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Morphometric analysis of glomeruli, clinical features and outcome in obese and non-obese patients with focal segmental glomerulosclerosis patients

Morfometrijska analiza glomerula, klinički tok i ishod bolesti kod gojaznih i negojaznih bolesnika sa fokalno segmentnom glomerulosklerozom

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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 a decline of renal function. The present study aimed to evaluate the glomerular morphometry, clinical features, and a two-year outcome in the obese and non-obese FSGS patients. **Methods.** The study included 35 FSGS patients (23 males, aged 46.5 ± 15.2 years) divided into two groups: obese [body mass index (BMI) ≥ 27 kg/m^2 (18 patients, aged 47.2 ± 15.5 years)] and non-obese [BMI < 27 kg/m^2 (17 patients, aged 45.7 ± 15.2 years)]. The serum concentrations of proteins, albumin, cholesterol, triglyceride, and creatinine were determined at the time of the biopsy, and 6, 12, and 24 months after the biopsy. Cockcroft-Gault (BMI < 27 kg/m^2) and Cockcroft-Gault$_{LBW}$ (BMI ≥ 27 kg/m^2) formulas were calculated. Glomerular radius (GR), glomerular volume (GV), and glomerular density (GD) were compared morphometrically between the two groups. **Results.** At the time of the kidney biopsy and 6 months later, the obese had significantly lower glomerular filtration rate (GFR) compared to the non-obese. After 24 months of follow-up, there were not any differences between the groups. The obese had a significantly higher GR (109.44 ± 6.03 μm vs. 98.53 ± 14.38 μm) and GV (3.13 ± 0.49 × 10^6 μm^3 vs. 2.26 ± 0.83 × 10^6 μm^3), and only slightly lower GD (1.91 ± 0.39/mm^2 vs. 1.95 ± 0.61/mm^2) compared to the non-obese. A significant positive association between GV and BMI (r = 0.439) was found. After 12 months of follow-up, a significantly higher percentage of the non-obese patients reached complete remission compared to the obese (71.4% vs. 37.5%, respectively; $p = 0.041$), but after 24 months there were no significant differences. **Conclusion.** Obese patients, at the time of the kidney biopsy and 6 months later, had already a significantly lower kidney function compared to the non-obese ones. However, 12 and 24 months after, this difference was not statistically significant. Also, 24 months after, there was no significant difference between the two groups in the percentage of patients with complete remission of the nephrotic syndrome.

Key words:
biopsy; glomerular filtration rate; glomerulosclerosis, focal segmental; kidney glomerulus; obesity; risk assessment; treatment outcome.

Apstrakt

Uvod/Cilj. U poslednje tri decenije fokalno segmentna glomeruloskleroza (FSGS) je predstavljena kao oblik glomerulopatije uslovljene gojaznošću (GUG), poseban entitet karakterisan proteinurijom, glomerulomegalijom, progresivnom glomerulosklerozom i smanjenjem bubrežne funkcije. Cilj ove studije bio je odrediti morfometriju glomerula, klinički tok i ishod nakon dve godine praćenja gojaznih i negojaznih FSGS bolesnika.

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Metode. Studija je obuhvatila 35 FSGS bolesnika (23 muškaraca, starosti od 46,5 ± 15,2 godina), podeljenih u 47,2 ± 15,5 godina] i negojazni [BMI < 27 kg/m² (17 bolesnika, starosti od 45,7 ± 15,2 godina)]. Merena je serumska koncentracija proteina, albumina, holesterola, triglicerida i kreatinina u momentu biopsije, kao i 6, 12 i 24 meseca nakon biopsije. Jačina glomerulske filtracije (JGF) procenjena je pomoću formula Cockcroft-Gault (BMI < 27 kg/m²) i Cockcroft-Gault$_{LBW}$ (BMI ≥ 27 kg/m²). Između dve grupe morfometrijski su poređeni poluprečnik glomerula (PG), volumen glomerula (VG) i gustina glomerula (GG) **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 ± 0,49 x 10^6 μm³ vs. 2,26 ± 0,83 x 10^6 μm³), ali nižu GG bez značajne razlike u poređenju sa negojaznim (1,91 ± 0,39/mm² vs. 1,95 ± 0,61/mm²). dve grupe: gojazni [body mass index (BMI) – indeks telesne mase ≥ 27 kg/m² – 18 bolesnika, starosti od Pronađena je značajna, pozitivna korelacija između VG i BMI (r = 0,439). Nakon 12 meseci praćenja, značajno viši procenat negojaznih bolesnika ušlo je u kompletnu remisiju u poređenju sa gojaznim (71,4% vs. 37,5%; p = 0,041), ali, nakon 24 meseca nije bilo značajne razlike između grupa. **Zaključak.** Gojazni bolesnici su u vreme biopsije bubrega i nakon 6 meseci praćenja imali značajno nižu JGF u poređenju sa negojaznim bolesnicima. Međutim, nakon 12 i 24 meseca, ova statistički značajna razlika se izgubila. Takođe, posle 24 meseca praćenja nije bilo značajne razlike između dve grupe u procentu bolesnika sa kompletnom remisijom nefrotskog sindroma.

