Case Report

Renal Disease in Bodybuilders

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ABSTRACT

We report our experience with the development of focal segmental glomerulosclerosis (FSGS) in two adult bodybuilders who had consumed high protein diet, anabolic steroids and growth hormone for years in their attempt to gain more muscular appearance. The first patient presented with severe renal failure (serum creatinine 700 umol/l) and kidney biopsy showed advanced FSGS. The second had moderate renal disease (serum creatinine 170 umol/l) and hypercalcemia (3.8 mmol/l) due to his high intake of milk instead of water to promote more protein intake. His kidney biopsy showed early FSGS with diffuse interstitial fibrosis associated with extensive calcium deposition in the tubules. Both patients were instructed to avoid such attitude and were treated conservatively. However, the first patient required kidney transplantation four months later. The second one improved gradually, over the past two months, with decrease of serum creatinine to 96 umol/l. He is currently on telmisartan 40 mg daily to decrease his glomerular pressure. In conclusion, athletes and bodybuilders should consider the risks involved with the use of high protein diet, anabolic steroids and growth hormone alone or in combination to avoid the development of serious renal disease such as FSGS.

KEY WORDS: anabolic steroids, bodybuilders, focal segmental glomerulosclerosis

INTRODUCTION

Many athletes use high protein diet and pharmacological doses of anabolic steroids and growth hormone to enhance their physique and improve their achievements. The anabolic steroids are banned drugs for their serious side effects on the immune system, blood, sterility, atherosclerosis, blood pressure, lipid, heart, liver, immune system and mood[1]. The renal complications of such practice are emerging yet remain rare[2,3]. In this case report, we present two adult bodybuilders who presented with advanced and moderate renal failure subsequent to consumption of such diet and drugs for years.

CASE REPORTS

Case 1

A 41-year-old Philipino man presented to our renal unit with progressive lower limbs edema and shortness of breath over the past few weeks. He denied fever, chest or abdominal pain, skin rash and joint pains. The patient was a coach for body building and he had used for years, high protein diet (20 - 30 g/kg/day), multiple anabolic steroids and growth hormone for > 20 years. The patient had muscle aches and laboratory tests six months ago showed serum creatinine at 135 umol/l and albumin at 34 g/l. Otherwise, he did not have significant medical illness, surgery, and allergy. On his initial physical examination, the patient was conscious and oriented. He did not have pain yet was in distress due to shortness of breath. Blood pressure was 150/90 mmHg and temperature was normal. He was very muscular with a body weight of 89 kg and height 1.82 meters. He did not have lymphadenopathy or goiter. However, he had jugular venous distension and edema. Systemic examination did not show abnormality except for bilateral basal crepitations. Laboratory investigations showed normal peripheral leucocytic and platelets counts. Hemoglobin was 110 g/l with normal MCV. ESR was 20 mm/h. Serum

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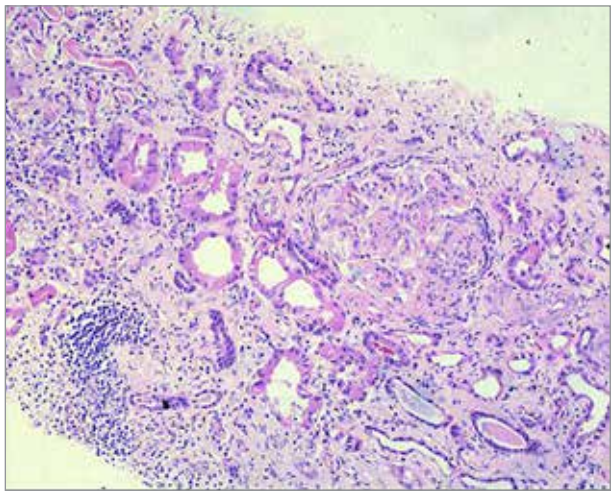

Fig. 1: Photomicrograph of kidney biopsy of the first patient showing global sclerosis of 80% of the glomeruli. The remaining glomeruli show segmental sclerosis. There is mild mesangial cell proliferation, interstitial infiltration by lymphocytes, plasma cells and few neutrophils around the global sclerosis glomeruli. (H&E, original magnification x 200)

