LIPID AND NON LIPID CARDIOVASCULAR RISK FACTORS AMONG BLACK PATIENTS WITH IDIOPATHIC NEPHROTIC SYNDROME

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Résumé

Objectif
Déterminer la prévalence des facteurs lipidiques et non lipidiques de risque cardiovasculaire chez des patients porteurs du syndrome néphrotique idiopathique.
Méthodes
Analyse rétrospective des données de 66 patients (34 hommes et 32 femmes ; âge moyen 33 ± 13 ans) pour la présence des facteurs lipidiques et non lipidiques de risque cardiovasculaire. La dyslipidémie athérogène était définie par un taux de triglycérides (TG) > 1.07 mmol/L et/ou HDL-C < 1.03 mmol/L ; l'hypertension artérielle (HTA) par une pression artérielle (PA) ≥ 140/90 mm Hg ou une notion de traitement antihypertenseur; la dysglycémie par un taux de glucose plasmatique à jeun > 5.10 mmol/L et la maladie rénale chronique (MRC) par une clairance de la créatinine (CCl) < 60 mL/min selon les recommendations de « National Kidney Foundation ».
Résultats
Les valeurs moyennes des facteurs de risque étudiés se présentaient comme suit : PA 132 ± 22/86 ± 13 mm Hg, cholestérol total (TC) 9.43 ± 5.38 mmol/L, LDL-c 4.65 ± 2.24 mmol/L, HDL-c 0.92 ± 0.07 mmol/L, TG 3.42 ± 2.37 mmol/L, CCl 76 ± 41 mL/min et protéinurie/24h 4.47 ± 2.89 g. Les lésions histologiques observées consistaient en glomérulosclérose focale et segmentaire (GSFS, 50%), lésions glomérulaires minimes (LGM, 17%), glomerulonéphrite chronique (GNC, 17%) et glomérulonéphrite proliférative (GNP, 14%).
L'hypercholestérolémie, l'hypertriglycéridémie et la dyslipidémie athérogène étaient observées, respectivement, chez 49 (71%), 45 (68%) et 17 patients (41%). La MRC, l'HTA et la dysglycémie étaient présentes, respectivement, chez 36 (54%), 25 (38%) et 4 patients (6%). En plus de la protéinurie, la coexistence de 1, 2 et 3 facteurs de risque cardiovasculaire était observée, respectivement, chez 24 (36%), 25 (38%) et 15 patients (23%).
Conclusion
La protéinurie est associée dans la présente série à plusieurs facteurs lipidiques et non lipidiques de risque cardiovasculaire exposant ces patients à un risque élevé de maladies cardiovasculaires et de progression de l'atteinte rénale sous jacente. De ce fait, une approche intégrée ciblant ces différents facteurs de risque et associant le contrôle des facteurs du mode de vie et le traitement pharmacologique devrait être encourager dans le syndrome néphrotique.
Mots clé : Facteurs de risque cardiovasculaire, Prévalence, Syndrome néphrotique

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SUMMARY

Objective: to assess the prevalence of lipid (total cholesterol, LDL-C, HDL-c, triglycerides) and non lipid (blood pressure, plasma glucose, creatinine clearance) cardiovascular risk factors among black patients with idiopathic nephrotic syndrome (NS) at presentation or the time of percutaneous biopsy.

Methods: retrospective analysis for the presence of lipid and non lipid risk factors of data from 66 nephrotic patients (34 men and 32 women aged 30 ± 13 years) admitted at the University of Kinshasa Hospital. Atherogenic dyslipidemia was defined as serum triglycerides > 1.70 mmol/L or HDL-c < 1.03 mmol/L, hypertension as blood pressure ≥ 140/90 mm Hg or history of antihypertensive medication, impaired fasting glucose (IFG) as FG > 5.10 mmol/L and chronic kidney disease (CKD) as creatinine clearance (CCl) < 60 mL/min according to National Kidney Foundation (NKF) guidelines.

