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MORFOMETRIJSKA ANALIZA GLOMERULA, KLINIČKI TOK I ISHOD BOLESTI KOD GOJAZNIH I NEGOJAZNIH BOLESNIKA SA FOKALNO SEGMENTNO GLOMERULKLEROZOM

Authors Elena Jordanova†, Radmila Janković§, Radomir Naumović*, Dejan Ćelić++, Bojana Ljubičić−, Sanja Simić-Ogrizović**, Gordana Basta-Jovanović§, Vojnosanitetski pregled (2019); Online First March, 2019.

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MORPHOMETRIC ANALYSIS OF GLOMERULI, CLINICAL FEATURES AND OUTCOME IN OBESE AND NON-OBESE FOCAL SEGMENTAL GLOMERULOSCLEROSIS PATIENTS

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Abstract

**Background/Aim.** In the past three decades focal segmental glomerulosclerosis (FSGS) was commonly regarded as a part of obesity related glomerulopathy (ORG) a distinct entity featuring proteinuria, glomerulomegaly, progressive glomerulosclerosis and renal functional decline. The aims of the present study were to evaluate the glomerular morphometry, clinical features and two years outcome in obese and non-obese FSGS patients. **Methods.** The study included 35 FSGS patients (23 males, age 46.5±15.2 years); divided in two groups: obese (BMI ≥27 kg/m² - 18 patients, age 47.2±15.5 years) and non-obese (BMI <27 kg/m - 17 patients, age 45.7±15.2 years). The serum concentrations of proteins, albumin, cholesterol, triglyceride and creatinine were determined at the time the biopsy, 6, 12, 24 months after the biopsy. Formulas Cockcroft- Gault (BMI <27 kg/m) and Cockcroft-Gault..(BMI ≥27 kg/m) were calculated. Glomerular radius (GR), glomerular volume (GV) and glomerular density (GD) were compared morphometrically between two groups. **Results.** At the time of kidney biopsy and 6 months later the obese had significantly lower GFR compared to non-obese. After 24 months follow-up there wasn’t any difference between groups. Obese had significantly higher GR (109.44±6,03 μm vs 98.53±14,38 μm) and GV (3.13±0.49 x10⁶ μm³ vs 2.26±0.83 x10⁶ μm³), only midly lower GD (1.91±0.39/mm² vs 1.95±0.61/mm²) compared to non-obese. Significant positive association between GV and BMI (r=0.439) was found. After 12 months follow-up significantly higher percentage of non-obese patients reached complete remission (71.4% vs 37.5%) compared to obese (χ²=0.041), but after 24 months there were no significant difference. **Conclusion.** Obese patients at the time of kidney biopsy and 6 months later had already the significant lower kidney function compared to non-obese. However, after 12 and 24 months, this difference was still lower and without significance as well as after 24 months percentage of patients with complete remission between two groups.

**Key words:**
FSGS, ORG, morphometry, remission, patient outcome.

Apstrakt

**Uvod/Cilj.** U poslednje tri decenije fokalno segmentna glomeruloskleroza (FSGS) je predstavljena kao oblik gojaznošću uslovljene glomerulopatije (GUG) poseban entitet karakterisan proteinurijom, glomerulomegalijom, progresivnom glomerulosklerozom i smanjenjem bubrežne funkcije. Cilj ove studije je odrediti morfometriju glomerula, klinički tok i ishod nakon dve godine praćenja gojaznih i negojaznih FSGS pacijenata. **Metode.** Studija je obuhvatila 35 FSGS pacijenata (23 muškaraca, starosti 46,5±15,2 godina); podeljenih u dve grupe: gojazni (BMI ≥27 kg/m - 18 bolesnika, starosti 47,2±15,5 godina) i negojazni (BMI <27 kg/m - 17 bolesnika, starosti 45,7±15,2 godina). Merena je serumsk koncentracija proteina, albumina, holesterola, triglicerida i kreatinina u momentu biopsije, 6, 12, 24 meseca nakon biopsije. Jačine glomerulske filtracije (JGF) procenjena je pomoću formula: Cockcroft-Gault (BMI <27 kg/m) i Cockcroft-Gault..(BMI ≥27 kg/m). Morfometrijski su poređeni poluprečnik
glomerula (PG), volumen glomerula (VG) i gustina glomerula (GG) između dve grupe.

