Original article **UDC: 616.61-008.61-07:616.12-008.331.1**
doi:10.5633/amm.2014.0403

ESSENTIAL ARTERIAL HYPERTENSION AND RISK FACTORS ASSOCIATED WITH HYPERTENSIVE NEPHROPATHY

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Arterial hypertension is a major risk factor that predisposes to cardiovascular disorders and is responsible for most of the morbidity and mortality in patients. Hypertension is closely associated with the kidney, because kidney disease can be both the cause and consequence of increased blood pressure. Elevation of blood pressure is a strong independent risk factor for hypertensive nephropathy and development of ESRD. The pathogenesis of ischemic hypertensive nephropathy (IHN) is multifactoral, and in addition to blood pressure other factors contribute to the development of this renal pathology and its progression to end-stage renal disease. These include obesity, smoking, male gender and other still unknown risk factors.

The aim of this paper was to analyse the association between essential arterial hypertension and renal hypertensive disease and prevalence of other atherosclerotic risk factors in patients with developed hypertensive renal disease.

In this prospective cross sectional study 283 patients of both genders with diagnosed essential hypertension and hypertensive renal disease were analysed. The anamnestic data related to age, duration of hypertension, history of smoking, presence of hypertensive retinopathy, hypertrophy of the left chamber and data about previous renal diseases were collected through conversation and medical documentation. The clinical examination comprise determination of blood pressure, body mass index (BMI), lipid parameters (total cholesterol, LDL cholesterol, HDL cholesterol and triglycerides), serum urea and creatinine, urine, albumin and protein concentration.

The total number of 283 patients (185 males and 98 females) with HN was analyzed. The analysis revealed significantly higher proportion of males aged over 60 years with IHN. The mean age of examined hypertensive patients with IHN is 62.6±8.8 years with duration of hypertension 19.8±5.9 years. All examined patients had hypertensive retinopathy and hypertrophy of the left chamber. The majority of the examined patients (78.6%) with IHN were overweight with BMI 25-30kg/m^2. Dyslipidemia was registered in 75% and smoking in 75% of hypertensive patients with IHN. The most common lipid disorders were hypertrigliceridemia with hypercholesterolemia found in 42.1% and combined dyslipidemia in 25.8%. Family history of arterial hypertension (AH) was registered in all patients with IHN.

The correlation analysis revealed a significant positive association between family history of HTA ($r=0.62$, $p<0.05$), smoking ($r=0.53$, $p<0.05$) and combined dyslipidemia (hypertrigliceridemia and hypercholesterolemia) ($r=0.45$, $p<0.05$) with occurrence of IHN.

Hypertensive nephropathy predominatly occurs in older hypertensive males with developed hypertensive micro- and macrovascular complications. The family history of AH, smoking and lipid disorders, especially combined dyslipidemia are very common risk factors associated with hypertensive nephropathy. Prevention and therapy of these risk factors is an important task in reduction of hypertensive nephropathy. *Acta Medica Medianae 2014;53(4):15-21.*

Key words: *hypertensive nephropathy, arterial hypertension, smoking, dyslipidemia*

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Introduction

According to the European Society of Hypertension (2003), patients are considered hypertensive if their blood pressures reach or exceed 140/90 mmHg (1). In many European countries the prevalence of hypertension appears to be around 30–45% of the general population,

with a steep increase with ageing (2). Arterial hypertension is highly prevalent in the elderly. In this regard, according to NHANES III Study, its prevalence rate for subjects >60 years old (white not Spanish speaking Americans) is estimated to be >60% (3). Secondary hypertension accounts for approximately 5-10% of all cases of hypertension and results from an underlying, identifiable cause. In the remaining 95% of the cases, no known cause is being recognized despite the extensive medical examination (idiopathic or primary hypertension) (4).

Arterial hypertension is a major risk factor that predisposes to cardiovascular disorders and is responsible for most of the morbidity and mortality in patients. Hypertension acts through its effects on target organs such as the brain, heart, and kidney. Structural alterations in the microcirculation form a major link between hypertension and target organ damage. Essential hypertension is a common health problem world-wide which remains asymptomatic until late in its course (5). Through its effects on target organs, hypertension is one of the most relevant risk factors for cardiovascular morbidity and mortality. Besides its effects per se other mechanisms such as oxidative stress, inflammation, or endothelial dysfunction have appeared to play a key role in the pathogenesis of target organ damage, and therefore represent another important pathway for therapy.

As a consequence of elevated blood pressure, arterial elasticity is reduced and wall damage appears, which can lead to cholesterol and fat deposition on those lesions and eventually to obstruction of the vessels. This is the basis of most of the target organ damages induced by hypertension.