Results: average data were 136 ± 22/86 ± 13 mm Hg for blood pressure, 9.43 ± 5.38 mmol/L for cholesterol, 4.65 ± 2.24 mmol/L for LDL-c, 0.92 ± 0.07 mmol/L for HDL-c, 3.42 ± 2.37 mmol/L for triglycerides, 5.33 ± 1.66 mmol/L for glucose, 76 ± 41 mL/min for CCl and 4.49 ± 2.89 g/d for 24h proteinuria. The histologic picture consisted of focal segmental glomerulosclerosis (FSGS), minimal change disease (MCD), chronic glomerulonephritis (CGN) and proliferative GN (PGN) in 50%, 17%, 17% and 14% of patients, respectively. Hypercholesterolemia, hypertriglyceridemia and atherogenic dyslipidemia were observed in 49 (74%), 45 (68%) and 27 patients (41%), respectively. CKD, hypertension and IGT were present in 36 (54%), 25 (38%) and 4 patients (6%), respectively. In addition to proteinuria, the coexistence of 1, 2 and 3 risk factors was observed in 24 (36%), 25 (38%) and 15 patients (23%), respectively.

Conclusion : nephrotic patients have multiple cardiovascular and renal risk factors and could be at high risk for both CVD and renal disease progression. Multiple risk factor intervention based on therapeutic life style changes and pharmacological therapy should be encouraged in these patients to prevent and/or delay the development and progression of both CVD and renal disease.

Key words: Cardiovascular risk factors, Prevalence, Blacks, Nephrotic syndrome

INTRODUCTION

Patients with the Nephrotic syndrome (NS) are thought to be at high risk for both cardiovascular disease (CVD) and renal disease progression[1]. Increased prevalence of dyslipidemia and other non lipid risk factors such as proteinuria, high blood pressure (HBP), impaired glucose tolerance (IGT), renal dysfunction and insulin resistance/ hyperinsulinemia has been reported to partly explain the trends toward high CV and renal risk in nephrotic patients[2-5]. These trends negatively impact on patients'quality of life and place a huge financial burden on the healthcare system[6-9]. Therefore, it becomes of utmost importance to identify CV and renal risk factors in nephrotic patients and to devise effective strategies that prevent the development and/or the progression of both CVD and renal disease[6-9]. In this regard, multiple risk factor intervention should be encouraged in nephrotic patients[10-13].

In the Democratic Republic of the Congo (DRC), NS accounts for 38% of admission in the Division of Nephrology of the University of Kinshasa Hospital[14-16] and is associated with multiple complications[17-19]. Focal segmental glomerulosclerosis (FSGS) is the leading cause of NS with HIV/AIDS accounting for 30% and the remaining cases were idiopathic [14-16]. Data from 2001-2004 indicate an overwhelming increase in admission for chronic kidney disease (CKD) among young adults in Kinshasa; 78% of them presented at late stages of the disease in a setting with limited access to dialysis facilities[14]. NS contributes partly to this increased prevalence of CKD. We retrospectively analyzed for the presence of lipid and non lipid cardiovascular and renal risk factors data from nephrotic patients admitted at the Division of Nephrology of the University of Kinshasa Hospital.

PATIENTS AND METHODS

To elucidate the cardiovascular risk profile of patients with idiopathic NS, we retrospectively analyzed, for the presence of lipid and non lipid cardiovascular risk factors, the records of nephrotic patients seen at the Division of Nephrology of the University of Kinshasa Hospital when proteinuria was first noted or at the time of renal percutaneous biopsy. For each record, demographic (age, gender), anthropometric (weight), clinical (blood pressure or BP, heart rate, treatment), biological (24h proteinuria, serum albumin, cholesterol and its fractions, triglycerides, creatinine, plasma glucose) parameters and histologic protocol were drawn. LDL-c and creatinine clearance (CrCl) were calculated according to Friedewald[20] and Modification of Diet in Renal Disease (MDRD)[21] equations, respectively. Hypertension was defined as BP $\geq$ 140/90 mm Hg and classified according to JNC VII[19] as following: BP 140-160/90-100 mm Hg and $\geq$ 160/100 for grades 1 and 2, respectively. According to National Kidney Foundation (NKF) guidelines[22], CrCl < 60 mL/min defined chronic kidney disease (CKD) that was stratified as following: CrCl 30-60 mL/min as stage 3, CrCl 15-30 mL/min as stage 4 and CrCl < 15 mL/min as stage 5. Total cholesterol (TC) > 5.20 mmol/L, triglycerides (TG) > 1.70 mmol/L, plasma glucose > 5.10 mmol/L defined hypercholesterolemia, hypertriglyceridemia and impaired fasting glucose (IGT), respectively. The risk was considered borderline in the presence of a TC of 5.20 through 6.20 mmol/L, LDL-c of 3.34 through 4.14 mmol/L, TG of 1.70 through 2.25 mmol/L and TC/HDL-c of 5 through 10; it was high with a TC $\geq$ 6.20 mmol/L, LDL-c $\geq$ 4.14 mmol/L, HDL-c < 1.03 mmol/L, TG $\geq$ 2.25 mmol/L and TC/HDL-c $\geq$ 10. Atherogenic dyslipidaemia was defined as TG $\geq$ 2.25 mmol/L and/or HDL-c < 1.03 mmol/L according to NCEP-ATP III guidelines [23].

