

Eczema craquelé associated with nephrotic syndrome



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INTRODUCTION

Eczema craquelé, also referred to as *asteatotic eczema*, is characterized by intersecting superficial fissures of the skin that lead to a distinctive “dried river bed” or “crazy-paving” appearance. It most commonly affects the distal lower extremities of older individuals during cold winter months and develops within a background of xerosis. Occasionally, eczema craquelé is a reflection of an underlying malignancy (eg, lymphoma, leukemia, solid organ tumors),^{1–3} malnutrition (eg, anorexia nervosa),^{4,5} decreased sweat gland activity (eg, chronic graft-versus-host-disease, Sjögren’s syndrome, hypoesthetic skin),^{6–8} or the use of a systemic medication (eg, retinoids). The possibility of a secondary association should be considered when there are atypical features (eg, developing in an adolescent) or in unusual sites (eg, upper back, abdomen). In this article, we report the appearance of eczema craquelé in a young woman with anasarca caused by hypoalbuminemia and review the disorders that can have eczema craquelé as a dermatologic finding.

CASE REPORT

A 28-year-old white woman with poorly controlled insulin-dependent diabetes mellitus complicated by diabetic nephropathy and gastroparesis presented with a new-onset pruritic rash in the setting of a 3-month history of worsening edema. Physical examination found 3+ pitting edema on her abdomen and bilateral lower extremities. In these

same areas, there was eczema craquelé characterized by superficial pink fissures (Fig 1); on the lower extremities, the fissures were more prominent and were edged by white scale and peripheral erythema.

Laboratory studies were notable for a serum albumin of 1.8 g/dL (normal, 3.5–5.0) and creatinine of 1.9 mg/dL (normal, 0.5–1.2). She had nephrotic-range proteinuria, excreting 12.5 g over 24 hours. Serology findings for hepatitis B and C viruses and human immunodeficiency virus were negative; no antinuclear antibodies were detected. Thyroid-stimulating hormone was elevated at 12.2 mIU/mL (normal, 0.3–4.2) with normal triiodothyronine and mildly decreased thyroxine of 4.9 µg/dL (normal, 5.0–10.6). Computed tomographic scan of the abdomen and pelvis showed no neoplasms, lymphadenopathy, or evidence of lymphatic obstruction.

The patient was aggressively diuresed with intravenous furosemide, and her malnutrition was addressed with supplemental feedings via a percutaneous endoscopic gastrostomy. Medium-potency topical corticosteroids and petrolatum ointment were applied twice daily for approximately 3 weeks with improvement of her pruritus and eczema craquelé.

DISCUSSION

Eczema craquelé is usually observed in older individuals, especially those with xerosis who are living in a cold, low-humidity environment.² In some patients, it is caused by an overuse of soaps. There were several atypical features in this patient—her

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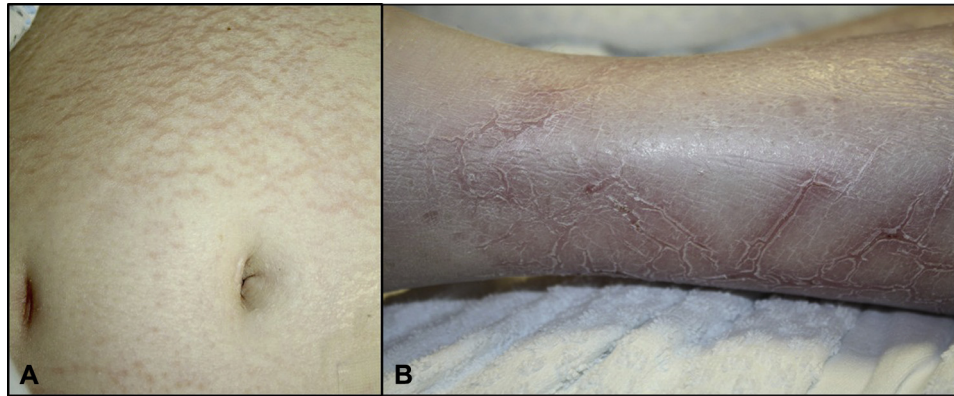


Fig 1. Pink superficial fissures of eczema craquelé on the edematous abdomen (**A**) and lower extremity (**B**). Note the background of xerosis on the leg.

