nated glove (4H) appears relatively impermeable to MMA, but widespread use is limited by cost and diminished dexterity.²,³

Allergic contact dermatitis induced by MMA may result in significant discomfort and is seen frequently after occupational exposure among dentists, dental technicians, orthopedic surgeons, and other health care workers. Second- and third-digit fingertips are commonly involved.³ Nail dystrophy and fingertip paresthesias have also been reported.⁴ Severe cases of MMA-induced dermatitis can even necessitate profession changes.³

Purpuric contact dermatitis has been reported in patients sensitized to agents such as textile dyes, formaldehyde, and epoxy resins.⁵ We report herein the second case, to our knowledge, of MMA-induced PCD.⁶ Although patch testing is considered the diagnostic gold standard for suspected allergic contact dermatitis, this patient’s strong contact history and rapid, sustained resolution of symptoms with allergen avoidance allowed for clinical diagnosis. Fingertip purpura may appear alarming to unsuspecting clinicians and may prompt an extensive workup. We encourage physicians to consider PCD in patients with potential contact exposures.

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Elastosis Perforans Serpiginosa: A Case of a Penicillamine-Induced Degenerative Dermatosis

Report of a Case | A man in his 60s presented in reduced general condition and with asymptomatic brownish-red papules organized in multiple arcuate to annular formations on his upper trunk and arms (Figure 1). A prominent cutis laxa and cutis rhomboidalis nuchae were noted. At the time of presentation, he had been treated for Wilson disease with daily doses of D-penicillamine (1.0-1.5 g/d) for more than 40 years.

Histopathologic analysis revealed channels through the epidermis formed by follicular epithelium (Figure 2A). The infundibula were filled with granular cellular debris, neutrophils, and corneocytes (Figure 2A and C). The interfollicular tissue showed a mixed inflammatory infiltrate. Elastic van Gieson staining demonstrated an accumulation of altered elastic fibers (Figure 2B) within the upper part of the dermis.

From the clinical and histopathologic findings, the diagnosis of elastosis perforans serpiginosa (EPS), caused by long-term ingestion of D-penicillamine, was made.
Wilson disease is a rare autosomal recessive condition caused by a genetic defect in the copper-transporting ATPase ATP7B. Copper accumulation in the liver and the basal ganglia of the brain may lead to hepatocerebral degeneration. In our patient, liver transaminase levels were slightly elevated, and neurologic symptoms included ataxia and rigid dystonia.

An effective treatment for Wilson disease is D-penicillamine, a chelating agent that depletes copper. In addition, successful treatment has also been reported with trientine dihydrochloride, zinc, tetrathiomolybdate, and liver transplantation. Long-term D-penicillamine therapy can induce EPS by reducing the activity of lysyl-oxidase, a copper-dependent enzyme that cross-links dermal elastic fibers, and by formation of complexes with precursors of elastic fibers, thus impairing their maturation. As a result, abnormal elastin aggregates promote a foreign body reaction with subsequent transspidermal elimination.

Clinically, EPS lesions present with multiple keratotic papules arranged in arcuate or circinate patterns. Lesions are typically found on the neck and upper extremities. Much less frequently cutis laxa has been described as an adverse effect of D-penicillamine. Morphologic changes of the elastic fibers of arteries and pulmonary tissue are a matter of concern. In our patient, the findings of chest and cardiovascular examinations were unremarkable.

Histopathologically, an acanthotic, hyperkeratotic epidermis with a mixed dermal inflammatory infiltrate with few giant cells is present in EPS. Epidermal invaginations with keratotic plugs at the surface form perforating channels filled with basophilic material. Atrophy of the skin may be found at the center of lesions. Elastic fiber stain shows a thickened and coarse morphology giving rise to the typical “lumpy-bumpy” picture.

Our patient had been taking D-penicillamine for more than 40 years, well above the average 10-year interval after which EPS may be acquired. For patients with cutaneous or systemic D-penicillamine adverse effects, an alternative copper-chelating agent like trientine dihydrochloride may be used. To our knowledge, no elastolytic dermatoses have been associated with trientine dihydrochloride therapy. Therefore, we recommended that the patient shift oral treatment from D-penicillamine to trientine dihydrochloride. 

