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Authors

Young, Kelly Z

Kolli, Sree S

Kwa, Michael C

et al.

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Case Report

Fluoroscopic radiation-induced skin reactions: radiation dermatitis and radiation-induced morphea

Kelly Z Young, MD, PhD¹, Sree S Kolli, MD^{2a}, Michael C Kwa, MD², Chauncey McHargue, MD²

¹ University of Michigan Medical School, Ann Arbor, Michigan, USA, ² Henry Ford Health, Department of Dermatology, Detroit, Michigan, USA

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Abstract

Given the rise of radiation based medical procedures, cutaneous radiation reactions are increasing in frequency. Diagnosis of fluoroscopic radiation-related cutaneous injuries are challenging, as patients are often unaware of or cannot recall radiation exposure. It is important to maintain clinical suspicion of radiation-induced skin injuries in patients with persistent morpheaform areas and localized areas of dermatitis or ulceration. Several cutaneous radiation-induced injuries have overlapping clinical presentations. Histopathology may be required to help differentiate between these distinct disorders. Treatment of cutaneous radiation reactions may vary, dictated by a variety of factors, including the disease process, the severity of the lesions, and the presence of comorbidities. Herein, we present two cases to highlight the spectrum of fluoroscopic radiation-induced cutaneous injuries.

Introduction

With the growing application of fluoroscopic procedures, awareness of iatrogenic cutaneous radiation related injuries has been amplified.^{1,2} The effects of radiation on the skin may occur acutely, chronically, or both with reactions occurring as quickly as within 24 hours or delayed by as much as a year,³ progressing from a rash to a non-healing ulcer with potential fibrosis.⁴ Radiation affects several cutaneous cellular populations including basal keratinocytes, stem cells, melanocytes, and fibroblasts.^{3,5} The effects of radiation likely occur via inflammation and alterations of redox states through the generation of free radicals.^{4,6}

Cutaneous radiation reactions vary based on duration or frequency (ie, fractionation) of exposure and dosage.

Low doses (2-5Gy) may cause transient erythema whereas doses of 5-15Gy may cause prolonged erythema, long-term induration, and dermal atrophy.⁷ Doses >15Gy may result in acute edema and ulceration followed by dermal atrophy, secondary ulceration, and/or dermal necrosis.⁷ Signs of high doses include dry or moist desquamation and ulceration.⁷ Disease severity may also be influenced by comorbidities such as obesity or the effects of smoking.⁵ Herein, we present two cases to highlight the spectrum of fluoroscopic radiation-induced cutaneous injuries.

Case Synopsis

Case 1

A 68-year-old man presented with an 8-month history of a progressive, nonhealing, painful ulceration of his left flank. Nine months prior, he was hospitalized for ischemic colitis necessitating a right hemicolectomy. His course was complicated by superior mesenteric artery thrombosis that failed repeated endovascular attempts at revascularization involving fluoroscopic visualization and ultimately required small bowel resection. One month after surgery, he developed an enlarging, painful ulceration that progressed over several months despite multiple debridements, antibiotic courses, and a 14-day course of prednisone. The examination revealed a large, tender ulcer with a fibrinous yellow base and a broad violaceous indurated border of the left flank (**Figure 1A**).

Biopsy disclosed extensive necrosis with mixed inflammatory cells in the dermis. Communication with the patient's surgeon revealed that the patient received 12-20Gy of radiation cumulatively, supporting a diagnosis of fluoroscopic induced radiation injury with necrosis. Referral to the plastic surgery department for debridement followed by an advancement flap closed the wound, which healed with resolution of pain (**Figure 1B**).

a Corresponding Author: Sree S Kolli MD, Department of Dermatology, 3031 West Grand Boulevard, Department of Dermatology, Suite 800, Detroit, MI 48202. Tel: 313-916-2600, Email: skolli1@hfhs.org

