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Review Article

Radiation Induced Skin Injury Including Cancer Versus Application of UV Radiation Therapy for Skin Disease

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Received: April 10, 2019; **Published:** August 06, 2019

Abstract

Skin Cancer is very common in Europe rather than African and Asian. Fair people are more vulnerable for skin cancer like squamous cell carcinoma and basal cell carcinoma. Repeated exposure to radiation can cause skin cancer. Avoiding sun light and use of sunblock is must to keep away of skin cancer. Skin biopsy helps to confirm the diagnosis of skin cancer. Early aging can be avoided by keeping away from sun light. Early diagnosis and early treatment, which may be surgery or topical medication could be a good option for avoidance of mortality and morbidity.

Keywords: *Cancer; Radiation; Melanoma; Malignant; Ultraviolet Ray*

Abbreviation

UV: Ultra Violet; RT: Radio Therapy; ROS: Reactive Oxygen Species; FDA: Food and Drug Administration; EMA: European Medicine Agency; OV: Oncolytic Viruses; NMSC: Non Melanoma Skin Cancer; HPV: Human Papilloma Virus

Introduction

In the recent past years there is marked increase in NMSC incidence with increasing age resulting from cumulative sun exposure, which reflects both the intensity and duration of this exposure. Excessive sun exposure is carcinogenic in humans and that it has a causal role in development of NMSC [non melanoma skin cancer] [1]. The skin is our body's largest organ, but also the most exposed part of body to the environment. With the crucial job of protecting our internal organs from damage, the elements, and other threats to our body, we need to do everything that we can protect it! Unfortunately, our skin is exposed to harmful ultraviolet rays from the sun every day, which puts us at high risk of developing skin cancer.

Discussion

Skin provides a major interface between the environment and the body and is constantly exposed to an array of chemical and physical environmental pollutants (Athar, 2002). In addition, a large number of dietary contaminants and drugs can manifest their toxicity in skin (Sander., *et al*. 2004). These environmental toxicants or their metabolites increase ROS production. ROS are short-lived which are produced during regular aerobic metabolism [2].

There is persuasive evidence that each of the three main types of skin cancer, basal cell carcinoma (BCC), squamous cell carcinoma (SCC) and melanoma, is caused by sun exposure. The incidence rate of each is higher in fairer skinned, sun-sensitive rather than darker skinned, less sun-sensitive people; risk increases with increasing ambient solar radiation; the highest densities are on the most sun exposed parts of the body and the lowest on the least exposed; and they are associated in individuals with total (mainly SCC), occupational (mainly SCC) and non-occupational or recreational sun exposure (mainly melanoma and BCC) and a history of sunburn and presence of benign sun damage in the skin. UV radiation specifically causes these skin cancers depends on indirect inferences from the action spectrum of solar radiation for skin cancer from studies in animals and the action spectrum for dipyrimidine dimers and evidence that presumed causative mutations for skin cancer arise most commonly at dipyrimidine sites. Sun protection is essential if skin cancer incidence is to be reduced [3]. Skin cancer is becoming an increasingly important public health problem. Multiple studies have now demonstrated a relationship between ultraviolet exposure and increased risk of developing skin cancer. However, the specifics of that association are somewhat different for malignant melanoma, basal cell carcinoma, and squamous cell carcinoma. A better understanding of the mechanisms that allow cutaneous ultraviolet radiation to induce neoplasia will result in the development of better future sun-protection agents and strategies [4].

