Chronic Radiodermatitis Following Percutaneous Coronary Interventions

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The incidence of radiodermatitis after percutaneous coronary interventions is rising with the increasing number and complexity of these procedures. This is a case report of a male patient with 3-vessel coronary artery disease in whom chronic radiodermatitis developed following four difficult and prolonged coronary interventions. The skin eruption was characterized by a well-demarcated, atrophic, rectangular plaque presenting as mottled hyper-and hypopigmentation with telangiectasia. Generally, the skin lesions can appear several weeks to a decade after the procedure. The variable onset of these clinical features renders the association with previous angiographic procedures difficult. Even though the risk of radiation injury in most of the patients undergoing cardiac catheterization is low, the potential for radiodermatitis should not be ignored, especially in patients receiving interventional and complicated procedures. (Dermatol Sinica 22 : 148-152, 2004)

Key words: Cardiac catheterization, Chronic radiodermatitis, Percutaneous coronary angioplasty, Percutaneous coronary intervention, Radiation, Radiation injury
INTRODUCTION

Fluoroscopy and cineradiography used during coronary angiography expose patients to some of the highest doses of ionizing radiation in diagnostic radiology.\(^1\)\(^-\)\(^2\) Even higher levels of radiation may result from percutaneous coronary angioplasty (PTCA), which is a more complicated and lengthier procedure. Recently, as the number of diagnostic cardiac catheterization and interventional procedures increased, radiation-induced skin injuries following prolonged fluoroscopic exposure (including acute and chronic radiodermatitis, radiation-induced morphea, radiation keratosis, and skin squamous cell carcinoma) have been reported.\(^3\)\(^-\)\(^9\) Radiodermatitis is dose-dependent and triggered by known threshold doses.\(^10\) The skin lesions encompass a wide spectrum ranging from erythema, telangiectasia, atrophy, hyperpigmentation, and hypopigmentation to necrosis, chronic ulceration, and squamous cell carcinoma.

We describe a patient in whom chronic radiodermatitis developed following repeated cardiac catheterization and PTCA with prolonged fluoroscopic imaging.

CASE REPORT

A 50-year-old man presented to the dermatology department with a skin lesion just

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Fig. 1
There is a palm-sized, square-shaped, sharply-demarcated, hyperpigmented plaque with telangiectasia and atrophy within it.

Fig. 2
(a) There are pigment incontinence and thickened collagen fibers with several dilated vessels in the upper dermis.
(b) There are diffuse dermal sclerosis with thickened collagen bundles, mild perivascular lymphocytic infiltration and stellate fibroblasts in the dermis.

Fig. 3
Gray area: Post-PTCA radiodermatitis could be seen anywhere in the right subaxillary region and upper back. A.B.C.D: Radiation injuries of these areas caused by cardiac catheterization have been reported in the literature. A: The injury site of the presenting case.
below the right axilla for 10 months. He recalled having had a similar sized brownish patch in the same region initially, but in the past month itching, tenderness, and a firm mass had developed. On physical examination, a palm-sized, square-shaped, sharply-demarcated, hyperpigmented plaque with telangiectasia was noted. There was also a pigeon-egg sized, hypopigmented, dermal atrophic area within the plaque (Fig. 1). Histopathological findings included pigment incontinence, diffuse dermal sclerosis with atypical stellate fibroblasts, and thickened vessel walls with perivascular lymphocytic infiltration (Fig. 2(a)(b)). These findings were consistent with a diagnosis of chronic radiation injury.

The patient had been diagnosed with 3-vessel coronary artery disease and suffered from frequent angina. He reported having undergone coronary angiography with PTCA four times (12 months, 11 months, 4 months, and 3 months before developing the current skin lesion). He had noted a brownish patch below the right axilla after the second procedure, and the lesion was asymptomatic. The lesion became sclerotic with tingling sensation 2 months after the fourth procedure. A review of his medical records revealed that all 3 vessels (right coronary artery, left circumflex artery, and left anterior descending artery) had severe stenosis over several segments. The coronary angioplasties were performed strenuously, and the courses were prolonged because of severe stenosis and coronary tortuosities. Although the exact radiation doses were not specified in the medical records, the patient recalled it took 8, 6, 6, and 5 hours, respectively, in the catheterization laboratory for the above-mentioned four cardiac catheterization and PTCA procedures.

The biopsy wound within the plaque did not heal completely for several months. Surgical intervention (excision of the radiodermatitis area and replacement with a skin graft) was recommended because skin ulceration and pain insufficiently responded to local treatment with antibiotic (tetracyclin) ointment, biweekly intralesional injection of 10% triamcinolone acetonide (Kenacort), and systemic non-steroid anti-inflammatory drug (NSAID).

