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**RESEARCH ARTICLE**

# SECONDARY FOCAL SEGMENTAL GLOMERULOSCLEROSIS IN BODYBUILDERS

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**ABSTRACT**

**Background:** Many athletes use high protein diet, high pharmacological doses of androgen anabolic steroids (AASs), growth hormone to enhance their physique, improve achievements. The(AASs)are banned for their effects on immune system, blood, sterility, atherosclerosis, blood pressure, heart, liver, and secondary focal and global glomerulosclerosis.

**Objectives:** identify pattern, risk factors, etiology, histopathological features, prognosis and management of renal involvement in bodybuilders.

**Patients and Methods:** This study enrolled (33) male body builders who developed impaired kidney functions, proteinuria and were followed up for (1) year. All patients were subjected to clinical examination, recording of( body mass index(BMI), training sessions/ week, duration, protein intake per/day, creatine and amino acids intake /day, AASs, growth hormone intake/week).Laboratory investigations{HCV antibody, HBV surface and core Ag, HIVAb}, {ANA, anti-ds DNA,C3,C4,CH50}, {serum creatinine, BUN}, total proteins, serum albumin, urine analysis, quantitative 24 hours urinary proteinuria, lipid profile, {ALT,AST}, serum calcium, phosphorus, total vitamin D, serum PTH, TSH and hemoglobin levels. Renal biopsy was performed for all participants and estimated glomerular filtration rate (eGFR)was done.

**Results:** Association of secondary focal segmental glomerulosclerosis (FSGS) in 33 bodybuilders; mean age (31.42y± 1.44), mean BMI (46.18 kg/m$^2$ ±2.51) after long-term abuse of AASs with mean intake (1827.27 mg/wk ±426.66). Clinical presentation included renal insufficiency, mean serum creatinine (527.79 ummol/l ±231), mean proteinuria (19.30 g/d.±5.24). Renal biopsy revealed FSGS and end stage renal disease (ESRD) glomerulomegaly in 33 patients, with tubular atrophy and interstitial fibrosis in 21 of them. Upon follow-up; 12 patients progressed to ESRD and required renal replacement therapy (RRT); 3 were non compliant to medical advice, 9 started (RRT) at time of diagnosis. All patients were prescribed renin-angiotensin system blockers, advised to discontinue (AASs) leading to weight loss, stabilization or improvement in serum creatinine, reduction in proteinuria in the remaining 21 patients.

**Conclusion:** Increased BMI, abuse of (ASSs), high protein diet carry a great risk for developing ESRD secondary to (FSGS) in bodybuilders.



## INTRODUCTION

### *Background*

Androgen anabolic steroid (AASs) abuse is becoming increasingly common, with over 75% of bodybuilders admitted to regular use. Many athletes use high protein diet and pharmacological doses of anabolic steroids and growth hormone to enhance their physique and improve achievements (1). Anabolic steroid abuse adversely affects the endocrine system including (testicular atrophy, decreased fertility, and gynecomastia), blood lipids (increase in LDL and decrease in HDL), neuropsychiatric disturbances, recurrent hepatic adenomas, but renal injury has not been described. The link with renal injury, and in particular focal segmental glomerulosclerosis (FSGS), is less well-recognized. (2,3-6). The anabolic steroids are banned drugs for their serious side effects on the immune system, blood, sterility, atherosclerosis,

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blood pressure, lipid profile, heart, liver, immune system and mood (7,8). The renal complications of such practice are emerging yet remain rare (2,3).Focal segmental glomerulosclerosis is a condition in which there is focal scarring of some glomeruli within the kidneys. The majority of FSGS is primary. Secondary FSGS is associated with hyper filtration from increased body mass or reduced renal mass, illicit drug use and HIV infection. Most patients with FSGS progress to end-stage renal disease (ESRD) within 6 to 8 years (9).

## PATIENTS AND METHODS

This study enrolled thirty three (33) adult male body builders who developed impaired kidney functions and proteinuria and were followed up for one (1) year. All patients were subjected to complete history taking and detailed clinical examination, recording of age, demographics, body mass index calculation ($kg/m^2$), recording of (training sessions/week and duration, protein intake /day, creatine and amino acids intake per day, anabolic steroids and growth hormone intake/week, systolic and diastolic blood pressure measurements). Renal biopsy was performed for all patients at start of the study after a written consent was signed. Their age ranged from (29-36 years) with a mean of (31.42y±1.44) and a mean BMI of (46.18 $kg/m^2$ ±2.51).All of them used to take anabolic steroids with an average of (1827 mg/week ±426.6)in addition to high protein diet with an average(17.30 gm/kg/day ±3.40)ranging from(10-25 gm/kg/day), average intake of growth hormone (115.45 IU/week ±41)ranging from (40-200 IU/week), creatine and amino acid intake with an average of (14.58gm/kg/day±2.72) ranging (8-20gm/kg/day). All of them were engaged in weightlifting for bodybuilding, with training experience ranging from (10-24 years) and training sessions from (5-7 times /week).

