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ESSENTIAL ARTERIAL HIPERTENSION (AHT) AND RENAL CHRONIC SUSPENSION

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Abstract

Arterial hypertension (AHT) is a high risk factor for patients with chronic renal failure. Over 72 million people are suffering from AHT in the US. AHT usually affects most patients with chronic renal disease (Nwankwo et al., 2011). Starting in 2011, a large number of clinical practice guidelines have been published on AHT treatment in patients with chronic renal insufficiency who are involved in the maintenance of arterial pressure <140/90 mm Hg (Chobanian *et al.,* 2003, Bansal *et al.,* 2015)A number of major studies in patients with chronic kidney failure have verified and documented that AHT treatment significantly affects rapid prevention of disease progression. According to the guidelines of the National Kidney Disease Outcome Quality Initiative (NKF / KDOI), the AHT treatment should be <130/80 mmHg to albuminuria and when the ratio of albumin to urine - creatinine> 30 mg / g which has shown high effects on clinical results and progression slowdown compared to the results of patients with arterial pressure above 140/90 mmHg (Chobanian *et al.,* 2003, Bansal *et al.,* 2015). The purpose of medically-mediated treatment for a blood pressure of <140 / 90 mm Hg or the lowest possible value tolerated by the patient should be the target of primary and secondary doctors. Adequate blood pressure control usually requires pharmacological therapy (with one or two antihetertonic drugs as well as diuretic therapy) which should be adjusted according to arterial pressure values.
Purpose: The purpose of this research is to verify the influence and effects of antihypertensive in the treatment of essential AHT in patients with CRF.
Material and Methods: In the "cross-section" study, 300 patients (170 males and 130 females) with mean median age of 55.7 ± 9.5 years and 200 healthy individuals (100 males and 100 women) with mean age: 56.8 ± 12 years who served as a control group for comparing values obtained from patients with CRF and HTA. Patients with CRF and AHT were treated with antihypertensive (ACE inhibitors and ARBs) within 12 meals - with three measurements (every 4 months). Patients were divided according to the degree of hypertension and according to the JNC-VII joint report of the national committee for the prevention and detection of AHT in 2003. The examined patients examined proteinuria, serum urea, serum creatine, uric acid, electrolyte and lipid profile in order to verify their influence on the appearance of arterial hypertension as complementary factors in the etiology of CRF.
Conclusion: In conclusion we can conclude that early detection of AHT etiology by AHT and quality treatment should be the first step of primary and secondary doctors a powerful AHT control in order to prevent rapid progression of the disease and its complications.

Keywords: arterial hypertension, chronic renal failure.

INTRODUCTION

Chronic Renal Insufficiency (CRF) represents an important global public health problem with a high prevalence of 20.00-43.35% of the population. In the paper we will present the objectives of treating the essential AHT patient with CRF that should be achieved to slow the progression of chronic kidney failure, proteinuria reduction, and renoprotective effects with antihypertensive therapy mainly angiotensin converting enzyme inhibitors (ACE- i) and angiotensin receptor blockers (ARBs). Essential AHT is accounted for as one of the major causes of CRF progression and cardiovascular disease risk (SEA). In physiological conditions, arterial pressure fluctuations may represent a homeostatic response to the nerves (e.g., central

sympathetic movement and its reflexes in arterial and cardiopulmonary reflexes), humoral (cateholamines, insulines, angiotensin II, bradykinins, endothelin-1 and nitric oxide), vascular (eg, elastic properties of the arteries) and blood viscosity to the environment (weather changes), physical activity, sleep, fatty food, and emotional stress (eg psychological stress). Under specific conditions, such as CRF, steady increase in arterial pressure may reflect significant changes in regulatory mechanisms (eg, sympathetic and impaired barrier function) with right-to-right effects on the cardiovascular system (SEA) such as left ventricular hypertrophy. There is documented evidence that between AHT and chronic renal failure (CRF) accompanied by hypercalcemia there is a high positive correlation between the patient, and the main therapeutic treatment and goal of AHT in these patients is the normalization of hypercalcemia. Patients with CRF without taking the basic illness or especially those with diabetes are recommended to reduce body weight, physical activity, fat reduction and salt in food. Evidence from a large number of clinical studies has clearly shown that effective antihypertensive treatment significantly affects the slowing of rapid IRR prognosis from essential hypertension (Parati *et al.,* 2013). Chronic kidney disease, regardless of cause, tends to progress, albeit at different rhythms. Blocking the renin-angiotensin system and blood pressure control are the pillars of current management guidelines to prevent the progression of chronic kidney disease. In recent years, a number of studies have verified and documented that there is a high positive correlation between HTA, CRF progression and hypertriglyceridemia. Common HA effects of hyperlipidemia significantly affect the modification and reduction in renal function by causing nephroangiosclerosis with glomerulosclerosis (Appel *et al.*, 2010, Sinclari *et al.,* 1987). Essential HTAs and dyslipidemia also remain a high-frequency problem and pose a difficulty for treating patients with CRF taking into account electrolyte (especially sodium) and water balance. AHT is present in 80% of cases of patients with CRF. For this reason, essential AHT treatment should consist of limiting the consumption of crypto, fatty foods, sodium and eliminating liquids or excess water.