Hypertension is intimately linked with the kidney, because kidney disease can be both the cause and consequence of increased blood pressure. According to the 2011 US Renal Data System (USRDS) data, in the year 2009, ischemic hypertensive nephrosclerosis (IHN) accounted for 28% of patients reaching end-stage renal disease (ESRD). The rate of ESRD attributed to hypertension has grown to 8.7% since the year 2000. Hypertensive nephrosclerosis is reportedly the second most common cause of ESRD in white people (23%) and is the leading cause of ESRD in black people 46% (6). Renal risk appears to be more closely related with systolic than diastolic blood pressure, and black men are at greater risk than white men for developing ESRD at every level of blood pressure. Clinically, macroalbuminuria (a random urine albumin/creatinine ratio >300mg /g) or microalbuminuria (a random urine albumin/ creatinine ratio 30-300 mg/g) are early markers of renal injury. These are also risk factors for renal disease progression and for cardiovascular disease (7).

There is no doubt that elevations of blood pressure are strong independent risk factors for hypertensive nephropathy and development of ESRD. The Multiple Risk Factor Intervention Trial (MRFIT) study, including 332.544 men, showed that a strong, graded relationship between systolic and diastolic blood pressure and the subsequent development of ESRD exists, independent of associations between ESRD and age, race, income, diabetes mellitus, history of myocardial infarction, serum cholesterol, and cigarette smoking (8). However, the risk of ESRD was greatly increased by the presence of myocardial infarction or heart failure. This study also showed the now well-recognized fact that lowering blood pressure decreases the risk of ESRD; a 20 mmHg decrease in systolic blood pressure reduced the risk by two thirds (9).

Aim

The aim of this paper was to analyse the association between the essential arterial hypertension and renal hypertensive disease, and prevalence of other atherosclerotic risk factors in patients with developed renal hypertensive disease.

Methodology

In this prospective cross-sectional study, 283 patients of both genders with diagnosed essential hypertension and hypertensive renal disease were analyzed.

The diagnosis of essential hypertension was made on the basis of increased blood pressure - 140/90 mmHg or more in the absence of other possible causes of hypertension or taking antihypertensive medications based on recommendations of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) (5). Blood pressure was registered as average of 2 or more BP measurements on at least 2 subsequent visits. Essential, primary, or idiopathic hypertension is defined as high BP in which secondary causes such as renovascular disease, renal failure, pheochromocytoma, aldosteronism, or other causes of secondary hypertension or mendelian forms (monogenic) are not present.

Hypertensive nephropathy (or "hypertensive nephrosclerosis", or "hypertensive renal disease") is a medical condition referring to damage to the kidney due to chronic high blood pressure. It was distinguished from "renovascular hypertension" (I15.0), which is a form of secondary hypertension.

The diagnosis of hypertensive renal disease is made with urine and blood tests. Serum urea and creatinine was determined from blood samples while urine albumin and protein concentration was determined in urine samples. Urine albumin-to-creatinine ratio and creatinine clearance was calculated. Kidney Disease Outcomes Quality Initiative (NKF-K/DOQI) work group has defined chronic kidney disease (CKD) as the presence of markers of kidney damage (abnormalities in blood, urine, or imaging tests) for ≥3 months or a

glomerular filtration rate (GFR) <60 mL/minute/ 1.73m^2 for ≥3 months, with or without other signs of kidney damage (10).

The anamnestic data about age, duration of hypertension, history of smoking, presence of hypertensive retinopathy, hypertrophy of left chamber and data about previous renal diseases were collected through conversation and medical documentation. The clinical examination comprise determination of blood pressure, body mass index (BMI), lipid parameters (total cholesterol, LDL cholesterol, HDL cholesterol and triglycerides), serum urea and creatinine, urine, albumin and protein concentration.

The clinical and biochemical examination were done at the Institute of Nephrology and Hemodialysis, Clinical Center Niš and Biochemical Laboratory, Clinical Center Niš.

Continuous data were expressed as mean ±SD and categorical data as absolute numbers with percentages. Differences between two groups were compared by Student's t-test and Mann-Whitney U test for continuous variables and χ^2 test for categorical variables. The relationship between two categorical variables was assessed by a bivariate correlation method (Spearman's correlation). Data analysis was performed using SPSS version 16 (Statistical Package for Social Sciences for Windows, SPSS Inc., Chicago, IL). Statistical significance was set at $p<0.05$.

Results

The total number of 283 patients (185 males and 98 females) with IHN was analyzed. Analysis revealed significantly higher proportion of males aged over 60 years with IHN (Table 1.).

