

# Linear focal elastosis associated with exercise



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**Key words:** connective tissue disease; elastin; elastogenesis; elastotic striae; linear focal elastosis; pseudoanthoma elasticum; striae distensae.

## INTRODUCTION

Linear focal elastosis (also known as *elastotic striae*) is a rare, acquired disorder of increased elastic tissue in the skin. Initially believed to be limited to elderly white men, this condition has since been reported in women, children, and patients from a variety of ethnic backgrounds.<sup>1,2</sup> The possibility of inherited susceptibility has been suggested by cases with positive familial associations.<sup>2</sup> However, little is known about the pathogenesis of this condition or the events that trigger its development. Here, we present the interesting case of an elderly man in whom linear focal elastosis developed on the mid-upper and lower back 1 year after beginning a rigorous, indoor swimming exercise program.

## CASE REPORT

A white man in his 60s was referred to our dermatology clinic for the evaluation of new, progressive, linear lesions on his back. He stated that the skin lesions began 1 year after starting a rigorous, indoor swimming exercise program (ie, 75-minute routine, 30 to 35 laps × 25-meter pool length, primarily freestyle stroke, 5 days per week). The lesions were asymptomatic and first appeared on his mid-upper back followed by his lower back. He denied a history of endocrine disorders, recent weight changes, use of oral or topical steroids, or preceding skin changes such as a rash or striae. His medical history was remarkable for hyperlipidemia and benign prostatic hypertrophy. His medications included atorvastatin and aspirin, both of which he had been taking for more than 10 years at the time of presentation.

### Abbreviation used:

PXE: pseudoanthoma elasticum

On physical examination, the patient had unusual yellow, horizontally layered, symmetric, linear papules and cords on the mid-upper and lower back (Fig 1). No similar lesions were noted in the intertriginous areas. Results of a complete blood count, complete metabolic panel, lactate dehydrogenase, immunofixation electrophoresis, and lipid panel were all within normal limits. Punch biopsies of the linear cords were performed and sent for histologic examination. Routine hematoxylin-eosin staining revealed a normal-appearing epidermis with increased elastic fibers throughout the dermis (Fig 2, A). There was an increase in intact and fragmented elastic fibers in the papillary dermis with some aggregation of elastic fibers seen with a Verhoeff–Van Gieson stain (Fig 2, B and C). von Kossa staining was negative for calcified elastic fibers, confirming the diagnosis of linear focal elastosis. No additional testing was performed, and the patient had no new skin lesions at a routine skin check 4 months later.

## DISCUSSION

Originally described by Burket et al in 1989,<sup>3</sup> linear focal elastosis presents as linear, symmetric, palpable, yellow papules and cords arranged in a layered fashion on the mid-upper and lower back. Similar lesions have also been described on the extremities and face.<sup>4,5</sup> The diagnosis is frequently

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Funding sources: None.

Conflicts of interest: None declared.

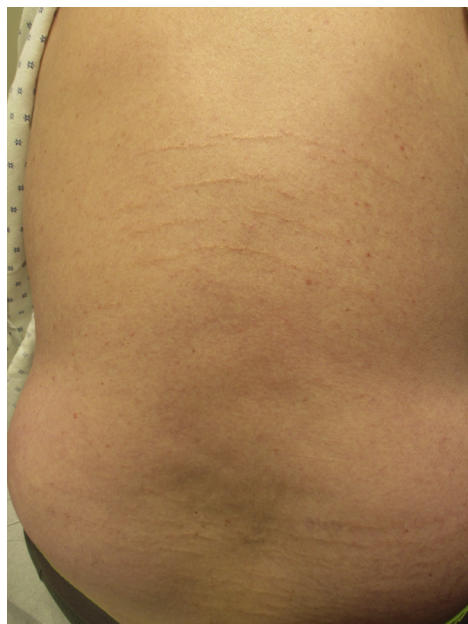
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JAAD Case Reports 2017;3:39-41.

2352-5126

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<http://dx.doi.org/10.1016/j.jidcr.2016.10.012>



**Fig 1.** Linear focal elastosis. Horizontally layered, symmetric, linear papules and cords on the mid-upper and lower back.

incidental given that the skin lesions are asymptomatic. No known risk factors or disease associations have been identified, and treatment of linear focal elastosis is unnecessary.