### *Analytic procedures*

A full blood film & differential cell count, full renal &hepatic profile {ALT,AST} were done for all participants, additionally; serum corrected calcium, phosphorus, total proteins &albumin, total vitamin D, TSH, iPTH were also done. Complete urine analyses with microscopic assay for RBCs, WBCs &urinary casts was also performed and quantitative 24 hours proteinuria was estimated. Also calculated was creatinine clearance, estimated glomerular filtration rate (eGFR)using the Modification of Diet in Renal Disease equation(MDRD) {$eGFR = 170\ X\ Serum\ Creatinine^{-0.999}\ X\ Age^{-0.176}\ X\ [0.762]\ if\ Female\ X\ [1.180]\ if\ Black\ X\ BUN^{-0.170}\ X\ Albumin^{+0.318}$}. Serological status for {HCV antibody, HBV surface and core antigens & HIV antibody} was done for participants, as well as collagen markers {ANA, anti-ds DNA antibody, serum complement C3, C4 and CH50}.

## ASSAY METHODS

Measurement of (serum creatinine, BUN, 24 hours urinary protein excretion, lipid profile, liver function test, serum calcium and phosphorus) were carried out using a fully automated analyzer unicell DXC 800 Beckman coulter (USA) with intra-assay and inter-assay CV for these tests ranged between (3.5-5.0 %). Measurement of (TSH) was carried out using a one step-sandwich immunoassay technique of a unicell DXI 600 Beckman coulter (USA),the analytical sensitivity is(0.003µIU/ml)and the expected value from (0.34-5.6 µIU /ml).HBs Ag detection was carried out using a one step-sandwich immunoassay technique. HCV Ab was carried out using indirect two step-sandwich immunoassay technique,HIV Ab was carried out using a two step-sandwich immunoassay technique, intact PTH was carried out using a one step-sandwich immunoassay technique, all on a unicell DXI 600 Beckman coulter (USA). Vitamin D 25(OH) assay was carried out by the 2nd generation platform of ECL (Electrochemiluminescence) technology of Cobas 4111 Hitachi-Roche diagnostics GmbH d-68298 Mannheim Germany, and the reference range:( < 50 nmol/L deficient), (50-75 nmol/L insufficient), (> 75 nmol/L sufficient). Testing for ANAs and ds-DNA was carried out using indirect Immune Fluorescence technique, C3 and C4 were carried out using light scattering technique on access Beckman coulter (USA).

## STATISTICAL METHODOLOGY

Analysis of data was done using SPSS (statistical program for social science) version 16. Quantitative variables were expressed as mean ± SD and range; whereas qualitative variables were expressed as number and percentage. Chi-square test was used to compare qualitative variables between groups and Unpaired t-test for comparison of two groups as regards quantitative variables. regression analysis was used for performing multivariate analysis of different variables, P value was expressed as; P value <0.05(S=significant),P value>0.05 (NS=non significant).

## RESULTS

At the time of presentation, it was found that the patients mean

**Table 1** Patients demographics, steroid & hormonal intake, training schedule.

| | Mean | SD | Min. | Max. |
|---|---|---|---|---|
| Age(yrs) | 31.42 | 1.437 | 29 | 36 |
| BMI($kg/m^2$) | 46.18 | 2.518 | 40 | 50 |
| Training experience(yrs) | 15.85 | 4.063 | 10 | 24 |
| Training session/wk | 6.48 | 0.619 | 5 | 7 |
| Protein intake (g/kg/d) | 17.30 | 3.405 | 10 | 25 |
| Creatine & A.A (gm/kg/d) | 14.58 | 2.728 | 8 | 20 |
| Anabolic steroid (mg/wk) | 1827.00 | 426.668 | 1000 | 2600 |
| Growth hormone (iu/wk) | 115.45 | 41.086 | 40 | 200 |

