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OLGU SUNUMU/CASE REPORT

A Case of Erythema ab igne Containing Atypical Squamous Cells in Epithelium

Epitelde Atipik Skuamöz Hücreler İçeren Eritema ab igne Olgusu

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ÖZ

Eritema ab igne (EAİ) genellikle kronik ve tekrarlanan düşük seviyelerde kızılötesi radyasyona ve çeşitli şekillerde ısıya anormal maruz kalmanın neden olduğu bir dermatozdur. Günümüzde nadir olmakla birlikte ısıtma yastıkları, araba ısıtıcıları, elektrikli alan ısıtıcıları ve dizüstü bilgisayarları içeren çoklu ısı kaynaklarının bu duruma neden olduğu bildirilmiştir. EAİ iyi prognozlu olmasına rağmen, skuamöz hücre ve Merkel hücreli karsinomlar için risk oluşturmaktadır. Radyant ısıtıcı etkisi ile oluşmuş EAİ nedeni ile histolojik olarak atipik skuamöz hücreleri yaygın olan bir kadın olguyu sunduk.

ABSTRACT

Erythema ab igne (EAI) is a chronic and recurrent dermatosis caused by long-time or excessive exposure to low levels of infrared radiation and various forms of heat. It is a rare condition today and multiple heat sources including heating pads, car heaters, electric space heaters and laptops have been reported to cause this condition. Although EAI has a good prognosis, it poses a risk for squamous cell and Merkel cell carcinomas. We present a woman diagnosed with EAI including extensive atypical squamous cells histologically caused by a radiant heater.

Introduction

Erythema ab igne (EAI) is a reticular, hyperpigmented, sometimes erythematous and telangiectatic dermatosis. Historically it has been associated with prolonged exposure to a stove or heat effect. It is usually caused by prolonged and repeated exposure to low levels of infrared radiation levels (1-3). EAI prevalence has decreased in the industrialized world since the use of central heating in homes (3-5). Contemporary heat sources reported cusing EAI to include radiant heaters, heated bed or massage chairs, heating pads, hot water bottles, car heaters and electric space heaters. The faces and forearms of bakers, glass blowers and casting workers are also at risk (1-4). Laptop-related EAI case reports have been increasing in recent years (1,2). Our case was directly exposed to long-term radiant heat.

Case Report

A 47-year-old woman applied to our dermatology outpatient clinic in February 2020, due to irregularly distributed blemishes on the knees and pretibial areas, in a red-brown color, with an interlocking appearance for 3-4 months. She stated that she used a radiant electric heater to warm her legs under her desk during the winter at work. In physical examination, there were reticular, erythematous and hyperpigmented macular patches extending from the lower legs to the knees and distal, especially on the front of the legs, scattered in the medial and lateral directions, interconnected mesh. There was no feature in the system examination (Figure 1). A punch biopsy was taken from the lesion on the lateral side of the right lower leg. In the follow-up of the patient, the heater was placed approximately three to four meters away from his legs. She

did not use his laptop on his lap. Itching and hyperpigmentation have regressed over time. In systemic evaluation, no pathological findings were observed. In laboratory data, complete blood count and extensive biochemical panel were normal. Consent form was taken from the patient.

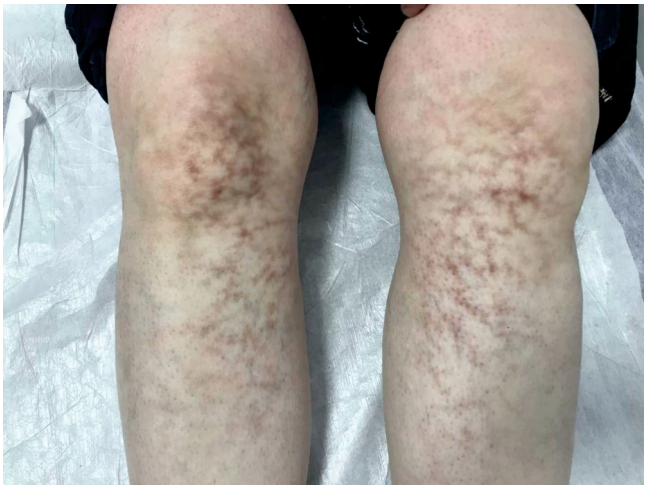

Figure 1. Reticulated, hyperpigmented, erythema areas with telangiectasies suggesting erythema ab igne in the skin.

