Bullae and Sweat Gland Necrosis After an Alcoholic Deep Slumber

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A 37-year-old man developed edematous areas and blisters on the right side of his face, chest, and arm after an alcoholic deep slumber. It was revealed that the affected body parts were those pressed during his alcoholic sleep. Histopathological findings of the patient's skin lesions showed typical sweat gland necrosis. Serum enzyme level studies of aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, and creatine phosphokinase were characteristic of muscular damage. This case report is an example of the typical findings of the effects of body pressure on soft tissue that can be seen in a dermatology clinic.

It is well known that patients in a coma due to accidents, illnesses, large doses of narcotic drugs, or carbon monoxide poisoning may show erythema and bullae at sites of pressure, as well as histopathologically characteristic eccrine sweat gland necrosis.[1-8] We discovered such cutaneous lesions in a patient who had an alcoholic deep slumber overnight and through noon the next day. The affected parts of the body had marked tender swelling in addition to characteristic sweat gland necrosis and marked elevation of skeletal muscle-derived serum enzyme levels. The patient's primary care physician misdiagnosed the lesions as either cutaneous infection or acute dermatitis. Considering the frequency with which people consume alcoholic drinks, it is our belief that dermatologists should be well informed about this condition.

Case Report

A 37-year-old man was transferred to the emergency department of the Chonbuk University Hospital from a local clinic, where he had been treated for 2 days under the clinical impression of soft tissue infection. The patient was an intermittently heavy drinker but otherwise was in good physical and mental condition.

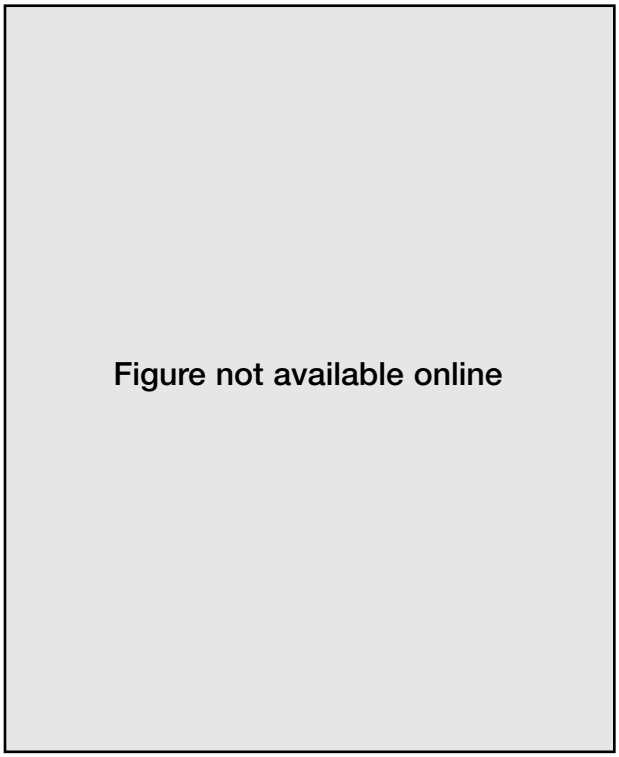

Figure 1. Tender swelling of the right cheek and eyelid, showing a still-fresh linear blister and a dried-up blister remnant on the lesion.

On admission, the patient had tender swelling of the right cheek and eyelid with a well-demarcated linear blister on the right lateral forehead and a dark brownish dried-up blister on the prominent area of the cheek (Figure 1). Physical examination also revealed well-demarcated edematous erythemas and blisters on the right upper inner arm and right anterior chest across the anterior axillary fold and diffuse tender edema of the right forearm that was

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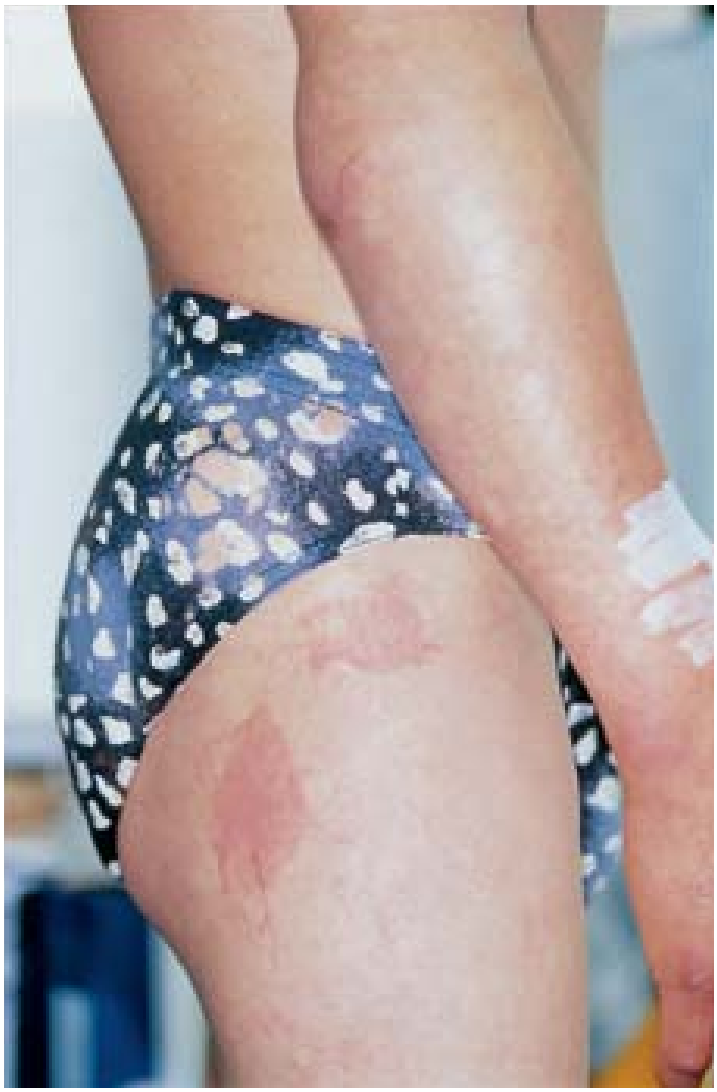

