CLINICAL VIGNETTE

Chronic Kidney Insufficiency with Proteinuria Associated with Androgenic Anabolic Steroid Abuse

Hamid Hajmomenian, M.D.

A 50-year-old male was referred by his primary physician for evaluation of protein in urine and elevated serum creatinine. His laboratory tests a few months prior to presentation had shown elevated creatinine of 1.4 mg/dl and 3+ proteinuria on dipstick urinalysis. His past medical history was remarkable for hypothyroidism; his family history and review of systems was unremarkable. The patient was a body builder and reported using testosterone supplements regularly over several years. His meds were Armour thyroid 60mg daily and Testosterone Cypionate 200mg intramuscularly monthly.

Physical examination was unremarkable. Repeat basic metabolic panel confirmed elevated creatinine of 1.4 mg/dl. His urinalysis showed 3+ proteinuria with a protein creatinine ratio of 4.08. Extensive laboratory evaluation for collagen vascular diseases, viral hepatitis, and HIV were completely within normal limits, while repeat urine testing showed continuing nephrotic range proteinuria. Ultrasound guided kidney biopsy showed focal and segmental glomerulosclerosis, hilar type consistent with secondary/compensatory FSGS, moderate interstitial fibrosis and tubular atrophy and moderate to severe arteriolar sclerosis, consistent with chronic hypertension.

Discussion

Anabolic androgenic steroids (AAS) are synthetic derivatives of testosterone, which are used by athletes and non-athletes to increase muscle mass and enhance endurance. Nearly four out of five users are non-athletes who use them for cosmetic reasons.[1]

The increasing prevalence of AASs use among non-athletes who are motivated to increase their muscle mass and enhance physical fitness and attractiveness has been associated with an alarming increase in the number of patients presenting with side effects associated with the use of these drugs, which has become a public health concern.[2]

Cardiovascular side effects including cardiac hypertrophy and sudden death,[3] gonadal dysfunction, hepatic side effects, and neuropsychiatric abnormalities are among the well-known side effects of using these drugs and are well-described. Over the past few years, cases of acute and chronic kidney injury associated with AAS drug abuse have been reported.[4,5] The clinical presentation of these cases included proteinuria, asymptomatic to nephrotic range, and reduced GFR and nephrotic syndrome. Kidney biopsies frequently revealed secondary form of focal segmental glomerulosclerosis, FSGS, with glomerulomegally in some cases. In the secondary forms of FSGS, the glomerular injury is the result of adaptive responses to elevated glomerular capillary pressures and flow rates. Conditions associated with either decreased nephron number or increased demand on a normal group of nephrons will result in increased single-nephron GFRs. These increased demands first manifest morphologically as glomerular hypertrophy but eventually become maladaptive, producing glomerulosclerosis. The increased demand associated with AAS abuse is secondary to increased muscle mass. As seen in well-described cases of obesity induced secondary FSGS,[6] kidney biopsy in patients with acute kidney injury associated with AAS abuse reveal the same features of peripheral glomerulosclerosis as in this case report, which is the predominant pathology pattern seen in secondary cases of FSGS. Furthermore, cases of highly muscular, non-obese patients with kidney biopsy consistent with secondary FSGS pattern support the same pathophysiology associated with increased demands and GFRs in previously normal nephrons with subsequent sclerosis seen in AAS drug abusers with kidney injury.[7]

In addition to increased demand on nephrons and secondary sclerosis, AAS has direct nephrotoxicity on glomerular cells leading to mesangial matrix accumulation and sclerosis have been suggested based on some animal studies.[8,9]

In patients with AAS abuse and kidney injury, discontinuation of AAS is the key treatment. In addition to discontinuation of AAS, renin-angiotensin system blockade and weight loss can be considered.

In conclusion, increased lean body mass and AASs drug abuse should be considered among the differential diagnosis in patients presenting with proteinuria and acute as well as chronic kidney insufficiency as a result of secondary FSGS.

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