Ključne reči:
biopsija; glomerulska filtracija, brzina; glomeruloskleroza, fokalna, segmentna; bubreg, glomerul; gojaznost; rizik, procena; lečenje, ishod.

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 several clinical and pathological syndromes that may be primary (idiopathic) or secondary, mediated by adaptive structural-functional responses. These adaptive forms include not only patients with congenital anomalies, but also patients with an acquired reduction of the functional nephron mass. Other secondary forms are associated with hemodynamic stress placed on an initially normal nephron population (hypertension, atheroembolism, sickle cell anemia, increased lean body mass, and obesity) [2]. In the past three decades, kidney biopsy findings of focal and segmental glomerulosclerosis were commonly regarded as a part of the obesity-related glomerulopathy (ORG), a distinct entity featuring proteinuria, glomerulomegaly, 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 obese persons develop ORG [3, 4]. A typical clinical feature of ORG is medium to massive proteinuria without reducing serum albumin levels or without developing 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]. Moreover, the 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, 6–8].

During the last 15 years, there has been an equivalent dramatic rise in the prevalence of obesity and ESRD, increasing the interest in the role of obesity-related kidney disease. Not only does obesity increase the risk of preexisting renal disease progression but is also in itself 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, especially in obese patients. On the one hand, the main histopathological features in ORG patients are FSGS with subtle differences from primary FSGS (perihilar FSGS variant, glomerulomegaly, foot process effacement usually in less than 50% of glomerular surface area) [4]. On the other hand, obesity can accelerate the progression of an already existing renal injury.

Methods

Patients

The study included 35 adult FSGS patients (23 males) with a mean age of 46.5 ± 15.2 years (range 21–72 years). Indications for kidney biopsy were: nephrotic syndrome, pathological proteinuria without the 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 ≥ 27 kg/m² and patients were divided into two groups: obese with BMI ≥ 27 kg/m² (18 patients, 14 males, mean age 47.2 ± 15.5, mean BMI 32.41 ± 3.47 kg/ 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 conformed with ethical guidelines, approved by the Faculty of Medicine, Belgrade University Ethics Committee (number 29/III-9), and informed consent was obtained from each participant.

After the histopathological diagnosis, the participants were treated according to the established protocols for FSGS. Some of them received oral corticosteroid therapy 1 mg/kg with symptomatic therapy, and some of them were only

symptomatically treated. The symptomatic therapy included angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor type 1 blockers (ARBs) [10]. The special nutrition diet for obese patients was not strictly recommended. All the patients were carefully followed up 6, 12, and 24 months after the kidney biopsy. Complete remission of the 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 was determined on the biochemical analyzer DXC 800, Beckman Coulter. The serum creatinine level was measured according to the Jaffe method. The proteinuria was determined by spectrophotometry with the pyrogall red protein assay. Only samples with a sterile urine culture were processed. Urine sediments with more than 3 red blood cells (RBC)/hpf or 5 white blood cells (WBC)/hpf were defined as clinically significant erythrocyturia or leukocyturia.

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

A.Cockcroft-Gault – for participants with BMI < 27 kg/m^2 [11]:

eGFR = [(140–age) × body weight/(72 × serum creatinine)] × 0.85 (correction factor for female);

B.Cockcroft-Gault$_{LBW}$ – for participants with BMI ≥ 27 kg/m^2:

Cockcroft Gault$_{LBW}$ = (140–age) × lean body weight (LBW)/serum creatinine × correction factor (correction factor for male = 1.23; correction factor for female = 1.04)

Lean body weight (LBW) = 9720 × body weight/6680 + 216 × BMI for male;

LBW = 9720 × body weight / 8780 + 244 × BMI for female [12].

