THE EFFECTS OF HYPERTENSION (HTA) IN PROGRESS OF CHRONIC RENAL FAILURE

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Abstract: In the past 20 years, doctors have clearly shown that antihypertensive therapy is very effective in reducing the incidence of myocardial infarction and stroke. However, little is known about the effects of blood pressure reduction in the end of renal disease stage (ESRD). Data from large clinical studies have clearly shown that patients with hypertension have an increased risk of developing ESRD.Black men and women with hypertension are at greatest risk;However, the incidence of ESRD has increased in all racial groups. Because hypertensive patients with ESRD require dialysis often, the cost of treatment of this disease is extremely expensive.The main effect of reducing blood pressure in patients with ESRD is not properly treated.Results from several studies show that lowering blood pressure may improve kidney function and that ACE inhibitors and calcium blockers in the United States,there is an increased incidence ofchronic renal failure and high cost due to treatment.Therefore, strategies aimed for identification,prevention and treatment of CKD and its associated factors with the risk of the disease are so important. In the discussion we focus on the role of hypertension in the development and progress of chronic renal disease.We will also present the high blood pressure objectives to be achieved to slow the progression of chronic terminal renal failure, the influence of proteinuria as well as on renoprotective effects antihypertensive therapy -mainly angiotensin-converting enzyme inhibitors (ACE-i) and blockers angiotensin receptor (ARB) -and achieved level of arterial pressure. Arterial hypertension (HTA) is counted as one of the main causes that affect the progression of chronic kidney disease (CKD) and the risk of cardiovascular diseases (CVD) .Numerous studies verify thatthere is a strong link between arterial hypertension (HTA) and chronic kidney failure .Arterial hypertension is an important cause of IRK,there are some other diseases too that are contributing for the progression of the disease.On the other hand, hypertension is very spread in patients with IRK (Kidney Chronic Disease-CKD), having a very important role in CVD appearance and high degree of mortality in patients with terminal chronic renal failure (ESRD End-Stage Renal Disease). This chapter will focus on the pathogenesis of HTA and the impact of it in progress of IRK.The etiology of arterial hypertension is multifactorial(close to 20-25% of cases with HTA know their etilogjy while other cases are due to many other disorders : hormonal, renal, cardiac, infectious, congenital or inherited kidney RVU etc).Except HTA that affects in the progress of chronic failure kidney the disorders of lipid metabolismalso affect in patients with CKD which are described for the first time ,in 1827 by Dr. Bright, especially in patients with nephrotic syndrome (1).it is known that patients with CKD present early atherosclerosis and cardiovascular, cerebrovascular complications more frequent and in younger age compared to healthy population.Arterial hypertension is an independent risk factor for the rapid pace of high CKD consequences to cardiovascular disease and high mortality increase (2,3,4,5) . The prevalence of CKD in fact is defined by the level of renal injuries randomized by values of instance glomerular filtration (GFR-Glomerular Filtration Rate) by Coccroft& Gault formul .HTA is counted as the second cause of chronic renal failure patients .There are documented facts that the high blood pressure significantly accelerates the weakening of the rate of glomerular filtration especially in patients with diabetic nephropathy and proteinemia. High positive correlation between hypertension and chronic renal failure for the first is verified in the 19th century .

Key words : Aterial hypertension(HTA) , IRKT (ESRD), lipid profile .

1 INTRODUCTION

Arterial hypertension (HTA) further remains one of the moston all the preferences and the World Health Organization all the common factors of disease worldwide. Between normotensive andvalues of systolic> 140 mmHg and diastolic> 90 mmHg are treated arterial hypetension persists not any precise definition, but basedas arterial hypertension. In recent years a number of studies have