Rezultati. U vreme biopsije i nakon 6 meseci gojazni su imali značajno nižu JGF u poređenju sa negojaznim. Nakon 24 meseca praćenja nije bilo razlike između grupa. Gojazni su imali statistički značajno viši PG (109,44±6,03 μm vs 98,53±14,38 μm) i VG (3,13±0,49x10 μm vs 2,26±0,83x10 μm), ali nižu GG bez značaja (1,91±0,39/mm vs 1,95±0,61/mm) u poređenju sa negojaznim. Pronađena je značajno pozitivna korelacija između VG i BMI (r=0,439). Nakon 12 meseci praćenja značajno viši procenat negojaznih bolesnika ulazi u kompletnu remisiju (71,4% vs 37,5%) u poređenju sa gojaznim (χ²=0,041), ali, nakon 24 meseca nije bilo značajne razlike.

Zaključak. Gojazni pacijenti su u vreme biopsije bubrega i nakon 6 meseci praćenja imali značajno nižu JGF u poređenju sa negojaznim. Međutim, nakon 12 i 24 meseca, ova statistički značajna razlika se gubi. Takođe, posle 24 meseca praćenja nije bilo značajne razlike u procentu bolesnika sa kompletnom remisijom nefrotskog sindroma između dve grupe.

Ključne reči: FSGS, GUG, morfometrija, remisija, ishod bolesti.

Introduction

Focal segmental glomerulosclerosis (FSGS), with the increasing prevalence worldwide, describes both a common lesion in progressive kidney disease, and a disease characterized by marked proteinuria and podocyte injury (1). Thus, FSGS defines a number of clinical and pathological syndromes that may be primary (idiopathic) or secondary that are mediated by adaptive structural-functional responses. These adaptive forms include not only patients with congenital anomalies but also patients with acquired reduction of the functional nephron mass, while other secondary forms are associated with hemodynamic stress placed on an initially normal nephron population (hypertension, atheroembolismus, sickle cell anemia, increased lean body mass and obesity) (2). In the past three decades kidney biopsy findings of focal and segmental glomerulosclerosis was commonly regarded as a part of obesity related glomerulopathy (ORG), a distinct entity featuring proteinuria, glomerulomegalia, progressive glomerulosclerosis and progressive renal functional decline. This pathohistological entity is described as a secondary form of glomerular disease in obese patients with morphological characteristics of FSGS and enlargement of the glomeruli, or only by enlargement of the glomeruli. Fortunately, not all the obese persons develop ORG (3, 4). A typical clinical feature of ORG is medium to massive proteinuria without reducing serum albumin levels, or without the development of nephrotic syndrome. This clinical feature is important in the differential diagnosis of ORG from primary FSGS in which massive proteinuria is followed by the development of full blown nephrotic syndrome (5). Also, progression of ORG to end stage renal disease (ESRD) is slower than in primary FSGS (5 years renal survival 75% vs 50%), even though 10% to 30% of ORG patients start the dialysis treatment (4, 6, 7, 8).

During the last 15 years there has been an equivalent dramatic rise in the prevalence of obesity and ESRD increasing the interest on the role of obesity - related kidney disease. Obesity not only increases the risk of preexisting renal disease progression, but is itself also an independent risk factor of renal injury (9).

Usually, in everyday clinical practice it is not easy to distinguish primary from secondary forms of FSGS, specially in obese patients. At the one side in ORG patients the main histopathological features is FSGS with subtle differences from primary FSGS (perihilar FSGS variant, glomerulomegalia, foot process effacement usually in less than 50% of glomerular
surface area) (4). On the other side, obesity can accelerate the progression of already existed renal injury.

Having in mind all of the previously mentioned, the aims of the present study were to evaluate the glomerular morphometry and clinical features in obese and non-obese FSGS patients and to assess the implication of obesity and glomerulomegaly on 24 months FSGS patients’ outcome.

**Methods**

**Patients**

The study included 35 adult FSGS patients (23 males) with mean age of 46.5± 15.2 years (range 21-72 years). Indications for the kidney biopsy were: nephrotic syndrome, pathological proteinuria without nephrotic syndrome or abnormal urinary sediment. Renal biopsies from patients with secondary FSGS other than ORG and with diabetic nephropathy were cautiously excluded.