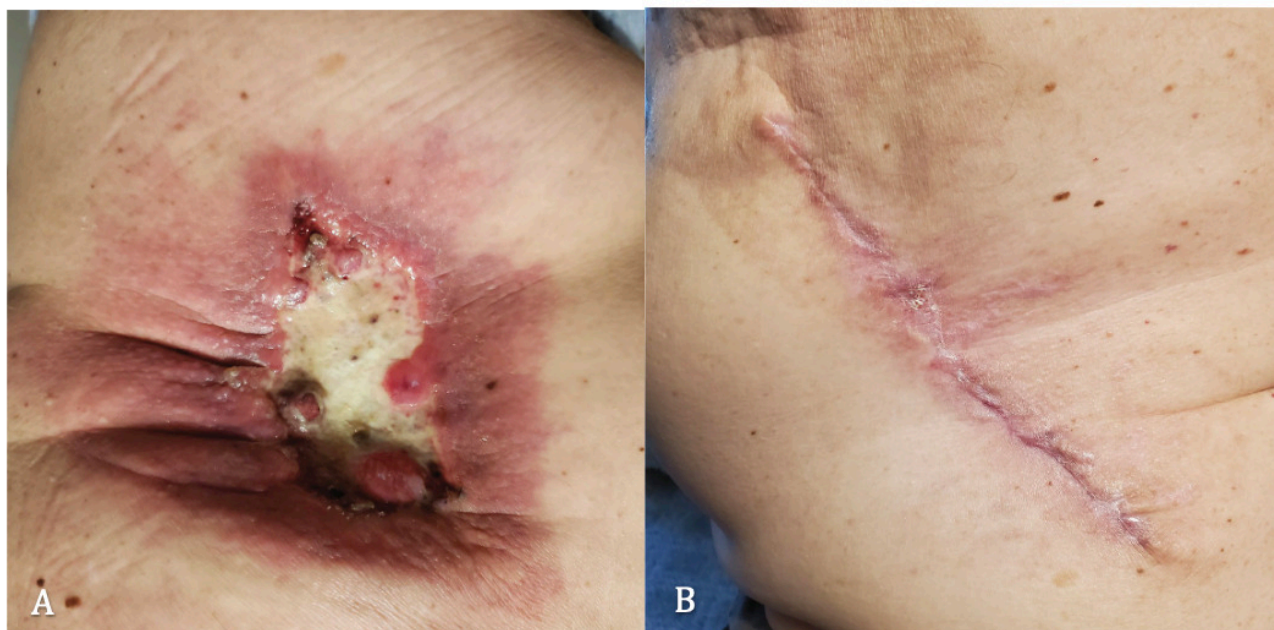


Figure 1. A) At presentation to the dermatology clinic 9 months following a fluoroscopic procedure, there was a large, tender ulcer with a fibrinous yellow base and a broad violaceous indurated border on the patient's left flank. **B)** The wound healed following debridement and complex wound closure with an advancement flap.

Case 2

A 65-year-old man presented with a history of skin changes on his left hip that had begun after an attempted endovascular repair of a right common iliac artery aneurysm. The procedure was terminated early owing to prolonged radiation exposure, with the peak skin dose estimated to be 13Gy. Three days after the procedure, he developed dark discoloration of the left hip with an itchy, tingling sensation. Examination through 6 weeks post-procedure was significant for a 15cm x 12cm sharply-demarcated rectangular patch of hyperpigmentation within which there was a 6.5cm x 9cm area of lichenification and erythema ([Figure 2A-C](#)). The differential diagnosis included radiation dermatitis, radiation-induced morphea (RIM), irritant or allergic contact dermatitis, and lichen simplex chronicus.

A biopsy disclosed dermal sclerosis with accompanying lymphocytic inflammation, hyperkeratosis, absence of radiation fibroblasts, and focal interface dermatitis ([Figure 3A-B](#)), most consistent with an early-evolving RIM. The patient was started on topical clobetasol with notable improvement at his one-month follow-up ([Figure 2D](#)).

Case Discussion

The diagnosis of fluoroscopic radiation related cutaneous injuries are challenging as patients are often unaware of intraoperative radiation use or may not recall radiation exposure by the time they develop cutaneous findings.¹ The variability in clinical presentation and mechanisms of radiation injury may further complicate

diagnosis. There is a spectrum of fluoroscopic induced injuries, as demonstrated by these two cases. Case 1 illustrates fluoroscopy-induced chronic radiation dermatitis.⁴ When fluoroscopy-induced chronic radiation dermatitis is suspected, ascertaining the intraoperative radiation dose is essential, as unlike radiation therapy, that is not predetermined.