verified and documented that between HTA and lipid abnormality progress of CKD, there is a positive correlation .Common effects of HTA and uremic hyperlipidemia clearly affect in modification and lowering renal functioons causing nephroangiosclerosis with glomerulosceloris.It is rated that 10-13% of elderly eight patients in the US suffer from CKD and HTA regardless the level of CKD.HTA during IRK is more (with manifestations of cardiovascular complications with cardiomyopathy hipertenzive etc. (6),even sometimes can be consequence of other mechanism for example hypernatremia etc. Numerous clinical studies have shown that adequate treatment and in the right time of HTA and dyslipidemia in patients with chronic CKD significantly have lower frequency of myocardial infarction, hypertrophy of the left ventricle, inadequacy of heart disease, peripheral arterial disease, retinopathy, thrombo- lytic processes and the presence of cerebrovascular stroke.According to etiology in internal medicine arterial hypertension is divided into: 1. secondary HTA which appears as a result of paren-chyma diseases ,nephrosclerosis, pheochro- mocytoma, primary aldosteronism,Cushing syndrome, etc.And 2. essential arterial hypertension (idiopathic factor) causes of which are unknown etiology and this group of HTA includes near 80- 90%,although in its etiology are counted many different factors such as genetic predisposition,adiposity, age, gender, sedentarity,stress,socioeconomic status etc.Arterial hypertension and dyslipidemia further remain as the most frequent and difficult problem to treat patients with CKD considering the imbalance of electrolytes especially in the report of renin-angiotensin aldosteronsystem.Inpatients with CKD and HTA are verified high value od LDL ch and TG and low HDL ch compared with healthy control group.Due the values of high concentrations of LDL-ch it appears injuries of in the endothelial cells, in the wall of blood vessels,with decreasing the synthesis of prostaglandin 2 (PGI 2) (with its fibrinolytic and antithrombotic effect)and with exfoliation and collection effect of ox- LDL (oxidized cholesterol) in macrophages and smooth muscle cells (7,8,9).

As the cause of lesions endothelial and stratification of LDL-ox to the walls of blood vesselsexcept other causes important role has HTA too, especially excessiveoscillations causing circular movement of blood with endothelial damages and the beginning of exfoliation the lipids in the walls of blood vessels.

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There are facts that in patients with CKD and HTA concentrations of atherogenic LDL-ch ,triglycerides are (TG) are significantly elevated, while concentrations of HDL-ch defense (antiaterogenic) are significantly lower (which was also verified in our studies)and the the consequences of cardiovascular diseases, cerebrovascular and early atherosclerosis that are highly elevated compared with the healthy population and the control group of healthy volunteer patients (10,11,12,13,).The determination of lipid abnormalities in patients with CKD to accompanied with HTA in the early stages of the disease,also the discovery ofetiopathogenic mechanisms can significantly help in preventing their consequences, which significantly will decrease the appearance of cardiovascular diseases, cerebrovascular and vascular atherogenicprocesses .Atherosclerotic lesions begin with damageof vascular endothelial cells (14,15).The prevalence of HTA to patients with CKD is higher among patients with overweight or BDMx (Body-Mass -Index, BMIx = TT / kg / x TV-2> 24.3±5.2 kg / m. Approximately one in three adults in the United States suffers from HTA, which represents the soci-economic problem for the country (16) .The prevalence of Prevalenca hypertension also varies in relation with CKD and has a high positive correlation. Various associations which deal with HTA and CKD studies have verified that the highest prevalence of CKD patients is with renal artery stenosis of approximately - 93% to 87%-diabetic nephropathy, kidney and polycystic adult disease with 74% (17) If HTA in patients with CKD is associated also with any glomerulopathy or diabetic nephropathy then the impact will be manifested by an very large kidney injury accompanied with vascular injury,nephroloarthero sclerosis and glomerular proliferation, with a speed toward uremia,when the only medication is the treatment with HD intermittent. It defines the intensive loss of kidney tissue and leads to progression of CKD. HTA is present in 80% of cases in patients with CKD.In fundamental illnesses of CKD associated with HTA there is an inability of kidney to eliminate the proper amount of natrium.HTA has a huge impact on the cardiovascular system with symptoms of left ventricular insufficiency,which can be accompanied with dyspnoea and edema pulmonar.The treatment of arterial hypertension affects in slowing progression of CKD especially patients who have proteinuria> 1 g / 24h. In recent years it has proved that the best effects during treatment HTA showed ACE-inhibitors (lisinopril, captopril, Ramipri, Perindopril, Skopril, Enalapril, l, ...)as well as a new group of drugs ARB- antagonists-angiotensin receptor of Angiotensin1 (Irbersartan, Candesartan, Valsartan, losartan, Cossaar ... etc.)compared to other antihypertonics.In the progress of IRK also affect:free radicals, Growth factors (PDGF-platelet Derived Growth Factor, TGF-b-Transforming Growth Factor-b), coagulation,prostaglandins,, age , gender, race, genetic factors, consumption of tobacco,renin angiotensin system(SRA), MIA syndrome (Malnutritio-Inflamatio-Atherosclerosis),renal anemia, nflammation,the impact of proinflammatory cytokines-interleukin, diabetes, diabetic nephropathy, disorders of glomerular hemodynamics,uremic dyslipidemia,dietary proteins, hyperfosfa- temia,renal anemia, food and eating excessive amounts of calories, horrmones, etc.From all the causes which can lead to CKD we can clearly see that in its appearance there is not only one mechanism,but the etiology of CKD is multifactorial therefore early detection of all mechanisms leading to CKD,prevention hygienic-dietary and medication in early stages can affect positively in slowing,therhythm,the speed and the progress od CKD and its complications to the cardiovascular system, brain and early atherosclerosis.In SMKV genesis of patients with HTA and IRK are counted: the concentrations of oxidized cholesterol Lara-LDLox, oxidative stress,impact vasoconstrictor mechanisms as:high plasmatic concentration of endothelin-1 potent vasoconstrictor.The synthesis of nitric oxide (NO) which is counted as the strongest and the most effective vasodilator in uremia is blocked due to accumulation of excess and due reduced synthesis of nitrogen oxide (NO) by Dimenthyl-L- Argenine asymmetric (DMAA) (18-22). There verified facts and arguments that the kidneys play an important role in long-term regulation of arterial pressure Guyton and that HTA can't be presented if there are not renal injuries and sodium disorders.In fact, almost all forms of experimental and human hypertension manifest concentration disorders of sodium secretion with or without normal blood pressure (23,24). In experiments using large and isolated animals

