Background: Fluoroscopy-induced chronic radiation dermatitis (FICRD) resulting from prolonged exposure to ionizing radiation during interventional procedures has been documented in the radiology and cardiology literature. However, the phenomenon has been rarely reported in the dermatologic literature. Since patients with FICRD often see a dermatologist or a primary care physician to treat their injuries, the diagnosis of FICRD is perhaps often overlooked.

Observations: A 62-year-old man with type 2 diabetes mellitus and severe coronary artery disease was seen with a 2-year history of a pruritic, tender, telangiectatic patch lesion over his left scapula. Over the next 2 years, the lesion became indurated and eventually ulcerated. A skin biopsy specimen demonstrated changes consistent with a chronic radiation dermatitis. The patient was unaware of radiation exposure, but persistent questioning from his dermatologists revealed that he had undergone multiple fluoroscopy-guided cardiac procedures. This was confirmed by a review of his medical records.

Conclusion: The diagnosis of FICRD should be considered for any patient who is seen with an acquired vascular lesion, a morphealike lesion, or an unexplained ulcer localized over the scapula, the back, or lateral trunk below the axilla.

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More than 700,000 fluoroscopy-guided procedures are performed each year in the United States. These procedures include many diagnostic studies as well as fluoroscopy-guided interventional therapeutic procedures such as coronary angioplasty and stent placement, cardiac radiofrequency ablation, and transjugular intrahepatic portosystemic shunt placement. While most of these procedures do not result in perceptible radiation-induced skin injury, the potential risk to the general patient population for exposure to a significant dose of ionizing radiation increases as the indications for use, complexity, frequency, and duration of these interventional therapeutic procedures increase.2

Examination of the data obtained from studies of fluoroscopy-induced skin injury in the interventional literature reveals that, in the appropriate clinical setting, the cutaneous and histopathologic findings are fairly characteristic, and the diagnosis of fluoroscopy-induced chronic radiation dermatitis (FICRD) should not be problematic. However, there are several confounding factors that contribute to the difficulty in establishing the diagnosis in the routine office setting. First, the incidence of FICRD is extremely low, which contributes to a lack of awareness and/or recognition among dermatologists and dermatopathologists. Moreover, the patients generally do not seek consultation for their skin lesions from the cardiologist or interventional radiologist who performed the fluoroscopy-guided procedure, and these physicians do not routinely screen their patients prospectively for long-term dermatologic adverse effects.3

Often when patients finally present to a dermatologist with symptoms of cutaneous radiation injury, many months to several years have elapsed between the most recent fluoroscopy-guided procedure to the onset of significant skin changes. These factors might also lead to underreporting, further contributing to the low prevalence of known cases of FICRD.4 Even when radiation dermatitis is suspected, a lack of association between radiation injury and a fluoroscopic procedure leads to a denial of radiation exposure. Furthermore, many patients are unaware of what procedures they have undergone. Knowledge of the most common entrance sites of ionizing radiation associated with cardiac procedures (scapula, back, and lateral trunk under the axilla) is a very important clue to the diagnosis.1 However, this is not intuitive to noncardiologists. All of these factors contribute to the difficulty of establishing the diagnosis of FICRD.
A 62-year-old man presented with an approximately 2-year history of a pruritic, tender, painful skin lesion located over the left scapula. He described having had some trauma to the area and subsequent bruising. Physical examination revealed a 15 × 15-cm erythematous patch on the left posterior shoulder composed of reticulated, confluent matlike telangiectasia (Figure 1). The initial working clinical diagnosis was of an acquired vascular lesion.

Histopathologic examination of a full-thickness skin biopsy specimen revealed several anastomosing, widely dilated, thin-walled vascular spaces lined by a single layer of flattened endothelium within the superficial dermis. Throughout the middle and deep reticular dermis, the collagen bundles were thickened, pink, and homogeneous. There was no inflammatory cell infiltrate (Figure 2). These findings were interpreted as possibly representing an acquired or congenital venous ectasia overlying hyalinized scar tissue. The patient denied radiation exposure or contact with radioactive materials, although he did list an angioplasty under the surgical history portion of the patient questionnaire.