Treatment of acute radiation dermatitis and fluoroscopy-induced chronic radiation dermatitis is challenging, dictated by a variety of factors, including the severity of the lesion and the presence of comorbidities. In general, initial management involves supportive care including maintenance of personal hygiene and prevention of trauma.⁴ As radiation generates free radicals causing inflammation, topical corticosteroids to suppress inflammation and superoxide dismutase to scavenge free radicals may be beneficial.^{6,8} For refractory chronic lesions, surgical interventions and hyperbaric oxygen therapy may be considered.⁸

Cutaneous radiation fibrosis may manifest as a late complication of chronic radiation dermatitis, caused by radiation-induced overproduction of connective tissue as a result of increased profibrotic cytokines.⁴ Management includes education, limiting further radiation exposure, rehabilitation, and physical therapy.⁹ Some studies demonstrated that antioxidants, pentoxifylline, and vitamin E, can improve tissue compliance when given for prophylaxis in patients receiving radiation.¹⁰ Given the potential role of transforming growth factor-beta causing radiation fibrosis, small molecules targeting transforming growth factor-beta have been proposed as therapeutic options.⁹ Research is ongoing in examining the efficacy of procedures, such as fat-grafting with and without deferoxamine in improving radiation fibrosis.⁹

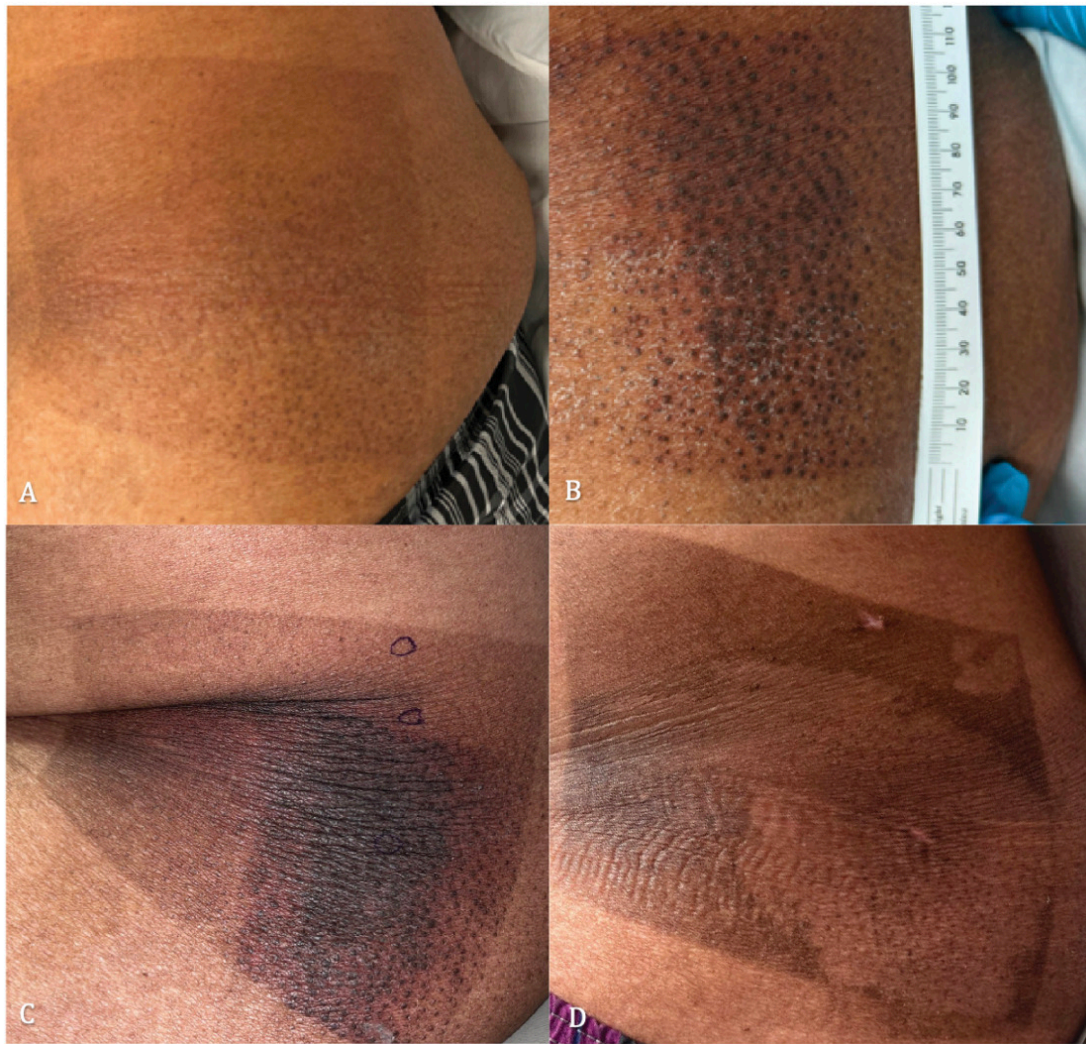


Figure 2. A 15cm x 12cm well-demarcated rectangular patch of hyperpigmentation was noted at **A)** two weeks, **B)** four weeks, and **C)** 6 weeks postoperatively. **D)** The patient started topical clobetasol with improvement in pigmentation at one-month follow-up.