Average age of examined hypertensive patients with IHN is 62.6±8.8 years with duration of hypertension 19.8±5.9 years. All examined patients had hypertensive retinopathy and hypertrophy of the left chamber. The baseline characteristics are shown in Table 2.

The majority of the examined patients (78.6%) with IHN were overweight with BMI 25-30 kg/m^2. Data are shown in Table 3.

Dyslipidemia was registered in 75% of hypertensive patients with IHN. The most common lipid disorders were hypertrigliceridemia with hypercholesterolemia found in 42.1% and combined dyslipidemia in 25.8% (Table 4.).

Table 1. Patients' gender and age in IHN

	Male		Female		All patients with IHN	
	N	%	N	%	N	%
Patients	185	65.4*	98	34.6	283	100
Age <60 years	67	23.7	31	10.9	98	34.6
Age ≥60 years	118	41.7*	67	23.7	185	65.4

Data are presented as N and %
*Hi^2=5.4; $p<0.05$ in relation to ideal gender and age distribution

Table 2. Baseline characteristics of IHN patients

Age (years)	62.6±8.8
Duration of hypertension (years)	19.8±5.9
SBP (mmHg)	163±11.3
DBP (mmHg)	101±10.1
Creatine (μmol)	203.2±62.4
C creatine (ml/s)	0.7±0.1
Proteinuria (g/l)	1.3 ±0.3
Hypertensive retinopathy	100%
Hypertrophy of left chamber	100%
Data about previous renal diseases	-

Table 3. Body mass index of examined patients with IHN

(kg/m2)	Male		Female		All patients with IHN	
	N	%	N	%	N	%
BMI <25	26	9.1	18	6.3	44	15.4
BMI 25-30	149	52.7*	73	25.8*	222	78.6
BMI >30	10	3.5	**7**	2.5	17	6.0

Data are presented as N and %
*Hi^2=4.04; $p<0.05$ vs. BMI<25 kg/m^2

Table 4. Lipid profile in patients with IHN

	N	%
Normal	71	25.0
Dyslipidemia total	212	75.0
↑TC+↑LDL-C+↓HDL-C	73	25.8
↑TC+ ↑TG	119	42.1
↑TG	20	7.1

Data are presented as N and %

Table 5. Smoking status in patients with IHN

	N	%
Nonsmokers	71	25.0
Smokers total	212	75.0
Active	111	39.3
Passive	101	35.7

Data are presented as N and %

Table 6. Risk factors for developing of IHN

	Positive	Negative	p
Dyslipidemia	212 / 75.0	71 / 25.0	NS
Family history of AH	283 / 100.0	0 / 0.0	<0.01

Data are presented as N / %

Active smokers were registered in 39.3% of patients with IHN, while passive smokers accounted for 35.7% (Table 5.).Family history of AH was registered in all patients with IHN, while dyslipidemia was presented in 75%.

The correlation analysis revealed significant positive association between family history of AH ($r=0.62$, $p<0.05$), smoking ($r=0.53$, $p<0.05$) and combined dyslipidemia (hypertrigliceridemia and hypercholesterolemia) ($r=0.45$, $p<0.05$) with occurrence of IHN.

Discussion

Based on the results of the MRFIT cohort, predictions showed that 5300 Americans come to dialysis with ESRD from hypertension yearly. Nevertheless, progression to ESRD is rare in persons with hypertension-related renal disease, and factors other than blood pressure probably play an important role. This observation is important because it suggests that other interventions in addition to blood pressure reduction may be effective (11).

The pathogenesis of hypertensive nephropathy is multifactoral and in addition to BP, other factors contribute to the development of this renal pathology and its progression to end-stage renal disease. These include genetic predisposition and increased pleasure level of homocysteineintermediate protein catabolism product known to induce kidney injury (12).

Hypertension and proteinuria are the major factors contributing to the progression of kidney disease. Essential hypertension is frequently associated with renal damage, for example, renal arteriolar thickening, fibrinoid deposition in the glomeruli, and proteinuria. The detrimental effects of systemic hypertension on renal vascular bed depend on the degree to which the microcirculation is exposed to elevated pressures. Renal injury occurs when the preglomerular autoregulatory mechanism is insufficient to maintain flow and pressure in the kidney (13). The pathophysiology of nephropathies results from an increased glomerular and systemic hypertension, permeability, and proteinuria.