The morphometric analysis of glomeruli

A percutaneous biopsy of the inferior pole of the left kidney was done under ultrasound control. The samples were relatively equal in the number of glomeruli and approximately of the same size. All tissue samples were routinely processed, cut into 5 μm thick sections, and stained using the Periodic Acid-Schiff method (PAS). Whole tissue sections were analyzed (Olympus BX51Tokyo, Japan) and captured (Olympus DP70 camera) at magnification x 12.5. The number of glomeruli in each section is determined. All present glomeruli were also captured at magnification × 400. Microphotographs 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 (MPA) method (V_{GMA}) identifying the profile of each glomerulus with the largest area. An ideal radius r_o was derived from the area of the largest profile (APmax) based on the assumption that the profile was a circle:

$$r_o = \sqrt{APmax/\pi}.$$

The volume corresponding to the MPA (V_{GMA}) was then calculated based on the assumption that the glomerulus was a sphere:

$$V_{GMA} = 4/3\ \pi\ r_o^3.$$

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

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 χ^2 test (for nominal data). Relationships between variables were estimated 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 ≥ 27 kg/m^2 (18 patients) and non-obese with BMI < 27 kg/m^2 (17 patients). There was no significant difference between the groups in age and gender. In both groups, the 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 1

Age, gender, and indications for kidney biopsy in two patient groups

Groups	Age (years) mean ± SD	Gender (m/f), n	Proteinuria n (%)	Eritrocyturia and proteinuria n (%)	*Syndroma nephroticum* n (%)
Obese	47.2 ± 15.5	14/ 4	4 (22.2)	1 (5.6)	13 (72.2)
Non-obese	45.7 ± 15.2	9/ 8	3 (17.6)	2 (11.8)	12 (70.6)
Total	45.5 ± 15.2	23/ 12	7 (20.0)	3 (8.6)	25 (71.4)
p	0.773	0.212		0.783	

m – male; f – female; SD – standard deviation.

Table 2 shows clinical and laboratory data for the two patient groups at the time of kidney biopsy, and 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 the non-obese patients. In other measured parameters, there were no significant differences. Six months later, eGFR was still lower in the obese than non-obese patients, daily proteinuria was lower in the non-obese patients but not significantly, and there were no other differences between the 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 concentrations compared to the obese patients. After 24 months of follow-up, no statistically significant difference in the examined variables between the groups could be found (Table 2).

Table 2

Clinical and laboratory data in two patient groups

Variable before kidney biopsy	Obese (mean ± SD)	Non obese (mean ± SD)	*p*
Hemoglobin (g/L)	136.33 ± 25.20	135.35 ± 21.56	0.903
Serum protein (g/L)	54.14 ± 11.29	56.72 ± 9.95	0.556
Serum albumin (g/L)	27.16 ± 10.40	29.11 ± 9.02	0.559
Cholesterol (mmol/L)	8.22 ± 2.68	7.47 ± 2.27	0.381
Triglyceride (mmol/L)	2.96 ± 1.48	2.54 ± 1.36	0.398
Serum creatinine (μmol/L)	144.83 ± 84.98	95.52 ± 43.22	0.040
Cockcroft- Gault (mL/min)#	62.22 ± 31.30	95.01 ± 49.23	0.032
Proteinuria (g/day)	8.29 ± 6.73	7.17 ± 7.83	0.654
6 months after kidney biopsy			
Hemoglobin (g/L**)**	138.05 ± 20.81	140.26 ± 15.49	0.725
Serum protein (g/L)	54.69 ± 10.09	57.76 ± 8.22	0.332
Serum albumin (g/L)	31.89 ± 7.20	34.91 ± 6.35	0.198
Cholesterol (mmol/L)	7.57 ± 2.09	7.70 ± 1.68	0.843
Triglyceride (mmol/L)	2.56 ± 0.95	3.03 ± 1.31	0.237
Serum creatinine (μmol/L)	105.33 ± 69.54	82.21 ± 31.49	0.218
Cockcroft-Gault (mL/min)#	67.9 ± 29.07	114.4 ± 40.03	0.009
Proteinuria (g/day)	5.04 ± 4.69	3.41 ± 3.60	0.259
12 months after kidney biopsy			
Hemoglobin (g/L)	138.00 ± 13.36	143.23 ± 8.99	0.190
Serum protein (g/L)	62.97 ± 9.28	62.48 ± 15.44	0.911
Serum albumin (g/L)	35.68 ± 5.22	35.58 ± 9.06	0.971
Cholesterol (mmol/L)	6.40 ± 1.46	6.06 ± 1.17	0.461
Triglyceride (mmol/L)	2.76 ± 1.02	2.70 ± 1.13	0.870
Serum creatinine (μmol/L)	115.20 ± 80.92	109.76 ± 44.29	0.809
Cockcroft- Gault (mL/min)#	61.87 ± 32.73	74.0 ± 35.19	0.711
Proteinuria (g/day)	4.08 ± 5.65	2.23 ± 1.92	0.210

