Acquired Elastotic Hemangioma: A Case Report of Multiple Lesions Following Progesterone Therapy

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INTRODUCTION

Acquired elastotic hemangioma is a relatively newly described cutaneous lesion that presents as an erythematous, well defined, asymptomatic plaque on sun damaged skin of upper extremities. Characteristically it is described as a slow growing, solitary lesion, without history of trauma. We report a unique case of acquired elastotic hemangioma in which the patient presented with multiple lesions following initiation of progesterone therapy

CASE REPORT

A 57-year-old woman presented for evaluation of multiple, asymptomatic, erythematous plaques on her arms bilaterally. The patient denied prior trauma to the areas. She noted the onset of the first plaque correlated with initiating progesterone therapy. The lesions slowly became more numerous over five years. Her medical history was notable for hypertension and rosacea. Her family history was unremarkable.

Physical examination revealed seven erythematous, well-defined, nontender, slightly elevated, nonblanching plaques on her arms bilaterally (Figures 1 and 2). The lesions ranged in size from 0.5 cm to -3 cm, with the largest lesion on the right lower forearm.

Based on the clinical presentation and history, our initial differential diagnosis included Kaposi's sarcoma, targetoid hemosiderin hemangioma, and pupura annularis telangiectoides. A punch biopsy was performed for histologic examination with hematoxylin and eosin (H and E) staining. The biopsy revealed solar elastosis in the epidermis with thin walled vessels in the upper dermis (Figure 3). No cytologic atypia or mitotic figures were seen. Inflammation was absent. Despite the 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.

FIGURE 1

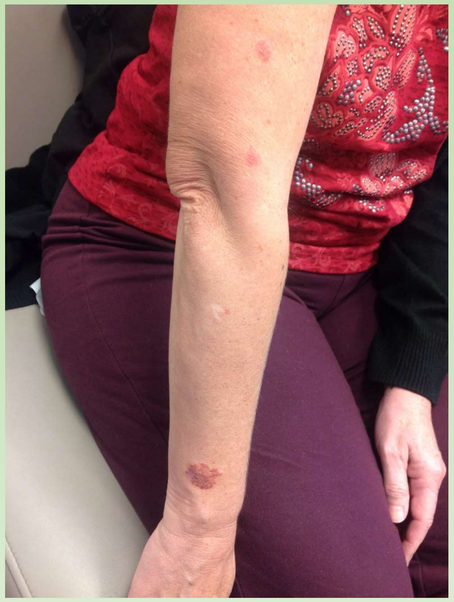


FIGURE 2

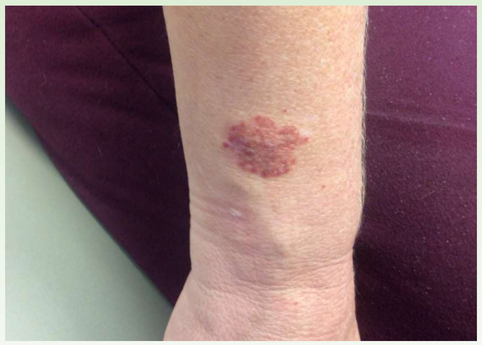
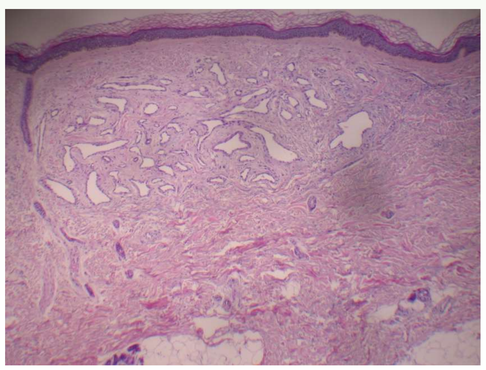


FIGURE 3



DISCUSSION

Acquired elastotic hemangioma was first described in 2002 as a clinicopathologic variant of an acquired hemangioma.[1] This lesion classically occurs in middle aged or elderly women, however, one report showed a slight male predominance.[2] An acquired elastotic hemangioma presents as an irregularly shaped, well defined, non-blanching, erythematous to violaceous plaque. Generally the lesions are asymptomatic, but occasionally they can be painful.[1] The lesion is usually solitary and very 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 also be found on the lower lip, shoulder, nose, and neck.[2] Clinically the lesion may be confused with a superficial basal cell or Bowen's disease.[1]

Histologically, an acquired elastotic hemangioma shows several characteristic features. The classic finding is a band-like proliferation of capillary blood vessels arranged parallel to the epidermis and confined to the superficial dermis. [3] 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.[4] Scant lymphocytic infiltrate may be present, but is typically absent.[1,2]

Immunohistochemically, the endothelial cells strongly express CD31 and CD34.[1,2,4] 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.[2] Proliferating markers Ki-67 and MPM2 stain only a few nuclei of the endothelial cells of the vessels.[1,4]

The histopathological differential diagnosis includes Kaposi's sarcoma (patch stage), acquired tufted angioma, and targetoid hemosiderotic hemangioma. Kaposi's sarcoma histologically exhibits jagged, vascular spaces lined by thin endothelial cells with a lymphoplasmacytic infiltrate, and red blood cell extravasation. The promontory sign, thin walled vessels surrounding preexisting capillaries and adnexal

DISCUSSION CONTINUED

structures, is a characteristic finding for Kaposi's sarcoma. An acquired tufted angioma shows a "cannon-ball" histopathological 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 hobnail endothelial cells and anastomosing collagen bundles with hemosiderin deposits.[1] None of these entities show band-like capillaries arranged along the superficial dermis with solar elastosis characteristically seen in acquired elastotic hemangioma.

The etiology is not completely understood, but the finding of solar elastosis supports the role of long-term sun exposure as an inciting cause. 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 on elastotic hemangiomas, however estrogen has been reported as an inciting factor for targetoid hemosiderotic hemangiomas.[6]

CONCLUSION

Acquired elastotic hemangiomas are benign, asymptomatic plaques seen on sun damaged skin. Treatment is unnecessary, 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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