Waxy fingers and skin tethering

The patient sought care for a burn he hadn't felt when he'd held a hot cup of coffee. His history, and the appearance of his hands, suggested a systemic disease at work.

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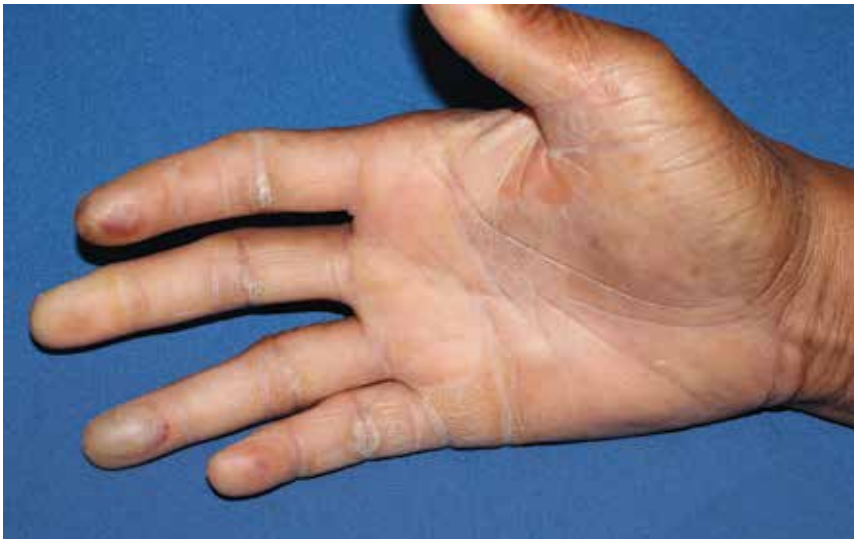
A 73-YEAR-OLD MAN with longstanding, poorly controlled type 1 diabetes (T1D) and worsening paresthesia presented to the dermatology clinic following a painless thermal burn of his fingertips from holding a hot cup of coffee. The patient's paresthesia in a stocking-and-glove distribution was attributable to diabetes-associated polyneuropathy. Two years prior, he had been diagnosed with mildly symptomatic, diabetes-associated scleredema of his upper back and treated with topical corticosteroids.

Physical examination revealed tense bullae on the pads of all 5 digits of his right hand (**FIGURE 1**). Localized, waxy tightening of the skin was noted on all digits of both hands, along with mild tethering of thickened skin on the right palm.

- WHAT IS YOUR DIAGNOSIS?
- HOW WOULD YOU TREAT THIS PATIENT?

FIGURE 1
Thermal burn of fingertips, as well as …



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Tense burn bullae attributed to holding a hot coffee cup were noted on the fingertips. The patient's skin was also firm and taut, with tethering on the central palm.

Diagnosis: Diabetic hand syndrome

Subtle, early signs of diabetic sclerodactyly and Dupuytren contracture (DC) were observed in the context of an existing diagnosis of T1D, leading to a diagnosis of diabetic hand syndrome.

Sclerodactyly, a thickening and tightening of the skin, is a characteristic component of limited and systemic sclerosis. Sclerodactyly is not commonly observed in association with type 1 and type 2 diabetes; however, when it does occur, it is typically found in patients who have had uncontrolled diabetes for some time.[1-3] (In the context of diabetes, this skin manifestation is known as *pseudoscleroderma* and *scleredema diabeticorum*.) In 1 study of 238 patients with T1D, the prevalence of this diabetes manifestation was 39%, with a range of 10% to 50% also reported.[3]

Diabetic hand syndrome is an umbrella term for the constellation of debilitating fibroproliferative sequelae of the hand rendered by diabetes.[3] In addition to diabetic sclerodactyly, diabetic hand syndrome includes limited joint mobility (LJM), or *diabetic cheiroarthropathy*, which typically manifests with either the "prayer sign" (the inability of the palms to obtain full approximation while the wrists are maximally flexed) or the "tabletop sign" (the inability of the palm to flatten completely against the surface of a table) (**FIGURE 2**).[4,5] The prevalence of LJM has been reported to range from 8% to 50% of patients diagnosed with longstanding, uncontrolled diabetes.[4]

Other musculoskeletal abnormalities seen in this syndrome include: DC, often found clinically as a palpable palmar nodule that ultimately results in a flexion contracture of the affected finger; stenosing tenosynovitis, or *trigger finger*, in which a reproducible locking phenomenon occurs on flexion of a finger, typically in the first, third, and fourth digits; and carpal tunnel syndrome, a median nerve entrapment neuropathy that results in pain and/or paresthesia over the thumb, index, middle, and lateral half of the ring fingers.[3-5]

FIGURE 2
"Tabletop sign"

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The patient's fingers were slightly flexed, and he was unable to fully contact the opposing surface. Also notable was the waxy tightening of his fingers—particularly over the distal interphalangeal joints.