Data were analysed using SPSS for

Windows version 12.0 at the Department of Epidemiology and Biostatistics of the Kinshasa School of Public Health and expressed as mean ± standard deviation or relative frequency (in percent). Pearson correlation coefficients were used to evaluate the relation between proteinuria, serum creatinine and other variables of interest. P value ≤ 0.05 defined the level of statistical significance.

RESULTS

General characteristics of nephrotic patients are summarized in table 1. Their mean age was 30 ± 13 years ; average data were 136 ± 22/86 ± 13 mm Hg for BP, 9.43 ± 5.38 mmol/L for cholesterol, 4.65 ± 2.24 mmol/L for LDL-c, 0.92 ± 0.07 mmol/L for HDL-c, 3.42 ± 2.37 mmol/L for triglycerides, 5.33 ± 1.66 mmol/L for glucose, 2.03 ± 0.66 g/dL for albumin, 76 ± 41 mL/min for CCl and 4.49 ± 2.89 g/d for 24h proteinuria. Percutaneous renal biopsy was performed in 56 patients and the histologic picture consisted of focal segmental glomerulosclerosis (FSGS), minimal change disease (MCD), chronic glomerulonephritis (CGN) and proliferative GN (PGN) in 28 (50%), 10 (17%), 10 (17%) and 8 patients (14%), respectively. Proteinuria was positively correlated ($r = 0.37$; $p \leq 0.001$) to total cholesterol.

The presence and magnitude of lipid risk factors are depicted in table 2. Hypercholesterolemia was observed in 49 patients (74%), 29 men and 20 women, of whom 44 (66%) had high risk levels. Boderline high risk and high risk LDL-c levels were found in 5 (8%) and 44 (66%) of patients with hypercholesterolemia. High risk HDL-c levels were present in 20 patients (30%). Hypertriglyceridaemia was observed in 45 patients (68%), 26 men and 19 women, of whom 36 (54%) had high risk levels. Boderline high risk and high risk TC/DL-c levels were observed in 13 patients (19.5%), respectively. Twenty seven patients (41%) presented with atherogenic dyslipidaemia. Dyslipidaemic patients were unaware of their condition and none was receiving lipid lowering therapy.

Table 3 summarizes the distribution of non lipid risk factors. Twenty five patients (38%), 17 men and 8 women, had hypertension of whom 17 (26%) were at stage 2. None of hypertensive patients was aware of his condition; apart from loop diuretic used to combat fluid retention, antihypertensive therapy was not prescribed to these patients. CKD was present in 36 patients (54%) with 10 (15%) of them at stages 4 and 5, respectively. IGT was observed in 4 patients (6%). Serum creatinine was positively correlated to age ($r = 0.28$; $p \leq 0.05$), SBP ($r = 0.43$; $p \leq 0.001$), DBP ($r = 0.38$; $p \leq 0.001$) and PP ($r = 0.32$; $p \leq 0.001$).

Clustering of risk factors is given in table 4. In addition to proteinuria, the coexistence of 1, 2 and 3 risk factors was observed in 24 (36%), 25 (38%) and 15 patients (23%), respectively. For patients with 1 additional risk factor, dyslipidaemia ($n = 20$) was the most common associated risk factor; dyslipidaemia and CKD were most frequent combination in patients with 2 additional risk factors.

DISCUSSION

Nephrotic patients had on average moderate proteinuria, high levels of total cholesterol (TC), LDL-c, triglycerides and low levels of HDL-c. Mean levels of TC and TG in the present study were within the range reported by a survey of 20 studies of nephrotic patients that showed a mean TC of 10.07 ± 1.03 mmol/L and TG of 2.71 ± 0.67 mmol/L; these are about 125% and 200 % of control values, respectively[25]. Proteinuria was positively correlated with total cholesterol. The relationship between proteinuria and abnormal lipid metabolism could be explained by at least two mechanisms. At moderate proteinuria (< 10g/day), LDL-c catabolism is impaired either due to reduced receptor activity or defective ligand-receptor interaction. Above a proteinuric threshold of 10 g/day, increased LDL-c (and possibly VLDL-c) synthesis

