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Research Article

**ROLE OF PROTEIN RESTRICTION AND RENIN-
ANGIOTENSIN-ALDOSTERONE SYSTEM IN THE
MANAGEMENT OF CHRONIC KIDNEY DISEASES**Dr. Anbreen Shabbir¹, Dr. Maheen Zafar¹, Dr Adeela Sarfraz Khan²¹Bahawal Victoria Hospital, Bahawalpur, ²Basic Health Unit Aliwah, District Vehari (PHFMC)

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Abstract:

Introduction: Chronic kidney disease (CKD) is a major public health problem, and preventing CKD and/or delaying progression of CKD patients to end-stage renal disease (ESRD) is a major task for the nephrology community.

Aim and objectives: The basic aim of the study is to analyze the role of protein restriction and Renin-Angiotensin-Aldosterone System in the management of chronic kidney diseases.

Methodology of the study: This cross sectional study was conducted at Bahawal Victoria Hospital, Bahawalpur during 2019. This study was basically conducted for the analysis of role of RAAS in the management of CKD. For this purpose we make a thorough analysis and study of RAAS.

Results: The RAAS is directly involved in the regulation of CKD, fluid volume, and vascular response to injury and inflammation. The inappropriate activation of this system causes hypertension, fluid retention, and inflammatory, thrombotic, and atherogenic effects that may contribute to end-organ damage in the long term. Although aldosterone (Aldo), renin, and several breakdown products of angiotensin I (AI) are also involved, most of the effects of the RAAS on target tissues are mediated by AII, which is generated both in the circulation and in the tissue.

Conclusion: It is concluded that RAAS plays very important role in the management of CKD in local population of Pakistan. However there are some evidence that in patients with diabetic nephropathy that the antiproteinuric effect of mineralocorticoid receptor blockade (MRB) is at least in part mediated by a direct effect on the glomerular basement membrane and is not dependent solely on reduction in systemic BP, glomerular filtration or dietary factors.

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INTRODUCTION:

Chronic kidney disease (CKD) is a major public health problem, and preventing CKD and/or delaying progression of CKD patients to end-stage renal disease (ESRD) is a major task for the nephrology community [1]. This looks like an achievable target, in particular because of the availability of reno-protective drugs that may interfere with disease progression such as the inhibitors of the renin-angiotensin-aldosterone system (RAAS). After the first inhibitor of angiotensin II (AII) system, the angiotensin-converting enzyme (ACE) inhibitor captopril, became available for clinical use in the early 1980s [2], other drugs have become progressively available that interfere with RAAS activity, such as AII-type 1 receptor blockers (ARBs) and the aldosterone (Aldos) antagonists that inhibit AII and Aldos activity by competitively antagonizing their binding to specific receptors [3]. In a short future, novel agents that interfere with renin activity (such as aliskiren) will also become available for clinical use [4], which will further increase the armamentarium of drugs that may interfere with the sequence of events, eventually resulting in AII and Aldos production at different levels and that, used in combination, may achieve an almost complete inhibition of the RAAS. In addition, to identify the optimal regimens to maximize reno-protection, major efforts should be made in identifying and treating all patients at risk, with the final aim to delay or even prevent the onset and progression of chronic renal disease and related complications [5].

The renin-angiotensin aldosterone system (RAAS) is a well-known regulator of blood pressure (BP) and determinant of target-organ damage. It controls fluid and electrolyte balance through coordinated effects on the heart, blood vessels, and Kidneys. Angiotensin II (AII) is the main effector of the RAAS and exerts its vasoconstrictor effect predominantly on the postglomerular arterioles, thereby increasing the glomerular hydraulic pressure and the ultrafiltration of plasma proteins, effects that may contribute to the onset and progression of chronic renal damage [6].

AIM AND OBJECTIVES:

The basic aim of the study is to analyze the role of protein restriction and Renin-Angiotensin-Aldosterone System in the management of chronic kidney diseases.

Methodology of the study:

This cross-sectional study was conducted at Bahawal Victoria Hospital, Bahawalpur during 2019. This study was basically conducted for the analysis of role of RAAS in the management of CKD. For this purpose, we make a thorough analysis and study of RAAS. The purpose of the present review is to present a general view of the multiple actions of RAAS and their more relevant components in the development and progression of CVD and CKD, together with an analysis of present treatments based on RAAS blockade including new drugs and their combinations as a fundamental strategies for reducing their impact on mortality and morbidity cause by these entities.

