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Basal Cell Carcinoma—A Preventable Disease

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Introduction

Basal cell carcinoma (BCC) is a non-melanoma skin cancer. According to the Skin Cancer Foundation, "BCC is the most frequently occurring form of all skin cancers. More than one out of every three new cancers is a skin cancer, and the vast majority are BCC's" (www.skincancer.org).

Exposure to ultraviolet (UV) rays is one of the main risk factors in developing a BCC. People with a lighter skin tone are at a higher risk of developing a BCC than those with a darker skin tone.

Signs and Symptoms

Patients should be on the look-out for a lesion that looks like a pimple, but does not heal like a pimple should. Any lesion that lingers for over a month should be evaluated. BCCs are typically found on sun-exposed areas such as the face, scalp, neck, ears, and nose (Thompson, 2010). The appearance of BCCs is most commonly "a pearly pink or white, dome-shaped papule with prominent telangiectatic surface vessels that develop as the lesion changes" (Firnhaber, 2012, p.163). Telangiectasias are appreciated using dermoscopy (dermatoscope with polarized light). BCCs can have an ulcerated or crusted appearance, or may look excoriated if there has been trauma to the area (Thompson, 2010). On the trunk and extremities, BCCs may occur as a plaque that is similar to eczema or psoriasis (Firnhaber, 2012).

Underlying Pathophysiology

Basal cell carcinomas arise in basal keratinocytes found in the epidermis, eccrine sweat ducts, and hair follicles (Firnhaber, 2012). These cells "have high nuclear-to-cytoplasmic ratio, and the nuclei are hyperchromatic with small, in conspicuous nucleoli and scant eosinophilic cytoplasm" (Thompson, 2010, p.420). Microscopically, mitotic figures and numerous apoptotic bodies are identified (Thompson, 2010). BCCs are unlikely to metastasize through blood or lymphatics, this is due to its surrounding stroma (supportive network) that is necessary for the lesion to grow (Firnhaber, 2012).

Histologically, there are five major patterns of BCC: nodular, superficial, micronodular, infiltrative, and morpheaform. In approximately 40% of cases, there is a "mixed pattern, containing two or more major histologic patterns" (Firnhaber, 2012, p.163). "Basaloid cells are usually arranged in palisades at the tumor periphery" (Kasper et al., 2012, p.455).

Effects of Ultraviolet (UV) Rays

UV radiation is considered to be a "complete carcinogen" because of its ability to be both a mutagen and a non-specific damaging agent (D'Orazio et al., 2013). Properties of a tumor initiator and tumor promoter are found in ultraviolet light and excessive exposure can lead to "profound health risks, including atrophy, pigmentary changes, wrinkling, and malignancy" (D'Orazio et al., 2013, p. 12222)

One's skin tone is determined by how much melanin is produced by the body. Keratinocytes found abundantly in the epidermis accumulate melanin pigments as they are maturing. The epidermal melanin functions to bloc UV rays from penetrating the skin (D'Orazio et al., 2013). According to D'Orazio et al., "melanin exists in two main chemical forms: (1) eumelanin, a dark pigment expressed abundantly in the skin of heavily pigmented individuals, and (2) pheomelanin, a light-colored sulfated pigment resulting form incorporation of cysteines into melanin precursors" (2013, p.12224). The main determinant of skin complexion and sensitivity to UV rays is the amount of melanin ("natural sunscreen") in the skin along with the type of melanin found in the skin (D'Orazio et al., 2013).

UV rays are separated into UV-A, -B, and –C. These classifications are based on different wavelengths and energy levels. UV-C is mainly absorbed by the atmosphere, so only UV-A and –B are absorbed by the skin. The dermis absorbs UV-A rays, and UV-B is absorbed by the epidermis (D'Orazio et al., 2013)

- Once a threshold has been exceeded by UV exposure, keratinocytes activate apoptotic pathways and die (D'Orazio et al., 2013).
- Damage to keratinocytes activates response pathways like p53 activation where keratinocyte physiology is altered, cell cycle arrest is mediated, DNA repair is activated, and apoptosis is induced if there is substantial damage (D'Orazio et al., 2013).
- Several hours after exposure, damage response signals diminish, and keratinocytes accumulate causing thickening of the develops more protection against UV rays (D'Orazio et al., 2013).
- Mutations from UV rays generate reactive oxygen species (ROS).
- ROS "can cause mispairings of nucleotide bases resulting in mutagenesis that can be carcinogenic" (D'Orazio et al., 2013, p.12230).
- Nucleotide base pairings in DNA are directly affected by UV rays (D'Orazio et al., 2013).
- "UV-induced photolesions (cyclobutane pyrimidine dimers or (6,4)-photoproducts) impair transcription, block DNA replication and base pair abnormally" (D'Orazio et al., 2013, p.12231).
- Photolesions also cause "characteristic transition mutations." These mutations are known as "UV signature mutations" (D'Orazio et al., 2013, p.12231).