In hypertension, the permeability of the glomeruli is altered, which leads to an excess of protein infiltration. The toxicity of this protein load generates tubular damage, inflammation and scarring. This leads to renal accumulation of immune cells and oxidative stress and further nephropathy development (14). Indeed, by inactivating endothelial nitric oxide (NO), ROS impairs vasodilation (15). Additionally, oxidative stress has proinflammatory effects: firstly, it activates NFnB, a transcription factor for proinflammatory genes, which promotes infiltration of the leucocytes by increasing the expression of adhesion molecules (16); secondly, it induces the expression of heat shock proteins that results in cell death and apoptosis in an inflammatory environment (17).

In the analyzed males, older age, high sistolic and diastolic blood pressure with long duration of hypertension were very frequent findings in patients with IHN. Proteinuria, increased serum creatinine concentration, presence of microvascular and hypertensive complications (hypertensive retinopathy and hypertrophy of left chamber) were also the most common findings in patients with IHN (Table 1. and 2.).

This is in line with results that high-normal albuminuria in uncomplicated essential hypertensive men is associated with an adverse cardiovascular and metabolic risk profile. Furthermore, hyperfiltration in the presence of minimally increased albuminuria may underlie an augmented glomerular blood flow and hydraulic pressure conducive to glomerular hypertension and, eventually, renal insufficiency. Overall, these data confirm the appropriateness to shift downward the limits for diagnosing microalbuminuria in essential hypertension, as indicated from previous prospective studies (18). Also, a strong, graded relation between both systolic and diastolic blood pressure and end-stage renal disease was identified, independent of associations between the disease and age, race, income, use of medication for diabetes mellitus, history of myocardial infarction, serum cholesterol concentration, and cigarette smoking were observed in some studies (8).

Obesity presents independent risk factor for the development of cardiovascular diseases and is usually associated with hypertension, insulin resistance and dyslipidemia in syndrome X. Similar finding is registerd in examined patients with IHN who were mostly overweight and dyslipidemic (Table 3. and 4.).

Few studies have examined the association between obesity and markers of kidney injury in a chronic kidney disease population. Large meta-analysis showed that obesity measured by BMI is independently associated with proteinuria and albuminuria which represent markers of chronic kidney disease progression in African Americans with hypertensive nephrosclerosis, particularly in younger patients (19).

The prevalence of smoking (active and passive) was very high in patients with IHN and reached 75% (Table 5.).

Although it is undoubted that smoking is the number one preventable cause of death in most countries, smoking as an independent progression factor in renal disease has been questioned against the background of evidence-based criteria. More recent data together with evidence from experimental studies clearly indicate that smoking is a relevant risk factor, conferring a substantial increase in risk for renal function deterioration (20). The study of Briganti et al. (21), involving 11.247 randomly selected, population-based Australians, showed that lifetime smoking exposure was significantly associated with CKD stage 3 or more in men but not in women. Smoking is associated with renal impairment and proteinuria in a population without hypertension or abnormal glucose metabolism. A dose-response relationship was found between cumulative amount of smoking and indicators of kidney damage. In conjunction with other studies and plausible biological mechanisms, this study suggests that smoking may cause kidney damage, even in a healthy population. This finding raised the hypothesis that men may be more susceptible to the adverse renal effects of smoking (22). Furthermore, lifetime smoking exposure was paralleled by an increment of urine protein excretion, independent of gender. This increase was particularly marked in individuals with high-normal systolic BP and postload glucose concentrations.

The weakness of the study

Hypertension is closely linked with the kidney, as kidney disease can be both the cause and consequence of increased blood pressure. There are many problems with clinical diagnosis of hypertensive nephropathy which cause over-estimation of its prevalence. Renal biopsy and morphological examination as the gold standard of diagnosis is rarely performed in such patients. The majority of cases of hypertension are primary or essential. Renal parenchymal diseases and renovascular hypertension are important causes of secondary hypertension. Fibrous dysplasia and atherosclerosis constitute the majority cases of renovascular hypertension. Renal diseases with hypertension are divided into the benign nephrosclerosis and malignant nephrosclerosis. Benign nephrosclerosis is characterized by hyaline arteriolosclerosis and intimal fibrosis and reduplication of internal elastic lamina of arcuate and interlobular arteries. Malignant hypertensive nephropathy is characterized by hyperplastic arteriolitis and fibrinoid necrosis of arterioles and glomeruli (23). Bearing in mind that clinical diagnosis of IHN in examined patients was not made by biopsy but with biochemical and clinical indicators, we could not exclude the presence of secondary hypertension in some of the examined patients.