Histological Findings

In the atrophic epidermis, thin lamellar-like orthokeratosis was present in the corneal layer. No pathological feature was observed in the granular layer. Atypical keratinocytes with nuclei, with a slightly hyperchromatic, pleomorphic, large eosinophilic prominent nucleolus in the spinous and basal layers were observed. In the immunohistochemical examination, the Ki-67 proliferation index rate was high in atypical keratinocytes. With Melan A, only basal melanocytes were positive. The rete ridge pattern has disappeared. In the basement membrane area, collagen tissue degeneration and occasional capillary proliferation were observed. Mild edema and rare lymphocyte reaction were observed in the perivascular areas of lamina propria (Figure 2).

The patient was diagnosed with erythema ab igne with clinical and histological findings.

Discussion

EAI is a reticular, hyperpigmented, sometimes erythematous and telangiectatic dermatosis. Historically it has been associated with prolonged exposure to a stove or heat effect. The most effective factor in etiology is prolonged and repeated exposure to low levels of infrared radiation (1-3). Its prevalence has decreased with central heating in residences (3-5). Radiant heaters, heated beds or massage chairs, heating pads, hot water bottles, car heaters and electric space heaters are other risk factors in etiology. Workers in areas exposed to heat for a long time, such as bakers, glass blowers, and casting workers, are at risk (1-4). Laptop-related case reports with EAI have been increasing in recent years (1, 2). Our case was directly exposed to long-term radiant heat.

The diagnosis of EAI is made clinically based on its characteristic appearance on the skin. It appears in the form of painful, macular erythema, which initially transient. In repeated exposures, the lesions become hyperpigmented and permanent. There is also a bullous variant and

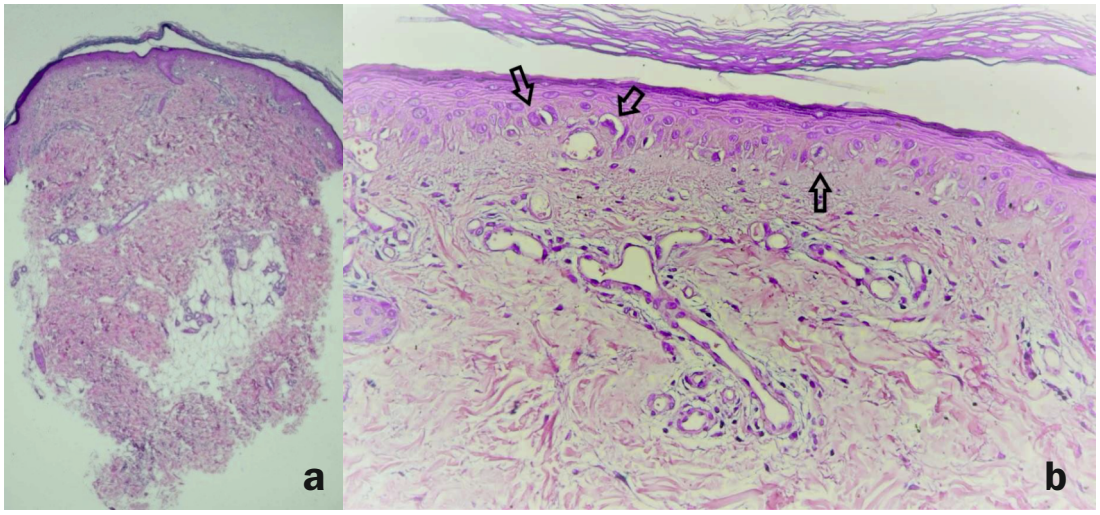

Figure 2. (a) A punch biopsy of lesional skin reveals a slightly attenuated epidermis, but otherwise unremarkable skin (H&E, 40x). (b) Atrophic Epidermal rete-ridge pattern is lost. Keratinocytic atypia is common in the basal and spinous layers (arrows). The superficial dermis has slightly dilated, thin-walled blood vessels. High power view reveals a nonspecific sparse perivascular lymphocytic infiltrate (H&E, 200x).

