To the Editor:

Since the publication of the article by Darling et al. [1] entitled “Oral Elastofibromatous Lesions: A Review and Case Series”, an additional case of this apparently rare oral lesion was submitted to our biopsy service. The patient was a 62-year-old male with an asymptomatic, exophytic, smooth-surfaced, pink nodule, 3 mm in diameter, of the left hard palate mucosa. There was no specific history of trauma to the area. The lesion was surgically excised and submitted for microscopic examination with a clinical diagnosis of “fibroma”. Microscopically, on routine hematoxylin and eosin (H&E) stained tissue sections, the lesion consisted of a sub-epithelial, finely fibrillar and granular, eosinophilic, sparsely cellular connective tissue matrix lacking the typical morphology and staining quality of collagen (Fig. 1). The matrix appeared to abut directly on the basement membrane of the overlying epithelium in some areas, and in the deeper adjacent fibrous tissue, perivascular deposits of this material could be seen. (Fig. 1 inset) A Masson’s elastic trichrome (MET) stain confirmed that the lesion was composed predominantly of elastic fibers (Fig. 2), resulting in a diagnosis of elastofibroma. The perivascular distribution was easily demonstrated by this stain. (Fig. 2 inset).

Our experience suggests that elastofibromas are more common orally than has been previously appreciated. Most are likely diagnosed as fibromas, based on the H&E appearance only and supported by the usual clinical diagnosis of fibroma or fibro-epithelial polyp. Microscopic clues to alert the pathologist of a possible elastofibroma at low magnification include the relatively homogenous, sparsely cellular matrix of the lesional tissue which extends to the immediate sub-epithelial zone, occasional “petaloid” fibre structure and the lack of typical collagen bundles. At higher magnification, clues include the granular and finely fibrillar matrix resembling solar elastosis, and the deeper perivascular distribution of the elastic tissue.

The condition appears to be a defect in perivascular connective tissue matrix with progressive expansion in the subepithelial zone, eventually forming a clinical mass. These observations raise the following question: could this represent some form of focal microangiopathy associated with smooth muscle-like activity of myofibroblasts, possibly in an area of mild trauma? Smooth muscle cells of elastic arteries replace elastic fibers efficiently, while subepithelial fibroblasts have difficulty degrading and turning over elastin, resulting in accumulation of defective elastin in such conditions as solar elastosis. In elastofibroma, there...
appears to be both an increase in perivascular elastic fiber production and a decrease in the ability to degrade damaged elastic fibers.

Reference


Fig. 1 A more homogenous subepithelial zone appears morphologically distinct from the collagenous tissue of the deeper connective tissue. In many areas, the homogenous material abuts directly on the overlying epithelium. (H&E, objective lens ×5). *Inset:* the finely fibrillar and granular material can be seen in a perivascular distribution even in the deeper collagenous connective tissue. (H&E, objective lens ×40)

Fig. 2 Masson’s elastic trichrome (MET) stain shows the lesional tissue to be elastic fibers. (MET, objective lens ×10). *Inset:* MET stain clearly shows the perivascular distribution of the elastic fibers in the deeper connective tissue, suggesting a microangiopathy as a possible pathogenesis of the lesion. (MET, objective lens ×20)