Conclusions

Hypertensive nephropathy predominantly occurs in older hypertensive males with developed hypertensive micro- and macrovascular complications. The family history of HTA, smoking and lipid disorders, and especially combined dyslipidemia, are very common risk factors associated with hypertensive nephropathy. Prevention and therapy of these risk factors are important tasks in the reduction of hypertensive nephropathy.

Acknowledgement

This paper was supported by the Ministry of Science, Republic of Serbia; Project No 41018.

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PRIMARNA ARTERIJSKA HIPERTENZIJA, FAKTORI RIZIKA I ISHEMIJSKA HIPERTENZIVNA NEFROPATIJA

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Arterijska hipertenzija je glavni faktor rizika za nastanak kardiovaskularnih poremećaja i predstavlja uzrok većine bolesti i mortaliteta. Postoji jaka veza između hipertenzije i funkcije bubrega, budući da bubrežne bolesti mogu da budu uzrok, ali i posledica povećanog krvnog pritiska. Povećanje krvnog pritiska je samo po sebi snažan faktor rizika za nastanak hipertenzivne nefropatije i razvoj završnog stadijuma hronične bubrežne bolesti ("end-stage renal disease" – ESRD). Patogeneza hipertenzivne nefropatije (HN) je multifaktoralna, tako da pored uvećanog krvnog pritiska, postoje i drugi faktori rizika koji doprinose razvoju ove bubrežne patologije i njene progresije do završnog stadijuma bubrežne bolesti. Tu spadaju gojaznost, pušenje, muški pol i ostali, još uvek neutvrđeni faktori rizika.

Cilj rada bio je analiziranje veze između primarne arterijske hipertenzije i renalne hipertenzije i prevalencije ostalih faktora rizika od ateroskleroze kod bolesnika sa razvijenom hipertenzivnom bolešću bubrega.

U prospektivnoj studiji preseka analizirani su podaci o 283 bolesnika oba pola sa dijagnozom primarne hipertenzije i hipertenzivnom bolešću bubrega. Anamnestički podaci o uzrastu, trajanju hipertenzije, pušačkom stažu, prisustvu hipertenzivne retinopatije, hipertrofiji leve komore i podaci o prethodnim bubrežnim bolestima prikupljeni su putem intervjua i medicinske dokumentacije. Klinička ispitivanja obuhvatala su merenja krvnog pritiska, određivanje indeksa telesne mase (ITM), parametara metabolizma lipida u krvi (koncentracija ukupnog holesterola, LDL-holesterola, HDL-holesterola i triglicerida), uree i kreatinina u serumu i koncentracije proteina i albumina u urinu.

Analizirani su podaci o ukupno 283 bolesnika (185 muškaraca i 98 žena) sa HN. Podaci dobijeni na osnovu analize ukazali su na značajno veći procenat muškaraca starosti preko 60 godina sa HN. Prosečna starost ispitivanih hipertenzivnih bolesnika sa IHN bila je 62.6±8,8 godina, pri čemu je prosečno trajanje hipertenzije bilo 19.8±5.9 godina. Kod svih ispitanih bolesnika utvrđena je hipertenzivna retinopatija i hipertrofija leve komore. Većina ispitanih bolesnika (78.6%) sa HN imala je povećanu telesnu masu sa ITM od 25-30 kg/m^2. Dislipidemija i pušenje zabeleženi su kod 75% hipertenzivnih bolesnika sa HN. Najčešći poremećaji metabolizma lipida bili su hipertrigliceridemija udružena sa hiperholesterolemijom zabeležena kod 42.1% i kombinovana dislipidemija kod 25.8% bolesnika. Porodična istorija HTA zabeležena je kod svih bolesnika sa HN.

Korelacionom analizom utvrđena je značajna pozitivna veza između porodične istorije HTA ($r=0.62$, $p<0.05$), pušenja ($r=0.53$, $p<0.05$) i kombinovane dislipidemije (hipertrigliceridemija i hiperholesterolemija) ($r=0.45$, $p<0.05$) i pojave HN.

Hipertenzivna nefropatija najčešće se javlja kod starijih hipertenzivnih osoba muškog pola sa razvijenim hipertenzivnim mikro- i makrovaskularnim komplikacjama. Porodična istorija HTA, pušenje i poremećaji metabolizma lipida, naročito kombinovana dislipidemija, predstavljaju jako česte faktore rizika za razvoj hipertenzivne nefropatije. Prevencija i terapija navedenih faktora rizika predstavlja veoma važan zadatak u cilju smanjivanja broja obolelih od hipertenzivne nefropatije. *Acta Medica Medianae 2014;53(4):15-21.*

Ključne reči: *hipertenzivna nefropatija, arterijska hipertenzija, pušenje, dislipidemija*

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