

RADIATION INJURIES RELATED TO NUCLEAR WEAPONS

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ABSTRACT

This final article in the series on the medical management of ionizing radiation injuries and illnesses focuses on the effects of acute ionizing radiation exposure to one of the largest organ systems of the body—the skin. These injuries may extend beyond the skin into deeper tissues and cause local radiation injury. There are numerous causes of these injuries, ranging from industrial incidents to medical procedures. In the present article, the authors characterize the clinical course, pathophysiologic process, sources of injury, diagnosis, and management of local radiation injury and describe a clinical scenario. This information is important for primary care physicians, to whom patients are likely to initially present with such injuries. Another treatment modality that may be helpful is hyperbaric oxygen therapy. This modality has been effective for delayed radiotherapy injuries, particularly, Hyperbaric oxygen therapy may result in improved quality of life, as exhibited in gynecologic oncology patients with delayed manifestation of radiotherapy-induced injuries, such as tissue necrosis and osteoradionecrosis.⁶⁶ The benefits of hyperbaric oxygen may include vasculoneogenesis, increased oxygenation of the tissues, and, possibly, increased production of various growth factors.

KEYWORDS: Radiation, Clinical Course, Pathophysiology, Terminology And Injuries .

INTRODUCTION

The skin is usually defined as the epidermis, dermis, and appendages (sebaceous, sweat, apocrine, mammary glands, and hair follicles). Injury to the skin due to ionizing radiation

(IR) can result in local radiation injury (LRI), which is not limited to the skin. Local radiation injury can be sustained from fluoroscopy, nuclear medicine, and computed tomography in disciplines such as radiology, nuclear medicine, interventional radiology, and interventional cardiology. As noted by Shope, the US Food and Drug Administration (FDA) first brought attention to fluoroscopy-induced cutaneous radiation injuries with the Safe Medical Devices Act of 1990. Since that time, much attention has been paid to LRI sustained from these injuries. However, it is primary care physicians who order these tests and follow up with patients after testing and, therefore, primary care physicians should be a patient's primary source for information on the risks of medical imaging. All clinicians should be aware of the signs and symptoms of cutaneous and deeper-tissue injuries caused by IR. Many other sources of LRI exist, resulting in pain, disability, and death. Industrial or commercial sources, medical sources, nuclear accidents, and, potentially, terrorist events can lead to cutaneous and deeper-tissue exposure to high doses of IR. ([Goldsmith et al. 2012](#))

To increase physician awareness of LRI, we review the terminology, clinical presentation, sources of injury, and evaluation and management of LRI, and conclude with a clinical scenario to illustrate the major concepts. Although these are not common injuries, they are difficult to diagnose without a known history of IR exposure, and they are difficult to manage. Similar to chemical and thermal burns, the extent of dermatologic injury has been shown to be a strong

AIM & OBJECTIVE

The objectives of radiation protection are to minimize the health effects due to radiation. Before formulating the appropriate countermeasures, we have to understand the [biological effects of ionizing radiation](#). Biological effects of radiation are mainly classified into "[Deterministic Effect](#)" and "[Stochastic Effect](#)". For the former, a [threshold level](#) of [absorbed dose](#) exists, above which the radiation will bring detrimental effect to the health. For the stochastic effect, a threshold does not exist, but the probability of having detrimental effect is proportional to the dose absorbed.

HYPOTHESIS:

The clinical course, pathophysiologic process, sources of injury, diagnosis, and management of local radiation injury and describe a scenario. This information is important for primary care physicians, to whom patients are likely to initially present with such injuries.

RESEARCH METHODOLOGY

Research paper is an explanatory research paper that's primarily based on secondary facts amassed from numerous sources. Distinct books, articles and web sites had been considered for the examination.