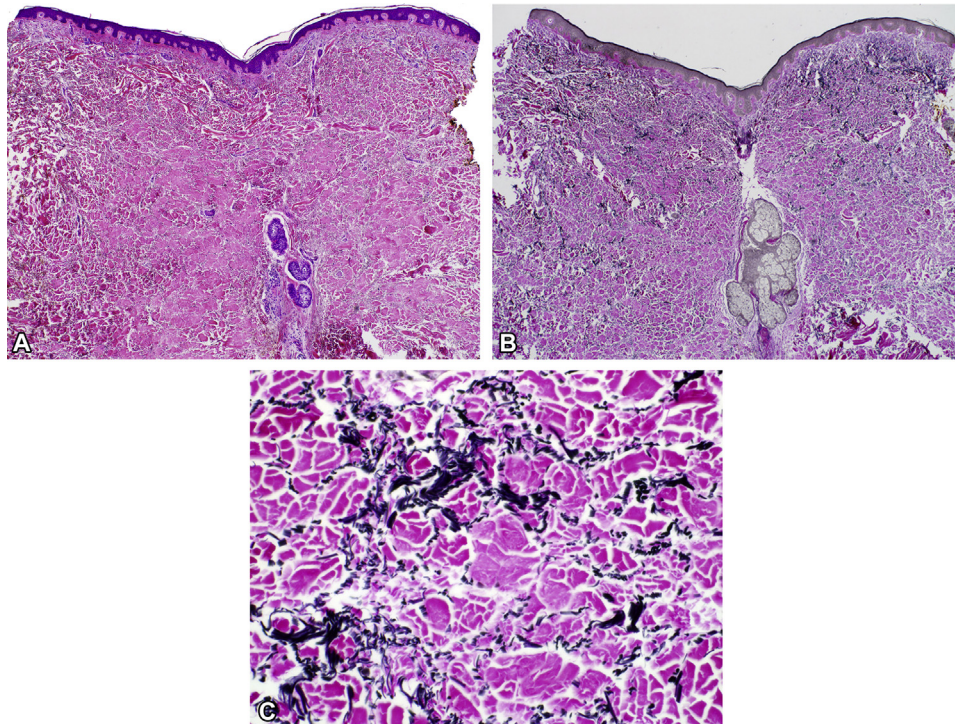
The differential diagnosis for linear focal elastosis includes striae distensae, solar elastosis, pseudoxanthoma elasticum (PXE), elastosis perforans serpiginosa, elastomas, dermatofibrosis lenticularis disseminata with osteopoikilosis (Buschke-Ollendorff syndrome), linear xanthomas, and mid-dermal elastolysis. In contrast to linear focal elastosis, the skin lesions of striae distensae are often depressed or atrophic. Additionally, patients with linear focal elastosis typically lack risk factors commonly associated with striae distensae (eg, Cushing syndrome, corticosteroid use, or recent weight changes). Elastoderma is associated with localized skin laxity (ie, increased elastic tissue), whereas mid-dermal elastolysis presents as fine wrinkling of the skin (ie, loss of elastic tissue). Buschke-Ollendorff syndrome and PXE have associated abnormalities, including osteopoikilosis and calcification of elastic tissues in other organ systems, respectively. A von Kossa stain is also helpful in distinguishing linear focal elastosis from the PXE, which contains an increased number of calcified elastic fibers.

The diagnosis of linear focal elastosis can be made clinically; however, it is confirmed microscopically. A punch biopsy of characteristic skin lesions finds a normal-appearing epidermis with abundant dermal

deposits of abnormal, wavy, pale-staining elastic fibers with feathered ends.<sup>3</sup> However, these histologic findings are unlikely to be recognized on routine hematoxylin-eosin staining unless the clinician has a high suspicion for linear focal elastosis or the dermatopathologist is instructed to perform staining for elastic fibers. Verhoeff–Van Gieson and Orcein-Giemsa stains are commonly used for better visualization of elastic fibers. Immunofluorescence of lesional skin for markers of elastin and elastin-related proteins are decreased or absent.<sup>4</sup> Electron microscopy of the elastic fibers show elongated or fragmented, mature and immature microfibrils providing evidence for active elastogenesis in skin lesions.<sup>6</sup>

The pathophysiology of linear focal elastosis is not entirely clear. However, several clinical and microscopic findings suggest that linear focal elastosis and striae distensae may represent different stages of the same process. First, electron microscopy of skin lesions in striae distensae finds thin elastic fibers, whereas older lesions primarily contain thick elastic fibers similar to those observed in linear focal elastosis.<sup>7</sup> Second, multiple cases have been described wherein striae distensae skin lesions subsequently develop into the indurated, palpable lesions characteristic of linear focal elastosis.<sup>8,9</sup> These findings suggest that linear focal elastosis may, therefore, represent an excessive regenerative or keloid-like repair of preceding striae distensae skin lesions. However, linear focal elastosis is not always seen in association with striae distensae leading others to conclude that the two processes are independent.

The temporal relationship between the development of linear focal elastosis and a new, rigorous exercise routine is curious, especially given our patient's lack of preceding skin changes or typical risk factors for striae distensae. One possible explanation for this association is the idea that mechanical forces or stretching of the skin during rigorous exercise or a rapid growth spurt, as previously described,<sup>10</sup> are sufficient to initiate the events that lead to the excessive regenerative process observed in linear focal elastosis. The linear configuration of lesions and their parallel arrangement with Langer lines is also suggestive of this mechanochemical hypothesis. Further studies exploring the relationship between the skin, external forces, and elastogenesis are warranted. These studies could shed light on the pathophysiology of linear focal elastosis or its relationship with striae distensae.



**Fig 2.** Microscopic findings. **A**, Routine hematoxylin-eosin stain; original magnification,  $\times 40$ . **B** and **C**, Verhoeff–Van Gieson staining. (Original magnifications: **B**,  $\times 40$  and **C**,  $\times 200$ .)

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