Poster Number 008. Scientific Abstract
Oral Presentation, Session 1

FASCIAL THICKNESS AND STIFFNESS IN HYPERMOBILE EHLERS-DANLOS SYNDROME

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BACKGROUND

There is a high prevalence of myofascial pain and chronic pain in hEDS patients (1). The mechanisms and pathophysiology behind myofascial pain are controversial. In recent years, the theory has been expanded to include fascial origin of pain with an increase in glycosaminoglycan content and increased thickness in ECM near trigger points (2).
Fascia is an innervated and metabolically active tissue, abundant in nociceptive free nerve endings. Inflamed fascia is thickened with laydown of extracellular matrix with glycosaminoglycan deposition by the fasciacyte and alteration in gliding properties. Alteration of gliding interactions strongly influences joint mobility and leads to impaired biomechanics, altered alignment, decreased coordination (2). These structural changes can be visualized under ultrasound as hypo-echogenicity and on elastography as increased stiffness in the muscle (3). Ultrasound also allows for the thickness of fascia to be measured and the visualization of gliding between various fascial layers (2). The deep fascia of the SCM is a studied area that is associated with neck pain.

METHODS

IRB approval was obtained. A series of 17 patients were examined prospectively 9 with confirmed/suspected hEDS by diagnostic criteria and 8 controls who had chronic intermittent neck pain without hEDS diagnosis.
Ultrasound examination of bilateral heads of the sternocleidomastoid were examined with B-mode scanning and strain elastography using the (Sonimage® HS-1, Konica Minolta Corporation, Japan) and a L18-4 transducer.
The thickness of the superficial deep fascia of the sternocleidomastoid was measured and stiffness was measured semi-quantitatively using elastography comparing the deep fascia with the underlying muscle.
Strain ratios were measured using built-in software and the strain index (SI) was calculated, as deep fascia (A) to muscle strain (B), respectively (SI = A/B ratio). See figure 1.
Differences in strain ratio and superficial deep fascial thickness were compared using unpaired t-test.
Statistical significance was set at $P < 0.05$.

RESULTS

hEDS subjects had a higher mean thickness (1.8 ± 0.5 mm) in the superficial deep fascia of the sternocleidomastoid compared with controls (1.4 ± 0.4 mm). There was no significant difference in the strain index. When strain index from only pathologically thickened areas of deep fascia (> 1.4mm) were compared, hEDS subjects had a strain index of 1.6 ± 1.0 compared with strain index of 2.1 ± 1.7 in controls. This difference approached statistical significance ($p=0.075$). See Table 1.

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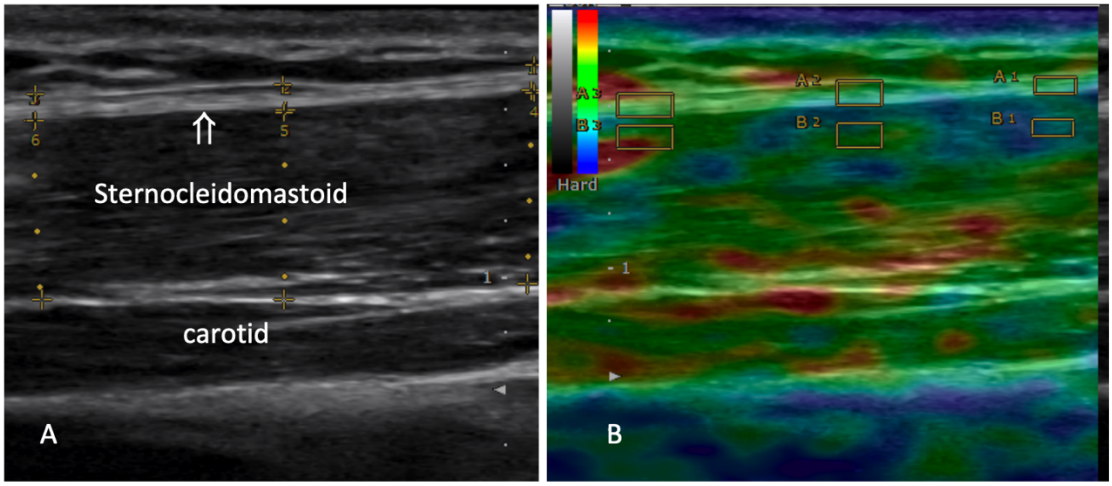
Figure 1



A. Three layers of dense connective tissue and two layers of loose connective tissue in the superifcial deep fascia of the sternocleidomastoid B. Ultrasound elastography comparing strain ratio of the deep fascia (A) to the muscle belly (B).

Table 1 Thickness and strain index of the superior deep fascia of the sternocleidomastoid

	Thickness of (mm)	Strain index = A/B *
Controls (n = 8)	1.4 ± 0.4	2.1 ± 1.7
Right	1.4 ± 0.4	
Left	1.4 ± 0.4	
hEDS (n = 9)	1.8 ± 0.5	1.6 ± 1.0
Right	1.7 ± 0.5	
Left	1.8 ± 0.5	
p-value	p < .001	p = 0.075
Right	p < 0.01	
Left	P < 0.001	

* A = strain deep fascia, B = strain sternocleidomastoid muscle belly
>1 strain index represents softening of the deep fascia in comparison to the muscle belly

CONCLUSION

hEDS patients exhibit increased superficial deep fascia thickness compared with prior reported norms of 0.49-1.1mm2 and greater compared to controls with neck pain. Increase in the loose connective tissue in the deep fascia may play a significant role in the pathogenesis of myofascial pain and may be amplified in hEDS.

Softening of the deep fascia may occur from increase in extracellular matrix content and relative increase in stiffness of the underlying muscle belly; this change is not as pronounced in hEDS possibly secondary to pathologic changes found in both the extracellular matrix

and dense connective tissue that is seen in hEDS. Thickened deep fascia with extracellular matrix deposition may alter gliding interactions and lead to impaired biomechanics, altered alignment, decreased coordination, and pain.

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DECLARATIONS OF INTEREST
None

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