A Unique Case of Subacute Radiodermatitis

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Practice Points

- Because of sharp circumscription, subacute radiodermatitis can mimic contact dermatitis. Subacute radiodermatitis should be considered for lesions in which radiation exposure is associated with apparent contact dermatitis, fixed drug reaction, or connective-tissue disease.
- Punch biopsy or shave biopsy with histologic examination by a dermatopathologist is needed to distinguish subacute radiodermatitis from contact dermatitis, fixed drug reaction, and connective tissue disease.
- Subacute radiodermatitis can present opposite the radiation source at the location of the grounding plate during prolonged fluoroscopy procedures.
- Minimizing radiation exposure is critically important to decreasing the incidence of radiodermatitis.

Subacute radiodermatitis is a rare cutaneous disease induced by ionizing radiation. It often is mistaken for contact dermatitis, fixed drug eruption, or connective-tissue disease. Routine use of fluoroscopy has flourished in many types of medical procedures. We present a case of subacute radiodermatitis stemming from prolonged fluoroscopic exposure during angiography; the lesion appeared only at the site of contact for the ground plate, remote from the field of radiation.

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Case Report

A 58-year-old man presented with an erythematous plaque on the back 7 weeks following cardiac catheterization. The area was sharply demarcated and presented in the form of a rectangle with poikilodermatous changes. It had areas of erythema, atrophy, dyspigmentation, and telangiectasia (Figure 1). Further questioning led to the discovery that the patient was placed

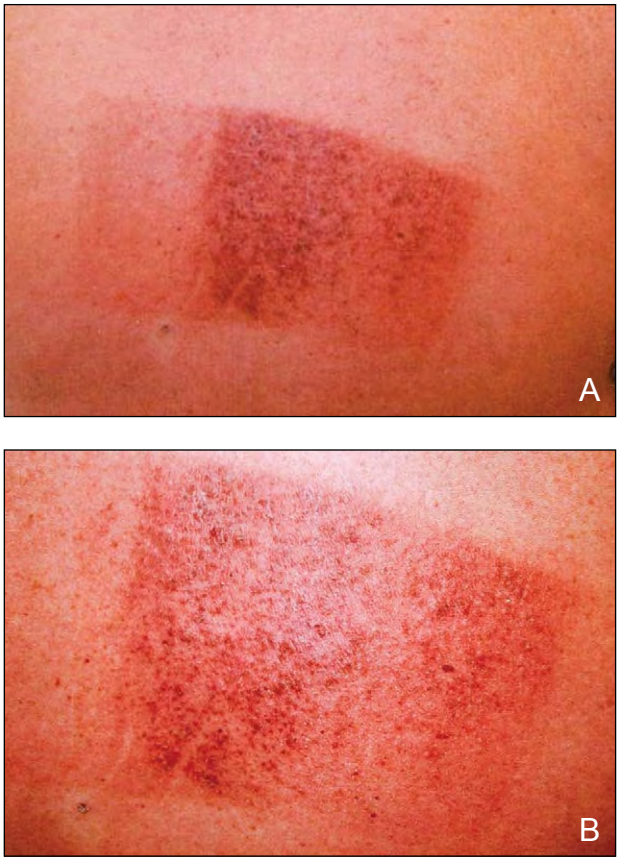

Figure 1. Poikilodermatous rectangular and sharply demarcated plaque that presented opposite to the fluoroscopy radiation site (A). Poikilodermatous changes included atrophy, telangiectasia, and dyspigmentation (B).

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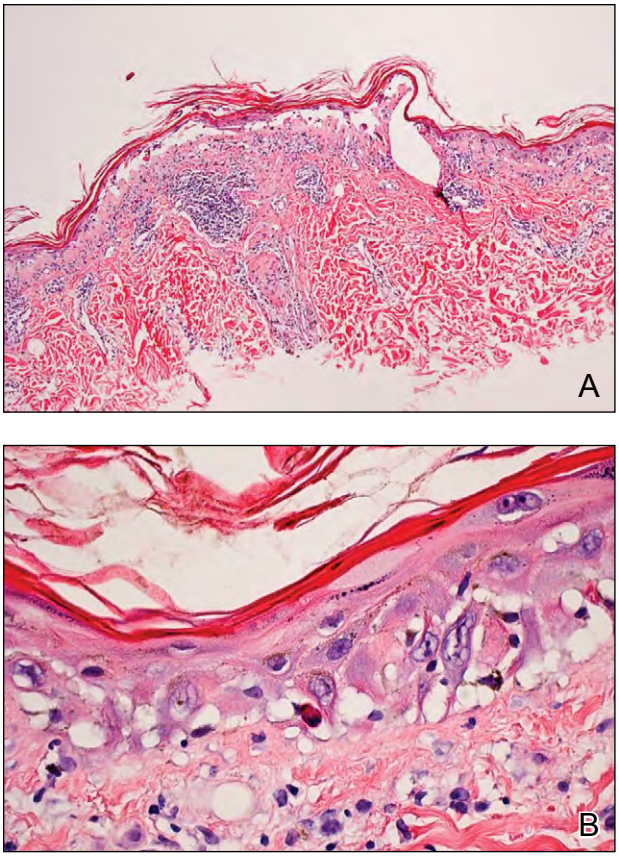

Figure 2. Biopsy revealed subacute interface dermatitis. High-power field showed a vacuolar interface infiltrate associated with mild atrophy, hyperkeratosis, and upper dermal perivascular and focal interstitial lymphocytic infiltrate (A)(H&E, original magnification ×10). Higher magnification showed marked vacuolar alterations of the epidermal basal layer and necrotic keratinocytes with surrounding lymphocytes (satellite cell necrosis)(B)(H&E, original magnification ×40).

on a grounding plate during fluoroscopy at the exact location where the lesion appeared and opposite the site of the radiation source. Contact dermatitis and fixed drug eruption were considered in the main differential diagnosis because of its sharp demarcation. The patient denied exposure to new drugs or any further exogenous trauma to the lesional area.