**Table 2** Clinical&laboratory findings of the participants in the study.

| | Mean | SD | Min. | Max. |
|---|---|---|---|---|
| SBP (mmHg) | 173.03 | 15.101 | 140 | 200 |
| DBP (mmHg) | 104.70 | 7.699 | 90 | 120 |
| S.Creatinine (ummol/l) | 525.79 | 231.002 | 180 | 1000 |
| BUN (mmol/l) | 17.58 | 6.471 | 10 | 46 |
| Total protein (g/l) | 12.85 | 2.181 | 8 | 16 |
| S. Albumin (g/l) | 44.67 | 3.416 | 34 | 48 |
| S. Cholesterol (mmol/l) | 6.812 | 1.0735 | 5.0 | 9.0 |
| S. Phosphorus(mmol/l) | 1.767 | 0.2689 | 1.4 | 2.6 |
| S.Calcium(mmol/l) | 2.976 | 0.3992 | 2.2 | 4.2 |
| S. Vitamin D (total) nmol/l | 507.64 | 341.995 | 80 | 1400 |
| TSH(IU/ml) | 0.891 | 0.1608 | 0.4 | 1.0 |
| Hb (gm/dl ) | 14.29 | 1.641 | 10 | 16 |
| 24 hours urinary proteins (g/d) | 19.30 | 5.241 | 10 | 28 |
| Urine Creatinine clearance (MDRD)mL/min/1.73 $m^2$ | 87.79 | 26.45 | 30 | 140 |

serum creatinine was (525.79 ummol/l ±231) with a range from (180-1000 ummol/l), mean 24 hours urinary protein excretion (19.30 gm/24 hours ±5.241) ranging from (10-28 gm/d), mean serum BUN was (17.58 mmol/l ± 6.471) ranging from (10-46 mmol/l), mean systolic blood pressure (SBP)was (173.03 mmHg ±15.101) ranging from (140-200 mmHg) and mean diastolic blood pressure(DBP)(104.70 mmHg ±7.6) ranging from(90-120 mmHg) Tables(1),(2).

Renal biopsy was done for all participants in this study and revealed that (100%) of patients showed glomerulosclerosis, glomerulomegaly and hypertensive changes, 21 patients (63.6%) showed tubular atrophy and interstitial fibrosis, 22 patients showed atherosclerotic changes (66.7%), whereas 6 patients (18.2%) showed calcium deposition. Immunoflourence stainining was unremarkable for IgA ,IgM ,IgG as well as C3,C4 Figures: (1,2,3,4,5,6 ).

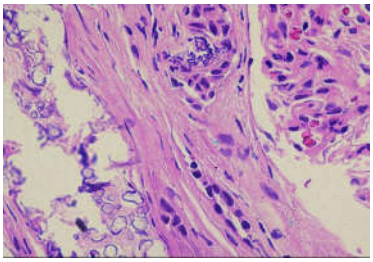

**Figure 1** H&E stain showing tubular calcification & most of the tubules contain calcium crystal depositions, also there is tubular atrophy.

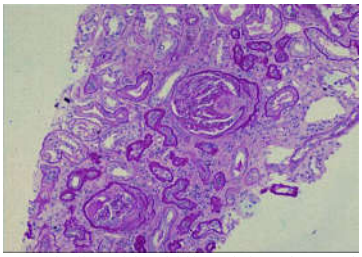

**Figure 2** PAS (Periodic acid Schiff) staining shows glomeruli with segmental hyaline deposits along with tubular atrophy.

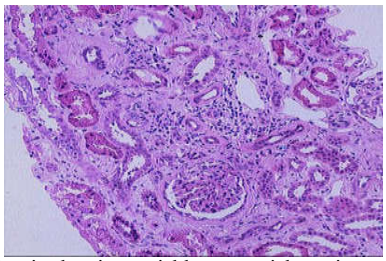

**Figure 3** H&E stain showing variable mesangial matrix expansion, with marked interstitial fibrosis and tubular atrophy, mild chronic interstitial inflammatory infiltrate.

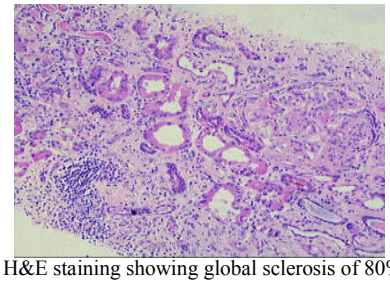

**Figure 4** H&E staining showing global sclerosis of 80% of the glomeruli, the remaining glomeruli show segmental sclerosis, there is mild mesangial cell proliferation, interstitial infiltration by lymphocytes, plasma cells & few neutrophils around the globally sclerotic glomeruli.