Table I. The development of eczema craquelé in patients with new-onset or worsening edema

Age (y) & sex	Etiology of edema	Distribution of eczema craquelé	Serum albumin level	Study
69 F	Congestive heart failure	Dorsal forearms, hands, legs, feet	Not reported	Caplan ²
73 M	Congestive heart failure	Right leg and ankle, stump of left leg	Not reported	Caplan ²
60 M	Congestive heart failure	Lower legs and ankles	Not reported	Caplan ²
24 M	Anaplastic carcinoma, origin unknown	Lateral legs and ankles	Not reported	Caplan ²
72 M	Unknown	Lower legs and ankles	Not reported	Caplan ²
80 M	Congestive heart failure	Lower legs	Not reported	Caplan ²
61 M	Acute myelogenous leukemia	Lower legs	Not reported	Caplan ²
75 M	Congestive heart failure	Lower legs	Not reported	Caplan ²
46 M	Chronic glomerulonephritis with nephrosis	Lower legs	Not reported	Caplan ²
55 M	Left knee swelling s/p popliteal aneurysm repair	Below left knee and along surgical scar	Not reported	Bhushan et al ⁹
42 F	Temporary disability after fractured left femur	Left ankle and dorsal foot	Not reported	Bhushan et al ⁹
67 M	Congestive heart failure exacerbation	Lower legs	Not reported	Bhushan et al ⁹
46 F	Congestive heart failure exacerbation	Lower legs	Not reported	Bhushan et al ⁹
82 M	Right ankle and foot cellulitis	Right dorsal foot and ankle	Not reported	Bhushan et al ⁹
47 F	Hepatic cirrhosis with recent reduction in diuretic dose	Abdomen, upper and lower extremities; also edema bullae	Not reported	Bhushan et al ⁹
52 M	Congestive heart failure exacerbation	Lower legs	Not reported	Bhushan et al ⁹
19 F	Several days after refeeding in patient with anorexia nervosa	Lower legs and feet; also xerosis & severe pain	2.8 g/dL*	Ishiguro et al ⁴
24 F	Several days after refeeding in patient with anorexia nervosa	Lower legs and feet; also xerosis & pain	2.4 g/dL*	Ishiguro et al ⁴
65 M	Several days after overinfusion of intravenous fluids in patient with lung cancer metastatic to the adrenal glands	Lower legs and feet	"Low"	Ishiguro et al ⁴
48 F	Anorexia nervosa with sudden bilateral lower leg swelling	Lower legs and dorsal feet	2.9 g/dL	Kishibe et al ⁵
28 F	Nephrotic syndrome (diabetic nephropathy)	Lower legs and abdomen	1.8 g/dL	Current case

Normal range for serum albumin level is 3.5–5.0 g/dL.

s/p, Status post.

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young age, onset during a humid summer month, and involvement of the abdomen. Therefore, the possibility of a secondary associated disorder was considered.

The development of eczema craquelé was previously reported in the setting of new-onset or worsening edema from congestive heart failure, hepatic cirrhosis,⁹ or the refeeding of patients with anorexia nervosa (Table I). The latter is termed *acute edema/cutaneous distension syndrome*.^{5,10} In addition, deposition of mucin, as in pretibial myxedema, has also been associated with eczema craquelé.¹¹

Our patient had significant edema caused by hypoalbuminemia from a combination of nephrotic syndrome and poor nutrition. Of note, hypoalbuminemia was described in several of the patients with anorexia nervosa who had edema and eczema craquelé. In most of the patients described in Table I, the eczema craquelé resolved with correction of the peripheral edema^{4,5,9}; the remaining died of their underlying medical conditions before resolution of their edema.^{2,3}

The questions raised by this patient and those outlined in Table I include the role distention of the skin plays in producing fractures of the stratum corneum and epidermis and the relative contribution of the rate of distention. Some authors have suggested that the rapidity of edema development plays a greater role than the actual amount of edema.^{5,9} The appearance of isolated eczema craquelé around a dermatofibroma¹² in the setting of acute leg edema suggested that decreased extensibility leading to greater dermal tension served as an inciting factor. Lastly, the possibility exists that even for the classic presentation of eczema

craquelé — older individual with xerosis — the preference for the distal lower extremities may reflect the common occurrence of edema from venous hypertension in this location and age group.

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