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Case report

Focal segmental glomerulosclerosis as a rare side effect of combined anabolic-androgenic steroid use, high-protein diet and high-intensity anaerobic training

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Abstract

Introduction: **Focal segmental glomerulosclerosis (FSGS) is a serious condition causing glomeruli damage and leading to nephrotic syndrome and renal failure.** One of its causes is the prolonged use of anabolic-androgenic steroids (AAS), which has become a common trend among young athletes who are in a majority not aware of its side effects.

Aim: This study aimed to present an influence of performance-enhancing drugs (including AAS) combined with high-intensity training on renal failure, referring to a clinical case report.

Case study: **The study presents a 25-year-old male who was admitted to Nephrology Clinic with hematuria, proteinuria, elevated creatinine values, and hypertension.** During hospitalization, a kidney biopsy was performed, which confirmed FSGS.

Results and discussion: **In our patient's case, FSGS was caused by overusing performance-enhancing drugs (including AAS) for 10 years, a high-protein diet, and high-intensity training.** When the patient had discontinued the use of all the performance-enhancing drugs and had begun appropriate treatment, the renal function parameters improved.

Conclusions: We conclude that overusing multiple performance-enhancing drugs may lead to renal failure and FSGS even in young adults with no congenital or immunological contributing factors. Thus such therapy should be disadvised especially to young athletes wanting to quickly improve their muscle mass.

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1. INTRODUCTION

The latest data suggest that the use of anabolic-androgenic steroids (AAS) has become common among bodybuilders, athletes, as well as adolescents, and young adults who want to increase their muscle mass for aesthetic or athletic reasons. The desire to achieve athletic success in a short time to improve performance, increase muscle growth, and lean body mass is a key factor influencing doping. Prolonged misuse and abuse of AAS can affect kidneys and may determine the development of focal segmental glomerulosclerosis (FSGS). FSGS usually presents with nephrotic syndrome and is a major contributor to end-stage renal disease (ESRD) among patients who do not achieve remission in proteinuria. ESRD is fatal unless treated properly.[1]

2. AIM

We report a rare case of FSGS and nephrotic syndrome in a man after long-term abuse of AAS and other performance-enhancing drugs combined with high-intensity anaerobic training and high-protein diet.

3. CASE STUDY

A 25-year-old, unemployed, previously untreated male was admitted to Nephrology Clinic with hematuria, proteinuria, and elevated serum creatinine level. The symptoms began a month ago, where the patient discovered blood in the urine. Blood panel including creatinine and urinalysis were conducted at the general practitioner's command. On admission to the Nephrology Clinic, the heart rate was 99 bpm, while the blood pressure (BP) was 142/96 mm Hg. The heart, abdomen, lungs, and neurological examinations were unremarkable. He distinguished himself by his markedly muscular physique and acne on the back on physical examination. Anamnesis revealed borderline values of BP in the past few years. Two-hour high-intensity physical training at the gym 6 times a week and running from 2 to 3 times a week were performed. The patient has had this workout schedule for the past 10 years. Additionally, he had been taking AAS to increase muscle mass (750 mg of intramuscular testosterone weekly, 200 mg of intramuscular injections of trenbolone enanthate 3 times a week, and 60 mg of oral trenbolone enanthate once a week). The patient also admitted taking approximately 40 units of insulin (1 unit to cover 10 g of glucose) and 240–260 g of protein powder per day. One month before submission to Nephrology Clinic, he ceased to take all of these performance-enhancing drugs, resulting in 10 kg of unintentional weight loss. However, he did not quit smoking (30 cigarettes daily) nor consuming alcohol.

Laboratory tests were conducted on admission and revealed impaired renal function, proteinuria in the nephrotic range, hematuria, an increase of hematocrit, hemoglobin, red blood cell count level, and the decrease of serum albumin value (Table 1). Electrocardiogram demonstrated features of left ventricular hypertrophy. Additionally, the patient underwent a chest X-ray and abdominal ultrasound, which showed enlargement of both kidneys (size of the left kidney: 140 × 50 mm; size of the right kidney: 130 × 50 mm) and bilaterally increased parenchymal echogenicity. During hospitalization, the renal biopsy was performed, which revealed focal segmental glomeruli sclerosis and fibrous emboldening of Bowman's capsules. Neither IgA, IgG, IgM, C3 nor light chain deposits were found, which confirmed the diagnosis of FSGS caused by performance-enhancing drugs, high-protein diet, and high-intensity training.

Table 1. Patient's laboratory results on admission.

