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Case Presentation

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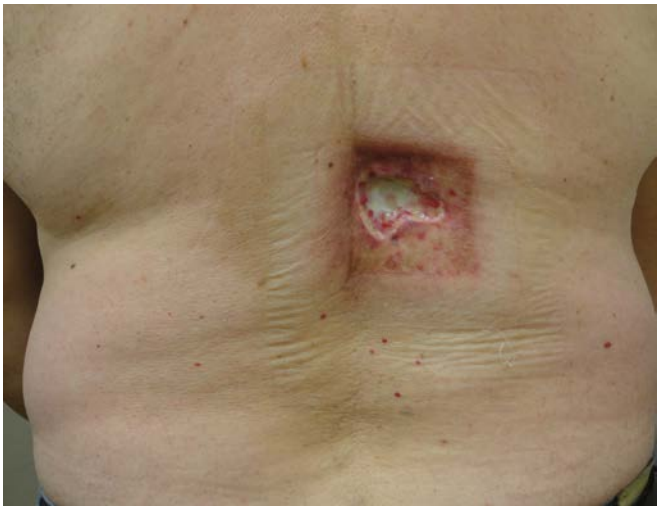
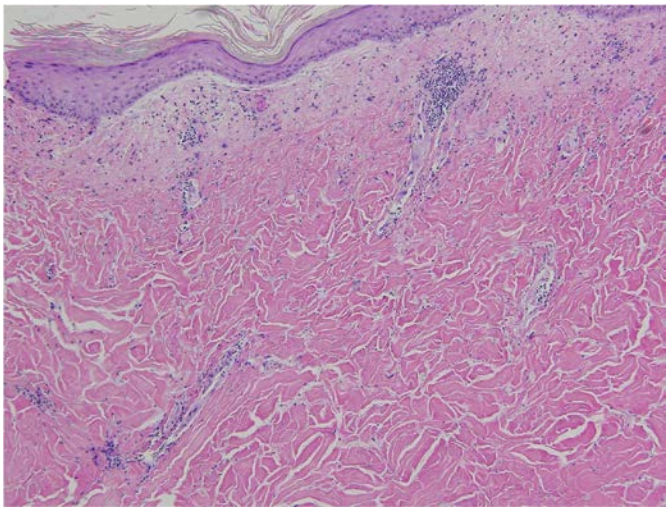
Abstract

We present a 67-year-old man with an ulcerated, indurated plaque on the right mid back with a presumed diagnosis of morphea that was complicated by an allergic contact dermatitis. Further clinical and histopathologic data elucidated the diagnosis of fluoroscopy-induced radiation dermatitis. We present a brief review of the common locations, clinical characteristics, pathophysiology, and management options for fluoroscopy-induced radiation dermatitis.





Case synopsis

A 67-year-old man was referred to the Skin and Cancer Unit for evaluation of a painful ulcer within a well-demarcated, firm, square-shaped area of skin on his right mid-back that had been present for seven months. The ulcer was preceded by a change in texture and color of his skin. The patient underwent a skin biopsy in the center of the involved area at an outside facility. The biopsy site evolved into a wider and deeper ulcer. The presumed diagnosis was morphea. The patient was applying bacitracin ointment to the area and it was thought that an allergic contact dermatitis to bacitracin was complicating healing. Minimal improvement was noted with the use of topical and intralesional glucocorticoids. Past medical history included hypertension, hyperlipidemia, diabetes mellitus, and coronary artery disease. Although the patient denied any prior radiation exposure to the area, additional review of his history disclosed that he had undergone a cardiac catheterization and coronary artery bypass graft surgery in 1994 and two additional cardiac catheterization procedures with placement of multiple stents, five and three months prior to onset of skin changes, respectively. The procedure performed five months prior was considered a complex intervention.

Physical Examination: On the right mid-back, there was a 7-by-8-cm, well-demarcated, erythematous, indurated, atrophic plaque with telangiectases within which there was a 3.8-by-4.5-cm ulcer.

Laboratory Data: A wound culture grew *Pseudomonas aeruginosa*.

Histopathology: There is a perivascular and patchy, band-like infiltrate that is comprised predominantly of lymphocytes. In the dermis; there is evidence of early sclerosis and scattered enlarged fibroblasts, several of which are stellate. There are telangiectatic blood vessels within the superficial dermis, some of which are lined by enlarged endothelial cells.

Diagnosis: Fluoroscopy-induced radiation dermatitis

Discussion: Interventional fluoroscopy is becoming an increasingly important tool in the diagnosis and management of disease. Increasing complexity of such procedures, greater fluoroscopic times, and prolonged radiation exposure increase the risk for iatrogenic skin damage. In 1994, the Food and Drug Administration alerted physicians of the possibility of severe radiation-induced skin injury in patients undergoing procedures with extended fluoroscopic exposure times [1].