with HTA Guyton showed that there is a rapid normalization of arterial pressure after rapid stimulation and high sodium renal excretion. On the other hand, the sodium loading showed an increase in arterial pressure when the renal excretion was conditioned by inhibition of sodium excretion or from the influence of mechanisms angiotensinit or aldosterone In these circumstances, the increase in blood pressure was initially mediated by overload of the volume of extracellular fluid (ECF),, despite a reduction in total peripheral resistance.In this case, the increase in blood pressure is manifested by enlargement of the heart and increased systolic pressure .There are documented facts during the fatal accidents that hypertensive patients had less functional nephrones compared with individuals who have been dead but had normal pressure in autopsy(25) .The nature of renal defects which are responsible for excretion of sodium inappropriate,or factors that mediate the subsequent increase of peripheral resistance are yet unkonown.Critical role of enlargement of the extracellular fluid volume in patients with CKD and HTA with frequent manifestations on the cardiovascular system has the influence of ultra filtrationtoo,hypernatremia,and the amount of excess of salt in body.The positive balance of salt is dominant ,but not the only factor in the genesis of hypertension in patients with CKD. [26).In patients with HTA and CKD who are treated with hemodialysis (HD) or peritoneal dialysis (to regimes of 3 times a week, 5-6 hours) was found in a significant improvement of HTA and improvement of left ventricular hypertrophy and decrease the prevalence of mortality. [27.28).As mentioned above, experimental evidence have clearly demonstrated that in patients with CKD HTA due to salt retention and excess water in the body appears as a result of increased peripheral resistance and impact of the renin - angiotensin – aldosteron system - RAAS (29) . There are facts verified that although renal function is saved activation of the RAAS is an important factor in the pathogenesis of HTA in patients with polycystic kidney and is supposed that it appears as a result of vascular overload from permanent growth of cysts(30).

II.THE AIM OF THIS RESEARCH

The aim of this research is to verify the impact of HTA in the rate of CKD process and the manifestations of HTA on the appearance of cardiovascular diseases (CVD) . This research also aims the detection of positive effects and impact of ACE inhibitors in preventing the progress of the rapid progression of CKD and treatment of arterial hypertension (HTA) treated in the Department of Internal Diseases in Hospital of Tetova and in Special Hospital of Nephrology and Hemodialyse ,, Vita Medical Group of Tetova".