Treatment of essential AHT affects the slowing progression of CRF especially in patients with proteinuria> 1g / 24 hours. Of all the causes leading to CRF, it can be understood that there is not only one mechanism in its presentation, but the etiology of CRF is manifold, therefore the early detection of all mechanisms leading to CRF, its prevention and treatment therapeutic early stages can positively influence the prevention of rapid progression of CRF and its complications in the cardiovascular system (Coresh *et al.,* 2007, KDIGOKBPW 2012, James *et al.,* 2014).The purpose of this research is to verify the influence and effects of antihypertensive in the treatment of essential AHT in patients with CRF.

Materials and Means: In the "cross-section" study, 300 patients (170 males and 130 females) with an average median of 55.7 ± 9.5 years and 200 healthy individuals (100 males and 100 women) with mean age: 56.8 ± 12.0 years who served as a control group for comparing values obtained from patients with CRF and HTA. Patients with CRF and AHT were treated with antihypertensive (ACE inhibitors and ARBs) within 12 measurements-with three measurements (every 4 months). Patients were divided according to the hypertension rate and according to the JNC-VII joint report of the national committee for the prevention and detection of AHT in 2003. The examined patients examined proteinuria, serum urea, serum creatine, uric acid, electrolyte and lipid profile in order to verify their influence on the appearance of arterial hypertension as complementary factors in the etiology of CRF.

Table 1. Presentation of patients according to gender, average age (N^O= 300) and control group N^O = 200

Gender	Total number N ^{o} = 300 (100%)	The average age ± SD	The average age of the control group=200 (F-100+M-100) ± SD
Male	**N ^{0} = 170 (55%)**	**55,70 ± 9,50**	**56,80 ± 12.00**
Female	**N ^{0} = 130 (45%)**	**55,70 ± 9,50**	**56,80 ± 12,00**

RESULTS

Results from measurements obtained are presented in tabular.

Table 2. The definition of progress, remission and regression of the chronic nephropathies manifested by proteinuria

Settings	Progression	Remission	Regression
Proteinuria	> 1.80 g / 24 h	<1.05 g / 24 h	<0.80 g / 24 h
GFR	GFR reduced	GF stable	GF increased
Kidney structure	GFR deteriorated	GF stable	GF improved

Table 3. Presentation of proteinuria values of 300 patients obtained from patients before use and after use of ACE inhibitors p r j 20 mg and values control group of 100 healthy individuals.

Proteinuria	Before therapy with ACE inhibitor 20 mg	After 24 months therapy with ACE inhibitors	Group controller - (healthy) N^{O}- 200
Male-170	> 3.40 g / 24 h	1.05 g / 24 h↓	< 0. 35 g / 24 h
Female-130	> 3.60 g / 24 h	1.15 g / 24 h↓	< 0. 35 g / 24 ^{h}

Table 4. Presentation of average values of patients (N^{O} = 300) for urea, uric acid, kreatinine and treatment before starting the arterial hypertension

Settings	Females N^{O}= 130 (45 %)	Males N^{O} = 170 (55%
	Average ± SD	Average ± SD
Urea	18.50± 4,50	17.90±4.00
Creatinine	320.00±10.00	290.80±14,00
Ac. uric	420,00±19,00	460,00±15,00

Table 5. Presentation of average values of patients (N ^{o} = 300) for urea, creatinine and uric acid after 12 months of treatment with AH

Settings	Females N^{O}= 130 (45 %)	Males N^{O} = 170 (%5%
	Average ± SD	Average ± SD
Urea	25,00 ± 3,50	24,60 ±4,00
Creatinine	380,00±14,00	390,00±12,50
Ac. uric	370,00±10.00	390,00±9,60

From the table itself is noticed a slight increase in nitrogen products (urea, creatinine uric acid but are non-significant and shows a chronic renal failure non progressive.

Table 6. Values obtained for GFR * ml / min / 1.73m^2 according to Cockroft & Gault formula after treatment with ACE inhibitor for 12 months

The value of AH before therapy	M=170	F=130	Therapy 20 mg ACE inhibitor	GFR * before the therapy	GFR values and AH after 24 months use of ACE in the dose of 20 mg
AH high risk 220/120 mmHg	20	10	2x1 plus diuretic	17,50	GFR-22,00 TA = 160/90 mmHg
AH very high 180/110 mmHg	40	50	2x1 plu s diuretic	42,80	GFR-58,50↑ TA = 140/95 mmHg
AH high 160/100 mmHg	80	40	2x1 plus diuretic	67,50	GFR-78,00 ↑ TA = 135/85 mmHg
AH mild 145/90 mmHg	30	30	1x1	70,60	GFR-84,00↑ TA = 130/85 mmHg

GFR - Glomerular Filtration Rate*

As can be seen from the table itself, we can confirm that with the treatment of AHT there is no progression of the disease with any statistical significance.

