

Two Cases of Linear Focal Elastosis

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We report two cases of linear focal elastosis. They are a 16-year-old girl and a 14-year-old boy who had had asymptomatic, several, yellow-red, slightly elevated, palpable, striae-like lesions on their middle and lower back since their early childhood. Light microscopic examinations of skin biopsy specimens demonstrated dermal thickening and focal increases of thin wavy fibers in the upper and mid-dermis but no changes in the epidermis. Verhoeff-van Gieson stains for elastic fiber revealed aggregated, clumped, curled, or fragmented elastic fibers.

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Key Words: Linear focal elastosis

Since the first report of linear focal elastosis (LFE) in 1989 by Burket et al.¹, at least sixteen males and three females have been reported²⁻¹¹. LFE is the term used to denote asymptomatic, palpable, striae-like lines extending horizontally across the middle and lower back. Histologically these lesions present a focal increase in elastic fibers separating the dermal collagen bundles. We report an additional two cases observed in Korea and review the literature.

CASE REPORTS

Our two cases (a 16-year-old girl and a 14-year-old boy) and those previously reported are summarized in table 1. Clinical findings of asymptomatic, several, yellow-red, slightly elevated, palpable, striae-like lesions on the middle and lower back were similar in both our cases (Fig. 1). These lesions had been present since the boy and girl's early childhoods. Their previous medical histories were uneventful and they had not taken any medications including topical and systemic corticosteroids. There was no evidence of striae distensae, connective tissue nevus, papular elastorrhexis or Buschke-Ollendorff syn-

drome. They had no family history of a similar dermatosis.

Light microscopic examinations of skin biopsy specimens demonstrated dermal thickening and focal increases of thin wavy fibers in the upper and mid-dermis but no changes in the epidermis in both cases (Fig. 2). Verhoeff-van Gieson stains for elastic fibers revealed aggregated, clumped, curled, or fragmented ones (Fig. 3).

DISCUSSION

LFE was first described by Burket et al.¹ in 1989. Since then, at least 19 cases have been reported¹⁻¹⁰. The middle and lower back are sites of predilection for the disease although two cases^{10,11} were exceptional. This disease has a preference for males.

Electron-microscopically, most areas of the lesion show numerous elongated or fragmented, mature elastic fibers composed of a central and peripheral component. The central component is composed of electron-lucent amorphous material. The peripheral component is composed of electron-dense material surrounding the periphery of the fibers⁷.

The etiology and pathogenesis are unclear. The initial reported four cases were exclusively elderly men with systemic disorders including myocardial infarction, peptic ulcer, nephrotic syndrome, etc.^{1,2} Therefore, a certain relationship between LFE and systemic disorders or the unidentified ageing

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Fig. 1. Yellowish, slightly elevated, palpable, striae-like lesions on the middle and lower back in a 14-year-old boy (A) and a 16-year-old girl (B).

Fig. 2. Dermal thickening and focal increases of thin wavy fibers in the upper and mid-dermis but no changes in the epidermis (H&E $\times 40$).

Fig. 3. Aggregated, clumped, curled, or fragmented elastic fibers (Verhoeff-van Gieson C: $\times 40$, D: $\times 400$).

Table 1. Reported cases of linear focal elastosis

Source	Age (yr)	Sex	Distribution	Onset (yr)	Concomitant disease
Burket et al. ¹					
Case 1	69	M	Middle & lower back	NM	BCE, MI, PU, HH.
Case 2	75	M	Middle & lower back	NM	Vitiligo, Kyphosis
Case 3	71	M	Middle & lower back	NM	MG, NS, RVT, UP
Hagari et al. ²	86	M	Lower back	60	MI, AO, PH, Erythroderma
White ³	Elderly	M	Upper back	NM	Striae distensae
Moiin et al. ⁴	29	M	Upper & lower back	Childhood	None
Trüeb et al. ⁵		21	F	Lower back	16 None
Vogel et al. ⁶					
Case 1	89	M	Lower back	NM	None
Case 2	69	M	Lower back	NM	None
Palmer et al. ⁷	61	M	Middle & lower back	19	None
Kim et al. ¹¹					
Case 1	12	M	Lower back	NM	None
Case 2	16	M	Lower back	NM	None
Case 3	12	M	Lower back	NM	None
Case 4	17	M	Lower back & both axilla	NM	None
Tamada et al. ⁸					
Case 1	17	M	Middle & lower back	Childhood	None
Case 2	20	M	Middle & lower back	Childhood	None
Case 3	29	M	Middle & lower back	Childhood	None
Hagari et al. ⁹	73	F	Lower back	16	Cardiomyopathy, Striae distensae
Breier et al. ¹⁰	13	F	Flexural aspect of the leg	11	None
Present cases					
Case 1	16	F	Middle & lower back	Childhood	None
Case 2	14	M	Middle & lower back	Childhood	None

NM: not mentioned in the original article, BCE: basal cell epithelioma, MI: myocardial infarction, PU: peptic ulcer, HH: hiatal hernia, MG: membranous glomerulonephritis, NS: nephrotic syndrome, RVT: renal vein thrombosis, UP: urticarial pemphigoid, AO: Angiosclerosis obliterans, PH: prostatic hypertrophy.

process of the skin could be postulated, but young healthy people have been affected in recent reported cases including ours^{4,5,8,10,11}. Some authors have suggested that LFE might represent a degenerative or regenerative process of striae distensae or unusual forms of striae distensae^{3,5}. However, coexisting or a previous history of striae distensae^{3,9} is an unusual finding in LFE and most reported cases were not combined with striae distensae. The possibility of a familial tendency has been suggested⁴. None of our patients had striae distensae or a family history of LFE. The connective tissue nevus is the major differential disease. It does not show elastic tissue fragmentation as does LFE. It seems that LFE is a degenerative nevus or hamartoma of elastic tissue.

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