**Discussion**

A, Overview, with black frame enclosing the affected area shown in panel B; the abnormal posture of the patient is a symptom of rigid dystonia. B, Close-up image of the framed area from panel A showing prominent cutis laxa and annular confluent patches with a central skin atrophy and a peripheral rim of red papules covered by white-yellow scales (scale bars indicate centimeters). Sun-exposed nuchal skin showed increased elastosis with severe cutis laxa and cutis rhomboidalis nuchae.

**Figure 1. Clinical Images of D-Penicillamine–Induced Elastosis Perforans Serpiginosa**

**Figure 2. Histopathologic Images of D-Penicillamine–Induced Elastosis Perforans Serpiginosa**

A, Transepidermal/follicular channels through the surface epithelium that contain basophilic necrotic material (hematoxylin-eosin, original magnification ×40). B, Elastic van Gieson–stained sections showing accumulation of abnormal elastic fibers in the upper part of the dermis; inset shows “lumpy-bumpy” fibers indicating induction by penicillamine (original magnifications ×40 for main panel, ×400 for inset). C, Suppurative folliculitis with transinfundibular elimination of altered elastic fibers (hematoxylin-eosin, original magnification ×100).
entine dihydrochloride in this interdisciplinary case. For the remaining lesions, we offered liquid nitrogen cryotherapy or ablative laser resurfacing to the patient. Unfortunately, the patient was subsequently lost to follow-up.

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Cutaneous Rosai-Dorfman Disease Successfully Treated With Low-Dose Methotrexate

In 1969, Rosai and Dorfman1 first described a series of patients with sinus histiocytosis with massive lymphadenopathy, characterized by histiocytic infiltration of lymph nodes and tissue. To our knowledge, as of 2006, there were a total of 86 reported cases of cutaneous Rosai-Dorfman disease (CRDD) in the literature,2-3 with several additional cases reported since then.

Report of a Case | An African American woman in her 50s reported a sudden eruption of dozens of facial papules and nodules 3 months previously and a 7-month history of a groin plaque. She had been treated with cefadroxil, topical clobetasol, oral prednisone, and a short course of oral isotretinoin without significant benefit.

Physical examination revealed approximately 75 pink-domed papules on the cheeks, upper lip, and chin (Figure 1) and dozens of red-brown papules becoming confluent on the right inguinal and suprapubic skin. Laboratory examination revealed a mildly elevated erythrocyte sedimentation rate (32 mm/h) and total triglyceride levels (145 mg/dL). Findings of complete and differential blood cell counts, a comprehensive metabolic panel, serum protein electrophoresis, and a light chains assay were within normal limits. Complete computed tomography of the chest, abdomen, and pelvis revealed no significant retroperitoneal, mesenteric, or pelvic lymphadenopathy. Biopsies from the groin revealed a dense infiltrate of lymphocytes and large histiocytes with abundant pale cytoplasm (Figure 2). The histiocytes showed emperipolesis of lymphocytes and occasionally red blood cells. The histiocytes seen in CRDD stain positively for macrophage marker CD68. The histopathologic differential diagnosis also includes Langerhans cell histiocytosis. While S-100 may stain positively in both CRDD and Langerhans cell histiocytosis, findings of CD1a staining are characteristically negative in CRDD. In our case, cells did not stain with melanocyte markers Melan-A or HMB45 or with cytokeratin AE1/AE or CD34 as in epithelioid sarcoma. Findings of acid-fast bacilli and Grocott methenamine silver stainings were negative.

The patient’s prednisone dose was tapered, and she began treatment with oral methotrexate, 15 mg once weekly, and significant improvement was noted over the next 11 months. Subsequently, the methotrexate dose was tapered to 5 mg.

Figure 1. Clinical Images Showing Gradual Improvement in Facial Papules