A Rectangular Dermatosis of the Left Back

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Background: Cardioversion and defibrillation have become widely used techniques aimed at restoring normal sinus rhythm in patients with cardiac arrhythmias. Following the procedure, cutaneous lesions are often seen at the site of the electrodes, but little has been reported regarding the evolution of such lesions over time.

Observations: Two patients presented with unusual, well-defined rectangular eruptions on the left back, and both reported a history of having undergone electrical cardioversion or defibrillation several years previously. The histologic characteristics of each lesion were distinct, and the management was symptomatic, with most of the relief coming from the recognition that the eruption was actually a self-limited manifestation of cardioversion and defibrillation.

Conclusions: The clinical cases and corresponding histologic findings represent possible long-term sequelae of electrical cardioversion or defibrillation. They are presented in order to enhance the diagnostic acumen of dermatologists and to avoid potential misdiagnosis.

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REPORT OF CASES

CASE 1

A 68-year-old man was seen at the dermatology clinic for evaluation of an intensely pruritic lesion on his left mid back. Examination demonstrated an 8 × 5-cm well-demarcated, depressed rectangular plaque with a heterogeneous center with waxy, crusted 3- to 5-mm papules and focal telangiectasias (Figure 1). Evidence of spontaneous breakdown was present on the superior aspect of the lesion. The patient reported that the lesion had developed at the site of a defibrillator pad burn that he sustained during an emergent defibrillation for ventricular fibrillation approximately 3 years previously. The lesion had healed uneventfully and remained an asymptomatic scar until 6 months prior to presentation, when it became pruritic and keratotic.

The remainder of the examination was notable only for an automatic implantable cardioverter defibrillator (AICD) insertion site at the left anterior chest. The patient’s medical history was clinically significant for hypertension, hypercholesterolemia, ventricular fibrillation with subsequent AICD insertion, chronic renal insufficiency secondary to type I membranoproliferative glomerulonephritis, and anemia and hyperkalemia secondary to chronic renal insufficiency.

A 5-mm punch biopsy specimen was taken from the right upper aspect of the rectangular edge of the lesion, and histologic findings demonstrated a focally parakeratotic epidermis overlying superficial papillary dermal and periadnexal lamellar fibrosis (Figure 2). The scar was present above grouped pilar erector muscles without histologic evidence of hair fol-
lices or sebaceous lobules. These histologic findings correlated with the clinically alopecic plaque. A mixed lymphohistiocytic and eosinophilic infiltrate was present adjacent to the scar (Figure 3). The remainder of the epidermis and reticular dermis was unaltered, and there was no histologic evidence of morphea or scleroderma. The patient was treated with topical clobetasol propionate and on follow-up had considerable clinical and symptomatic improvement of the lesion.

CASE 2

A 50-year-old man was referred for dermatologic evaluation of an asymptomatic lesion on the back that had been noted during a recent routine primary care visit. Physical examination showed a 10 × 3-cm atrophic plaque on the patient’s left mid back with telangiectasias (Figure 4). There was neither palpable induration nor tenderness. The patient had undergone elective direct current cardioversion 7 years previously for atrial fibrillation, and he recalled having experienced burns from the cardioversion paddles on his chest and back, both of which required only local therapies at the time. He had not had any medical follow-up in the interim. Findings from the remainder of the physical examination were not clinically significant. The patient’s medical history was notable for asthma, diabetes mellitus, and gastroesophageal reflux disease.

An excisional biopsy extending into the subcutaneous fat was performed on patient 2 to compare the atrophic plaque with the normal-appearing adjacent skin (Figure 5). On low power, the thickness of the entire dermis of the lesional skin compared with the normal-appearing skin (Figure 5) was considerably decreased, which was also observed clinically. There was a clinically significant decrease in the size of the sebaceous glands in the atrophic areas compared with those in the adjacent normal skin. In addition, only the infundibular portion of the hair follicle was present along the extremely atretic sebaceous lobule. There was also a complete absence of eccrine lobules, dermal ducts, and acrosyringea. Although vestigial, the sebaceous units appeared to be present at a normal frequency in this biopsy specimen; however, only a small portion of normal-appearing skin was present for comparison. On higher power (Figure 6), the histologic sections showed papillary dermal fibroblasts and scar formation overlying significantly atrophic sebaceous lobules with a mild, non-
specific, superficial perivascular lymphocytic infiltrate. An elastin stain (not shown) demonstrated a normal distribution of elastin fibers present in the reticular dermis and adjacent to the normal-appearing sebaceous lobules in the nonlesional skin, militating against a sclerodermoid disorder. Because the patient was asymptomatic, no treatment was initiated.

