Cutaneous botryomycosis diagnosed long after an arm injury

To the Editor: We report a 44-year-old Turkish man who presented with an expanding, brownish-reddish to hypopigmented, nodular, scarred skin lesion with multiple ulcerations on his right arm, primarily at the elbow and fingers (Fig 1). The fingers of the right hand showed flexion contractures, and the ulcerated nodules were located above the proximal interphalangeal joints (Fig 1). The patient reported that his skin problems were initiated by a work-related welding accident 25 years ago, wherein the lower aspect of his arm was burned by iron filings. Thereafter, the patient was seen at various dermatologic and surgical clinics. Tuberculostatic treatments were administered repeatedly because of the patient's history of lung tuberculosis 7 years before his arm injury, although tests were consistently negative for *Mycobacterium tuberculosis*. In addition, sarcoidosis, atypical mycobacteriosis, lupus vulgaris, Wegener granulomatosis, leprosy, and skin neoplasms were excluded by prior histopathological and microbiological examinations; the correct diagnosis remained unknown. Despite continuous medical care, the skin never properly healed. Treatment with alprostadil (20 μg intravenously, 20 infusions over 2 months, 3 times repeated within 6 years) temporarily improved his skin condition by decreasing the number of skin ulcers. He presented at our department approximately 13 years after his skin condition initially developed. Histopathological examination revealed the Splendore-Hoeppli phenomenon; eosinophilic material adjacent to the inciting grains was surrounded by inflammatory cells consisting of eosinophils, histiocytes, epithelioid cells, and giant cells as described by Hussein.[1] More importantly, clusters of gram-positive cocci (Fig 2), confirmed as *Staphylococcus aureus*, were observed. Fungi and acid-fast rods were not detected. A complete blood cell count showed hypochromic, normocytic anemia. Angiotensin-converting enzyme, glycosylated hemoglobin, and α1-antitrypsin levels were within normal ranges. Blood cultures produced negative results. Tuberculosis was not detected in skin samples (polymerase chain reaction) or gastric fluids or by puncture test (tine test) or enzyme-Linked Immunospot. Infectious diseases, including HIV, syphilis, and hepatitis (A, B, and C), were excluded. No signs of the following were detected: thrombophilia, cold agglutinins, cryoglobulins and cryofibrinogens, antinuclear antibodies, and neutrophil cytoplasmic antigens. The phagocytosis and chemotaxis of immune cells such as neutrophils was normal. Magnetic resonance imaging of the right arm showed inflammation of the cutis and subcutis and early signs of osteomyelitis. Chest x-rays and abdominal ultrasonography revealed normal findings. A psychosomatic examination revealed an auto-aggressive personality. Detection of *S aureus* in the skin lesions confirmed the diagnosis. The lesion had been partially maintained through the patient's self-manipulation to attain his pension claim. Clindamycin matched the antibiogram, and a fusidic acid lotion was applied topically. Iloprost

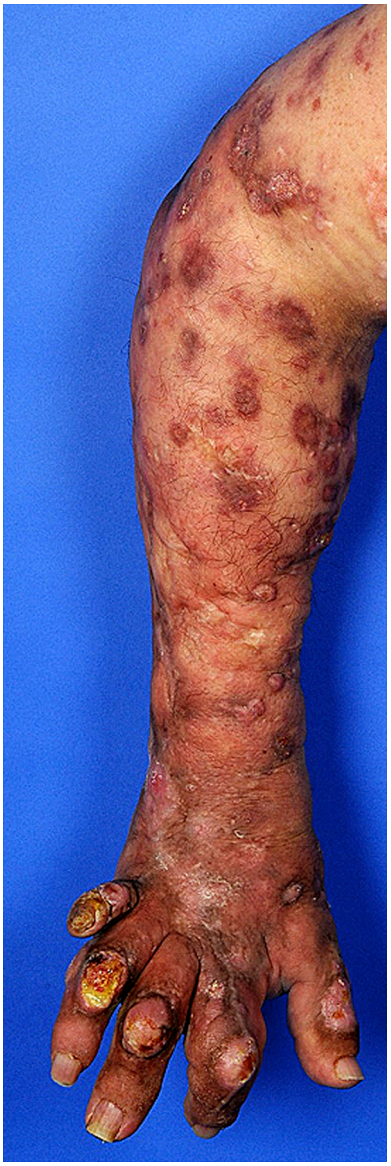

Fig 1. Cutaneous botryomycosis on the right arm. Brownish-reddish and hypopigmented, partly scarred, nodular skin lesions in the right arm with multiple ulcers, primarily at the elbow and above the proximal interphalangeal joints. The fingers of the right hand showed flexion contractures.