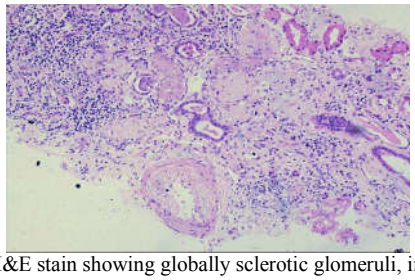

**Figure 5** H&E stain showing globally sclerotic glomeruli, interstitial infiltration around the sclerotic glomeruli, there is medial hypertrophy of the blood vessels & atherometous changes.

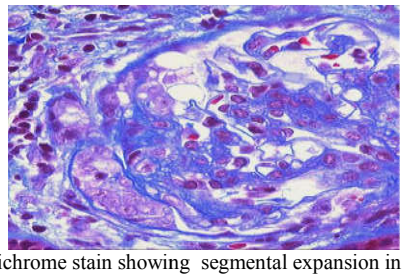

**Figure 6** Trichrome stain showing segmental expansion in mesangial matrix.

Also within this study, 9 patients (27.3%) were diagnosed as ESRD and started renal replacement therapy (RTT) in the form of hemodialysis or transplantation at the time of diagnosis; however the remaining 24 patients (69.7%) were advised to stop anabolic steroids and other supplements, decrease protein intake and were given medical treatment in the form of renin-angiotensin system blockers. Among these 24 patients with a follow up of one year, 3 of them (9.1%) were non compliant and started anabolic steroid reuse again along with high protein diet to maintain their body shape and subsequently started suffering again from higher proteinuria and rising kidney functions and they progressed to ESRD and required RTT; however the remaining patients (21 patients; 63.6%) showed improvement in their kidney functions (mean serum creatinine 250 ummol/l ±63), decrease in proteinuria and weight loss. A statistically significant result (P value <0.05) was revealed upon comparing {body mass index, training experience, DBP, total vitamin D, tubular atrophy and atherosclerotic renal biopsy changes}between patients who developed ESRD and required RTT (Group A) and those patients whom were compliant on medical advice and showed improvement of their kidney functions (Group B) Table(3).

**Table 3** Comparison between patient groups who required renal replacement therapy and those who didn't in reference to different variables.

| | Group A | Group B | P value |
|---|---|---|---|
| BMI kg/m$^2$ | 47.67±1.61 | 45.33±2.57 | 0.008S* |
| Training experience (yrs) | 18.50±3.77 | 14.33±3.45 | 0.003S* |
| DBP mmHg | 109.17±7.93 | 102.14±6.43 | 0.009S* |
| Total vitamin D | 668.33±358.60 | 415.81±303.41 | 0.003S* |

Tubular atrophy was detected in all patients (100%) who developed ESRD(Group A), and presented in 9 patients (42.9%) out of 21 patients who improved(Group B), which was statistically significant (P value <0.05) when comparing between patients of both groups Table (4). Whereas regarding atherosclerotic renal changes; they were detected in all patients who developed ESRD(Group A), and presented in 10 patients (47.6%) out of 21 patients who improved, which was statistically significant as well(P value <0.05) among both groups Table (5).

**Table 4** Renal tubular atrophy in both groups

| | | Outcome | | Total | P value |
|---|---|---|---|---|---|
| | | Group A | Group B | | |
| Tubular atrophy | Normal tubules | 0 | 12 | 12 | 0.001 S* |
| | | 0% | 57.1% | 36.4% | |
| | Tubular atrophy | 12 | 9 | 21 | |
| | | 100.0% | 42.9% | 63.6% | |
| | Total | 12 | 21 | 33 | |

**Table 5** Renal Atherosclerotic changes in both groups.

| | | Outcome | | | P value |
|---|---|---|---|---|---|
| | | Group A | Group B | Total | |
| Atherosclerosis | No atherosclerosis | 0 | 11 | 11 | 0.002 S* |
| | | 0% | 52.4% | 33.3% | |
| | Atherosclerosis | 12 | 10 | 22 | |
| | | 100.0% | 47.6% | 66.7% | |
| Total | | 12 | 21 | 33 | |