III. Methodology and materials

On the prospective cohort study (,, cross-section ")were total included N0 = 120 (of whom 66 were male with an average age of: 58.60± 14:00)while 54 were female with an average age of: 56.00±12.50).In the study there was also a control group who had N^{O}-120 healthy individuals (66 male and 54 female with an average age of 55.80 ± 12.60)that served for comparing the values obtained from examined parameters.Patients with HTA and IRK treated with ACE inhibitors in Department of Internal Diseases in Hospital of Tetova and in Special Hospital of Nephrology and Hemodialyse ,, Vita Medical Group of Tetova".We devided the patients according to the level of hypertension ,according to criteria report of VII të JNC-Joint National Committe on Prevention Detection , Elevation Treatment of High Blood Pressure year 2003.At examined patients we did examination of proteinuria, the presence of urea in serum,creatinine in serum,,uricacid,electrolytes,profile of lipids for total lipids(TL), total cholesterol (CHT), triglycerides (TG), HDL-ch, LDL-ch with intending to verify their impact on the presentation of arterial hypertension as complementary factors in the display the etiology of CKD.

Table number.1: Presentation of patients by gender, age average (NO = 120) and control group N^{O} = 120

Gender	Total number N^{o}= 120(100%)	Average age ± SD	Average age of control group± SD
Male	N^{0}=66 (55 %)	58.60± 14.00	55.80 ± 12.60
Female	N^{0}=54 (45 %)	56.00±12.50	55.80 ± 12.60

The average age of male patients was 58.60± = 14.00 while the female gender was = 56.00±12.50 ,the difference of average age between male and female according to statistics is not significant with p = 0.0005 , which indicates for a homogeneous groups (table number 1) .
In table No. 2 , there are identified normal and pathological values of albumin and protein.

Values:	Microalbuminuria	Proteinuria
M= 17-250 mg/L	M= < 17mg/L	M= > 250.0
F = < 25 mg/L	F= 25- 355 mg/L	F= 355.0mg/L

IV. RESULTS

The results of measurements obtained are presented in tables and graphs .
Table nr.3 The definition of the progression,remission, and regression of chronic nephropathymanifested by proteinuria

Parameters	Progression	Remissoin	Regression
Proteinuria	>1.8 g/24 hours	< 1.0 g/24 hours	<0.30 g/24 hours
Grade of FG	LowFG	FG stabil	High FG i
Kidney structure	High FG	FG stabil	Improved FG

Table number 4 : Presentation of average values of patients with acquired HTA of total- (N^{o} -120) for Total Lipid (LT) ,Total Cholesterol (CHT) , triglycerides (TG) , HDL - ch , LDL – ch

Examinded parametres	Total number of patients	Average ± SD
LT	120	6.40 ± 1.20
TG	120	3.80± 1.50 ↑
ChT	120	5.20 ± 2.70 ↑
HDL-ch	120	1.04 ± 0.80↓
LDL-ch	120	4.80 ± 0.70↑

In the table number 4 is noted that in the examined patients there is a significant increase with $p < 0.0001$ membership of LDL - ch factions , CHT , and TG , while a decrease in a significant difference with $p < 0.0001$ for HDL-ch.with referent values of the lipids

.Table number 5 : Presentation of proteinuria values of 120 patients (66 male , 54 female) received by patients before use and after use of ACE inhibitors of 20 mg and values of the controller group of 120 healthy individuals .

	Proteinuria before the therapy ACE inhibitor from 20mg	Proteinuria after the therapy of 12 with ACE inhibitor	(Healthy)- Controller group N^{O}=120
Male N^{O}=66	>3.40 g/24 hours↑↑	1.20 g/24 hours ↓↓	<0.4 g/24 hours
Female N^{O}=54	> 3.20 g/24 hours↑↑	1.22 g/24 hours ↓↓	

Table No. 6 : Presentation of average values of patients (No = 120) for Blood sugar , urea , creatinine and uric acid before the treatment of HTA.

Parametres	Female-54 (45 %)	Male=65 (55 %)
	Average value ± SD	Average value ± SD
Glycemia	6.50 ± 2.80	6.40 ±3.60
Urea	17.60 ± 2.45	18.00 ± 4.20
Creatinine	340.0 ± 12,60	275.00 ±26.30
Uric acid	390.50 ± 3.50	365.00 ±35,80

Table 7 : Presentation of average values of patients (No = 120) for Blood sugar , urea , creatinine and uric acid after 12 months of treatment of HTA.

