

Exuberant allergic contact dermatitis leading to hand compartment syndrome



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INTRODUCTION

Contact dermatitis (CD) is a common dermatologic condition mediated by a chemical irritant or via a delayed type IV hypersensitivity reaction.¹ Complications of contact dermatitis are unusual and typically mild, including post inflammatory hyperpigmentation and secondary bacterial infections.² More serious complications are rare.

Compartment syndrome is caused by increased pressure within a myofascial compartment, leading to circulatory and functional compromise.³ Compartment syndrome of the hand may be difficult to diagnose and, if delayed, can lead to loss of function and permanent tissue damage.³ We present a striking case of CD of the hand leading to compartment syndrome and digital necrosis.

CASE REPORT

A 71-year-old female smoker with hypertension presented to the emergency department (ED) with severe right hand swelling, pain, and pruritus. These symptoms developed approximately 1 hour after gardening. She denied trauma and other notable exposures. Physical examination by dermatology and hand surgery revealed large, tense bullae geometrically distributed on the right hand and fingers, with pallor of the distal digits (Fig 1). Radial pulses were 2+, with intact sensation and motor function. At that time, a diagnosis of allergic contact dermatitis (ACD) due to an exposure while

Abbreviations used:

ACD:	allergic contact dermatitis
CD:	contact dermatitis
CTA:	computed tomography angiography
ED:	emergency department
HCS:	hand compartment syndrome

gardening was favored, and the patient was discharged with high potency topical steroid ointment.

The following day, the patient returned to the ED with worsening pain, decreased sensation, and cool fingers. Repeat dermatology examination revealed worsening of edema and pallid, cool digits with dusky discoloration and absent capillary refill (Fig 2, A). Radial pulses remained 2+. Sensation to light touch was markedly diminished. X-ray of the hand showed soft tissue edema. Bulla fluid was aspirated and sent for culture, and the patient received one dose each of vancomycin and piperacillin-tazobactam. The swelling and other findings were again attributed to ACD, and, following derroofing of bullae, the patient was discharged home on prednisone 60 mg daily and cephalexin 500 mg 3 times daily for 10 days.

The patient next presented for outpatient dermatology follow-up 14 days after the initial ED evaluation. She reported worsening burning pain and numbness of the fingertips, and examination revealed evolving necrosis of the distal digits (Fig 2, B). Given the evident vascular compromise and

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Fig 1. Initial (day 1) presentation of geometrically distributed tense bullae on the (A) dorsal and (B) ventral hand, with pallor of the fingertips.



Fig 2. Evolution of tense bullae and pallor on day 2 (A), resulting in avascularity and digital necrosis noted on day 15 (B).

significant functional implications, the patient was sent to the ED for urgent evaluation by hand surgery. Digital pulses were undetectable by doppler; computed tomography angiography showed patency of the visualized arteries. However, digital brachial indices were decreased with a normal wrist brachial index. The patient was discharged home with wound care and gabapentin for pain relief.

One week later, the patient was readmitted due to uncontrolled pain. At that time, laboratory evaluation was unrevealing for autoimmune inflammatory disease, including negative ANA, RF, and ANCA, and normal C3 and C4. Magnetic resonance imaging and magnetic resonance angiography of the right hand showed no evidence of vasculitis, inflammatory arthritis, or thromboangiitis obliterans. For relief of

symptoms, the patient was treated with botulinum toxin injections and sildenafil 20 mg daily. Given the unrevealing workup, and with improved pain, the patient was discharged home with close follow-up and eventual autoamputation of involved areas.

DISCUSSION

CD is a frequently encountered dermatologic condition that can result in decreased quality of life and occupational consequences.⁴ CD is characterized by erythema, edema, and vesiculation and often involves the hands or other exposed sites.¹ Complications, if any, are typically mild and due to sequelae of local cutaneous inflammation.² In this case, compartment syndrome and digital necrosis developed as a severe complication of CD-related edema of the hand and fingers. To our knowledge, one prior case of hand compartment syndrome (HCS) occurring as a complication of CD has been reported.⁵

The acute onset of geometric bullae, edema, and pruritus after gardening are suggestive of a diagnosis of ACD secondary to plant-derived allergens, though phytophotodermatitis may also be considered. Negative infectious, rheumatologic, and vascular imaging make other possibilities less likely. The most likely exposure is to the plant compound urushiol from *Toxicodendron radicans*, which is prevalent in the eastern United States and produces a highly allergenic reaction with up to 50 million cases yearly.² Prognosis is favorable with prolonged corticosteroids. However, in this case, a delay in the diagnosis of HCS as a consequence of ACD resulted in ischemia and necrosis of multiple digits.

HCS is a clinical diagnosis initially presenting with tense edema and pain out of proportion to examination, exacerbated by passive stretching.⁶ Some suggest bullous edema should be a diagnostic criterion.^{6,7} Late findings include pulselessness, pallor, and sensory and motor deficits.⁵ There is no consensus regarding objective compartmental pressures, and the importance of careful clinical evaluation and diagnosis is emphasized. Despite pulses measurable by doppler, the early findings of severe pain with signs of distal ischemia, such as pallor, warrant consideration for urgent fasciotomy.⁶

In this case, the patient's history and initial diagnostic label of ACD, a typically benign dermatologic condition, may have resulted in framing bias among providers from dermatology and hand surgery who, perhaps, downplayed the severity of the situation and failed to consider HCS or intervene due to the expectation of a benign and skin-limited course. The patient's care was further compromised by the lack of prompt outpatient follow-up. We speculate that reassurance by providers, fatigue from multiple ED visits, and low health literacy may have contributed to the 14-day delay before the patient sought out additional care.

This case highlights the possibility of a rare but devastating complication of ACD resulting from exuberant inflammation and edema, and underscores the importance of recognizing potential early signs of compartment dysfunction in order to avoid tissue ischemia and death. It is critical to avoid framing bias by considering potential sequelae or "end-stage" complications of even the most routine or banal diagnoses.

Conflicts of interest

None disclosed.

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