(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. It primarily results from the inoculation and persistent infection of injured skin by bacteria such as *S. aureus* and *Pseudomonas aeruginosa*. T cell–related immune deficiency and diabetes can promote the disease.

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. 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. 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...
iatrogenic—according to the original etiology of the symptoms. Dystrophic calcinosis cutis, including that of PXE and calcific elastosis, occurs in damaged or inflamed skin secondary to mechanical, chemical, or other factors; serum calcium and phosphate remain within normal limits in such cases.4 Localized calcific elastosis, although histologically indistinguishable from PXE, is a distinct, often skin-limited entity.2 It is most frequently described as lax, reticulated, or cobble-stoned plaques often in the periumbilical region of obese multiparous women. Mechanical stress, such as that of pregnancy and/or surgery, with subsequent elastic fiber degeneration is thought to produce localized disease.2,5 Calcific elastosis has been reported in the setting of ascites, anasarca, chronic renal failure, and tumefactive lipedema.7,5 Certain chemicals, such as Norwegian saltpeter (calcium-ammonium-nitrate salts) fertilizer and penicillamine have also been associated with calcific elastosis.

To definitively rule out PXE, the patient underwent ophthalmologic evaluation, cardiovascular consultation, and routine health screening. Molecular analysis of ABCC6 was not performed in this case, in that these clinical evaluations were all within normal limits.6 Localized calcific elastosis has not been previously described secondary to weight gain alone. As evidenced by our findings, when confronted with cutaneous elastic tissue abnormalities, it is important that clinicians consider and pursue a comprehensive diagnostic workup to rule out underlying systemic involvement when appropriate for long-term clinical management and counseling of affected patients.

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REFERENCES
Atypical scleromyxedema with prominent nodular lesions associated with immune thrombocytopenia: An unusual presentation

To the Editor: Scleromyxedema (SM) is a rare cutaneous mucinosis that usually occurs with monoclonal gammopathy (>83.2%), predominantly an IgG lambda subtype.1,2 SM may show a great variety of extracutaneous manifestations (gastrointestinal, musculoskeletal, neurologic, pulmonary, cardiac, and renal involvement) leading to significant morbidity and mortality.1,2 We report a case of SM with prominent scalp involvement, presence of nodular lesions, and immune thrombocytopenia.

A 69-year-old woman presented with a 2-week history of facial edema and progressive eruption. The patient also reported severe scalp pruritus and increased hair loss.

On physical examination, skin-colored firm papules, nodules, and edema of the face and both hands were observed. The papules were initially located on the scalp, neck, and back of the auricular area, then gradually involved upper aspect of the trunk and the surrounding skin showed scleroderma-like induration in these areas. Several nodules, 4 to 10 mm in size, were present on the scalp, forehead (Fig 1), and side portions of the chin.

Pathological examination of biopsy specimens from a nodular scalp lesion and of a neck papule (Fig 2) revealed an increase in fibroblasts, collagen, and deposits of mucin in the papillary and mid-dermal dermis. Laboratory analysis showed a progressive low platelet count (60-10^10/L, normal range 150–400 x10^10/L, platelet count within normal limits 6 months earlier), with a normal peripheral blood smear result and a normocellular bone marrow with trilineage hematopoiesis. Serum protein electrophoresis, immunofixation electrophoresis of serum and urine, and immunoglobulin free light chain assays did not show paraproteinemia. Other laboratory examination findings including thyroid function were within normal limits and autoantibody screening produced negative results. Computed tomographic scans of the chest, abdomen, and pelvis showed no relevant abnormalities. Electromyography detected signs consistent with a right carpal tunnel syndrome and peripheral sensory and motor neuropathy. Based on clinical manifestations, and histopathological and laboratory data, the diagnosis of SM with a secondary immune thrombocytopenia was made. The patient was treated with 3 methylprednisolone pulses of 1 g, followed by 1 mg/kg/d of prednisone for 1 month, and intravenous immunoglobulins (2 g/kg per cycle). Administration of a second course of intravenous immunoglobulins, delivered after 4 weeks, dramatically improved skin manifestations; however persistence of low platelet count prompted the addition of romiplostim, a thrombopoietin-receptor agonist. The prescribed corticosteroids were gradually tapered off and romiplostim was withdrawn. After 12 months of follow-up, she remains asymptomatic and platelet count was within normal limits

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