Obesity was defined as BMI ≥ 27kg/m² and patients were divided into two groups: obese with BMI ≥27kg/m² (18 patients, 14 males, mean age 47.2± 15.5, mean BMI 32.41± 3.47kg/m²) and non-obese with BMI< 27 kg/m² (17 patients, 9 males, mean age 45.7± 15.2, mean BMI 23.99± 2.11 kg/m²).

The study protocol was in conformity with ethical guidelines, approved by School of Medicine, University of Belgrade ethical comity (number 29/III-9) and informed consent was obtained from each participant.

After the histopathological diagnosis, the participants were treated according to established protocols for FSGS, some of them received oral corticosteroid therapy 1mg/kg BW with symptomatic therapy and some of them were only symptomatically treated. The symptomatic therapy was included angiotensin- converting enzyme- ACE inhibitors or angiotensin receptor type 1 blockers- ARBs (10). The special nutrition diet for obese patients wasn’t recommended strictly. All the patients were carefully follow-up 6, 12 and 24 months after the kidney biopsy. Complete remission of nephrotic syndrome was defined with daily proteinuria less than 1 g/day with normalization of protein, albumin and lipids serum concentration and partial remission with daily proteinuria between 1-3 g/day.

**Laboratory methods**

Hematological, as well as biochemical analyses, were done at the time of kidney biopsy, as well as 6, 12 and 24 months after the biopsy. A hematological analyzer (The Beckman Coulter HmX) was used to provide a complete hematological profile. The serum concentration of protein, albumin, cholesterol, triglyceride and creatinine were determined on biochemical analyzer DXC-800 Beckman Coulter. The serum creatinine level was measured according to the Jaffe method. The proteinuria was determined by spectrophotometry with pirogal red. Only samples with a sterile urine culture were processed. Urine sediments with more than 3 RBC/hpf or 5 WBC/hpf were defined as clinically significant erythrocyturia or leukocyturia.

The estimated glomerular filtration rate (eGFR) was calculated according to formulas:

A. Cockcroft-Gault- for participants with BMI <27 kg/ m (11)

\[
\text{eGFR} = \frac{((140-\text{age}) \times \text{body weight}))}{(72 \times \text{serum creatinine})} \times 0.85 \quad \text{(correction factor for female)}
\]

B. Cockcroft-Gault, - for participants with BMI ≥27 kg/m
Cockcroft Gault. = (140 - age) x LBW / serum creatinine x correction factor
(correction factor for male =1.23; correction factor for female = 1.04)

LBW = 9720 x body weight/ 6680+ 216 x BMI for male
LBW= 9720 x body weight/ 8780+ 244 x BMI for female (12)

The morphometric analysis of glomeruli

A percutaneous biopsy of the inferior pole of left kidney was done under ultrasound control. The samples were relatively equal in the number of glomeruli and approximately the same size. All tissue samples were routinely processed, cut in 5 μm thick sections and stained using Periodic Acid-Schiff method (PAS). Whole tissue sections were analyzed (Olympus BX51Tokyo, Japan) and captured (Olympus DP70 camera) at magnification x12.5. The number of glomeruli in each section is determined. All present glomeruli were also captured at magnification x400. Microphotographies were analyzed using a computer-assisted image analysis system, ImageJ (13).

The volumes of all glomeruli contained entirely within the serially sectioned material were measured in each case (N= 20± 10 glomeruli). Glomerular volume was calculated by the maximal profile area methodMPA (V...) by identifying the profile of each glomerulus with the largest area A... An ideal radius r o was derived from the area of the largest profile based on the assumption that the profile was a circle:

\[ r_o = \sqrt{A_{\text{max}}/\pi} \]

The volume corresponding to the MPA was then calculated based on the assumption that the glomerulus was a sphere:

\[ V_{\text{gma}} = \frac{4}{3} \pi r_o^3 \]

Glomerular density was expressed as the average area of tissue in the biopsy sample per one glomeruli in a group of obese and non- obese patients (14).