 # for obese patients – Cockcroft-Gault$_{LBW}$ (mL/min).

Figure 1 presents mean glomerular volume and density in both obese and non-obese patients. Not only did the obese patients have significantly higher glomerular radius (109.44 ± 6.03 μm vs. 98.53 ± 14.38 μm) compered to the non-obese ones (t = 2.729; p = 0.011) but they also had higher glomerular volume (Figure 1 A) (3.13 ± 0.49 x 10^6 μm^3 vs. 2.26 ± 0.83 x 10^6 μm^3) in comparison with the 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).

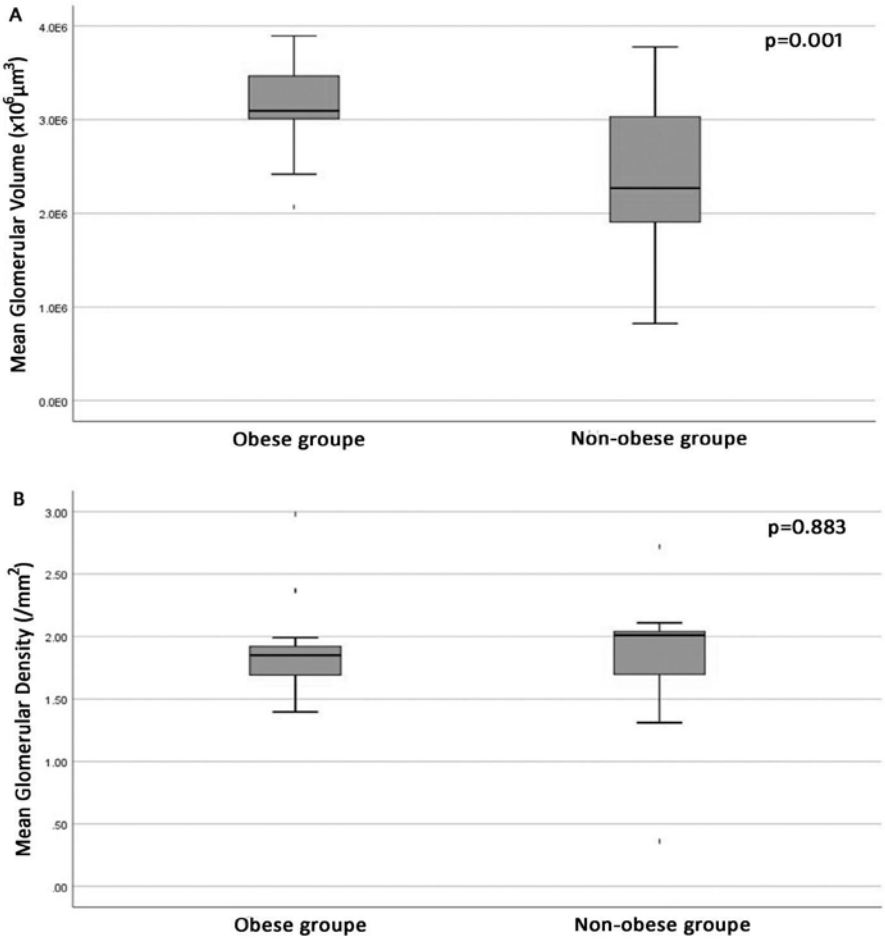

Fig. 1 – Mean glomerular volume (A) and mean glomerular density (B) in obese and non-obese focal segmental glomerulosclerosis (FSGS) patients.

