[TH-PO163] Development of FSGS Following Anabolic Steroid Use in Bodybuilders

Leal C. Herlitz, Glen S. Markowitz, Alton B. Farris, Joshua A. Schwimmer, Michael B. Stokes, Cheryl Kunis, Robert B. Colvin, Vivette D. D'Agati Dept. of Pathology, Columbia University Medical Center, New York, NY; Dept. of Pathology, Massachusetts General Hospital, Boston, MA; Dept. of Medicine, Columbia University Medical Center, New York, NY

Purpose: Anabolic androgenic steroids (AAS) have multiple adverse effects, however renal injury has not been described previously. We report a novel association of FSGS in bodybuilders who abuse AAS.
Methods: Ten patients (6 Caucasian, 4 Hispanic) were identified from the archives of the Columbia University Medical Center and Massachusetts General Hospital between 1999 and 2009 with: 1) highly muscular physique and bodybuilding; 2) longterm AAS abuse; 3) proteinuria (≥ 1g/d) and 4) renal biopsy diagnosis of FSGS and/or glomerulomegaly.
Results: Average BMI was 34.7 kg/m^2 (range 27-43). Presentations included proteinuria (mean 10.1 g/day, range 1.3-26.3 g/day) and renal insufficiency (mean serum creatinine 3.0 mg/dl, range 1.3-7.8 mg/dl). Five of 10 patients presented with full nephrotic syndrome. Renal biopsy revealed FSGS in 9 patients, of which 4 also had glomerulomegaly, and glomerulomegaly alone in 1 patient. Three biopsies had collapsing lesions of FSGS, 4 had perihilar lesions and 7 showed ≥40% tubular atrophy and interstitial fibrosis. Follow-up (mean 2.2 yrs) was available in 8 patients. One patient progressed rapidly to ESRD. The other 7 received RAS blockade, and 1 also received corticosteroid therapy. All 7 discontinued AAS and reduced exercise, leading to weight loss, stabilization or improvement in serum creatinine and decrease in proteinuria. In 1 patient, restarting AAS led to progressive proteinuria and renal insufficiency. When compared to historical controls with obesity related glomerulopathy, FSGS in bodybuilders is a more severe disease with higher creatinine and proteinuria at presentation and more glomerular and tubulointerstitial scarring.
Conclusion: We hypothesize that FSGS results from a combination of post-adaptive glomerular changes driven by increased lean body mass and potential direct nephrotoxic effects of AAS.

Thursday, October 29, 2009 10:00 AM

Poster Session: Clinical Nephrology: Clinical Advances in Glomerular Diseases I (10:00 AM-12:00 PM) Poster Board Number: TH-PO163

Location: Exhibit Halls A/B/C