Parametres	Female-54 (45 %)	Male=65 (55 %)
	Average value ± SD	Average value ± SD
Glycemia	6.40 ± 1.40	6.80 ±1.50
Urea	18.50 ± 3.60	17.80 ± 4.60
Creatinine	370.00 ± 20.00	320.00 ±26.30
Uric acid	340.50 ± 26.00	370.00 ±38.60

From the table itself it is noticed a slight increase in the products of nitrogen (urea , creatinine ,uric acid but not significant and it shows about chronic renal insufficiency but not progredient.

Table 8.Division of patients (NO = 120) of the HTA examined by the degree and the report for VII of JNC (Joint National Committe-2003).

Category of hypertension	Male N^{o}= 66	Female N
High HTA	26	18
HTA first degree easy	10	13
HTA second degree average	12	8
HTA third degree heavy isolated systolic	8	9
HTA isolated systolic	10	6

Table number 9. Values earned for GFR * ml / min / 1.73m2 by Cockroft& Gault formula after ACE inhibitor therapy after 6 months

Values of HTA before therapy	M*N^{O} =66	F*N =54	Therapy with ACE inhibitor 20 mg	GFR * Before therap	Values of GFRand HTA after 8 monthstof using ACE inhibitors from 20 mg
Dangerous HTA 220/120 mmHg	28	21	2x1 plus diuretic	GFR-20	GFR-27↑; TA=150/95 mmHg
Very high HTA 180/110 mmHg	17	14	2x1 plus diuretic	GFR-40	GFR-54↑; TA=140/90 mmHg
High HTA -160/100 mmHg.	12	12	2x1	GFR-65	GFR-78 ↑; TA=135/90 mmHg
Easy high HTA -145/90 mmHg	9	7	1x1	GFR-70	Gfr-87; TA=130/80↑ mmHg

***M**-male ;F-female; * GFR-Glomerular Filtration Rate.

V. DISCUSSION

Arterial hypertension (HTA) remains the most common hemodialysis with the purpose of normalization of HTA and cardiovascular manifestation during the IRK .On the terminal reduction of cardiovascular consequences ,with this also will phase of uremia known as comes to hyperhydration,respectively decrease the mortality rate (50-6 % as a result of CVD (31).All increases the volume of extracellular fluid which results in HTA the instructions by many national associations (JNC VII report of - and edema syndrome.HTA during IRK often is of volumetrically Joint National Committe on Prevention Detection, Elevation type even if sometimes it may be as a result of other Treatment of High Blood Pressure - 1993), European Society of mechanisms such as during hypernatremia ,disorders of the renin Hypertension (ESH 2003) and European Society of Cardiology- - angiotensin – anldosteron system etc.Patients with HTA and ESC 2003 K / DOQI- Kidney Disease Outcomes Quality Initiative) CKD are potential candidates with a high level of cardiovascular on prevention,early diagnosing, evaluation, and early treatment of disease (CVD) compared with the general population and in the CKD and HTA recommend that the purpose of treatment to terminal phase is more than required treatment with be:systolic blood pressure <130 mmHg while diastolic = 80 mmHg