2.3 Statistics

Data are presented as mean values and standard deviation (SD). The Kolmogorov- Smirnov test was used to check the normal distribution of the variables. Data were analyzed using Student's t test (or Mann Whitney due to distribution) and Pearson's χ² test (for nominal data). Relationships between variables were estimated using using Pearson’s parametric correlation method. Statistical analysis is performed using SPSS software 17.0. Statistical significance is defined as the conventional p-value with the effects being considered significant at p < 0.05.

Results

The study included 35 FSGS patients. The patients were divided into two groups: obese with BMI ≥27kg/m² (18 patients) and non-obese with BMI< 27 kg/m² (17 patients). There was no significant difference between the groups in age and gender. In both groups, nephrotic syndrome was the major indication for kidney biopsy (72.2% obese vs 70.6% non-obese); all of the patients had some levels of pathological proteinuria (Table 1).

Table 2 shows clinical and laboratory data for two patient groups at the time of kidney biopsy, 6, 12 and 24 months after the biopsy. At the time of kidney biopsy, the obese patients had only significantly higher serum creatinine concentration and significantly lower eGFR compared to non- obese patients. In other measured parameters there were no significant difference. Six months later, eGFR was still lower in obese than non-obese patients, daily proteinuria was lower
in non-obese patients but not significantly and there were no other difference between groups. Twelve months after the kidney biopsy the non-obese patients had significantly lower daily proteinuria as well as cholesterol serum concentration and higher serum protein and albumin concentration compared to obese patients. After 24 months of follow-up we could not found any statistically significant difference in examined variables between the groups (Table 2).

Figure 1 presents mean glomerular volume and density in both obese and non-obese patients. The obese patients had not only significantly higher glomerular radius (109.44±6.03μm vs 98.53±14.38μm) compared to non-obese (t = 2.729; p = 0.011) but also glomerular volume (Figure 1 A) (3.13±0.49x10^6 μm^3 vs 2.26±0.83x10^6 μm^3) in comparison with non-obese patients (t = 3.545; p = 0.001). Obese patients had lower glomerular density (1.91±0.39/mm^2 vs 1.95±0.61/mm^2) but without significant difference (Figure 1 B).

Significantly positive association between mean glomerular volume and BMI was found (r = 0.439; p = 0.008) (Figure 2). There is no significant correlations between glomerular volume and daily proteinuria as well as with age, gender and eGFR.

After 6 months of follow-up there was no significant difference in patients outcome between obese and non-obese patients. Complete remission reached 23.1% of obese and 36% of non-obese patients, while partial remission was reached in 15.4% of obese and 9.1% of non-obese patients. Without remission were 61.5% of obese and 54.5% of non-obese patients. After 12 months of follow-up significant higher percentage of non-obese patients reached complete remission (71.4% vs 37.5%) compared to obese patients (χ^2 = 0.041; p = 0.041). After 24 months follow-up there was no significant difference in patients outcome between obese and non-obese patients. Complete remission reached the same percentage of obese and non-obese patients (33.3%), partial remission was accomplished in 11.1% of obese and 16.7% of non-obese patients. Almost half of examined patients in both groups were without remission after two years of follow-up (55.6% vs 50%) (Figure 3).

Discussion

The study included 35 patients with FSGS, 18 obese patients with BMI ≥27kg/m^2 and 17 non-obese patients with BMI <27 kg/m. In both study groups, nephrotic syndrome was the major indication for kidney biopsy (72.2% obese vs 70.6% non-obese). In Danilewicz et al. (15) study almost the same huge percent of ORG and primary FSGS patients had nephrotic syndrome as major indication for the kidney biopsy.

In the present study morphometric glomerular analysis in FSGS patients showed that obese patients had significantly higher radius and significantly higher glomerular volume as well as lower glomerular density, although without significance, in comparison with non-obese patients. Morphometric study on glomerular parameters confirmed the earlier findings of Praga et al. (7) that glomerulomegalia and lower glomerular density in obese FSGS was significantly increased compared to non-obese patients as well as the results of Kambham et al. (6) and Danilewicz et al. (15). Although pathogenesis of ORG was not clearly defined, it has been shown that the enlarged glomeruli, found in animal models of rats, may have a close relationship with intraglomerular hyperfiltration and hypertension. It has been suggested that relative reductions in the number of nephrons, as a result of increases in body size, can play a role in the pathogenesis of ORG. Decreased nephron mass in experimental animal models is clinically analogous to congenital renal agenesis or nephrectomy (3). Praga et al. (16) showed that obese patients could develop significant proteinuria after unilateral nephrectomy. Fukuda et al. (17) demonstrated that
hypertrophy of the glomerular podocytes could be a compensatory mechanism for renal injury associated with obesity. ORG. It could be suggested that appearance of FSGS in obese patients depends not only on obesity-related increases in glomerular volume but on podocyte hypertrophic responses. Also relative reduction in the coating area of glomerular podocytes on the glomerular surface could be found in ORG patients.

