ISAKOS

International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine

12th Biennial ISAKOS Congress • May 12-16, 2019 • Cancun, Mexico

Paper #127

Absence of Ligament Progenitor Cells in the Pediatric Knee Anterolateral Complex

Tomoya Iseki, MD, UNITED STATES
Ben Rothrauff, MD, PhD, UNITED STATES
Shinsuke Kihara, MD, PhD, UNITED STATES
Kevin G. Shea, MD, UNITED STATES
Freddie H. Fu, MD, UNITED STATES
Peter G. Alexander, PhD, UNITED STATES
Volker Musahl, MD, UNITED STATES

University of Pittsburgh
Pittsburgh, PA, UNITED STATES

Summary:
Absence of genetically defined ligament structures in pediatric knee anterolateral complex (ALC would be observed

Abstract:

Introduction

The presence of ligamentous components within the knee anterolateral complex (ALC) is controversial. Scleraxis is a basic helix-loop- helix (bHLH) transcription factor that genetically marks tendon and ligament tissues throughout development, regulating the expression of tissue-specific molecules such as Tenomodulin. However, there are no report that genetically the presence of ligamentous components in ALC. The objective of the present study is to confirm the presence of tenogenic progenitor cells in pediatric knee ALC. We hypothesized that the absence of genetically defined tendon or ligament structures in pediatric ALC would be observed.

Methods

15 pediatric ALCs (age 6.3 ± 3.3) and 5 pediatric lateral collateral ligaments (LCLs; age 3.4 ± 1.3) from unpaired, fresh cadaveric knees were used in this study. First, the LCL was identified and dissected after dissection of the overlying skin and muscluro soft tissue including ITB. Then, the ALC was harvested from the anterolateral region of the knee anterior to the LCL. After harvesting, the band samples were fresh-frozen and subsequently sectioned at 7 μm thickness and post-fixed in acetone. The sections were then stained with hematoxylin & eosin as well as analyzed immunohistochemically for the presence of scleraxis and tenomodulin.

Results

The histological sections of the pediatric LCL showed well-organized, dense collagenous tissue fibers with elongated fibroblasts on HE staining. No such structural organization was observed within the ALC. Scleraxis staining was apparent in pediatric LCL within regions of aligned fibers, while the comparatively disorganized structures of the ALC were negative for scleraxis (Fig. 2C-D). The pediatric LCL also stained positive for tenomodulin, while the ALC was only sparsely positive for this tendon/ligament extracellular matrix molecule.

Discussion

In this study, a distinct ligamentous structure could not be visualized in the ALC. Neither histologically, nor

Paper #127

immunohistochemically, any structure could be identified that was indicative of a ligament or expression of tendon and ligament genetic markers including scleraxis and tenomodulin. The ALC appears to develop post-natally in the absence of genetically defined tendon or ligament structures.