Data collection:

For this study the data was collected from 50 patients who was suffering from kidney disease. For this purpose, we make two groups of study. One group was control group and the other group was suffering from kidney problems. Then we collect the socio-economic status and therapy status of both groups. Then we analyze the data and find that either statin therapy is helpful for patients or not.

Analysis:

A chi-square test was used to examine the difference in the distribution of the fracture modes (SPSS 19.0 for Windows, SPSS Inc., USA).

RESULTS:

The RAAS is directly involved in the regulation of CKD, fluid volume, and vascular response to injury and inflammation. The inappropriate activation of this system causes hypertension, fluid retention, and inflammatory, thrombotic, and atherogenic effects that may contribute to end-organ damage in the long term. Although aldosterone (Aldo), renin, and several breakdown products of angiotensin I (AI) are also involved, most of the effects of the RAAS on target tissues are mediated by AII, which is generated both in the circulation and in the tissue.

Table 01 of the data shows the basic values of control group and patients. It shows the BMI, age, Total cholesterol level and other basic values. We can find that cholesterol level is high in patients as compared to normal values. We also shows the comparison of statin group and normal group.

Table 01: General values of Control group and diseased group

Variable	Diseases Group	Control Group	t Value	p Value
Age (Year)	56.56±8.46	53.64±8.36	1.716	0.081
BMI (kg/m ²)	24.31±2.26	23.37±2.09	2.195	0.031
SBP (mmHg)	140.36±15.70	116.53±13.46	8.248	0.000
DBP (mmHg)	87.94±10.69	75.81±9.94	5.967	0.000
PP (mmHg)	52.42±12.87	40.72±8.74	5.426	0.000
FBG (mmol/)	5.12±0.65	5.06±0.49	1.764	0.081
TG (mmol/L)	1.74±0.75	1.69±0.86	1.838	0.071
TC (mmol/L)	4.95±0.76	4.88±0.82	1.712	0.090
HDL-	1.30±0.43	1.31±0.56	1.717	0.089
LDL-C	3.46±0.58	3.38±0.66	1.139	0.266

Tale 02 shows the values of analysis of statin therapy in patients. It shows the comparison between two groups on the basis of functional values.

Table 02: Comparison between two groups in structural and functional parameters

Group	IMT (μm)	CC (mm ² /KPa)	α	β
CKD Group	694.88±77.63	0.89±0.13	5.68±1.23	11.25±1.01
Control Group	586.87±62.12	0.96±0.08	4.77±0.62	9.24±1.24
T value	7.818	-3.115	4.712	9.004
P value	0.000	0.002	0.000	0.000

DISCUSSION:

The renin-angiotensin-aldosterone system regulates renal vasomotor activity, maintains optimal salt and water homeostasis, and controls tissue growth in the kidney. However, pathologic consequences can result from overactivity of this cascade, involving it in the pathophysiology of kidney disease [7]. An activated renin-angiotensin-aldosterone system promotes both systemic and glomerular capillary hypertension, which can induce hemodynamic injury to the vascular endothelium and glomerulus. In addition, direct profibrotic and proinflammatory actions of angiotensin II and aldosterone may also promote kidney damage. The majority of the untoward effects associated with angiotensin II appear to be mediated through its binding to the angiotensin II type 1 receptor. Aldosterone can also induce renal injury by binding to its receptor in the kidney [8]. An understanding of this system is important to appreciate that inhibitors of this cascade can reduce the progression of chronic kidney disease in proteinuric disease states. Pharmacologic agents that can interfere with this cascade include angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and aldosterone receptor antagonists [9].

CONCLUSION:

It is concluded that RAAS plays very important role in the management of CKD in local population of Pakistan. However there are some evidence that in patients with diabetic nephropathy that the antiproteinuric effect of mineralocorticoid receptor blockade (MRB) is at least in part mediated by a direct effect on the glomerular basement membrane and is not dependent solely on reduction in systemic BP, glomerular filtration or dietary factors.

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