(32,33).In prevention of progress of CKD in patients with HTAwhich promote the releasing of cytokines and the growth factor of except the normalization of HTA it is also needed the evaluationmesangial cells andcylindrical epithelial cells causing vascular of proteinuria and its correction.Several studies have documentedlesions. The prevalence of HTA which is counted as main risk that patients with HTA and CKD(especially in patients> 70 yearsfactor of cardiovascular related illness (CVD) occurs in 28% of the old with CKD might cause high risk to cardiovascular diseasesadult population.Iin the US from HTA suffer 37-39% while in the especially to suffer from acute myocardial infarction if systolicworld from HTA total of 1 million residents.It is supposed that the blood pressure decreased <120 and diastolic <80 mm Hg. prevalence of appearance of HTA is increasing,it will also increase (34).The double therapy with ACE inhibitors and ARBs) of HTA inthe prevalence of CKD,cardiovascular diseases and cerebral patients with CKD affects in reduction of proteinuria a higherstroke(36, 37, 38).In basic way it defines the loss of quantity of degree ,but there are no verified and documented studies thatkidney tissue and leads into glomerular filtration rate (FG) .Exact double medication can impact on the preservation of renalmechanisms of kidney damage in patients with hypertension still function, or the prevention of cardiovascular injuries, comparedremain unclear.Two additional pathogenic mechanisms in the end with therapy ACE inhibitors combined with any diuretic (whichfinish in kidney fibrosis ranging changes in macrosystem and affects handling overload volume or hypercalemia (35).For moremicrovasculare , auto-regulation disorders of capilaro- intraglo-adequate treatment of the HTA in patients with CKD we alwaysmerular pressure mediated from hyperfiltration. Hyperfiltra-tion must take in consideration the nature of basic kidney disease.Theleads to transglomerular loss of protein that stimulates release of purpose of of treatment with ACE inhibitors or ARBs should aim acytokines and the growth factor from mesangial cellsand epithelial pressure <130/80 mm Hg while the blood pressure of <140/90 mmcells causing vascular lesions.The prevalence of HTA which is Hg is acceptable for a large number of patients but with othercounted as main risk factor of cardiovascular related illness (CVD) forms of CKD. Double or triple treatment of HTA should generallyoccurs in 28% of the adult population. In the US from HTA suffer be avoided.Studies on HTA treatment of patients with CKD aremore than 37-39% while in the world from HTA suffer total of 1 controversial and still it is not known exactly what level should gomillion people.It is supposed that the prevalence of appearance of down arterial pressure that kidney to be maximally protected fromHTA is increasing also will increase the prevalence of of CKD, its impact.Uncertainty of mentioned postulates also exists aboutcardiovascular diseases and cerebral insults (36, 37, 38).It the quantitative relationship between blood pressure levels anddefines in a fundamental manner the kidney tissue loss and leads progressive renal failure. Treatment of HTA and normalization ofto progression of CKD.Hypertension of volumetrically type that hyperlipidemia has shown positive effects on slowing the progressoccurs in uremia brings also the presentation of cardiovascular of CKD and reducing hypercreatinemia in patients who takecomplications with hypertensive cardiomyopathy with left carefully and in time the antihypertensive therapy while preservingventricular hypertrophy.HTA damages in silent way kidney the values of pressure from 120 ... 