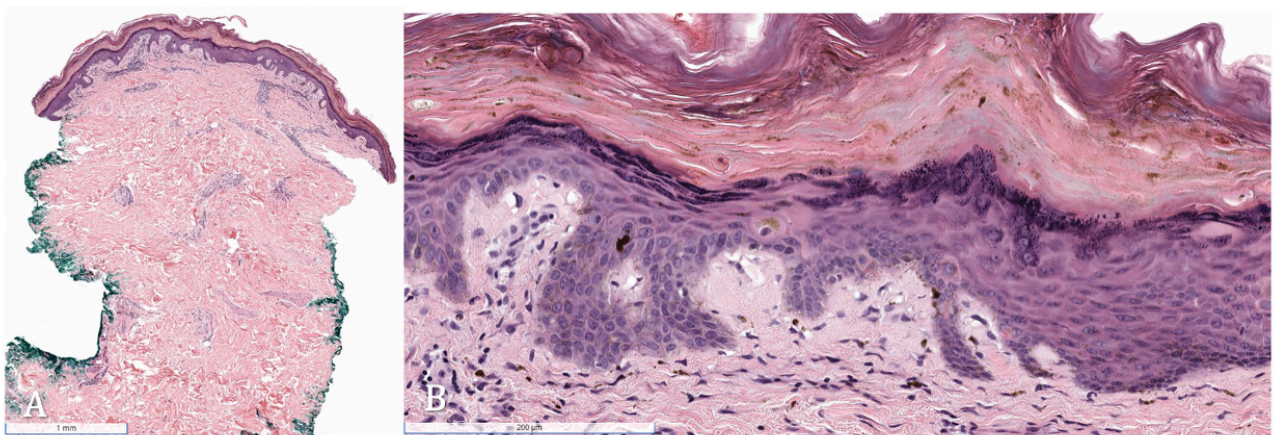


Figure 3. A) A representative punch biopsy taken 6 weeks post-radiation exposure is depicted (2x magnification), which revealed irregular hyperkeratosis, superficial perivascular and periadnexal lymphocytic inflammation, absence of radiation fibroblasts, focal interface dermatitis, and sclerotic dermis. **B)** The same biopsy at increased magnification (10x).

Radiation-induced morphea is a distinct, rare, chronic inflammatory condition characterized by progressive, painful, scleroderma related to fibrotic transformation of the skin and underlying tissue.¹¹ Although its pathogenesis remains unclear, increased secretion of T helper 2 cytokines (ie, interleukin-4 and interleukin-5) have been implicated resulting in increased transforming growth factor-beta and collagen production with subsequent activation of fibroblasts.¹¹ Onset from radiation exposure typically occurs within one year but can vary, with reports ranging from one month to 32 years after radiation therapy.¹¹

In contrast to radiation dermatitis resulting in fibrosis, there is no clear relationship between radiation dosage and RIM occurrence. Radiation-induced morphea can be differentiated from other radiation related injuries through histopathology showing features of morphea including sclerosis, thick collagen bundles, a “trapped” appearance of the eccrine glands from loss of adventitial fat, sparse inflammation, and loss of CD34.¹¹⁻¹³ In contrast, histological features of radiation dermatitis exhibit sclerosis and show papillary dermal pallor, fibrin depo-

sition in the upper dermis, dilated ectatic vessels, large stellate radiation fibroblasts, and destruction of the adnexal structures.^{1,14} Available therapies for RIM include topical corticosteroids, calcineurin inhibitors, vitamin D analogues, phototherapy, and systemic immunosuppressants.¹¹

Conclusion

Given the rise of radiation-based procedures, it is important for dermatologists to recognize radiation related skin injuries. In patients with persistent morpheaform areas and localized dermatitis, a thorough clinical history about radiation exposure, including fluoroscopy, should be obtained.

Potential conflicts of interest

The authors declare no conflicts of interest.

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