Figure 2. Firm bulky edema of the entire right forearm and circumscribed erythematous areas on the right lateral buttock.

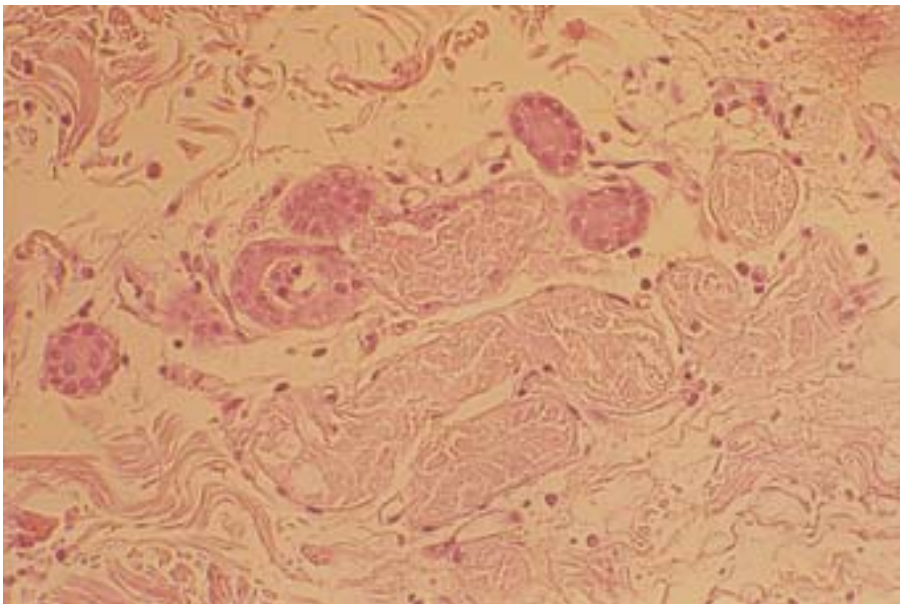

Figure 3. Secretory cells of the sweat glands show necrosis characterized by eosinophilic homogenization of their cytoplasm and absence of their nuclei. Sweat ducts are preserved (H&E, original magnification ×200).

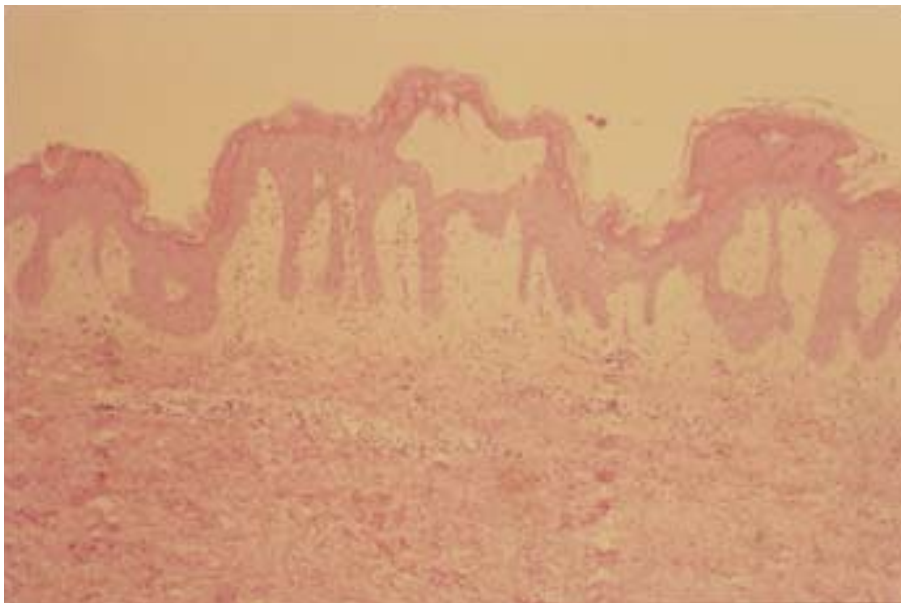

Figure 4. Intraepidermal blisters with eosinophilic degeneration of overlying epidermis and marked edema of papillary dermis (H&E, original magnification ×40).

firm and bulky (Figure 2). In addition, circumscribed erythemas and swelling of the right lateral buttock (Figure 2) and well-demarcated edematous erythemas on the fingertips and hypothenar area of right hand were found. Because the cutaneous lesions were located only on the right side of the body, we determined the lesions might have formed on the body parts that were pressed during the patient's alcoholic slumber. Family members who witnessed his sleeping position supported our belief that the patient had slept on his right side.