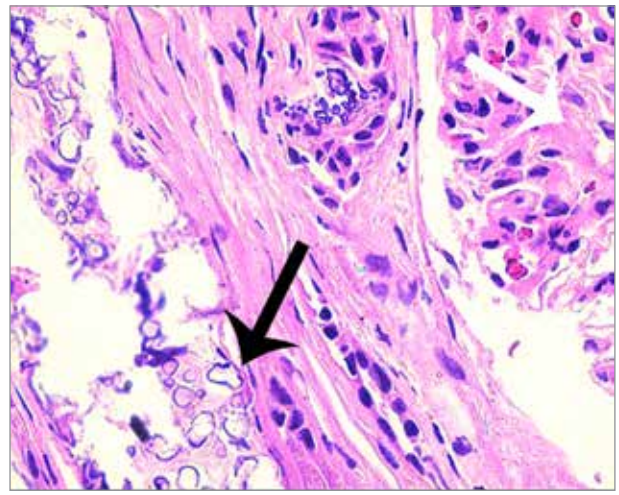

Fig. 2: Photomicrograph of the kidney biopsy of the second patient showing a glomerulous with mesangial sclerosis (white arrowhead) next to tubules containing calcium deposits (black arrowheads) which had extended into the interstitium. (H&E, original magnification x 400)

glucose was normal. Serum urea and creatinine were elevated at 40 mol/l and 700 umol/l, respectively. Serum electrolytes were normal except for phosphate at 2.5 mmol/l. Liver functions were normal except for albumin at 27 g/l. Serum CPK was 250 iu and serum cholesterol 6 mmol/l. His TSH was normal. Urine routine and microscopy showed 3+ protein with 25 RBCs / HPF and yet no pyuria. Serum complements (C3 & C4) and protein electrophoresis was normal. The ANA, anti-ds DNA, ANCA, RA, hepatitis B surface antigen (HBsAg) and anti-HCV antibodies were negative. Twenty four hour urine showed protein excretion at 6 g/day. Stool testing for ova, parasites and occult blood was normal. Chest X-ray and ECG were also normal. Abdominal and pelvic ultrasound was normal except for echogenic cortex of both kidneys. Percutaneous kidney biopsy showed a total of 11 glomeruli, out of which nine had global sclerosis (Fig. 1). The remaining ones showed focal and segmental glomerulosclerosis. There was mild diffuse interstitial infiltrate of lymphocytes and plasma cells. Blood vessels showed mild medial hypertrophy. Immunoflourescent stains were negative. The patient was instructed to have low protein diet and avoid the use of anabolic steroids and GH. Four months later, he received successful kidney transplant and since then, is well.

Case 2

A 36-year-old Iraqi man was referred to our renal unit for high serum creatinine (170 umol/l). His main complaint was bilateral loin pain and lower limbs edema for six months. He denied fever, skin rash and joint pains. The patient was a body builder and had used, for years, high protein diet (30 - 50 g/kg/day) and had used milk up to 10 liters/day instead of water. He had used testosterone injections up to 250 mg/day intramuscularly in addition to growth hormone on an average of 100 mg/day. He was a heavy cigarette smoker. He did not have any other significant medical illness, surgery, and allergy. On his initial physical examination, the patient was conscious and oriented. He looked very muscular without pain or shortness of breath. Blood pressure was 120/80 mmHg and temperature was normal. He was very muscular with a body weight of 76 kg and height of 1.78 meters. He did not have jugular venous distension, lymphadenopathy or goiter. However, he had pallor and bilateral lower limbs oedema. Systemic examination did not show any abnormality. Laboratory investigations showed normal peripheral leucocytic and platelets counts. Hemoglobin was 129 g/l with normal MCV. ESR was 20 mm/h. Serum sugar was normal. Serum urea and creatinine were elevated at 11 mmol/l and 170 umol/l, respectively. Serum electrolytes were normal except for phosphate at 2.8 mmol/l and corrected calcium at 3.8 mmol/l. Liver functions were normal except for albumin at 34 g/l. Serum CPK was 970 iu. Serum cholesterol was 5.2 mmol/l. TSH and parathyroid hormones were normal. Urine routine and microscopy did not show pyuria. Yet he had proteinuria 3 +, 12 - 15 RBCs/HPF and excess calcium oxalate. Serum complements (C3 & C4) and protein electrophoresis was normal. ANA, anti-ds DNA, ANCA, RA, HBsAg, anti-HCV and anti-HIV antibodies were negative. A twenty-four hour urine