Non-malignant conditions are easily diagnosed by interventional and fluoroscopic radio imaging. Over exposure of radiation procedure may cause serious hazards to children at their late stage of life. Repetition of low radiation to children is same as high dose. Children are more sensitive than adults to carcinogen and have long life expectancy [5]. The rising Incidence and morbidity of non-melanoma skin cancers has generated great interest in unravelling of their pathogenesis and in the search for new non-invasive treatments. Whereas the role of cumulative sun exposure in pathogenesis of squamous-cell carcinoma seems clear, the relation between sun-exposure patterns and subtypes of basal-cell carcinoma remains undetermined. Several complex genotypic, phenotypic, and environmental factors contribute to pathogenesis of non-melanoma skin cancers. Unlike basal-cell carcinoma, squamous-cell carcinomas can arise from precursor lesions. Diagnosis of non-melanoma skin cancer is made clinically and confirmed by histological testing. Surgical excision with predetermined margins is the mainstay of treatment for squamous-cell carcinoma and for most basal-cell carcinomas. Of the new non-invasive treatments, only photodynamic therapy and topical imiquimod have become established treatments for specific subtypes of basal-cell carcinoma, and the search for more effective and tissue-salvaging therapies continues [6]. Skin cancer is less common in persons with skin of color than in light-skinned Caucasians but is often associated with greater morbidity and mortality. Thus, it is crucial that physicians become familiar with skin cancer in persons of color so as to maximize the likelihood of early detection of these tumors. In dark-skinned ethnic groups, squamous cell carcinoma is most common; squamous cell carcinoma and melanoma usually occur on non-sun-exposed sites; and ultraviolet radiation is not an important etiologic factor for skin cancer with the exception of basal cell carcinoma [7]. Immediate effects of UV irradiation on normal skin can cause erythema, tanning and immunosuppression. At the molecular level, UV irradiation causes DNA damage such as cyclobutane pyrimidine dimers and photoproducts, which are usually repaired by nucleotide excision repair (NER). Chronic exposure to UV irradiation leads to photoaging, immunosuppression, and ultimately photocarcinogenesis [8]. The advantageous effects of repeated low-dose UVR on the skin barrier, and epidermal cell proliferation and function, were described, in addition to a range of largely beneficial effects modulated through innate and adaptive immunity. UVR is a well-known treatment for the prevalent inflammatory skin conditions psoriasis and AD (Atopic dermatitis). Benefit is also gained in vitiligo, in which UVB is effective in restoring melanocytes alongside inducing immunosuppression, and in the cutaneous manifestations of GvHD (Graft Versus Host Disease). High-dose UVR injury to the skin can trigger both psoriasis and vitiligo (the Koebner phenomenon), while even low-dose UVR exposure is detrimental in the photodermatoses. In these, exposure of the skin to sunlight causes the condition (e.g. PLE= polymorphous light Eruption) or aggravates the disorder (e.g. in SLE= Systemic Lupus Erythematosus and subsets of patients with psoriasis and AD), while UVB is effective in PLE, only the deeply penetrating wavelengths of UVA1 can regulate SLE. UVR suppresses the skin's adaptive immune system, the courses of only a few infections are recognized to be affected by UVR exposure, perhaps reflecting UVR's augmenting effects on innate immunity and AMP (Antimicrobial Peptide) production and the complex interactions between adaptive and innate elements of cutaneous immunity. It has been proposed that exposure to suberythemal doses of UVR is sufficient for local and systemic immunomodulation; UVR stimulates

normalizing pathways-characterized by the induction of innate immunity and suppression of adaptive immune responses and involving the stimulation of Tregs and Bregs-and immunomodulatory and anti-inflammatory molecules. UVR can predominantly regulate inflammatory T lymphocyte-driven diseases, such as the organ-specific autoimmune diseases MS and T1D, in a manner similar to the regulation of psoriasis and AD. A reduction in blood pressure through UVR exposure that limits cardiovascular disease and lengthens life expectancy is not brought about by the induction of vitamin D metabolites [9]. Benign and malignant disease can spread lesion to breast but not always. So many invasive procedure can be avoided by breast imaging findings. Leukemia, lymphoma and malignancy of other organs can do secondary changes in breast [10]. FDA and EMA approved oncolytic viral therapy for the treatment of melanoma since 2015. So many oncolytic viruses have developed for the inhibition of melanoma [11].