Fluoroscopy is an X-ray based imaging modality that uses a fluorescent screen instead of radiographic film to view images in real time. The flow of radio-opaque contrast agents injected into the body area is visualized [2]. Fluoroscopic X-rays are rapidly attenuated in tissue. The skin at the beam entrance point receives maximal radiation. The rapidly dividing nature of the basal cells of the epidermis makes them susceptible to radiation damage [3]. Ionizing radiation causes damage to basal layer keratinocytes, which initiates an inflammatory cascade and interrupts the cell proliferation cycle [4].

Over 70 cases of fluoroscopy-induced radiation skin injury have been reported. As in our patient, most have undergone cardiac procedures, which reflects the relatively higher number of fluoroscopic cardiologic interventions that are performed when compared with other procedures [5]. However, fluoroscopy-induced radiation dermatitis continues to remain under-recognized and under-reported. A high index of clinical suspicion is necessary to establish the correct diagnosis and early diagnosis may prevent the associated morbidity.

Diagnosis relies on clinical findings and a relevant history of radiation exposure to the affected area. The distribution of lesions correlates with the type of procedure that was performed. Radiation dermatitis that results from coronary procedures most commonly is found on the mid portion of the back, scapula, right anterolateral aspect of the chest, and below the right axilla [6]. The diagnosis is challenging because of the variable latency period, which ranges from days to months; the subtle progression of the condition; and its morphologic similarity to other disorders of the skin. Fixed drug eruption [7-9] and morphea [9-11] are the most frequent clinical mimickers of radiation-induced dermatitis. Patients, such as ours, often are unaware of prior radiation exposure. Although the differential diagnosis of fluoroscopy-induced radiation dermatitis is rather extensive, owing to the clinical heterogeneity, the timing of onset of the eruption in relation to the fluoroscopic procedure may be a helpful guide.

Histopathologic features of acute radiation dermatitis include intracellular edema, necrotic keratinocytes, fibrin thrombi in small vessels, and hemorrhage. Chronic radiation dermatitis is characterized by epidermal atrophy, telangiectatic vessels, and dermal sclerosis exhibiting atypical stellate fibroblasts and atypical endothelial cells. Adnexal structures are destroyed. Subacute radiation injury is not well characterized clinically. However, histopathologic features are distinctive and include interface dermatitis with basal vacuolar changes, dyskeratotic keratinocytes, dermal melanophages, and a superficial, perivascular, lymphocytic infiltrate [12].

Although a skin biopsy may be helpful in some cases, the histopathologic findings are not pathognomonic and vary depending on the phase of injury. Morphea shares the common histopathologic features of an atrophic epidermis and hyalinized dermal collagen with chronic radiation dermatitis. Morphea, however, typically does not present with ulceration or hyperkeratosis. Other histopathologic features of morphea are atrophy of adnexal structures, thick and narrow blood vessels, a variable lymphoplasmacytic infiltrate, and absence of atypical fibroblasts. A loss of CD34 positive dermal dendritic cells also is observed in morphea [13]. In radiation dermatitis, expression of CD34 is preserved in background normal dermal dendritic cells, but atypical fibroblasts are CD34 negative [14].

The total dose, the interval between radiation exposures, the size and location of the exposed area, and patient comorbidities may affect the expression and severity of radiation injury [15]. During coronary angiography, the patient is exposed to a radiation dose that averages 0.02 to 0.05 Gy/minute [5]. Peak skin dose is the highest radiation dose that is received by a patient's skin during any part of the procedure. Fluoroscopic time has been used as an alternative measure for the skin dose of radiation, but it does not account for factors such as radiation dose from radiographic or fluoroscopic images, differences in fluoroscopic dose rate, or movement of the radiation field on the patient's skin. The anterior aspect of the neck is the most sensitive site, followed by the flexor surfaces of the extremities, trunk, back, extensor surfaces of extremities, the nape of the neck, scalp, and palms and soles [15].

Other patient-related factors, such as smoking and poor nutritional status, compromise skin integrity. Obesity and overlapping skin folds may result in a bolus effect of radiation exposure [16,17]. Individuals with light-colored hair and skin are most susceptible [15]. Co-existing conditions are important considerations. Patients with connective tissue disease may experience late subcutaneous fibrosis after radiotherapy [18]. Patients with defects in the ability to repair damaged DNA are predisposed to increased radiation sensitivity. Diabetes mellitus and hyperthyroidism also have been associated with increased skin sensitivity to

radiation [16]. Certain medications, which include chemotherapeutic agents, simvastatin, ciprofibrate, and carbamazepine, have been reported to play a role in radiation-induced skin injury [16,19,20].

There is insufficient evidence to make any conclusive recommendations regarding prevention and treatment of radiation dermatitis [21]. For superficial radiation damage, debridement and skin grafting may be sufficient [22]. However, skin grafting often fails because of a poorly vascularized wound bed. In more extensive injury, debridement and reconstruction with a skin flap is a viable option [23].

In our patient, a surgical consultation was obtained for excision of the diseased tissue followed by musculoskeletal skin flap coverage. However, surgical management was deferred because the ulcer was re-epithelializing with local wound care measures, which include gentamicin ointment and petrolatum-impregnated gauze. Whereas other therapeutic options are available, it is important to consider patient preferences, comorbidities, and the trajectory of clinical improvement.

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