aggravates the dyslipidemia and TG levels rise, often to become the dominant abnormality[25]. Hypertriglyceridaemia contributes to low HDL-c levels by increasing the exchange, mediated by the enzyme cholesteryl ester transfer protein (CETP), of esterified cholesterol from HDL-c to TG-rich lipoproteins and of TG from HDL particles with subsequent enrichment of HDL-c particle core with TG. Enriched HDL-c particles have a faster catabolic rate than normal HDL-c with subsequent lowering of circulating HDL-c particles[26]. A decrease in circulating albumin or plasma oncotic pressure by stimulating a non specific increase in hepatic protein synthesis may be the precipitating factor of lipid abnormalities observed in the nephrotic syndrome[25].

Lipid profile in the present study was characterized by a higher proportion of patients with hypercholesterolemia, hypertriglyceridaemia, TC/HDL-c ratio, low HDL-c and atherogenic dyslipidaemia. The majority of patients with hyper-cholesterolemia and hypertriglyceridaemia had high risk levels of LDL-c and TG, respectively. The observed lipid profile could expose these patients to high risk for cardiovascular and renal disease. A dose-response relationship between the level of TC and the risk of coronary artery disease (CAD) has already been reported[27]. Age-adjusted death rate for CAD rose from about 3.5/1,000 men at TC of 3.62 mmol/L to about 5/1,000 men at TC of 5.16 mmol/L; the slope of the association in this range was shallow. Between a TC of 5.16 mmol/L and 6.20 mmol/L the risk rose (from 5/1,000 to about 9/1,000) in a linear fashion. Above a TC of 6.20 mmol/L, the risk curve became exponential with an additional doubling of risk[27]. However, predictive power of TC changed with both age and time since measurement with steady diminution of predictive power with increasing age and recent cholesterol measurement in comparison to remote one [27,28]. The relationship between TC and CAD is thought to rely upon the increased susceptibility of LDL-c to oxidation[28]. LDL-c is the principal atherogenic lipoprotein and is a better predictor than TC of CAD in both men and women[27]. On average, about 70 % of TC in an individual is LDL-c, although interindividual variation may exist. Thus, the association of TC with CAD may reflect the fact that TC can serve as a surrogate for LDL-c. LDL-c levels observed in the present study met current guidelines threshold (LDL-c $\geq$ 4.13 mmol/L) for lipid lowering treatment[23]. In this regard, inhibitors of 3-hydroxymethyl-3-glutaryl CoA reductase (Statins), through lipid lowering effects and lipid independent mechanisms, have been proved useful and effective in preventing and delaying the development and progression of both CV and renal disease [29].

The role of hypertriglyceridaemia in the pathogenesis of atherosclerotic CVD remains controversial[30,31]. Hypertriglyceridaemia is clearly associated with CVD in univariate analysis. However, many multivariate studies have shown that hypertriglyceridaemia-associated CV risk is markedly attenuated after adjustment for other strong CHD risk factors namely low HDL-c levels and increased small, dense LDL-c particles[30,31]. These findings have led some researchers to believe that hypertriglyceridaemia serves more as a proxy for abnormal cholesterol levels and cholesterol subfractions to which hypertriglyceridaemia is frequently associated[30,31]. In contrast to the aforementioned findings, many other studies have shown hypertriglyceridaemia to be an independent risk factor for CHD even after adjustment for HDL-c and LDL-c[30,31]. Furthermore, National Cholesterol Education Program (NCEP)-Adult Treatment Panel III (ATP III) considers hypertriglyceridaemia as an independent risk factor for CHD and calls for medical treatment with fibrate alone or in combination with statins in cases where therapeutic lifestyle changes (TLC) are not adequate to reduce TG to appropriate levels[23].

A low HDL-c has been reported to be the most common lipid abnormality in up to 30% of high CV and renal risk patients[32,33]. In the present study, 30% of nephrotic patients presented with low HDL-c and could be at high risk for both CV and renal disease. A

0.02 mmol/L (1 mg/dL) decrement in HDL-c is associated with an approximately 2-3% increase in fatal CAD in both men and women[27]. The protective effects of high HDL-c levels rely upon several mechanisms including reverse cholesterol transport of cholesterol, inhibition of LDL-c oxidation and subsequent vascular and tissue inflammation, proliferation and fibrosis, inhibition of cellular adhesion and/or platelet activation[27,32,33]. Therefore, increasing HDL-c has become a therapeutic goal in primary and secondary prevention of CVD and renal disease[33,34]. Since lifestyle changes, statins, fibrates and niacin induce only moderate (5-25%) increase of HDL-c, inhibition of cholesteryl ester transfer protein (CETP), the key enzyme in cholesterol reverse transport, has emerged as an attractive alternative therapy[32].