In the present study, the level of proteinuria was the same in both groups at the time of kidney biopsy. Six months later daily proteinuria was lower in non-obese patients but without significance, 12 months after biopsy proteinuria was significantly lower and protein and albumin serum concentrations were higher than in the obese patients. Therefore, significantly higher percentage of non-obese patients had complete remission compared to obese patients. However, 24 months after the kidney biopsy there were no statistical difference in percentages of patients with complete remission.

Forty years ago, association between proteinuria and obesity was first reported (18). In the 1980s there were several case reports and autopsy series study of ORG (19, 20). In 2001, Kabham et al. (6) published the first large study on this entity. In obese patients, the degree of proteinuria can be variable, but it can reach nephrotic range (≥3.5g/24h) in a significant number of cases. Interestingly, the obese patients with ORG hardly ever develop hypoproteinemia, hypoalbuminemia, oedema or the other typical findings of nephrotic syndrome even in presence of massive proteinuria (5, 7). This occurrence could be very useful in differential diagnosis with other proteinuric renal diseases (idiopathic FSGS, membranous nephropathy, minimal change disease) that can also affect obese patients (7). The reason why ORG patients do not develop oedema and have a lower incidence of nephrotic syndrome when compared to idiopathic FSGS patients is unclear. One of the explanation could be the slow progression of proteinuria in ORG patients that may allow the development of hepatic compensation for protein synthesis, and the other one may relate to lower grade of podocyte injury, selectivity of proteinuria and ability of the tubules to reabsorb and catabolize the filtered protein in a different manner (7). Several studies have shown that weight loss either induced by low calorie diets, physical exercise or bariatric surgery (21) as well as pharmacotherapy (ACE inhibitors or AT1 receptor antagonists) are associated with important antiproteinuric effect (22). At the present study, at the time of kidney biopsy, the same percentage of obese and non-obese patients had nephrotic range proteinuria with full blown nephrotic syndrome.

In the current study clinical and laboratory analyses showed that obese patients at the time of kidney biopsy and 6 months later had significantly lower kidney function than non-obese patients; but after 12 and 24 months, with the progression of chronic kidney disease in non-obese patients, there were no significant difference between groups. Also, only 12 months after biopsy significantly more patients in non obese patient group had complete remission compared to obese patients, but after 24 months there were no differences in clinical outcome. Some studies pointed that obesity can accelerate the progression of chronic kidney disease. Bonet et al. (23) reported that a BMI >25 kg/m² or more is a significant risk factor for the progression of chronic renal failure in IgA nephropathy patients and Morales et al. (24) found that weight loss is effective for attenuating the progressive loss of the kidney function in obese patients with diabetic and non-diabetic kidney diseases. Bertoux et al. (25) have demonstrated in a cohort of 331 IgA nephropathy patients that normal or elevated BMI status at the time of biopsy was associated with a worse presentation at diagnosis in the overweight/obese IgA nephropathy patients: more patients with hypertension; more patients with proteinuria ≥1 g/day. Also, the absolute renal risc (ARR) score for dialysis/death was also significantly worse in obese patients.
compared to non-obese. As expected, the final outcome was globally worse in obese Ig A nephropathy patients. Praga et al. (7) followed patients 5 and 10 years after the renal biopsy and the conclusion was that the estimated probability of renal survival in obese FSGS patients, was significantly higher compared to non-obese FSGS patients. On the other hand, some studies revealed slower chronic kidney progression in obese patients with FSGS compared to non-obese FSGS patients (5,7).