During the next 2 years, the patient’s symptoms progressively worsened, and the area of involvement became thicker, indurated, and sclerotic. He was unresponsive to intralesional injections and topical application of triamcinolone acetonide. In addition, a 1-cm ulceration developed within the central portion of the lesion, corresponding to the previous biopsy site. The patient complained of persistent pain at the site of the ulcer. While hospitalized for chest pain and cardiac workup, the patient developed a 4.5-cm ulcer covered by a thick eschar with foul odor and surrounding erythema (Figure 3). Findings of a tissue culture were positive for Pseudomonas species. The patient was treated for the bacterial infection and additional biopsy specimens were taken.

Histopathologic examination demonstrated that the biopsy specimens were devoid of fatty tissue despite the fact that the full-thickness skin biopsy procedure was performed sufficiently deep to obtain the underlying subcutaneous adipose tissue in the specimen (Figure 4). Hyperplasia of the squamous layer was found and slight irregular elongation of the rete pegs with thickening of the basement membrane zone and squamationization of basal layer keratinocytes. Numerous markedly dilated thin-walled vessels were noted within the superficial dermis. Throughout the superficial, middle, and deep reticular dermis, the collagen was markedly thickened, pink, and homogeneous and lacked the normal clefs that usually separate them into small, wavy, fibrillar bundles. There was very sparse and widely spaced proliferation of spindle-shaped fibroblasts, some of which were quite plump and demonstrated some hyperchromatism of the nucleus. A marked reduction, and in some areas complete absence, of epithelial adnexal structures was noted, including hair follicles, erector pili muscles, sebaceous glands, eccrine coils, and eccrine ducts. Evaluation of the previous biopsy specimen also revealed marked reduction and focal absence of adnexal structures. Despite repeated denials from the patient of a history of exposure to ionizing radiation, the presumptive diagnosis of chronic radiation dermatitis was made.
A subsequent medical history revealed 12 interventional cardiac procedures, including cardiac catheterizations and percutaneous coronary interventions, several of prolonged duration. A focused review of medical records revealed that the patient underwent an estimated 2393.5 rad (23.935 Gy) of total fluoroscopy exposure (5 rad [0.05 Gy]/min) and an unknown cineradiography exposure in an 8-year time frame. This is well beyond the 1000-rad (10-Gy) cumulative threshold for FICRD. The longest procedure was a complicated 168.6-minute percutaneous transluminal coronary angioplasty (estimated dose, 5 rad [0.05 Gy]/min or 843 rad [8.43 Gy]) that occurred 4 years prior to presentation. This investigation, revealing cumulative doses of greater than 1000 rad (10 Gy), combined with the clinical characteristics (eg, site and appearance of lesion) and histopathologic features of FICRD confirmed the diagnosis. A surgical consultation was obtained for excision of the diseased tissue followed by musculoskeletal skin flap coverage.

**COMMENT**

The diagnosis of FICRD should be considered in any patient with a spontaneous ulceration involving the upper back or axilla. However, without clear evidence of radiation exposure, FICRD is often misdiagnosed. Complicating the diagnosis is the insidious and variable onset of symptoms ranging from erythema to dermal necrosis. Diagnosing FICRD is established by correlating a patient history of a fluoroscopy-guided procedure to a skin lesion with the appropriate cutaneous characteristics located at the entrance site of ionizing radiation for that particular procedure. Thus, awareness of these locations is crucial. The most common sites of skin injury associated with these procedures include the right scapular or subscapular area, left scapular or subscapular area, right lateral trunk below the axilla, midback, and the right anterolateral chest.

Fluoroscopic radiation skin injury can be distinguished as either chronic, acute, or subacute. Acute radiation injury is characterized by erythema with vesication, erosion, and pain persisting up to 9 weeks following presentation, has a threshold dose of 200 to 800 rad (2-8 Gy), and a time of onset days after exposure. Chronic radiation injury has a longer time of onset (months to years), a cumulative dose threshold of 1000 rad (10 Gy), and is represented clinically as permanent erythema, dermal atrophy, and ulceration. The Table lists threshold doses and skin manifestations. While an episode of acute radiation injury increases the risk of developing chronic skin changes, FICRD is not always preceded by acute manifestations. Subacute radiation injury is a mixture of these clinical signs, and the diagnosis is based on the key histopathologic feature: lichenoid tissue reaction.