The histopathological findings of the skin tissue taken from the right anterior chest near the axillary fold revealed intraepidermal blisters with overlying eosinophilic degeneration of granular cells, marked upper-dermal edema, eosinophilic necroses of the eccrine sweat glands (Figures 3 and 4), and some perivascular neutrophils.

Blood chemistry results showed that aspartate aminotransferase (AST) was greater than 850 U/L, alanine aminotransferase (ALT) was greater than 259 U/L, lactate dehydrogenase (LDH) was greater than 4558 U/L, and creatine phosphokinase (CPK) was greater than 3540 U/L. Those data were compatible with acute muscular injury. Myoglobulinuria also was noted. Serologic markers for hepatitis and ultrasonography of the liver were all either negative or normal.

We treated the patient by elevating the affected body parts and administering massage and cold compresses. The patient was discharged after 7 days, without sequel, when his serum enzymes decreased to the following levels: AST, 49 U/L; ALT, 86 U/L; LDH, 944 U/L; and CPK, 571 U/L.

Comment

It is well known that the skin can change because of sustained autocompression by a patient's own body weight. Previously reported cases were largely caused by drug overdoses[1,2,4,6,7] or carbon monoxide intoxication.[8] The diagnosis of the condition has been reported by different names. It has been called *sweat gland necrosis* based on its characteristic dermatohistopathological features,[1,2] *coma blister*,[4] *bullae* (in comatose patients),[6,9] or, more inclusively, *cutaneous changes* (in drug-induced comatose patients).[3,7,10,11] These cutaneous findings also are found with compartmental syndrome, which is a more serious condition often reviewed in surgical literature.[12]

The cutaneous lesions reported in the literature were quite protean, showing erythema, blisters, and brawny edema. The observations of Arndt et al[5] showed that these lesions frequently appeared over bony prominences, were linear and unilateral, and were localized to sites of body apposition. These 4 characteristics were all observed in our patient. The linearly distributed blister running downward combined with the erythematous swelling on the patient's face led the hospital dermatologist to first suspect the case was contact dermatitis. Also, the sudden onset of multiple tender swellings and blister formations found in people who drink alcohol led the patient's primary care physician to suspect the condition was some type of bacterial infection, such as *Vibrio vulnificus*, which is often found where the patient lived.

Prolonged pressure on both the skin and underlying soft tissue normally causes people to feel

discomfort, which in turn causes them to change their position and thus avoid pressure damage. Even during sleep, intermittent nocturnal movements relieve localized sustained pressure on the body. In comatose patients, however, the inability to change position can lead to pressure damage of the skin. Ischemia induced by pressure is the main cause of cutaneous lesions in comatose patients.[5] Depression of respiration and general circulation in these patients also may contribute to their state of anoxia. Similar cutaneous lesions also appear on noncomatose patients who are immobilized.[5] In our case report, the patient fell into a deep sleep after alcoholic intake and remained on his right side throughout his slumber. Although we did not check his state of unconsciousness during the sleep, we think he was in a deep sleep rather than an actual coma.

Histopathologically, eccrine sweat gland necrosis has been regarded as either pathognomonic or the earliest change of the illness.[3,13] Because of this characteristic finding, many authors have titled the illness *sweat gland necrosis*. One study of the histopathology of cutaneous changes in drug-induced coma showed that the secretory portion of the eccrine sweat coil in the epithelium is most susceptible to necrosis.[1] Sweat gland secretion is mediated by the energy-dependent active transport of ions, so a continuous supply of metabolic energy is mandatory for sustained sweat secretion. Because the eccrine secretory cells are metabolically active, they seem to be highly vulnerable to the state of anoxia. In addition to sweat gland necrosis, varying degrees of eosinophilic degeneration of epidermal cells, intraepidermal and subepidermal blisters, and some polymorphs in dermis are other common findings of the illness.

In a country where hepatitis is prevalent, AST and ALT are often included in routine laboratory examinations. Considering only a history of alcohol intake and elevated AST and ALT levels, clinicians may suspect a patient has acute alcoholic hepatitis. Although both AST and ALT levels can be increased by either hepatic or muscular damage, an increase in ALT levels is more specific to the liver. When the ratio of AST to ALT is greater than 3 (in our patient it was 3.28) and the AST level is higher than 500 U/L (in our patient it was >850 U/L), muscular damage has occurred.[14] In addition to increased levels of AST and ALT, our patient had greatly increased levels of LDH and CK. Few reports on skin lesions in comatose patients showed these enzyme levels. Studying the enzyme levels of such patients would be needed to better understand a patient's overall condition.

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