is associated with the disease that develops in the late stage (3,4). Skin eruptions are asymptomatic. There may be a slight burning sensation. With the location of the heat source and the protection of the clothes, the distribution of the lesions varies (5,6). The lesions in our case were hyperpigmented and reticular in the form of patches connecting each other in the knee and pretibial areas.

Pathophysiology is unknown, but causes changes in actinically damaged skin, dermal elastic fibers (1-3). Early histopathological changes are epidermal atrophy and vasodilation in capillaries. Dermal melanin and hemosiderin accumulate in later-stage lesions. The lesions also contain atypical squamous cells with a nucleus in the vesicular chromatin pattern, which contains a size difference similar to that observed in actinic keratoses. Mitosis can be observed (1,2). EAI patients are at risk of developing skin malignant conditions after a delay that can last for decades.

These patients are at risk for both squamous cell and Merkel cell carcinomas (1-6).

In our case, atypical and atypical mitosis were observed histologically in squamous cells. The Ki-67 proliferation index was found to be high in this area (Figure 3).

Its treatment consists primarily of stopping the use of the heat source. While early-stage lesions typically disappear within months, hyperpigmentation associated with more chronic lesions can last for years. The topical 5-fluorouracil cream has been reported to successfully eliminate epithelial atypia associated with EAI and reflecting histopathological overlap with actinic damage (6).

Atypical squamous cells and high Ki-67 proliferation index rates observed in our case pose a risk for squamous cell carcinoma. For this reason, it will be appropriate to clinically close follow-up of patients like our case by removing them from heat exposure.

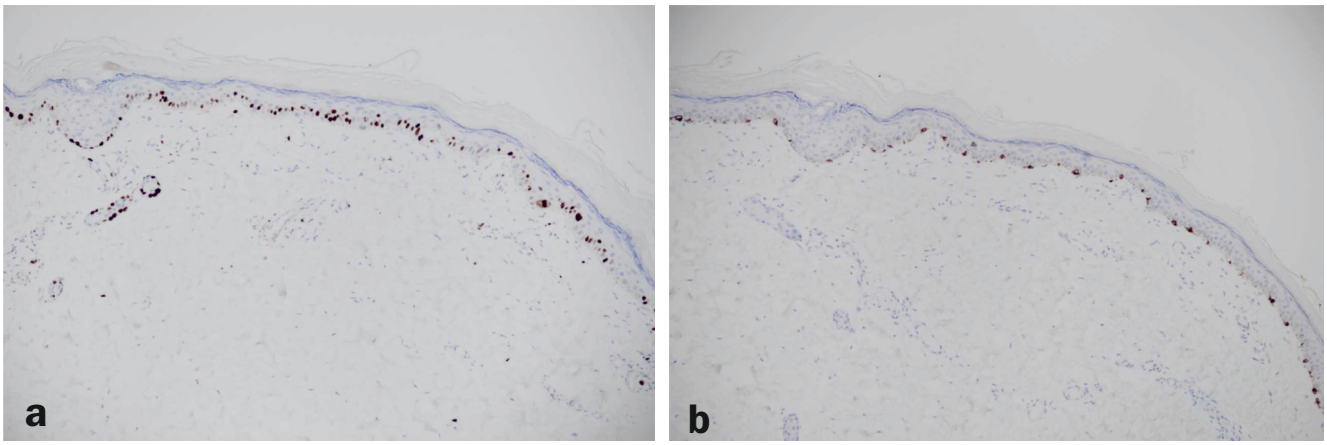

Figure 3. (a) In immunohistochemical examination, the Ki-67 proliferation index rate was found to be high in atypical squamous cells (100x). (b) A slight increase in melanocytes was observed in the basal layer with Melan A in immunohistochemical examination (100x).

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