130/85 mm Hg (values causing nephro atherosclerosis which leads up to CKD terminalis preferred for patients with renal impairment).Except the slowdownwhen the only treatment is with HD intermittent.HTA is present in of CKD pace its normalization significantly affects in the prevention80% of cases in patients with CKD.HTA incidence varies by type od cardiovascular disease (acute myocardial infarction, anginaof underlying primary renal disease for example.Patients with pectoris congestive heart failure, cerebrovascular stroke etc.)Lastchronic glomerulonephritis have higher incidence of HTA years in the prevention of progress of CKD and glomerularcompared with patients with interstitial tubules (eg pyelonephritis). filtration increase in patients with CKD and HTA important role hasTherefore, to treat HTA with a better effect should be considered also shown restriction of protein consumption , which with a morethe important role of Na balance in the pathogenesis of HTA.In rigorous diet affect in slowing the rhytm of CKD process.Similargeneral kidney diseases accompanied with HTA such as results are observed in hypertensive patients with CKD with aglomerulonephritis (GMN),accompanied by detention type of Na, minimum dosage of an antihypertensive before sleep.Thesewhile kidney disease who do not have the HTA frequent events results together are evidence that verify conclusions of many(illnesses tubulointersticiale),are not accompanied by detention scientists on positive correlation of salt consumption with HTA intype of Na .In fundamental illnesses of IRK associated with HTA patients with malignant hypertension and to patients with CKDexists an inability of kidney in elimination of the right amount of from strong reasons for reducing consumption of salt as aNa.Clinical symptoms of HTA that accompanies IRK do not differ mechanism to slow the pace of progress of diabeticfrom those with HTA with other etiology. Of other symptoms there nephropathy.The consumption of daily protein should not past aare : heart enlarge, at the end of the eye are observed hypertonic value of 0.5 - 0.6 g / kg / day .. Over diseases of thechanges (fundus hypertonicus)with different degrees depending of aforementioned ACE inhibitors are used as qualitative choiceduration of hypertension.The treatment of HTA is of great andconsidering that some ACE inhibitor have shown high positiveimportance to preserve residual renal function and prevent effects on other groups of drugs in slowing the pace of progress ofdamage of blood vessels.Important symptoms and complications CKD and reduce the morbidity and mortality of patients.Preventionwhich appear during chronic renal insufficiency are:congestive of progress of CKD requires treatment of arterial hypertensioncardiac insufficiency which appears during the excessive (HTA) also arterial pressure in patients with proteinuria of 0.25-1.0consumption of salt.It is proved from the fact that during use of g / 24 hours should be ≤ 130/80 while the of patients withHD and ultra filtration (extraction of fluids) congestive cardiac proteinuria ≥ 1.0 g / 24 org / 24 hours should be ≤ 125/75 mmHg. insufficiency condition is improved very good.Hypertension in Proteinuria also shows on efficiency of the action and impact ofpatients with renal damages is presented in 80%of them.This ACE inhibators while as parameter serves us eliminate thephenomenon is in positive correlation with the progressive loss of reduced protein in urine during 24 hours.We can say that thekidney function respectfully of nephrons.In appearance of regression is achieved when through urine the loss of proteinshypertensive syndrome affect several mechanisms: water is <0.3 g / 24 hours, and when the glomerular filtration rate (FG) iselectrolyte disorders and disorders of the relationship between the improved.Accurate mechanisms of kidney damage in patients withamount of sodium and renineangiotensinealdosteron hypertension still remain unclear.Two additional pathogenicsystem,function disorder of “autoregulation” and peripheral mechanisms in the end finish in kidney fibrosis by starting withresistance,excessive consumption of salt,dyslipidemiaetc .HTA macrosystem and microvascular changes,disorders of auto- remains a complication of permanent malignant,and most often it adjustment of intraglomerular pressure mediated byis the type of voluminous hypertension even if it can be with other hyperfiltration.Hyperfiltration leads to trans glomerular protein lossorigin and mechanism. Diagnosis is based not only on the