TERMINOLOGY

The terms used to describe IR injury to the skin and deeper tissues vary. Some authorities classify damage to the skin as a subsyndrome of acute radiation syndrome (ARS) and generally use the term *cutaneous radiation syndrome*(CRS) to refer to this injury. Cutaneous radiation syndrome is also used to describe the classic, clinical picture of IR injuries to skin without another organ system component of ARS *Local radiation injury* (LRI) is sometimes used to refer to injury to tissues or organs deeper than the skin, and this term will be used henceforth in this article to describe IR injuries to the skin and deeper tissues. *Beta burns* refer to injury to the skin by beta particles and may cause a partial or even a full-thickness burn, depending on the beta energy of the radionuclide. *Radiodermatitis* is often used to refer to radiotherapy-induced skin changes.[\(Gausche-Hill et al. 2007\)](#)

Clinical Course

Acute LRI often occurs when an individual handles or comes into close contact with a high-dose rate, sealed radiation source. Many of these accidents are reported to the Radiation Emergency Assistance Center/Training Site (REAC/TS) and managed in consultation with patients' primary care physicians. The 3 isotopes that cause the most concern for these injuries are 192 iridium, 60 cobalt, and 137 cesium. Local radiation injury is a deterministic effect, or an effect that varies with dose and for which a threshold is believed to exist. The *Table* presents the clinical dose thresholds for LRI as used at REAC/TS. It is important to note that this information serves as a guideline and that there is some variation among sources for dose threshold and timing of appearance.[\(Carlos 2009\)](#)

Pathophysiology

A prodrome of erythema may occur transiently within a few hours of exposure and reappear weeks later as a manifestation of the injury. The mechanism causing erythema includes arteriolar constriction with capillary dilation and increased vascular permeability. Early

erythema is highly variable, however, and may not occur at all, although the incidence increases with dose. With dry desquamation, there is diminished mitotic activity in the cells of the basal and parabasal layers, with thinning of the epidermis and desquamation of large macroscopic flakes of skin. Moist desquamation exhibits intracellular edema, coalescence of vesicles to form bullae, and a moist dermal surface. With doses greater than 25 Gy, overt radionecrosis may occur. ([Greenberg et al. 2012](#))

The classic presentation of LRI during the weeks to months after injury follows. Within the first week, the patient may present with a prodrome of transient erythema (which, as above, is highly variable), pruritis, and paresthesias of the skin. In subsequent weeks, true erythema develops along with progressive epilation, suppression of sweating, and diminished sebaceous gland secretion. As the injury evolves, the patient exhibits edema, pruritus, and blister formation, and he or she may have severe pain. There may be a spectrum of changes in pigmentation, ranging from redness to bronzing and blackening if necrosis develops. A higher dose leads to an earlier and more severe presentation. Delayed effects of LRI, which may occur from months to years after injury, include telangiectasia formation, atrophy, and fibrosis. Telangiectasias occur as a result of damage to the microvasculature and subsequent distortion of capillary loops. Fibrosis, one of the most consistent delayed effects, may occur in tissues and vessels. Fibroblasts are the main producers of extracellular matrix, which is necessary for normal wound healing and scar formation. Local radiation injury causes the fibroblasts to become atypical and enlarged, often called *radiation fibroblasts*. These atypical and dysfunctional fibroblasts may be responsible for the delayed fibrosis. This delayed and progressive fibrosis is 1 factor that makes LRI so different from chemical or thermal burns. ([Gómez et al. 2018](#))

The pathophysiology of LRI is still not fully understood and seems to be multifactorial. There is agreement that part of the reason LRI continues evolving is secondary to waves of various interrelated physiologic cascades. Inflammation is a major component. Many mediators are involved with and feed back to prolong these processes. Damage to the microvasculature consists of damage to endothelial cells and subsequent activation of many proinflammatory and proclotting cascades. In addition, IR induces free radical species that may lead to oxidative stress. It is important to recognize that patients may have a high dose of radiation to the skin with little to no dose to the whole body or to the bone marrow. As noted above, with deeper tissue injury, other organ systems may become involved in the area of injury and

present different subsyndromes of ARS. There may be enough damage to deeper tissues over enough area or even a whole-body distribution to cause ARS. Therefore, a suspicion of ARS is warranted in any case of radiation exposure, even if only LRI is initially evident. Ionizing radiation injury to more than 50% of the body surface area is a poor prognosticator for survival. Multiorgan failure and death may result. ([Gvozdjaková 2008](#))

Sources of Injury

Many LRI incidents occur in industrial settings. In the United States, most of these cases are known to be IR-related early in their course. However, given that the injury may not manifest until weeks later, patients may delay seeking care. Internationally, a number of "orphaned" sources have been handled by persons who did not know the devices were radioactive. These injuries are often misdiagnosed.