Dermatologists and interventionalists must be aware of the potential for skin injuries and recognize the characteristics of such injuries to avoid misdiagnosis, histopathology of skin is usually done. However, the results are usually normal beside radiological change. Fluoroscopy-induced injuries can be recognized by the location of the injury as being congruent to the entrance of the X-ray beam and by the temporal pattern of the injury in relation to the fluoroscopy. Additionally, the injury often shows well-margin, which occur during unchanged beam is applied for long time fluoroscopy. Histopathology is avoided due to chance of ulceration. Some patients may be at greater risk for injury because of preexisting health conditions like collagen vascular disease, high blood sugar, or ataxia telangiectasia, or past high radiation exposure history [12]. Intestinal tissues are highly sensitive to radiation and are among the most common sites of clinical radiation damage. Radiation-induced acute intestinal injury has been frequently reported in individuals exposed to nuclear accidents and radiation therapies for abdominal tumors, and there are currently no effective treatments for this damage [13]. Radiation induced necrosis and more severe injury, on the other hand, are extremely unlikely. These injuries may still occur as a result of secondary infection following moist desquamation [14]. Low platelet count due to radiation cause bleeding tendency, infection or fatal condition. In this study, we showed that radiation-induced endothelial cell injury weakened the migration and adhesion of MKs (Megakaryocytes) to HUVECs (human umbilical vein endothelial cell) which hindered platelet regeneration in the process of hematopoietic reconstitution and slow platelet recovery after radiation injury from the perspective of the hematopoietic microenvironment [15]. Radiotherapy with curative intent is given to many patients with cancer. In breast cancer, radiotherapy reduces the risks of recurrence and death, but incidental cardiac irradiation may increase the risk of heart disease. Thoracic radiotherapy can also increase heart disease risk in Hodgkin lymphoma, childhood cancer, esophageal cancer, and lung cancer. Ischemic heart disease (IHD) is the most common radiation-related heart disease, and radiation-related risk increases approximately linearly with mean whole-heart radiation dose. Radiation-related IHD (Ischemic Heart Disease) may be caused by microvascular myocardial disease or macrovascular coronary artery disease. Doses from radiotherapy to individual myocardial or coronary artery segments differ substantially depending on regimen, and regimens differ by tumor type, stage, and location and, for breast and lung cancer, laterality [16]. Carotid arteries frequently receive significant doses of radiation as collateral structures in the treatment of malignant diseases. Vascular injury following treatment may result in carotid artery stenosis (CAS) and increased risk of stroke and transient ischaemic attack (TIA) [17]. Sun exposure helps to get vitamin D. Sun exposure convert previtamin D3 to vitamin D3 with the help of body heat. Several factors are responsible for the synthesis of previtamin D3. More than half of world population is vitamin D3 deficient [18]. External and internal stress affect skin along with ROS (reactive oxygen species). UV ray exposure is responsible for aging and skin cancer. Exposure to UV radiation affect DNA, though it can last for hours to years. Oxidative stress occur by UV through defeat of antioxidant action. Less sun exposure and more antioxidant can lessen oxidative stress. Melanin is the internal skin protector along with antioxidant. Melanin is the primary skin protector which can't give full defence. Avoiding sunlight, sun blocks, protective dress and antioxidant can give protection from UV light [19]. Dermatologists using radiation therapy for the treatment of various skin conditions. Mohs surgery decreased the use of radio therapy by the dermatologists. Radiotherapy plays a role for nonmelanoma skin cancer used by the respective skin specialists [20]. UV radiation (UV) is classified as a complete carcinogen, because it is both a mutagen and a non-specific damaging agent and has properties of both a tumor initiator and a tumor promoter. UV radiation is the modifiable risk factor of environment which can cause skin cancer. White skin is more prone to skin cancer round the world. The incidence, morbidity

and mortality rates of skin cancers are increasing and, therefore, pose a significant public health concern. Ultraviolet radiation (UVR) is the major etiologic agent in the development of skin cancers. UVR causes DNA damage and genetic mutations, which subsequently lead to skin cancer. Unnecessary exposure to the sun and artificial UVR are important personal attributable risks [21]. Epithelial cancers are easy to identify and treat. They originate mostly from epidermal cells (e.g. sweat apparatus, hair follicle) [22].

Conclusion

Basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) are very common among all NMSC. SCC in situ is easy to treat before invasion. BCC in situ is rare though easy to find. Sunlight, radiation (UVR) and human papillomavirus (HPV) is responsible for skin cancer in fair skin. Solar keratoses is a type of SCC in situ, which is common in Europe due to over exposure of sunlight. UVR and HPV is the most common etiology for SCC. NMSC due to inflammation, arsenic, hydrocarbon are more harmful than UVR or HPV associated tumors. Radiation therapy is an excellent treatment choice for a variety of skin cancers in a wide number of anatomic locations, which frequently offers high rates of cure with excellent cosmesis and provides patients an alternative to aggressive and often disfiguring surgery. RT also fills an important role as an adjunctive therapy when combined with other systemic agents with or without surgery and should be included in the armamentarium of any multidisciplinary approach to skin cancer. By providing proper education, people will be more aware of the truths and consequences of excessive UV exposure and skin cancer. This will hopefully allow them to take preventive steps, to reduce their risk of skin cancer. Through public education, healthcare agencies/providers can increase the public's awareness of skin cancers, their causes and preventative measures, which might be both life changing and life-saving.

Conflict of Interest

Author has no conflict of interest.

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Volume 2 Issue 6 September 2019