DISCUSSION

Chronic radiodermatitis following cardiac catheterization was first reported in 1996 by Lichtenstein et al. Cutaneous side-effects are mainly dose-dependent effects of ionizing radiation. The calculated dose from fluoroscopy can range from 0.02 to 0.05 Gray (Gy) per minute, and in extreme circumstances, doses can reach up to 0.5 Gy per minute. Image recording involves even higher doses. Little time is spent to verify the proper location of the catheter tip during simple angiography with fluoroscopy. However, during some other procedures including PTCA, the fluoroscopy time is much longer because guidewires and balloons must be carefully placed in the stenosed portions of the coronary arteries before balloon dilatation is performed. A routine cardiac catheterization procedure exposes a patient to an average dose of 2.5 Gy, whereas percutaneous interventions result in an average dose of 6.4 Gy. However, the exact dose varies with the complexity of the procedure and the number of vessels treated. Specifically, the threshold doses for development of erythema, permanent epilation, desquamation, and necrosis are 3-10, 7-10, 12-25, and 25 Gy, respectively. Moreover, the cumulative dose necessary to induce chronic skin changes has been estimated to be greater than 10-12 Gy. In the presenting case, the estimated cumulative dose in the prolonged PTCA procedures might have been up to 25 Gy (as mentioned above, the average dose of a PTCA is 6.4 Gy x 4 times) thus leading to such severe chronic radiodermatitis and skin necrosis.

Chronic radiodermatitis may develop weeks to years after radiation exposure. It is often misdiagnosed because of the insidious and late onset of clinical symptoms, which in turn encompass a wide spectrum ranging from erythema to telangiectasia, atrophy, hyperpigmentation, ulceration, and necrosis. The variable onset time of these clinical features renders the association with previous angiographic procedures
even more difficult. Not all patients with chronic radiodermatitis have a history of an acute dermatosis immediately after exposure to X-rays in the same locations where chronic radiodermatitis may later develope. This may be attributed to two factors. First, acute radiodermatitis may have been too mild to be remembered or may not have been recognized. Second, although the cumulative dose of radiation was sufficient to induce chronic changes, the fractions in which it was delivered may have been too small to induce acute changes (approximately 3-Gy dose of a single 90-kV irradiation or 6-Gy to 8-Gy dose of a single 200-kV irradiation may cause erythema).

In addition to the onset time, there are several other elements in the medical history that help to raise the clinical suspicion of radiodermatitis. The location of radiation injury may correlate with the stenosed vessels and may be related to the position of the X-ray tube. The different degrees of radiosensitivity in different regions of the skin also account for the predilection areas. Whereas the neck is the least radiosensitive, the chest, abdomen, thigh, back, and face are progressively more radiosensitive.

In reviewing the literature, we found there are four predilection areas (sites A, B, C, and D) located on the back (Fig. 3). Chronic radiodermatitis in this patient was located on site A. Injury sites A and B mainly result from left anterior oblique projection in cardiac catheterization; whereas, injury sites C and D mainly result from right anterior oblique projection. During most cardiac angiographic procedures, the upper back receives the highest exposure. This is because left and right anterior oblique projections are used most frequently during fluoroscopy and angiography. Variable degrees of antero-posterior projections (used to visualize the segment of the vessel to be treated) can result in radiation damage anywhere between sites A and D (the gray patch on the Fig. 4).

The clinical manifestation of chronic radiodermatitis following percutaneous coronary interventions is characterized by a well-defined patch (which could be erythema, telangiectasia, atrophy, hyperpigmentation, ulceration, or necrosis) with burning sensation, most frequently located on the back. A history of several difficult and prolonged coronary interventions several weeks to years before the onset of this skin lesion may be identified. The histological findings are multiple dilated vessels surrounded by a lymphocytic infiltrate in the superficial dermis and thickening of collagen with stellate fibroblasts in the deep dermis. Sometimes the patients can be reminded of previous acute radiodermatitis on the same area, and this may assist in diagnosing chronic radiodermatitis.

When the lesion of chronic radiodermatitis is noted early in its development, it is usually misdiagnosed as a fixed drug eruption or morphea and therefore inappropriately treated. Radiation-induced morphea (RIM) can be differentiated from chronic radiodermatitis by several clinical and histological findings. RIM does not appear to be related to the radiation dose and is a rapidly progressive; whereas chronic radiodermatitis changes are dose-related and often take years to develop. Besides, RIM extends beyond the boundaries of the irradiated area in 20% of cases; whereas chronic radiodermatitis is confined to the irradiated area. The histological changes in RIM, fibrosis centered primarily in the dermis, are different from those in chronic radiodermatitis, fibrotic changes mostly involving deep subcutaneous fat, fascia, and muscle.

The clinical symptoms of pain and ulceration only appear several years later. The pain is usually a burning sensation and often the reason for the patient to seek medical help. Treatment is directed at using hydrocolloid dressing to stimulate wound healing, using topical and intralesional corticosteroids to inhibit local inflammation, using antibiotics to prevent infection, and using anagelsics to alleviate discomfort. Occlusive dressings may speed healing and can reduce the pain associated with radiation-induced skin reactions. However, wide surgical excision with covering skin graft is suggested when adequate local treatment to resolve skin lesions fails and the excruciating pain per-
Because pain often remains unresponsive to local treatments and because of the risk of developing squamous cell carcinoma, it is important to prevent these complications as much as possible. Many techniques are useful, including minimizing the time of both fluoroscopy and cineradiography and using monitoring devices that provide cumulative radiation exposure dose. Topical radioprotective agents such as prostaglandins have been shown to be effective. Since cardiac angiography is frequently used in diagnostic and therapeutic coronary procedures, it is important for physicians to prevent and diagnose chronic radiodermatitis early.

REFERENCES