Cutaneous Membrane

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The cutaneous membrane is the skin described in more technical terms. The skin's primary role is protecting the rest of the organs and tissues in the body from physical damage like chemical damage, biological damage, and abrasions. It is usually the first defense line from external factors. The skin, like most other body tissues, can be affected by cancer. There are several types of cancer that can affect the cutaneous membrane, including Kaposi sarcoma, Merkel cell carcinoma, cutaneous lymphoma, several sarcoma types, and skin adnexal tumors. This document discusses the cutaneous membrane as an essential part of the body; cancer affects it, and the various therapies that attempt to manage cancer.

Structure and functions

The cutaneous membrane is divided into several layers. The first one is the epidermis which is mainly composed of Keratinocytes. Keratinocytes produce keratin, a protein, which is the building block of the epidermis (Linder, 2020). The epidermis does not contain any blood vessels; therefore, it depends on waste disposal and nutrient delivery on the dermis. Its primary function is being a biological and physical barrier to the external environment. It prevents allergens and irritants from penetration. Additionally, it prevents water loss while maintaining internal homeostasis(Linder, 2020). The cutaneous membrane also contains the dermo-epidermal junction- an underlying structure between the dermis and the epidermis. It gives cohesion between the dermis and the epidermis. The dermo-epidermal junction has two layers: lamina densa and lamina Lucida. Lamina Lucida is connected to the dermis and undulates between the epidermis and the dermis through dermal papillas. The dermal papillas have capillary loops for supplying oxygen and nutrients to the epidermis. The dermo-epidermal junction significantly increases the exchange surface area for waste, nutrients, and oxygen.

The dermis forms the skin's inner layer and is thicker compared to the epidermis. Its primary role is to support and sustain the epidermis. It is built for cushioning internal structures from mechanical harm, protection, nourishing the epidermis, and has a significant role in the wound-healing process. The dermis contains collagen, an interlacing connective tissue network, and some elastin. The dermis also contains several scattered structures (nerves, sweat glands, blood vessels, and lymphatics) and fibroblasts, and mast cells(Linder, 2020). The dermis is made up of two layers: superficial papillary and reticular dermis. The papillary dermis consists of connective tissues and is the thinner layer. The papillary dermis contains some collagen, elastic fibers, and capillaries. The reticular dermis has a dense and thick layer of connective tissue that has large blood vessels that are interlaced closely with elastic fibres and collagen in thicker bundles. The reticular dermis also contains nerve endings, epidermal appendages, mast cells, and fibroblasts(Linder, 2020). These structures are encompassed by a viscous gel that allows the passage of hormones, waste products, and nutrients across the dermis. They also provide lubrication for elastics fibres and collagen networks. The surrounding viscous gel also provides bulk necessary for the dermis as a shock absorber. The hypodermis is another part of the skin and is below the dermis. Its primary function is shock absorption and insulating the body. The skin belongs to the epithelial type of tissues that provide covering.

Squamous cell carcinoma

Histology and gross pathology

SCC has squamous epithelial cells nests generating from the epidermis and reaching the dermis. The malignant cells are abundant with cytoplasm, eosinophilic and nucleus, vesicular, and variable keratinization(Abnet, Arnold, Wei, 2018). SCC grading is determined by the easy recognition of squamous epithelium- for instance, keratinization and intracellular bridges, mitotic

and pleomorphism activity characteristics (Abnet, Arnold, Wei, 2018). SCC infiltrates blood vessels' adventitia, lymphatics, and nerve sheaths. They can cause inflammation in this area, suggesting the presence of a tumor. Tumor cells can potentially a response like stromal desmoplastic.

Subcellular features disrupting the cell cycle.

Expression of E6 and E7 oncoproteins triggers cell immortalization through inhibitory impact on p53 and pRb protein tumor suppressors (Migden et al., 2018). Undermining the growth of pRb inhibitory role with E2F transcription factors release makes the cell mitogenic stimuli independent. The transcription factors' growth abundance offers infinite proliferative potential by graning products expression such as B, E, and A cyclins, DNA polymerase which stimulates celllcycles' various stages, and dihydrofolate reductase. There is disruption of G2-M and G1-S cells subsequently in the cell cycle checkpoint(Migden et al., 2018). Cyclin E overexpression causes chromosomal instability and potential genetic mutations unmasking which further allows the disease to progress. Cyclin A affords growth that is anchorage-independent, which facilitates tumor spread and tissue invasion(Migden et al., 2018). Growth inhibitory and apoptotic mechanisms are evaded with anchorage-independent growth. E6 degrades p53 and mdm2-its downstream protein. p21, like p53, the other downstream protein is ineffective against E7 cyclin-cyclin-dependant kinase units.

How therapies attempt to stop squamous cell carcinoma

Radiation therapy is an effective treatment for squamous cell carcinoma. The therapy is targeted precisely at the site of squamous cell carcinoma, and there is the direction of high-energy X-rays to the site. Exposure and lesion on surrounding areas is minimized in radiation therapy(Abnet, Arnold, Wei, 2018). Cancer cells divide and grow faster than a significant

number of normal cells. Radiation develops small breaks in the cancer cell's DNA. The breaks, in turn, prevent cancer cells from dividing or growing, which causes them to die**.** Surrounding cells can be affected, but they recover and continue with normal operations(Abnet, Arnold, Wei, 2018). Other therapies also try to inhibit the growth and division of cancer cells, like photodynamic therapy. Photodynamic therapy involves directing an intense light beam at a tumor which kills cancer cells.

The difference in body reaction from cancer injury and tissue injury

When there is a tissue injury, the cells damaged trigger local vasodilation and blood vessels widening by releasing inflammatory chemical signals. The blood flow increase causes apparent heat and redness. As a response to the injury, tissue degranulates mast cells release histamine, a potent vasodilator (Chandorkar and Basu, 2018). The primary response to tissue injury is inflammation. For cancer, the body reacts by sending T-cells to the tumor. The T-cells infiltrate and attack the tumor. T-cells are activated locally and can identify cancer cells. The activated T-cells attack cancer, which causes a release of more antigens that are tumor-specific(Chandorkar and Basu, 2018). Cancer cells do not die after being attacked; instead, they continue to grow and form new damaged cells invading other tissues.

In conclusion, the cutaneous membrane serves an essential role in the body. It mainly insulates and ensures that the internal organs of the body are protected from external harm. It is susceptible to various forms of cancer, but this document has focused on squamous cell carcinoma. The various forms of therapy treating this cancer, like radiotherapy and photodynamic therapy, attempt to kill the cancer cells by inhibiting growth and division. The main difference between the body’s reaction to a tissue injury and cancer injury is that for the

tissue injury, the body's reaction is inflammation. For a cancer injury, the body activates T-cells which work on destroying the cancer cells leading to disease progression.

References

Abnet, C. C., Arnold, M., & Wei, W. Q. (2018). Epidemiology of esophageal squamous cell carcinoma. *Gastroenterology*, *154*(2), 360-373.

Chandorkar, Y., & Basu, B. (2018). The foreign body response demystified. *ACS Biomaterials Science & Engineering*, *5*(1), 19-44.

Linder, K. E. (2020). Structure and Function of the Skin. *Feline Dermatology*, 3-21.

Migden, M. R., Rischin, D., Schmults, C. D., Guminski, A., Hauschild, A., Lewis, K. D., ... & Fury, M. G. (2018). PD-1 blockade with cemiplimab in advanced cutaneous squamous cell carcinoma. *New England Journal of Medicine*, *379*(4), 341-351.