Secondary symptoms can signal long-term degenerative disease

Stocking-and-glove distribution polyneuropathy with deterioration of tactile sensation is a common sequela of diabetes, especially as disease severity progresses.[2] Although the exact pathogenesis remains unclear, it has been proposed that both diabetic polyneuropathy and increased skin thickness occur secondary to long-term degenerative microvascular disease.

Specifically, prolonged hyperglycemia and secondary chronic inflammation set the stage for protein glycation, with formation of advanced glycation end products (AGEs). It is thought that these AGEs in cutaneous and connective tissues stiffen collagen, leading to scleroderma-like skin changes.[2]

These microvascular and fibroproliferative changes are also considered important contributors in the etiology of DC and trigger finger, ultimately leading to increased collagen deposition and fascial thickening.[4,5] In addition, increased activation of the polyol pathway may occur secondary to hyperglycemia, resulting in increased intracellular water and cellular edema.[5]

The differential is comprised of components of systemic disease

The differential diagnosis includes tropical diabetic hand, autoimmune-related scleroderma (also called *systemic sclerosis*), complex regional pain syndrome, and diabetic scleredema.

Tropical diabetic hand, a potentially dangerous infection, is generally found only in tropical regions and in the setting of injury.[5,6]

Autoimmune-related scleroderma may be diagnosed alongside other signs and symptoms of CREST: calcinosis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia. In the absence of other signs and symptoms, and in the presence of uncontrolled diabetes, biopsy would be needed to definitively diagnose it. Clinically, diabetic hand can be distinguished with concurrent involvement of the upper back.

Complex regional pain syndrome is characterized by chronic, disabling pain, swelling, and motor impairment that frequently affect the hand, often secondary to surgery or trauma.[5,7] This diagnosis differs from the generally painless skin hardening of diabetic hand.

The co-existence of diabetic scleredema and diabetic sclerodactyly has been previously reported, although the onset of each condition is often temporally distinct.[8] In contrast to diabetic sclerodactyly, the firm indurated skin characteristic of diabetic scleredema (which our patient had) initially involves the shoulders and neck and may progress over the trunk, including the upper back, typically sparing the distal extremities. Of note, the dermis in scleredema is thickened with marked deposition of mucopolysaccharide.[9]

Glycemic control is paramount

Studies of patients with diabetes who have thick, waxy skin and LJM have shown that tight glycemic control may reduce skin thickness and palmar fascia fibrosis.[3,5,9] Thus, in this patient with poorly controlled T1D, diabetic sclerodactyly, early DC, and second-degree burns attributable to advanced polyneuropathy, tightened glycemic control is logical and warranted. Such control could potentially impact the trajectory and morbidity of skin and musculoskeletal manifestations in this broad-reaching disease.

Although there are limited treatments for mobility-related symptoms of diabetic hand syndrome, physiotherapy is recommended in more severe stages of disease to increase joint range of motion.[4,5] More severe cases of DC and trigger finger have been successfully treated with topical steroids, corticosteroid injections, and surgery.[4,5] Simply stated—and in line with compulsive foot care—the diabetic milieu necessitates clinicians' close attention to the hands. Components of diabetic hand, LJM, DC, or trigger finger may indicate a need to screen not only for diabetes in a patient previously undiagnosed but also, importantly, for other sequelae of diabetes, including retinopathy.[4,5]

Our patient was treated with a moderate-potency topical steroid, triamcinolone 0.1% cream, and was advised to continue optimizing glycemic control with the aid of his primary care physician. It was unclear whether the patient improved with use, as he was lost to follow-up. JFP

The diabetic milieu necessitates clinicians' close attention to patients' hands.

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