The nuclear power plant accident at Chernobyl in 1986 is a well-known incident in which many of the victims had severe CRS caused by a mixture of beta particles and gamma-emitting radionuclides. Sixteen of the 28 acute deaths after the incident were attributed to CRS. Another source of LRI that has attracted much attention over the past 20 years is radiologic imaging techniques that deliver a large dose of IR. The average radiation dose received by patients in the United States has roughly doubled over the past 20 years, and the increase is primarily attributed to medical exposure (radiography, fluoroscopy, computed tomography, nuclear medicine, and external beam radiotherapy). Physician and patient education along with safety features on newer equipment have helped reduce the dose of radiation exposure. In the past, a lack of education resulted in patients not knowing that they were being exposed to radiation during their procedures or understanding the risk associated with exposure. A latent LRI presentation may not have been attributed to the procedure by patient or physician. Educational efforts among physicians are improving and are including many different disciplines. Discussing with patients the nature of a radiologic procedure, the radiation dose involved, and the risks and benefits of the procedure is necessary, especially if the procedure is potentially life-saving. Radiotherapy-induced LRI should be suspected if the wound has a grid-like pattern (*Figure*), if there are 2 locations of injury that correspond to the angles used in the procedure, or if the wound resembles a burn without a history of thermal or chemical burn. ([B. 2014](#))

Management

Most treatment regimens for patients with LRI have been derived from radiation oncology, traditional burn care, and past experiences with acute LRI. Acute LRI may differ dramatically from radiotherapy-induced injuries, because radiotherapy-induced skin injuries are the result of fractionated doses, not acute doses. Fractionation of a radiation dose allows for some tissue healing and repair to occur between treatments. Many of the incidents of accidental LRI are delivered much more rapidly or at high doses with little or no fractionation. ([B. 2014; Pawlicki et al. 2010](#))

Inflammation plays a large role in LRI, and methods to reduce inflammation are beneficial. There is consensus for topical steroidal treatment; class II and III topical steroids have been used historically. Some radiation oncologists have had success with intralesional steroids (A.L. Wiley, personal communication, September 2013). The World Health Organization consultancy was strongly against use of systemic steroids in the absence of a specific indication. Nonsteroidal anti-inflammatory drugs may also be indicated, but their use for LRI has not been addressed by the World Health Organization consultancy. Further, they should only be used if no contraindication is present (eg, gastrointestinal ulcer or bleeding, thrombocytopenia, coagulopathy, or aspirin allergy).

Recommendations have been fairly consistent on the use of topical antibiotics for LRI. The use of systemic antibiotics should be based on the clinical picture. Physicians should consider consulting with infectious disease specialists if there is suspicion of a high dose to deep tissues, a large percentage of affected body surface area, or another organ system is involved. The use of silver sulfadiazine and dressings may be helpful, as indicated. "Skin substitutes" and other dressing constructs should be used as indicated for thermal burns. ([Bronner 2005](#))

Combination treatment with 400 mg of pentoxifylline (not FDA-approved for this use) 3 times per day and α -tocopherol (a form of vitamin E) has shown success in decreasing radio fibrosis. Pentoxifylline alone may also help to decrease pulmonary damage due to lung and breast radiotherapy. Other antioxidants or antioxidant enzymes such as superoxide dismutase have been used to manage these injuries and are still areas with active research for further

development. Topical aloe vera seems to shorten healing time, has anti-inflammatory and antihistaminic properties, and is an excellent moisturizer. Aloe vera is often recommended to patients undergoing radiotherapy; however, the literature about its efficacy is mixed.

Traditional surgical management of LRI may be indicated, but surgeons must be aware that the margin of injury and nonviable tissue will not be grossly visible or evident. Imaging modalities or radiation dose mapping should be used to delineate the margin of the damage to the microvasculature or margin for necrosis before surgical intervention. If the microvasculature and infrastructure are adequate, and the dose is below the threshold for necrosis, successful skin-grafting may be achieved. Consultation with experts in radiation-induced injuries should be done before definitive surgical therapy.