COMMENT

Electrical cardioversion and defibrillation are methods commonly used to restore normal sinus rhythm to patients with cardiac arrhythmias. Cardioversion refers to the delivery of a synchronized current and is used for organized arrhythmias such as atrial fibrillation, whereas defibrillation refers to the delivery of an unsynchronized shock and is used in cases of ventricular fibrillation or pulseless ventricular tachycardia. In either case, the basic procedure is the same: an electrical current is delivered between 2 transcutaneous electrodes, resulting in depolarization of the myocardium. There are several different commercially available defibrillators, which have a variety of corresponding electrodes. The types of electrodes include self-adhesive pads preapplied with liquid gel, self-adhesive pads preapplied with polymer gel, traditional handheld electrode paddles coated with conductive paste, and handheld electrode paddles with preformed conductive gel pads that are placed between the paddles and chest.1

The electrodes are typically placed on the right anterior chest and left back but also may both be placed on the chest. Owing to the high resistance across the thorax, a relatively high level of energy is necessary to deliver adequate current density, with certain protocols discharging up to 360 J. Although each shock is only transmitted for 5 to 10 milliseconds, the energy is sufficient to cause superficial burns to the skin.2 Most patients who have undergone electrical cardioversion or defibrillation experience overlying significantly atretic sebaceous lobules with a mild, nonspecific, superficial perivascular lymphocytic infiltrate (hematoxylin-eosin, original magnification ×100).

The extent of cutaneous damage that occurs in association with defibrillator electrodes is variable, and, in
addition to superficial burns, more serious complications, including deeper burns and pectoral muscle necrosis, have also been reported. The initial severity of a lesion has been shown to be influenced by the peak and cumulative shock energies delivered, and burns tend to be greater at the periphery than at the center of the lesion. While the specific electrodes used affect the shape of the skin lesion, there does not seem to be an obvious difference among the various electrode types with regard to histologic damage.

Histologically, defibrillator electrode injuries biopsied within a week after the procedure have been described as demonstrating variable amounts of focal epidermal necrosis, which in many cases has involved sebaceous glands and which has been associated with multinucleated keratinocytes. Dermal edema associated with a perivascular lymphocytic infiltrate containing eosinophils and neutrophils has also been observed. To our knowledge, however, there have not been any reports of chronic lesions resulting from the electrical current-induced injuries.

By report, in the first patient the type of electrode used during the defibrillation was a pad with preapplied self-adhesive polymer gel. The mixed infiltrate containing eosinophils may represent a hypersensitivity reaction to the polymer gel in acute cases. The scar in this patient, however, was asymptomatic for several years; therefore the etiology of the eosinophilic infiltrate is unclear. We speculate that as these types of electrodes come to be used more frequently than traditional paddles, the incidence of postcardioversion skin lesions may increase, and awareness of this entity will greatly assist clinical management.

The available data regarding the nature of skin lesions caused by defibrillator electrodes primarily detail short-term effects. Our cases demonstrate superficial dermal and periadnexal scar formation with focal pilosebaceous unit and eccrine apparatus destruction. The finding of periadnexal scar formation may indicate that the sebaceous lobules may act as a conductor of the electrical current, and, therefore, the adnexal areas are more significantly affected than the surrounding tissue. In patient 2, no normal hair follicles, sebaceous lobules, or eccrine glands were present in the atrophic affected areas. Only the infundibular portion of the hair follicle and the atretic sebaceous lobules were present. In fact, the finding of complete loss of the pilosebaceous units and eccrine glands, as seen in patient 1, may suggest a greater duration or magnitude of current with subsequent adnexal destruction. The combined histologic and clinical findings of remote electrical current-induced injury are clinically significant because they represent novel changes observed in patients after remote electrical injury. Our cases demonstrate the course and potential evolution of skin lesions caused by defibrillator pads years following the acute injury and are presented to promote recognition of this under-reported finding and diagnostic pitfall.

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REFERENCES