A substantial proportion of nephrotic patients in the present study had elevated TC/HDL-c ratio. TC/HDL-c ratio has been reported to be an optimal screening strategy for subjects aged 30-59 years; at all levels of TC, low HDL-c levels identify those at increased risk[28]. TC/HDL-c ratio explains a greater proportion of variation in CV mortality than do HDL-c and TC alone[28]. TC at an individual level is not a highly specific marker for risk of CAD or CVD due to the substantial overlapping of TC curve distribution of men with and without CAD between TC levels of 3.87-7.75 mmol/L, range seen in most individuals[27]. This overlap together with the fact that cholesterol carried on different lipoprotein fraction has different prognostic significance have led to abandonment of the use of TC alone in risk assessment[27]. TC/HDL-c is the ratio most frequently used to evaluate CV risk and carries composite information with a numerator containing lipid fractions with a positive association with CAD and a denominator inversely associated with CAD[27]. A ratio < 3 indicates a risk below average, but not absent risk; a ratio of 3-5 indicates average risk and a ratio > 9, clearly a marked high risk. Thus, a reasonable therapeutic goal is a ratio < 4, although this may not be achievable in most patients[27].

Atherogenic dyslipidaemia was observed in nearly half of nephrotic patients and could expose them to high CVD and renal disease risk. Atherogenic dyslipidaemia is a marker of insulin resistance and subsequent hyperinsulinemia which are well known CV and renal risk factors[34]. Insulin resistance and subsequent hyperinsulinemia induce CV and renal disease through hemodynamic and structural changes through activation of renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system (SNS), production of oxidative stress, expression of proinflammatory and profibrotic cytokines, induction of a procoagulant state [35].

Nephrotic syndrome in the present study was also associated with non lipid risk factors including hypertension, impaired fasting glucose (IFG) and CKD. Hypertension and IGT are well known components of the metabolic syndrome and could enhance CVD and renal disease risk through underlying insulin resistance and hyperinsulinemia[35]. CKD is actually emerging as a potent and independent risk factor for both CVD and renal disease progression [36]. In the present study, age, SBP, DBP, PP were positively correlated with serum creatinine. Many observational studies evaluating the changing pattern of BP with the aging process have shown a concomitant increase in SBP, DBP and PP. Beyond the approximate age of 60 years SBP continues to increase but DBP reaches a plateau or gradually falls and this leads to accelerated rise in PP which reflects increased stiffness of the large arteries [37,38]. Aging as well as high BP-induced vascular remodelling process consisted of hypertrophy, fibrosis and altered macro and micro-circulation[39]. Several studies have shown that PP is a reliable prognostic factor for mortality and CVD in patients in CKD patients[40]. Control of each of these non lipid risk factors should contribute to improve nephrotic patients' quality of life[12]. Angiotensin converting enzyme inhibitors (ACEIs) and angiotensin II type 1 receptor blockers (ARBs) have been proved useful and effective in controlling both proteinuria, high BP and IGT

as well as delaying the development and progression CVD and renal disease [41,42].

Clustering of risk factors has been reported to exert multiplicative rather than additive effect on CV and renal disease risk[43]. In this regard, NCEP-ATP III has defined the metabolic syndrome (MS) clinically as any three of the following five traits: abdominal obesity, IFG, hypertension, hypertriglyceridemia and/or low HDL-c[23]. MS is now recognized as a potent and strong independent predictor of CV and renal disease than does each of its components [35]. In the present study, a substantial proportion of patients had 3 or more risk factors and could be considered as having MS and thus at high risk for both CV and renal disease. This increased risk could be explained by insulin resistance and subsequent hyperinsulinemia reported as the essential and common denominator of MS[43]. For patients with MS, a global approach targeting all risk factors based on combination of therapeutic lifestyle changes and pharmacological treatment is recommended to reduce effectively the risk of CV and renal disease[12]. With reference to evidence from other high risk patients, nephrotic patients should benefit from this multiple risk factor interventions.