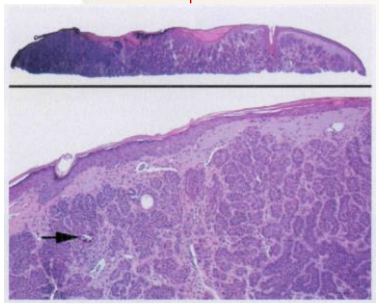

Figure 1 - H&E-stained sections of a micronodular type BCC show overlying ulceration and serum crusting (top). The tumor in this field is not attached to the surface, showing a micronodular pattern of growth. Calcifications (arrow) can be seen (bottom). (Thompson, 2010)

Melanocortin 1 receptor

"The melanocortin 1 receptor (MC1R) is a critical genetic locus involved in pigmentation, the adaptive tanning response and skin cancer susceptibility. MC1R is found on the surface of melanocytes where it binds to alpha-melanocyte stimulating hormone (MSH) and transmits differentiation signals into the cells through activation of adenylyl cyclase and generation of cAMP" (D'Orazio et al., 2013, p.12234. When cAMP is signaled, the protein kinase (PKA) cascade is activated. The activation of this cascade leads to increased levels and/or activity of various melanogenic enzymes. This in turn leads to enhanced production and export of melanin by the melanocytes (D'Orazio et al., 2013)

In fair-skinned, sun-sensitive and skin cancer prone populations, loss-of-signaling MC1R polymorphisms are commonly found (D'Orazio et al., 2013). "Loss of signaling MC1R alleles are associated with up to a four-fold increased lifetime risk of melanoma and other skin cancers" (D'Orazio et al., 2013, p.12234-35).

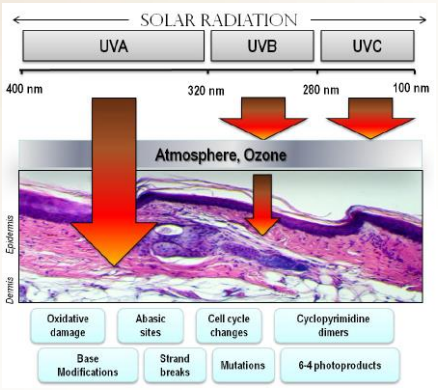

Figure 2 – Diagram of UV rays hitting the skin (D'Orazio et al., 2013)

Genetics & the Hedgehog (Hh) pathway

Since there are different appearances of BCCs, it is thought that the cell of origin could be a stem or progenitor cell (Kasper et al., 2012). "Stem and progenitor cells are thought to be the most probable sources of tumor initiation due to their longevity and ability to self-renew" (Kasper et al., 2012, p.456) In basal cell carcinomas, the hedgehog (Hh) pathways seems to be impaired. The Hh pathway is responsible for transmitting information and plays a role in embryonic development, but also plays a role in adults as well (medical-dictionary.thefreedictionary.com). With the impairment of the Hh pathway, this could explain why the immune system does not take care of the cancerous cells when they first develop.

Significance

Basal cell carcinoma is a type of cancer that is preventable. While there may be a genetic predisposition for some, many cases are caused by prolonged exposure to UV rays. By getting a tan, damage is being done to the skin. With each exposure to harmful rays, people are setting themselves up for issues down the road. As stated before, skin has a good memory, and bad habits cannot be erased after a certain amount of time. While physiologic changes happen internally, they are put into motion because of what has been done externally.

Implications for Nursing

For healthcare professionals, it is imperative to educate patients on the dangers of unprotected sun exposure. "Roughly 1 in 5 Americans will develop skin cancer in their lifetime" (D'Orazio et al., 2013, p.12233). Sun exposure is not the only thing that individuals should be educated on, healthcare professionals should also educate patients on the danger of using tanning beds.

Research shows that the younger a person is when they first start using a tanning bed, relative risk for developing a skin cancer steadily increases (Firnhaber, 2012). An important population to educate are adolescents. While many parents try to use proper sun protective measures with younger children, it can become more difficult to do once adolescence is reached. It is hard for a teenager to think about how what is done now can affect later in life. It is important to try to reach this population of the community in order to prevent these issues when they become older adults. "Preventive measures, such as using sunscreen and not using artificial tanning devices, are recommended to avoid developing skin cancer. Skin-protection measures are especially important for children and adolescents because sun exposure during childhood and adolescence directly influences the development of skin cancer later in life" (Basch et al., 2014). Education about sun protection and skin cancer is not solely for those involved in the field of Dermatology. Discussions regarding this topic can be done by any healthcare professional. Regardless of specialty.

Conclusion

There is an epidemic of skin cancer in our society. The obsession with getting a tan is setting society up for future health issues that could have been easily prevented. It is important to begin good sun protection practices at a young age, but it is never too late for individuals to start treating their skin better. With proper education, hopefully the number of those diagnosed with a skin cancer will rapidly decrease.

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