Parameter	Patient value	Normal range
Total protein, g/dL	5.80	6–8
Urine total protein, g/L	5.52; 3.50; 3.30; 3.65	0.05–0.08
Cholesterol, mg/dL	228	115–190
Creatinine clearance, mL/min	83.5	75.0–110.0
Creatinine, mg/dL	1.14	0.70–1.30
Red blood cell count, M/μL	5.80	4.30–5.60
Hemoglobin, g/dL	17.8	13.7–16.5
Hematocrit, %	51.3	40.1–51.0
Albumin, g/dL	3.39	3.50–5.50
Complement component 3, g/L	1.26	0.9–1.8
Complement component 4, g/L	0.23	0.1–0.4

After 4 days of well-tolerated renoprotective and hypotensive treatment (5 mg of ramipril and 10 mg of lercanidipine), the patient was discharged from the hospital in good general condition. The patient was ordered to continue the initiated therapy, cease the high-intensity training, performance-enhancing drugs, and protein powder supplements. The performance-enhancing drugs' discontinuation lasted for 8 weeks in which normalization of renal function was achieved with creatinine level at 1.03 mg/dL and stable proteinuria of 1.5 g / 24h. However, BP was still increased.

Despite explaining all the risks related to using performance-enhancing drugs, protein excessive consumption and informing that they are the direct trigger of FSGS, the patient refused further renoprotective treatment and decided to return to the previous lifestyle.

4. RESULTS AND DISCUSSION

Improving men's physical appearance and creating a more muscular build has become a factor in recent years contributing to overusing multiple performance-enhancing drugs in combination with excessive high-intensity trainings. A survey of 500 AAS users revealed that 78.4% participants were noncompetitive bodybuilders and nonathletes which confirms a non-medical trend of AAS use.[2] In this particular case, the patient combined AAS, insulin injections and high-protein diet. Basing on the study from Pereira et al. only less than half of interviewed athletes realized the adverse health effects related to anabolic steroids,[3] which may present as various psychiatric symptoms, acne, voice alterations, liver changes, hypertension, arrhythmias, left

ventricular hypertrophy,[4,5] decrease of reproductive hormones, potency or gynecomastia.[5,6] Left ventricular hypertrophy was observed in the patient's chest X-ray, which could cause systolic or diastolic function weakening in the future. Another important, albeit less reported side effect of prolonged usage of AAS, is FSGS.[7,8] Currently, FSGS has become the most common lesion among adults undergoing renal biopsy. Its prevalence is estimated from 0.2 to 1.8 cases per 100,000 population per year. FSGS is a progressive disease, which untreated may contribute to ESRD.[9] Kidney biopsy confirmed the diagnosis of FSGS and proved that no other congenital or immunological factor contributed to renal failure. Additionally, the patient suffered from hypertension, which combined with FSGS led to nephrotic syndrome manifesting with proteinuria and increased cholesterol values. Hematuria, not typical for FSGS, was probably triggered by all three factors equally and could be a reflection of glomerular filtration failure and renal hyperfiltration. After discontinuing all the performance-enhancing drugs, renal function parameters improved and a decrease in proteinuria was observed. It suggests that FSGS in such a young patient was caused by AAS and other used substances.

Acidosis, kidney hyperfiltration, glomerular injury, and proteinuria are thought to be partially caused by high-protein diet.[10] The patient's average dose of protein was 250 g per day, which significantly exceeded his daily basis protein intake (N: 0.8 g protein/kg a day[11]). Therefore, high-protein diet could collaterally exacerbate renal functions, and combined with AAS, contribute to FSGS.

AAS dependence syndrome has recently been reported and is described as occurring withdrawal symptoms and continued use despite them.[12] The etiology of AAS dependence is probably multifactorial and is thought to implicate muscular body-image and neuroendocrine factors.[13] Despite physical (acne) and mental side effects, the patient used AAS for almost 10 years. He even suffered from hypoglycemia involving medical assistance due to insulin injections. Not until hematuria occurred, did he temporally discontinue using all of the performance-enhancing drugs and began appropriate therapy. However, after a few weeks of hypotensive therapy and renal functions improvement, the patient abandoned the treatment. Not only was the patient aware of severity of his condition and potential implications related to the FSGS, but he decided to return to high-intensity training and using performance-enhancing drugs and protein powders.

5. CONCLUSIONS

This case report aims to emphasize the impact of performance-enhancing drugs, high-protein diet, and intensive exercise on renal functions. This combination may lead to the development of FSGS and nephrotic syndrome. Thus, further studies should be conducted to increase awareness in this matter, especially of the young athletes.

Conflict of interest

None declared.

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