The present study has some potential limitations. The sample size could not allow sufficient power to detect additional associations. Lack of insulin measurement constitutes a limiting factor to firmly establish the coexistence of insulin resistance and subsequent hyperinsulinemia. Finally, one wonders to which extent the conclusions of the present clinical based study could be extrapolated to the general population given the bias in the referral of patients.

CONCLUSION

Nephrotic Syndrome in the present study was associated with isolated as well as combined lipid and non lipid risk factors that could enhance the risk for both cardiovascular and progressive renal disease. With reference to current guidelines, nephrotic patients should benefit from a global cardiovascular and renal risk approach based on multiple risk factor intervention combining therapeutic lifestyle changes and pharmacological treatment to prevent or delay the development and progression of cardiovascular disease and renal disease.

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ANNEXES

Table 1. General characteristics of nephrotic patients (n = 66)
Table 2. Lipid cardiovascular risk factors in nephrotic patients (n = 66)
Table 3. Non cardiovascular lipid risk factors in nephrotic patients (n = 66)
Table 4. Clustering of cardiovascular risk factors in nephrotic patients (n = 66).

Table 1. General characteristics of nephrotic patients (n = 66)

Characteristic	NS
Gender, M/F	34/32
Age, years	30 ± 13
Weight, Kg	43 ± 24
SBP, mm Hg	136 ± 22
DBP, mm Hg	86 ± 13
PP, mm Hg	50 ± 11
Albumin, g/L	20 ± 7
Total chosterol (TC), mmol/L	9,43 ± 5,38
LDL-c, mmol/L	4,65 ± 2,24
HDL-c, mmol/L	0,92 ± 0,07
Triglycerides, mmol/L	3,42 ± 2,37
TCHDL-c	8,40 ± 2,41
Glucose, mmol/L	5,16 ± 1,66
Créatinine, µmol/L	133 ± 42
CrCl, ml/min	76 ± 41
24 h proteinuria, gl/day	4,49 ± 2,34

Data are expressed as mean ± SD or absolute frequency.
Abbreviations: NS, nephrotic syndrome M, male F, female SBP, systolic blood pressure DBP, diastolic blood pressure PP, pulse pressure LDL-c, low density lipoprotein-cholesterol HDL-c, high density liporpotein-cholesterol CrCl, creatinine clearance

Table 2. Lipid cardiovascular risk factors in nephrotic patients (n = 66)

Risk factor	n (%)
Hypercholesterolemia	49 (74)
- boderline high risk levels (5.20 – 6.19 mmol/L)	5 (8)
- high risk levels (≥ 6.20 mmol/L)	44 (66)
Elevated LDL-c	49 (74)
- boderline high risk levels (3.34 – 4.09 mmol/L)	5 (8)
- high risk levels (≥ 4.10 mmol/L)	44 (66)
Low HDL-c	20 (30)
- (< 1.03 mmol/L, high risk levels)	
Hypertriglyceridemia	45 (68)
- boderline high risk levels (1.70 – 2.19 mmol/L)	9 (14)
- high risk levels (≥ 2.20 mmol/L)	36 (54)
Elevated TC/HDL-c	26 (39)
- boderline high risk levels (5 – 10)	13 (19.5)
- high risk levels (≥ 10)	13 (19.5)
Atherogenic dyslipidemia	27 (41)

Data are expressed as absolute frequency (relative frequency in percent)
Abbreviations: n, absolute frequency %, relative frequency LDL-c, low density lipoprotein-cholesterol HDL-c, high density liporpotein-cholesterol TC, total cholesterol.

Table 3. Non lipid cardiovascular risk factors in nephrotic patients (n = 66)

Risk factor	n (%)
Hypertension	25 (38)
- stage 1	8 (12)
- stage 2	17 (26)
Chronic kidney disease (CKD)	36 (54)
- stage 3	16 (24)
- stage 4	10 (15)
- stage 5	10 (15)
Impaired fasting glucose (IFG)	4 (6)

Data are expressed as absolute frequency (relative frequency in percent).

Table 4. Clustering cardiovascular risk factors in nephrotic patients (n = 66)

Risk factor	n (%)
2 risk factors (proteinuria + 1 additional risk factor)	24 (36)
3 risk factors (proteinuria + 2 additional risk factors)	25 (38)
4 risk factors (proteinuria + 3 additional risk factors)	15 (23)

Data are expressed as absolute frequency (relative frequency in percent).