**Table 6** Multivariate logistic regression analysis of BMI, DBP, training experience, tubular atrophy and atherosclerotic renal changes

| P value | Exp(B) | 95% C.I.for EXP(B) | |
|---|---|---|---|
| | | Lower | Upper |
| BMI | 0.642NS | 1.262 | 0.474 | 3.355 |
| Training experience | 0.326 NS | 1.193 | 0.839 | 1.697 |
| DBP | 0.260 NS | 1.144 | 0.905 | 1.446 |
| Tubular atrophy | 0.998 NS | 0.000 | 0.000 | . |
| Atherosclerosis | 0.998 NS | 0.000 | 0.000 | . |

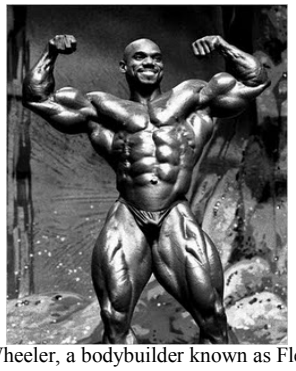

**Figure 7** Kenneth Wheeler, a bodybuilder known as Flex, 1996 (was 290 pounds), lost about 50 pounds, developed chronic kidney disease and had kidney transplantation & eventually was forced to retire in 2003 at age 37.

## DISCUSSION

The anabolic or "muscle-building" effects of (AASs) were first recognized in the 1930s. Initially, testosterone was used for its androgenic (masculinizing) effects in the treatment of hypogonadism. Later, uses included the treatment of anemia, burns, and malnutrition. However, as early as the 1940s, weightlifters started using testosterone for its anabolic (muscle-building) effects to enhance athletic performance. This was followed by the synthesis of new compounds related to testosterone that are longer acting (testosterone enthanate and testosterone cypionate), orally active as (methyl- testosterone and stanozolol), more potent as (nandrolone), having less incidence of conversion to estrogen resulting in less gynecomastia as (methandrostenolone), having fewer androgenic effects as (oxandrolone and oxymetholone), and are more difficult to detect by drug testing (10). By the 1970s, the use of anabolic steroids had become so widespread among amateur athletes, including adolescents, that they were banned from Olympic competition in 1975.The United States Drug Enforcement Agency classified them as scheduled drugs in 1991(11).

Anabolic steroids (AASs) promote growth of skeletal muscle and increase lean body mass (12). They are anti-catabolic and can convert a negative nitrogen balance to a positive balance by improving utilization of dietary protein and increasing protein synthesis(13).Androgen anabolic steroids are taken by athletes to improve performance. In certain sports, such as bodybuilding and power lifting, AASs abuse is quite common. One study of bodybuilders in Sweden found that 75% of competitive bodybuilders and 24% who engaged in bodybuilding solely to improve their sense of well-being used AASs (14).

Focal segmental glomerulosclerosis (FGS) is produced by many mechanisms (15). The etiologic classification of FGS is complicated and somewhat confusing (16). It's a pattern of glomerular injury seen in primary podocytopathies as well as in secondary forms of glomerular injury caused by adaptive responses to elevated glomerular capillary pressures and flow rates. Conditions associated with either decreased nephron number or increased demand on a normal endowment of nephrons will result in increased single nephron GFR. These increased demands first manifest morphologically as glomerular hypertrophy but eventually become maladaptive, producing glomerulosclerosis. Patients with normal numbers of nephrons may develop secondary FSGS as a result of morbid obesity, which causes an increased demand for glomerular filtration that parallels increased body mass (17,18). Secondary forms of FSGS typically have a lower incidence of nephrotic syndrome and a better overall prognosis when compared with primary (idiopathic) FSGS (19). Whereas the mainstay of treatment for primary FSGS is immunosuppressant, secondary (post adaptive) forms of FSGS are treated with RAS blockade and treatment of the underlying cause whenever possible (*e.g.*, weight loss in the obese patient) (20,21).

The mean GFR (MDRD) at the time of presentation in our study was (87.79 mL/min/1.73 $m^2$±26.45), these values can be explained by post adaptive form of FSGS, also our mean serum creatinine was (527.79ummol/dl ±231.002) which is disproportionate with GFR, and can be explained by the high protein intake in diet and also creatine supplement. Post adaptive forms of FSGS are usually due to structural-functional adaptations driven by increased hemodynamic stress on the glomeruli. Animal models suggest that podocyte depletion plays a key role in post adaptive models of FSGS. Increased body mass requires an increase in glomerular filtration. In an attempt to meet these demands, individual glomeruli adapt to hyperfiltration through hypertrophy. Podocytes are terminally differentiated cells that cannot proliferate, and in the process of compensatory glomerular hypertrophy, podocyte connections to the GBM become mechanically strained. If these conditions persist, then podocytes eventually detach from the GBM, leading to development of a segmental scar. (22).