DISCUSSION

Pathological processes of AHT resulting in progressive loss with the dominance of essential hypertension are still completely unexplored and multifactorial. From our study we documented a very positive effect on AHT treatment in patients with CRF after 12 treatment missions and a slowdown in the progress of the disease was also verified according to the glomerular filtration values determined with the help of the Coccfoft & Gault formulation. However, the slowest progressive vascular pathology of benign nephroszleroscopy eventually results in glomerular ischemia and nephron loss. By contrast, significant reductions in functional kidney size during chronic kidney disease are associated with nearby arteriolar vasodilation, impaired self-regulation, and increased blood pressure transmission in glomerular capillaries resulting in proteinuria, glomerulosclerosis, and faster progression of CRF . It is imperative that we prevent the progress of CRF in AHT patients in addition to the normalization of HTA, we should appreciate the proteinuria manifestation and its correction. Double Therapy (ACE inhibitors and ARBs) in treating AHT essential in CRF patients affects proteinuria decrease to a greater degree and prevents cardiovascular injury (Krause *et al.,* 2011, Weber *et al.,* 2014). For the treatment of essential AHT in patients with cardiovascular disease, we must always consider the nature of the underlying renal disease.
Treatment with ACE inhibitors or ARBs should target a <130/80 mm Hg. There are verified facts and arguments that the kidneys play an important role in long-term arterial pressure regulation. Critical role in extending the volume of extracellular fluid in patients with CRF with manifestations to SEQ has ultrafiltration, hypernatremia and excessive salt amount in the body. The positive balance of salt is dominant, but not the main factor in the AHT genesis in patients with CRF. As mentioned above, experimental evidence clearly indicates that AHT in patients with CRF due to salt and excess water in the body appears due to increased peripheral resistance and the effect of the renin-angiotensin-aldosterone system. In the aforementioned diseases, ACE inhibitors are used as a qualitative choice and given that some ACE inhibitors have shown high positive effects compared to other drug groups in slowing the pace of CRF progression and reducing the mortality and mortality of patients with CRF . Prevention of CRF progression requires treatment and treatment of AHT in patients with proteinuria of 0.25-1.0g

for 24 hours should be ≤ 130/80 while patients with proteinuria of ≥ 1.0 g per 24 hours should be ≤ 125/75 mmHg. In this paper we examine current evidence that support the treatment of essential hypertension in various forms of kidney cancer and that are consistent with recent studies on the treatment of essential AHT in patients with CRF.

The recent National Guidelines of the National Joint Commission on the Prevention, Detection, Evaluation and Treatment of High Voltage VII (JNC VII) and the Kidney Disease Quality Initiative (K / DOQI) recommend blood pressure <130/80 for the purpose of treatment for patients with cardiovascular disease. Arterial hypertension (HTA) still remains the leading cause of SEA in patients with CRF. AHT during CRF is most often of the voluminous type as a result of hypernatremia as well as disorders of the renin-angiotensin aldosterone system. To treat the essential AHT in the patient with CRF should always take into account the important role of the sodium balance because chronic renal disease is accompanied by sodium retention because there is a kidney disability to eliminate the proper amount of Na. In the manifestation of hypertensive syndrome there are several mechanisms: excessive amounts of water, electrolyte disturbances and relationship disorders between sodium and Renin-Angiotensine-Aldosterone, disorder of "auto-regulation" function and peripheral resistance, excessive salt consumption , diabetes, dyslipidaemia, etc.

There are a large number of studies on the effects and actions of antihypertensive drugs in AHT patients, the action and the effect of ACE inhibitors of 10mg or 20mg or ACE inhibitors in combination with diuretics however ACE remains one of the most preferred drugs during the AHT of patients with CRF. Medications of these action groups develop their hypotensive effect by blocking the effect of dipeptide-carboxy-peptidase and with increasing levels of inhibiting the conversion of Angitensine-I (AI) to Angiotensine-II, slowing the conversion of bradykinin and quinine to reducing the charming activity thus reduces and prevents the negative effects of kidney hypertension. In our paper we have verified that the qualitative, appropriate and timely treatment of essential AHT with ACE inhibitors in CRF patients significantly affects the slowing down of disease progression by reducing proteinuria and the side effects of AHT related to CRF. AHT treatment and treatment in patients with CRF should begin at the early stages of the disease. Inadequate AHT treatment in patients with CRF is apparently experiencing rapid progression of disease deterioration and manifestations to the cardiovascular system. In recent years, high ACE inhibitors (Perindopril, Ramipril, Lisinopril, Captopril, Quinapril, Enalapril, etc.) have been shown to have a very high positive effect on AHT treatment and a new group of angiotensin-IA receptor antagonists (Losartan, Irbesartan , Candesartan, Valsartan, Telmisartan, etc.) compared to other antihypertensives (Kunz *et al.,* 2008, Navaneethan *et al.,* 2009). In severe cases of AHT patients need to be treated with two or more antihimeonic and combined with diuretic.

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