absolute values of the arterial pressure but also under its/day.According to facts and studies made about the treatment of accompanying symptoms as they are :Cefalea , nausea,votimus,HTA in patients with IRK all groups of antihypertensive drugs are confusion,mitralinsuficiency,pulmonary edema etc.Theeffective but the last years more favorable effects in treatment of aforementioned effects grow if arterial hypertension isHTA and low side effects have shown ACE inhibators and accompanied by hyperlipidemia, diabetes,hyperfibrinogenemiacombined medications that block aT-1 receptors, angiotensin II[the and MIA syndrome (Malnutritio-infl-Matio-Atheroskle -rosis) .Asso called ARB :Losartan, Valsartan, Candesartan, Eprosartan, arterial hypertension is considered systolic level of 140 mmHg andIbersartan, Cossaar, Telmisartan) Xalec + HCT (Candesartan 8 diastolic pressure 90 mmHg.Numerous studies have shown thatmg + 12.5 hidrohlorothiazid)] with what modified the effect of the the increase in peripheral vascular resistance is closely related tosystem renin-angiotensin-aldosteron (RAAS), which during the renin angiotensin aldosterone system and distorted reportsHTA is disturbed (39.40).There have been many studies on the between concentrations of calcium and parathyroid hormonepositive effects of various antihypertensive,of their actions and (HPT) also disorders in vasodilatators ,kinins,prostaglandins,andeffects and all have verified that ACE inhibators angiotensin-disorders of neuromuscular system in level of arterioles.There areconverting-enzyme more preferred for the treatment of HTA with a number of studies on the effects and action of antihypertensiverenal origin and in patients with CKD.The different ways in drugs in patients with HTA and effect of ACE-inhibitors of 10 mg orcorrecting the HTA and crises in patients with IRK include the 20 mg or ACE inhibitor combined with a diuretic but ACE inhibitorsmost important part of prevention in the quickly the progress of further remain as one of the favorite drugs during hypertensivechronic renal failure (41.42).Therefore we can conclude that the crisis and treatment of HTA in patients with CKD.Drugs of thistreatment and normalization of HTA significantly affects reducing group their hypotensive action develop by blocking the effect oftheir effects on renal system, cardiovascular system,and use of dipeptide carboxy oxidase and increasing the level of obstructionACE-inhibitors are indicated with minimal side effects and highly conversion of Angiotensin I(AI) to angiotensin-II,slowing convertingeffective in the treatment of HTA and the benefits of them are that bradykinin and other quinine and reduce the activity ofthis group of drugs may be combined with calcium blocker and a sympathetic with what they reduce and prevent negative effects onlarge numberof diuretics.In markets there are ACE inhibators renal hemodynamic hypertension.The duration of hypertensioncombined with duretics whose advantage is using only once a day also seems to have an effect on the reduction of renaland highly awaited effect in reducing arterial pressure.The high activity.Except HTA and hypercholesterolemia also unbalancedmortality in patients with CKD often occurs due ischemic diabetes is presented as additional and dominant factor in thecardiomyopathy associated with hypertesnion (44,45,46).Ischemic progress and the rapid pace of CKD.In our work we verified thatcardiomyopathy is associated with: old age, male gender, arterial the quality of treatment,adequate and timely HTA with ACEhypertension, diabetes mellitus,smoking, anemia, oxidativestress, inhibitors in patients with CKD is closely correlated with thedyslipidemia,and hiperhomocisteinemia (47,48). Uremic cardiomy-slowdown in the pace of progress of the disease by reducingopathy is closely associated with the action of HTA,the action of proteinuria and side effects of HTA to CVD.Treatment of HTA inuremic toxins, proinflammatory cytokines, primary patients with CKD should be started in the early stages of thehyperparathyroidism,and MIA syndrome (Malnutritio-Inflamatio - disease.Except the drug therapy in preventing and slowing CKDAtherosclersis (49, 50.51, 52.53).It is verified that patients with also has an important role compliance dietary measures (reduceduremia, the occurrence of myocardial infarction is 10 times more consumption of excess fluids and salt,correcting dyslipidaemia,frequent than in patients with another primary disease regulation of sugar values in diabetics, adhering to(54.55,56.).Cardiovascular diseases (CVD -infarkti of heart hypoproteinemic diet of phosphates,limiting Ca.In patients withdisease, left ventricular hypertrophy, congestive heart failure, CKD and HTA restriction of proteins in diet helps reducing thecerebrovascular stroke)) in the form of epidemics occur in patients retencion of substances that come from increased catabolism ofwith CKD and HTA and are five times more frequent than people proteins in body.For this reason daily consumption of protein mustwith the same sex and age from the general population who not exceed the extractive capacity of urea from the kidneys . Dailysuffers from HTA but not with CKD-Fragmingham study intake of protein in grams is calculated by multiplying it three times(43).Statistical studies published in the US in 1997 on the with the amount of urea in the urine of 24 hours.Duringintroduction of mortality in patients with chronic renal insufficiency consumption of food in patients with CDK is recommended intaketreated with hemodialysis showed that 53% mortality caused due of the essential amino acids of who 1/3 should be of animalto cardiovascular disease, 16% due to infections, 4% of origin.With the progress of CKDthe amount of protein consumptioncarcinomas and 27% from other causes in patients up to age 64 should be gradually and continuously reduced. Daily proteinyears (57.58,).
consumption should not pass a value of 0.5 - 0.6 g / kg

VI CONCLUSION

Understanding the pathophysiologic mechanisms of the HTA ismanagement and treatment hypertension with the only purpose of necessary to manage more qualitative hypertension in order toprevention and preventing the impact of its own performance and reduce the negative effects and impact of the trailer in the pace ofaccelerated pace of chronic renal failure progress.
progress of the IRK.Patients with HTA and IRKT (ESRD) have often need for two, three , until four antihypertensive medicaments to achieve the goals of treatment and to minimize their risk of side effects of HTA.In addition , changes in food,lifestyle and physical activity should always be considered as a vital component of any regime and antihipertensivetreatment.In conclusion we can suggest and recommend that the treatment of arterial hypertension in early stages (in particular,in patients with chronic renal failure) should be the main objective of doctors in the

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