**INTRODUCTION & OBJECTIVE**

**INTRODUCTION**

- Lichen sclerosus (LS) is a poorly understood inflammatory condition of the genital skin affecting 1:300 adults.
- Men with LS develop severe urethral stricture due to profound fibrosis which fails surgical management in >50% of cases.
- Female cutaneous LS lesions display:
  - increased stromal hyaluronic acid (HA) deposition which is known to drive inflammation and fibrosis in other diseases.
  - decreased epidermal CD44 expression which is the principal receptor responsible for HA degradation.
- These changes have not been characterized in male patients nor urethral tissue.
- Therefore, we propose that decreased CD44 expression may cause HA accumulation and drive inflammation and collagen deposition in LS related urethral stricture.

**OBJECTIVE**

- To test the hypothesis that epithelial CD44 expression is decreased and stromal HA abundance increased in LS compared to non-LS tissues.

**Methods**

**Pathology Review**

- Lichen Sclerosus (n=48)
- Control (n=14)

- Excluded (n=16)
  - Inflammation (n=4)
  - Acneiform (n=4)
  - Herpes/genital herpes (n=4)
  - C Candidiasis (n=2)
  - Linear Erythema (n=2)

- Analyzed (n=62)

**Image Analysis**

- 6 ROIs per slide (2 ROIs per compartment)
- CD44 expression and HA abundance quantified as mean optical density in each compartment.

**Immunohistochemistry**

Quantify CD44 expression and HA abundance in three histologic compartments:

- Epithelial (+ e cadherin)
- Inflammatory (+ CD45)
- Stromal (- e cadherin, - CD45)

**Results**

We expect our quantitative approach to demonstrate downregulation of epidermal CD44 expression and increased stromal HA accumulation in human LS. These data will provide a foundation to support the overarching hypothesis that CD44 dysregulation drives HA accumulation and propagation of inflammation and fibrosis in LS and related urethral stricture disease.