sample showed protein excretion at 6 g/day. Stool testing for ova, parasites and occult blood was normal. Chest X-ray and ECG were normal. Abdominal and pelvic ultrasound was normal except for echogenic cortex of both kidneys with two non-obstructing calculi at the lower pole of the right kidney which were confirmed by plain KUB. Initial management included intravenous normal saline to correct his hypercalcemia. Serum calcium dropped to 2.6 mmol/l yet, without significant change in his kidney function. A percutaneous kidney biopsy showed a total of 13 glomeruli with variable mesangial matrix increase and yet, without cellular proliferation. Only one glomerulus showed segmental sclerosis. There was marked interstitial fibrosis with tubular atrophy. Most tubules contained calcium crystals (Fig. 2). The blood vessels were normal. Immunoflourescent stains were negative. The final diagnosis was FSGS with interstitial fibrosis associated with excessive calcium deposition in the tubules. The patient was instructed to have low protein diet and avoid dairy products, the use of anabolic steroids and GH. He received telmisartan (micardis) 40 mg daily to protect his kidney by decreasing glomerular pressure. Two months later, his blood pressure was normal and he did not have edema or muscular pains. Serum urea and creatinine had dropped to 6 mmol/l and 97 umol/l, respectively. His serum calcium remained 2.4 mmol/l.

DISCUSSION

FSGS is produced by many mechanisms[4]. In our patients (2 cases), lack of florid nephrotic state and IgM deposition are against a primary immunological disease. Moreover, the normal kidney size indicates lack of previous significant kidney injury leading to progressive hyperfilteration due to compensatory hypertrophy of the remaining glomeruli. Lastly, the patients did not have a family history of similar glomerulopathy, diabetes, congenital cyanotic heart disease, morbid obesity, familial dysautonomia, acromegaly, infections, IV drug abuse or HIV infection. The only relevant insult in their past medical history was the use of high protein diet, pharmacological doses of anabolic steroids and growth hormone. The high protein "myth" has been floating around for generations. Historically, it can be traced to *Milo* of Crotona in the sixth century B.C. He was a famous Greek athlete who was considered to be one of the strongest men in ancient Greece. He had won wrestling victories in five Olympic games as well as in other sacred festivals.

The optimal protein requirement for adults hardly exceeds 0.8 g/kg/day as recommended by the US food and nutrition agency[5]. Our patients had consumed 30 - 50 g/kg/day. High protein intake increases the renal blood flow and glomerular filtration rate in an attempt to excrete the nitrogenous by-products of its catabolism. Such chronic hyperfilteration may be a factor or may accelerate the development of FSGS[6]. The use of androgenic anabolic steroids (AASs) to promote wound healing in World War II and those in concentration camps is well documented. However, abuse of such drugs in sports started in 1940 and especially during the cold war between the west and east. Their emerging systemic side effects on the heart, liver and mood led to its ban in Olympic games in 1976. Interestingly, the direct toxic effect of such banned drugs on the glomeruli has been clearly documented and has been shown to be mediated *via* specific testosterone receptors in the glomeruli[7,8]. Moreover, the usual dose of replacement therapy of testosterone hardly exceeds 250 mg IM every three weeks. As has been shown, our patients had consumed pharmacological doses of such drug reaching up to 100 times the usual therapeutic one and for years. The last insult came with addition of GH to their regimen. Interestingly, the drug had limited success in treatment of short stature children and yet is being promoted as body building agent. Experimental reports in mice have shown its renal toxicity and induction of glomerulosclerosis through increase in renal blood flow and GFR[9]. Moreover, it has been shown that its target site was specifically on the glomerular podocyte[10]. In fact, a case report on an FSGS patient with acromegaly who had failed all therapeutic drug regimens improved after treatment with trans-sphenoidal microsurgery of the adenoma[11]. The deleterious effects of other factors such as hypercalcemia in our patient who used milk instead of water to promote his protein intake should not be underestimated as well as the effect of episodic severe elevations of blood pressure associated with weight lifting. Moreover, these patients are liable to use less water, diuretics, laxatives and non-steroidal anti-inflammatory drugs for pain. The latter combinations are harmful to the kidney, though not documented in our patients.

CONCLUSION

The idea of burning fat and feeding the muscle should not be on the expense of serious renal disease such as FSGS. Athletes and bodybuilders should not dismiss these facts as an "anti-anabolic steroid propaganda" and should consider the risks involved before losing their career and even their life with such banned practice.

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