To our knowledge, 42 cases of fluoroscopic-induced radiation skin injury have been reported in the dermatology literature. Of these 42 cases, 31 were defined as chronic radiation dermatitis (FICRD). 3 were subacute (1 of which developed into chronic disease), and 9 were acute dermatitis. There are a number of fluoroscopic procedures implicated as causative mechanisms for radiation injury. The vast majority of these are percutaneous coronary interventions and/or coronary angiography (30 cases). There were 2 cases of radiation injury after radiofrequency catheter ablation, following transjugular intrahepatic portosystemic shunt.
placement,12 and 6 cases related to neurovascular embolization.18

While conventional radiation therapy uses fractionated beam energies designed to spare the skin, fluoroscopic procedures have no skin-sparing qualities, and doses can be delivered in a single fraction.3 During a typical fluoroscopic procedure, the skin radiation dose is between 2 and 5 rad/min (0.02 and 0.05 Gy/min). However, doses as high as 5 rad/min (0.5 Gy/min) have been documented.10 As in the present case, most of the reported cases of FICRD are due to cardiac procedures. As summarized in the Table, our patient underwent 12 such procedures. Cardiac catheterizations generally expose patients to an average dose of 250 rad (2.5 Gy). Percutaneous transluminal coronary angioplasty procedures are particularly dangerous (average dose, 640 rad [6.4 Gy]) because radiation is focused only on those vessels with stenosis.6 While these represent standard exposure estimates, interventional procedures are highly variable. The machinery used, the particular facility, and most importantly the proficiency of the person performing the procedure (a 6-fold variance) all play a role in determining the dose of radiation for a given procedure.3 Finally, patients requiring cardiac catheterizations and percutaneous transluminal coronary angioplasty procedures often undergo multiple exposures, easily putting them at risk for exposure to the cumulative threshold dose required for FICRD (1000 rad [10 Gy]).4 While certain changes have been made to limit skin exposure during fluoroscopy, radiation exposure during fluoroscopic procedures remains a risk to patients.

For primary care physicians and dermatologists to whom these patients usually initially present, an emphasis must be on a history of fluoroscopic procedures in a patient with characteristic skin manifestations. The clinical and histopathologic signs of FICRD, particularly the site where lesions appear, are key elements in establishing the diagnosis. Patients are often unaware of previous radiation, and therefore additional information should be obtained from hospital records and other physicians when clinical suspicion for FICRD is high.

Table. Skin Manifestations of Radiation Exposure

<table>
<thead>
<tr>
<th>Effect</th>
<th>Typical Threshold Dose, rad (Gy)</th>
<th>Time of Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early transient ischemia</td>
<td>200 (2)</td>
<td>Hours</td>
</tr>
<tr>
<td>Temporary epilation</td>
<td>300 (3)</td>
<td>3 wk</td>
</tr>
<tr>
<td>Main erythema</td>
<td>600 (6)</td>
<td>10 d</td>
</tr>
<tr>
<td>Permanent erythema</td>
<td>700 (7)</td>
<td>3 wk</td>
</tr>
<tr>
<td>Dry desquamation</td>
<td>1000 (10)</td>
<td>4 wk</td>
</tr>
<tr>
<td>Invasive fibrosis</td>
<td>1000 (10)</td>
<td>NR</td>
</tr>
<tr>
<td>Dermal atrophy</td>
<td>1100 (11)</td>
<td>&gt;14 wk</td>
</tr>
<tr>
<td>Telangiectasia</td>
<td>1200 (12)</td>
<td>&gt;52 wk</td>
</tr>
<tr>
<td>Moist desquamation</td>
<td>1500 (15)</td>
<td>4 wk</td>
</tr>
<tr>
<td>Late erythema</td>
<td>1500 (15)</td>
<td>6-10 wk</td>
</tr>
<tr>
<td>Dermal necrosis</td>
<td>1800 (18)</td>
<td>&gt;10 wk</td>
</tr>
<tr>
<td>Secondary ulceration</td>
<td>2000 (20)</td>
<td>&gt;6 wk</td>
</tr>
</tbody>
</table>

Abbreviation: NR, not reported.

*This table is an adaptation of data found in text and tables in Koenig et al1 and Lee et al.3

REFERENCES