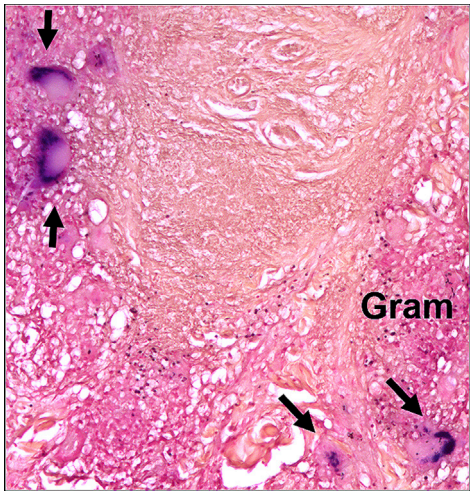

Fig 2. Histopathology of a biopsy specimen of the lower ventral aspect of the arm. Glandlike masses of gram-positive cocci (*arrows*) typical of staphylococcal botryomycosis were detected by Accustain Gram stain (Sigma, Steinheim, Germany). (Original magnification: ×100.)

(5 μg) was administered intravenously to enhance blood flow to the arm. Physiotherapy included manual lymphatic drainage of the right arm. In addition, compression bandages were applied to prevent further self-manipulation. Altogether, these treatments improved his skin condition.

Cutaneous botryomycosis is a chronic granulomatous bacterial skin infection characterized by nodules, abscesses, and ulcers on the hands, feet, genitals, and head.[2] It primarily results from the inoculation and persistent infection of injured skin[3] by bacteria such as *S aureus*[4] and *Pseudomonas aeruginosa*. T cell–related immune deficiency and diabetes can promote the disease.[2]

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Calcific elastosis in the setting of weight gain

To the Editor: A 51-year-old man presented for evaluation of stable, asymptomatic skin lesions that had been present for several years. His history was notable for Graves disease treated with thyroidectomy, and significant ensuing weight gain that predated the onset of his cutaneous findings. The patient was otherwise healthy, and his only prior medical treatment was levothyroxine. He was adopted and his family history was unknown.

Physical examination revealed an overweight man with asymptomatic hyperpigmented reticulated plaques and longitudinal striae over his bilateral flanks and legs (Fig 1). No other concerning cutaneous lesions were present. A 4-mm punch biopsy was performed of a right lower flank hyperpigmented plaque. Hematoxylin-eosin and elastic stains showed fragmented, mineralized elastic fibers in the reticular dermis. Elastic fibers with calcium deposition were present on von Kossa stain (Fig 2).

The patient's histologic examination, lesional morphology, and lack of systemic symptoms suggested a diagnosis of acquired calcific elastosis in the setting of weight gain. The differential diagnosis also included pseudoxanthoma elasticum (PXE), a rare heritable disorder associated with defects of the ABCC6 gene, causing progressive mineralization of elastic fibers and complications of the skin, eye, and cardiovascular system.[1,2] Cutaneous lesions may be the presenting sign; however, 85% of patients with inherited PXE demonstrate angioid streaks, the characteristic retinal findings resulting from breaks in Bruch's elastic membrane.[3] Vascular involvement is also common, manifesting as hemorrhage, intermittent claudication, and/or hypertension.

PXE-like features have been reported in calciphylaxis and inflammatory conditions, including lipodermatosclerosis, granuloma annulare, lichen sclerosis, morphea profunda, erythema nodosum, septal panniculitis, and nephrogenic systemic fibrosis. More broadly, 4 major types of cutaneous calcification exist—dystrophic, metastatic, idiopathic, and