Conclusion

Morphometric analysis of glomeruli, clinical features and treatment outcome in obese and non-obese FSGS patients showed that obese patients had significantly higher glomerular volume and insignificantly lower glomerular density. Obese patients at the time of kidney biopsy and after 6 month follow-up had significantly lower kidney function compared to non-obese patients. However, after 12 and 24 months, with the progression of chronic kidney disease in non-obese patients, this difference was lower and without significance. It can be speculated that the progression of FSGS in obese patients is slower than in non-obese patients. The lack of the present study is short time follow-up period and is in extension.

References

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<th>Groups</th>
<th>Age (years)</th>
<th>Gender m/f</th>
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Table 2 Clinical and laboratory data in two patient groups

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<th>Obese (mean ± SD)</th>
<th>Non Obese (mean ± SD)</th>
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<tr>
<td>Hemoglobin (g/l)</td>
<td>136.33± 25.20</td>
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<tr>
<td>Serum protein (g/l)</td>
<td>54.14± 11.29</td>
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<tr>
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<td>8.22± 2.68</td>
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<td>Proteinuria (g/day)</td>
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6 months after kidney biopsy

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<td>Serum creatinine (μmol/l)</td>
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<td>Proteinuria (g/day)</td>
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12 months after kidney biopsy

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<td>56.14± 5.57</td>
<td>63.65± 2.63</td>
<td>0.000-</td>
</tr>
<tr>
<td>Serum albumin (g/l)</td>
<td>33.05± 4.60</td>
<td>36.50± 2.88</td>
<td>0.013-</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>6.88± 1.72</td>
<td>5.76± 1.39</td>
<td>0.043-</td>
</tr>
<tr>
<td>Triglyceride (mmol/l)</td>
<td>2.38± 1.64</td>
<td>2.23± 0.91</td>
<td>0.745-</td>
</tr>
<tr>
<td>Serum creatinine (μmol/l)</td>
<td>107.72± 49.98</td>
<td>95.09± 31.02</td>
<td>0.379-</td>
</tr>
<tr>
<td>Cockcroft- Gault (ml/min)#</td>
<td>63.33± 27.16</td>
<td>93.83± 35.52</td>
<td>0.112-</td>
</tr>
<tr>
<td>Proteinuria (g/day)</td>
<td>4.15± 3.13</td>
<td>1.49± 1.86</td>
<td>0.005-</td>
</tr>
</tbody>
</table>

24 months after kidney biopsy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Obese (mean ± SD)</th>
<th>Non Obese (mean ± SD)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/l)</td>
<td>138.00± 13.36</td>
<td>143.23± 8.99</td>
<td>0.190</td>
</tr>
<tr>
<td>Serum protein (g/l)</td>
<td>62.97± 9.28</td>
<td>62.48± 15.44</td>
<td>0.911</td>
</tr>
<tr>
<td>Serum albumin (g/l)</td>
<td>35.68± 5.22</td>
<td>35.58± 9.06</td>
<td>0.971</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>6.40± 1.46</td>
<td>6.06± 1.17</td>
<td>0.461</td>
</tr>
<tr>
<td>Triglyceride (mmol/l)</td>
<td>2.76± 1.02</td>
<td>2.70 ± 1.13</td>
<td>0.870</td>
</tr>
<tr>
<td>Serum creatinine (μmol/l)</td>
<td>115.20 ± 80.92</td>
<td>109.76 ± 44.29</td>
<td>0.809</td>
</tr>
<tr>
<td>Cockcroft- Gault (ml/min)#</td>
<td>61.87± 32.73</td>
<td>74.0± 35.19</td>
<td>0.711</td>
</tr>
<tr>
<td>Proteinuria (g/day)</td>
<td>4.08± 5.65</td>
<td>2.23± 1.92</td>
<td>0.210</td>
</tr>
</tbody>
</table>

*p < 0.05  **p < 0.01

# for obese patients -Cockcroft- Gault_{LBW} (ml/min)
Fig. 1— Mean glomerular volume (A) and mean glomerular density (B) in obese and non-obese FSGS patients
Fig. 2 – Correlation between glomerular volume and body mass index (BMI).
Fig. 3 – Percentage of obese and non-obese patients with complete remission of nephrotic syndrome after 6, 12 and 24 months of follow-up.

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