ACQUIRED ELASTOTIC HEMANGIOMA
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A 57-year-old woman presented with multiple, asymptomatic, erythematous plaques on her arms bilaterally. She denied prior trauma to the areas and noted onset of the first plaque correlated with initiating progesterone therapy. Lesions slowly became more numerous over five years. Her medical history was notable for hypertension and rosacea. Family history was unremarkable. Physical examination revealed seven erythematous, well-defined, non-tender, slightly elevated, non-blanching plaques on her arms bilaterally (Figs. 1, 2). The lesions ranged from 0.5-3.0cm with the largest on the right lower forearm.

Initial differential diagnosis included Kaposi’s sarcoma, targetoid hemosiderin hemangioma, and purpura annularis telangiectodes. A punch biopsy performed for histologic examination with hematoxylin and eosin (H&E) staining revealed solar elastosis in the epidermis with thin walled vessels in the upper dermis (Fig. 3). No cytologic atypia or mitotic figures were seen. Inflammation was absent. Despite multiplicity of lesions, a diagnosis of acquired elastotic hemangioma was favored. Due to the benign nature, no further treatment was warranted. Following discontinuation of progesterone therapy, new lesions stopped occurring and some lesions showed mild regression.

Discussion
Acquired elastotic hemangioma was first described in 2002 as a clinicopathologic variant of an acquired hemangioma. It classically occurs in middle aged or elderly women, however, one report showed a slight male predominance. Acquired elastotic hemangioma presents as an irregularly shaped, well defined, non-blanching, erythematous to violaceous plaque. Generally the lesions are asymptomatic, but are occasionally painful. The lesion is usually solitary and slow growing. The plaques have a predilection for sun damaged skin and are most commonly seen on the dorsal aspect of the forearms, but may be found on the lower lip, shoulder, nose, and neck. Clinically the lesion may be confused with a superficial basal cell or Bowen’s disease.

Histologically, the classic finding is a band-like proliferation of capillary blood vessels arranged parallel to the epidermis and confined to the superficial dermis. A zone of non-involved papillary dermis separates the capillaries from the epidermis. The epidermis is unremarkable or atrophic. Solar elastosis is present surrounding the capillaries. Mitotic figures, cellular atypia, spindle cell proliferation, red cell extravasation, hemosiderin deposition, and fibrosis is not seen. Scant lymphocytic infiltrate may be present, but not typically.

Immunohistochemically, the endothelial cells strongly express CD31 and CD34. Alpha smooth muscle actin-positive (SMA) pericytes surround the vascular channels. Acquired elastotic hemangioma was initially thought to be a true vascular tumor, however research has recently proposed a lymphatic origin after noting expression of D2-40. Immunohistochemically, the endothelial cells strongly express CD31 and CD34. Alpha smooth muscle actin-positive (SMA) pericytes surround the vascular channels. Acquired elastotic hemangioma was initially thought to be a true vascular tumor, however research has recently proposed a lymphatic origin after noting expression of D2-40. Proliferating markers Ki-67 and MPM2 stain only a few nuclei of the endothelial cells of the vessels.

The histopathological differential diagnosis includes Kaposi’s sarcoma (KS, patch stage), acquired tufted angioma, and targetoid hemosiderotic hemangioma. KS histologically exhibits jagged, vascular spaces lined by thin endothelial cells with a lymphoplasmacytic infiltrate. The promontory sign, thin walled vessels surrounding preexisting capillaries and adnexal structures, is a characteristic finding for KS. An acquired tufted angioma shows a “cannon-ball” histopathologic pattern with multiple lobules of capillary tufts scattered in the dermis and subcutaneous fat. A targetoid hemosiderotic hemangioma displays dilated vascular spaces in the superficial dermis, lined by prominent (Continued on page 29)
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hobnail endothelial cells and anastomosing collagen bundles.¹
None of these entities show band-like capillaries arranged along
the superficial dermis with solar elastosis characteristically seen
in an acquired elastotic hemangioma.

The etiology is not completely understood, but the finding
of solar elastosis supports the role of long-term sun exposure.
In most cases, there is no history of previous trauma. Since the
lesions in our case occurred following progesterone therapy,
the question arises of hormonal influence in developing
acquired elastotic hemangiomas. This possible correlation has
not been described by previously published reports.

Acquired elastotic hemangiomas are benign, asymptomatic
plaques seen on sun damaged skin. Treatment is unnec
essary, but excision of solitary lesions has been successful
without local recurrence.¹ Our case of seven lesions, arising
following initiation of progesterone, makes this acquired
elastotic hemangioma presentation atypical and unique.

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4. Tong PL, Beer TW. Acquired elastotic hemangioma: ten cases with immunohistochemistry ruling a lymphatic origin in most lesions.

CSF RECOGNIZES TOP RESIDENT PRESENTATIONS

The top resident presenters at Cosmetic Surgery Forum 2015
were:
• Daniel Belkin: Atypical Fibroxanthoma
• Amy Chero: A Newborn with Kaposiform
  Hemangioendothelioma Causing Severe Aesthetic and
  Functional Deficits Treated With Sirolimus and Vincristine
• Sanja Galeb: Medical Spas
• Annie Genois: Polyarteritis Nodosa
• Morgan Godin: Photography
• Jolene Jewell: Blepharoaplasty Associated with M. Chelonae
• Dennis Kim: Junctional Nevus Secondary to Afamelanotide
• Ravi Anik: Juvenile Xanthogranuloma After Cosmetic
  Removal of a Cutaneous Growth of the Eyelid
• Alexis Stephens: Oral Polypodium Leucotomos
• Wesley Wu: Buried Half-Horizontal, Half-Vertical
  Mattress Suture