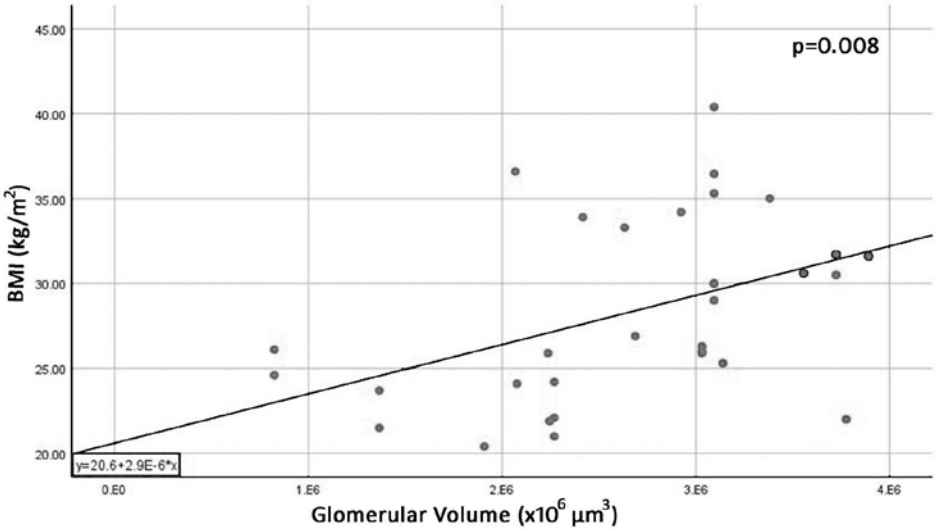

Fig. 2 – Correlation between glomerular volume and body mass index (BMI).

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

After 6 months of follow-up, there was no significant difference in the outcome between the 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, a significantly higher percentage of non-obese patients reached complete remission compared to obese patients (71.4% vs. 37.5%; p = 0.041). After 24 months of follow-up, there was no significant difference in the outcome between the obese and non-obese patients. Complete remission reached the same percentage of the 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 the examined patients in both groups were without remission after two years of follow-up (55.6% vs. 50%) (Figure 3).

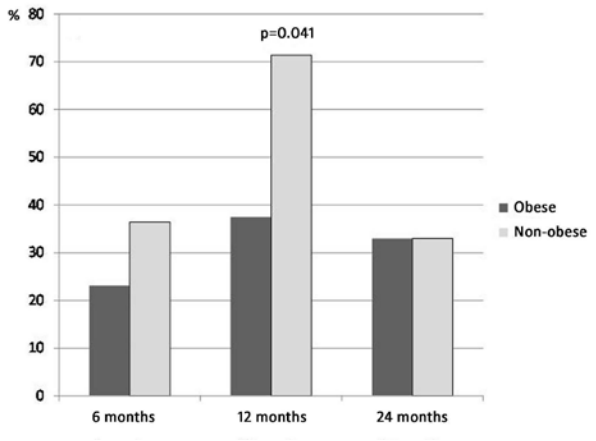

Fig. 3 – Percentage of obese and non-obese patients with complete remission of the nephrotic syndrome after 6, 12, and 24 months of follow-up.

Discussion

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

In the present study, the 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 with no significant difference in comparison with non-obese patients. The morphometric study on glomerular parameters confirmed the earlier findings of Praga et al. [7] that glomerulomegaly and lower glomerular density in obese FSGS patients was significantly increased compared to non-obese patients. Additionally, Kambham et al. [6] and Danilewicz and Wagrowska-Danielwicz [15] showed the same results. Although the 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 in body size increases, 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 the appearance of FSGS in obese patients depends not only on obesity-related increases in glomerular volume, but also on podocyte hypertrophic responses. Moreover, the 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 the non-obese patients but without significance, and 12 months after biopsy, proteinuria was significantly lower, and protein and albumin serum concentrations were higher than in the obese patients. Therefore, a 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 differences in percentages of patients with complete remission.

Forty years ago, the association between proteinuria and obesity was first reported [18]. In the 1980s, there were several case reports and autopsy series studies of ORG [19, 20]. In 2001, Kambham 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 the nephrotic range (≥ 3.5 g/24 h) in a significant number of cases. Interestingly, obese patients with ORG hardly ever develop hypoproteinemia, hypoalbuminemia, oedema, or other typical findings of nephrotic syndrome even in the presence of massive proteinuria [5, 7]. This occurrence could be very useful in the 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 explanations 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, the selectivity of proteinuria, and the 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] and pharmacotherapy (ACE inhibitors or ARBs) are associated with important antiproteinuric effect [22]. In 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 differences between the groups. Additionally, only 12 months after biopsy, a significantly higher number of patients in the non-obese patient group had complete remission compared to the obese patients, but after 24 months, there were no differences in the clinical outcome. Some studies pointed out that obesity can accelerate the progression of chronic kidney disease. Bonnet et al. [23] reported that a BMI > 25 kg/m^2 or higher 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 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 ≥ 1g/day). Moreover, the absolute renal risk (ARR) score for dialysis/death was also significantly worse in obese patients compared to the non-obese ones. As expected, the final outcome was globally worse in obese IgA 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 months of follow-up had significantly lower kidney function compared to non-obese patients. However, 12 and 24 months after, with the progression of chronic kidney disease in non-obese patients, this difference was without statistical 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 the short time of the follow-up period and is in extension.

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