Nordstrom’s
      - MAX Factor Pan-Stick

VI. Keloid and Hypertrophic Scarring
A. Keloids are the result of an overgrowth of dense fibrous tissue that usually develops after healing of a skin injury. The tissue extends beyond the borders of the original wound, does not usually regress spontaneously, and tends to recur after excision.
   1. Result of abnormal wound healing, characterized by local fibroblast proliferation and excessive collagen production in response to cutaneous injury.
   2. Although keloids are medically benign, they are often psychologically and/or socially devastating for patients.59

B. Cause
   1. The etiology of keloids is uncertain.60
   2. Proposed Etiology of Keloids
      a. Trauma
      b. Skin tension
      c. Infection
      d. Endocrine factors
      e. Genetic predisposition

C. Clinical Appearance
   1. Smooth surface and are firm to palpation.59,60
   2. They may be pruritic or painful and can even inhibit normal motion of adjacent tissues.59,60
3. Keloids may range in size from papules a few millimeters in diameter to football-sized or larger tumors.  

4. The color can vary from pink–purple (early lesions) to skin-colored to hypo- or hyperpigmented.

5. Elevated above the skin surface and extend beyond the original wound margin into adjacent normal skin, often with claw-like extensions resembling the pincers of a crab.

6. They can occur anywhere on the body, most often on the earlobes, shoulders, mid-chest and upper back, but rarely on the hands, feet, axillae, or scalp.

7. The growth is usually slow, but keloids sometimes will enlarge rapidly, tripling their size in months.

8. Malignant transformation of keloids is rare; most patients had had some type of radiation therapy prior to the development of the malignancy.

D. Incidence

1. There is a higher incidence of keloids in darkly pigmented individuals of African ancestry. From 4.5% to 16% of predominantly black and Hispanic populations have keloids, with an incidence of up to 16% in black Africans.

2. Keloids form more frequently in Polynesian and Chinese persons than in Indian and Malaysian persons.

E. Treatment

1. At Risk persons include personal or family history of keloid formation. The risk is lower among patients who have only earlobe lesions. Warn at-risk patients not to have their ears pierced, get tattoos, or undergo nonessential cosmetic surgery, such as breast reduction or augmentation, face-lifts, or tummy-tucks.

2. Although no one therapy works best for all keloids, the standard treatment is intralesional corticosteroids and/or topical corticosteroids.

3. If injections do not work, surgical removal is considered.

   a. The sutures are left in for 10–14 days because earlier removal may cause wound dehiscence.

   b. Prolonged pressure is often effective in preventing recurrence of keloids after surgical excision. An elastic garment covering the postoperative site should be worn 16 to 20 hours a day, beginning immediately after complete wound healing.

   c. Surgical excision alone has a recurrence rate of > 50%, so adjunct therapy is advised.

4. Occlusive dressings have been used with varied success.

   a. These include silicone gel sheets and dressings, non-silicone occlusive sheets, and Cordran tape.

   b. Anti-keloidal effects appear to result from a combination of occlusion and hydration, rather than from an effect of the silicon.

   c. Silicone Occlusive Sheeting - Previous studies have shown that in patients treated with silicone occlusive sheeting with pressure worn 24 h/d for up to 12 months, 34% showed excellent improvement, 37.5% showed moderate improvement, and 28% demonstrated no or slight improvement.

   d. Semipermeable, semiocclusive, non-silicone-based dressings - patients treated for 8 weeks, 60% experienced flattening of keloids, 71% had reduced pain, 78% had reduced tenderness, 80% had reduced pruritus, 87.5% had reduced erythema, and 90% were satisfied with the treatment.

   e. Cordran tape - is a clear surgical tape that contains flurandrenolide, a steroid that is uniformly distributed on each square centimeter of the tape, and it has been shown to soften and flatten keloids over time.

5. Compression
a. Compression therapy involves pressure, which has long been known to have thinning effects on skin. Reduction in the cohesiveness of collagen fibers in pressure-treated hypertrophic scars has been demonstrated by electron microscopy.63

b. Compression treatments include button compression, pressure earrings, ACE bandages, elastic adhesive bandages, compression wraps, spandex or elastane (Lycra) bandages, and support bandages.63

6. Several other keloid treatments include: cryosurgery, radiation therapy, laser therapy, interferon (IFN) therapy, 5-fluorouracil (5-FU), doxorubicin, bleomycin, verapamil, retinoic acid, imiquimod 5% cream, tamoxifen, tacrolimus, botulinum toxin, and over-the-counter treatments (e.g., combination of hydrocortisone, silicon, and vitamin E).59,60,63 Other promising therapies include antiangiogenic factors, including vascular endothelial growth factor (VEGF) inhibitors (e.g., bevacizumab), phototherapy (photodynamic therapy [PDT], UVA-1 therapy, narrowband UVB therapy), transforming growth factor (TGF)–beta3, tumor necrosis factor (TNF)-alpha inhibitors (etanercept), and recombinant human interleukin (rhIL-10), which are directed at decreasing collagen synthesis.63

Bibliography