Histologic examination revealed an interface dermatitis characterized by epidermal atrophy, mild hyperorthokeratosis, vacuolar alterations of the basal layer, mild papillary dermal pigment deposition, and a sparse dermal infiltrate (Figure 2A). Dyskeratotic epidermal cells and surrounding lymphocytes (satellite cell necrosis) also were observed (Figure 2B). Eosinophils and dermal sclerosis were not observed. Periodic acid–Schiff stain and mucin stains did not show basement membrane thickening or increase in dermal mucin, respectively. The combination of clinical and histologic findings and recent history of angiography resulted in the diagnosis of subacute radiodermatitis.

Comment

Radiodermatitis, usually arising after exposure to ionizing radiation, has increased in frequency over the last few years, coinciding with the large increase in fluoroscopy-based procedures in fields such as cardiology, orthopedics, and pulmonology. The extent of injury from radiation is determined by the dose of radiation[1]; duration of exposure; and host factors including connective-tissue diseases, diabetes mellitus, and hyperthyroidism,[2] and can be modulated by concomitant use of photosensitizers.[3] This disease state ultimately can lead to increased morbidity and mortality in patients. Ionizing radiation affects both the epidermis and dermis. It has classically been described as being comprised of acute and chronic radiodermatitis. Subacute radiodermatitis was described in the literature by LeBoit[4] in 1989.

The signs of acute radiodermatitis appear starting several days to several weeks following exposure to ionizing radiation. Patients may present with erythema, desquamation, and blistering that may mimic a burn injury. Less commonly, patients can develop xerosis, alopecia, epidermal and dermal atrophy, or necrosis.[4] Histologically, acute radiodermatitis resembles a phototoxic reaction pattern. It is characterized by pyknotic keratinocytes and epidermal edema. Dermal changes include edema, vasodilation, sparse inflammation, and preservation of eccrine glands. In severe cases there is epidermal or dermal necrosis and blister formation with desquamation.[4,5]

Chronic radiodermatitis occurs months to years after exposure and is characterized by atrophy, telangiectasia, pigmentary alterations, ulcerations, fibrosis of dermis or subcutaneous tissues, and in some instances malignant neoplasms. The microscopic features include epidermal atrophy, homogenization of dermal collagen, telangiectasia, fibrosis, absence of pilosebaceous units, and the appearance of atypical bizarre radiation fibroblasts.[6]

Subacute radiodermatitis is a form of ionizing dermatitis that usually presents weeks to months after exposure and can have overlapping features of both acute and chronic radiodermatitis.[5] It may clinically resemble contact dermatitis; fixed drug eruption; or connective-tissue diseases such as dermatomyositis, morphea, and subacute cutaneous lupus erythematosus. Histologically, subacute radiodermatitis is described by interface dermatitis with basal layer vacuolization and conspicuous necrotic keratinocytes. The characteristic satellite cell necrosis seen

in subacute radiodermatitis is a hallmark feature of acute graft-versus-host disease.[5] It may be indistinguishable from graft-versus-host disease and fixed drug eruption.[5,7] Clinical information, history, and histologic features differentiate subacute radiodermatitis from these other conditions mentioned.

Fluoroscopy-guided procedures usually cause radiodermatitis in areas directly in the field of radiation, but cases of radiodermatitis not directly in the path of the radiation beam have been mentioned in the literature.[4] Radiation injuries have been known to occur anywhere on the body from the head and neck to the buttocks. They can be located posteriorly, anteriorly,[8] or on the sides depending on the orientation of the fluoroscope or the direction of the radiation beam.[9] Similar to our case, radiation wounds described in the literature are well-demarcated and can be rectangular or square shaped.[9,10] Our case is unique because the patient presented with skin changes in a location that was remote from the direct pathway of the ionizing radiation beam and the lesion occurred at the contact site for the grounding plate. Our main differential diagnosis, contact dermatitis, was ruled out based on the patient's history and histologic findings showing absence of spongiosis and presence of satellite cell necrosis. We hypothesized that the grounding plate may have reflected the radiation beam, resulting in amplification of the local ionizing radiation dose.

Conclusion

Subacute radiodermatitis should be considered for lesions in which radiation exposure is associated with apparent contact dermatitis, fixed drug reaction, or connective-tissue disease. With increased use of fluoroscopy and other forms of ionizing radiation for diagnostic and therapeutic indications, dermatologists should be aware of the cutaneous consequences. Minimizing radiation exposure is critically important to decreasing the incidence of radiodermatitis.

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