A newer treatment approach that shows promise is mesenchymal stem cell therapy or adipose-derived stem cells. Japanese investigators, using adipose-derived stem cells injected into the wound and surgical debridement, showed good wound healing in a gynecologic oncology patient with late tissue and bone necrosis. French investigators¹⁰ successfully used bone marrow mesenchymal stem cell wound injections, with and without skin grafting, in a small series of patients. They used dose mapping techniques to determine the margins for excision of all of the necrotic or potentially necrotic tissue and then injected the area with the mesenchymal stem cells.¹⁰ Both aforementioned investigation teams are engaged in ongoing clinical trials of these methods, with continued success. Appropriate controlled studies need to be performed with long-term follow-up before these techniques can be recommended unequivocally. However, such results may be difficult to achieve with the relatively low incidence rate of LRI. ([Issues in Pharmacology, Pharmacy, Dru...](#))

Clinical Scenario

A 62-year-old man had chest pain while traveling alone overseas. His medical history included diabetes, coronary artery disease, and 2 previous percutaneous coronary interventions, with 1 stent placed each time. In addition, he had a 40 pack-per-year history of tobacco abuse and was obese (height, 5'9"; weight, 240 lb). He was rushed into the interventional cardiology suite of a large metropolitan hospital and, after several hours, a successful percutaneous coronary intervention was accomplished. The patient returned home

from his travels without further incident. Twelve days later, he experienced itching in his back, but it stopped. Twenty-five days after his return home, he began to have more itching, burning, and pain in his back. His primary care physician noted some erythema and desquamation on his left, lower scapular area and his right subscapular area (laterally) but was more concerned with establishing follow-up with his cardiologist. A month after the follow-up, his physician noted some blister formation in the left, lower scapular area and the right, subscapular area (laterally) (*Figure*). His primary care physician requested a consultation with a dermatologist, who tried conservative topical treatment without success. The dermatologist performed a punch biopsy, and the specimen showed morphea consistent with sclerosis or radiation injury. At that point, the primary care physician consulted REAC/TS. The patient was evaluated and started on a treatment protocol similar to the management recommendations outlined in the Management section in the current article. He received more than the standard recommended hyperbaric oxygen therapy (100 treatments). His wound care continued for 4 years, and then he underwent wide local excision of the nonhealing area of the lesion, with aggressive postoperative wound care (months of wound vacuum dressings, dressing changes, etc). He also had successful excision and skin grafting of the lesion on the left. One year after his surgical procedures and aggressive wound care, his wounds healed completely. ([Walker 2011](#))

Discussion

Local radiation injury has a classic presentation with a somewhat predictable course. Presenting signs include transient erythema, itching, and edema, resulting in a nonhealing wound, possibly with a grid-like pattern, that may progress to necrosis. The additional presence of nausea, emesis, and diarrhea should put the physician on alert that such a patient may have ARS. Furthermore, immunologic compromise may result in a complex and potentially life-threatening situation that requires intensive therapy. Many cases of occupational LRI involve the hands. While not necessarily life-threatening, LRI can cause significant disability and psychosocial problems. On-the-job LRI is further complicated by the unapproved, "experimental" therapies that may be required in these cases but are not covered by worker's compensation insurance carriers. Mesenchymal stem cell wound injections are not approved by the FDA.

Conclusion

Although uncommon, LRI is difficult to diagnose without a known history of radiation exposure. These injuries often have a delayed presentation that may make the history and dose estimation difficult to impossible. They may initially present as minor but evolve into a critical stage and are often associated with a high degree of disability and morbidity. An evolving wound resembling a burn in the absence of a history of thermal or chemical exposure should alert physicians to the possibility of LRI. As with most complex medical cases, specialty consultation should be obtained when dealing with IR-induced injuries of all types. Specialties that may be helpful include radiation oncology, nuclear medicine/radiology, hematologic oncology, surgical oncology, dermatology, burn surgery, and infectious diseases.

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