The link between obesity and FSGS is well established (20).The National Institute of Health defines obesity as BMI ≥30 kg/m2. The vast majority of people who meet this criterion have elevated body fat content; however, patients with dramatically increased lean body mass may have a significantly elevated BMI and subnormal body fat content. This study showed that the mean BMI was (46.18 kg/$m^2$ ±2.51), which

concord with several studies in that obesity is a widely recognized risk factor for renal disease and a common cause of secondary FSGS (3). In a univariate analysis, our study revealed that changes in BMI were statistically significant (P value < 0.05)upon comparing patients in both groups in the study, those who developed ESRD and required RTT(mean 47.67 kg/$m^2$±1.61); and those who were compliant to medical advice and showed improving in kidney function (mean 45.33 kg/$m^2$±2.57). Several studies suggest that androgens may exert a direct toxic effect on glomerular cells, leading to mesangial matrix accumulation and podocyte depletion independent of structural-functional adaptations (2).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 (23,24).

Experimental reports in mice have shown its renal toxicity and induction of glomerulosclerosis through increase of renal blood flow and GFR (25). Moreover, it has been shown that its target site was specifically on the glomerular podocyte.In fact, a case report on a FSGS patient with acromegaly who had failed all therapeutic drug regimens improved after treatment with trans-sphenoidal microsurgery of the adenoma (26).In our study, the mean anabolic steroids usage among our bodybuilders was (1827.00 mg/week ±426.668), and for a mean period of (15.85 years ±4.063).These findings were in agreement with those of Leal et al.,which showed that the greater severity of clinical and biopsy findings in bodybuilders suggests that additional factors are modifying the typical clinical and pathologic features seen in purely post adaptive FSGS, leading us to hypothesize that (AASs) abuse has direct nephrotoxic effects (2).

Also our study concur with the study of Herlitz *et al*., that there is an additional process occurring in patients known to abuse anabolic steroids, as they had a higher incidence of nephrotic syndrome at presentation and more advanced histological evidence of glomerulosclerosis, than other patients with secondary FSGS. This is in keeping with the advanced biopsy findings in our patients, and the rapid progression to ESRD requiring RRT(27). As this study showed, the mean 24 hours urinary protein excretion was (19.30 g/24hrs ± 5.241), and the histological findings in our patients revealed that 100% of them showed glomerulosclerosis, glomerulomegaly, 21 patients (63.6%) showed tubular atrophy and interstitial fibrosis, 22 patients showed atherosclerotic changes (66.7%), whereas 6 patients (18.2%) showed calcium deposits, which explains why 9 patients at the time of presentation were diagnosed as ESRD, and required renal RRT.The stoppage of anabolic steroids in this study provided strong evidence that they played a major role in the progress of renal disease, as 21 patients who stopped these drugs and were compliant to medical advice, showed improvement in their kidney function with reduction of proteinuria and their body weight.

As the optimal protein requirement for adults hardly exceeds 0.8 g/kg/day as recommended by the US food and nutrition (23); 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 in acceleration of development of FSGS (24).

Other additional factors that could have exerted stress on glomeruli in this study, was the high protein diet, as our bodybuilders were consuming high protein diet for a long duration of time, with a mean intake of (17.30 g/kg/day ±3.405), which caused marked changes in renal hemodynamics.

Travis et al., showed that mean diastolic blood pressure (DBP) at baseline in FSGS patients with kidney failure was significantly higher, compared to those without (P value<0.0001). This finding meets our results, as mean DBP was (104.70 mmHg ±7.6), and upon comparing DBP in patients who developed ESRD and required RTT (mean 109.17 mmHg ±7.93)with DBP in the other group of patients(mean102.14mmHg±6.43)whom were compliant to medical advice and showed improvement in their kidney functions the result was statistically significant(P<0.05)(28). Tubular atrophy was also a predictor of progress of renal disease and the development of ESRD, as all patients who required RRT showed marked tubular atrophy and interstial fibrosis, in comparison to patients who improved.

## CONCLUSION

The increase in BMI above normal values, the abuse of anabolic steroids, high protein diet and other nutritional supplement carries a great risk for developing ESRD secondary to Focal segmental glomerulosclerosis in bodybuilders. Infact, bodybuilders are exposed to forced muscle gain and hypertensive situations and should be considered a high risk group.

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