Original Research Article

The significance of proteinuria in essential hypertension

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International Archives of Integrated Medicine, Vol. 9, Issue 2, February, 2022.
Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 21-01-2022 **Accepted on:** 29-01-2022
Source of support: Nil **Conflict of interest:** None declared.

How to cite this article: E.A. Ashok Kumar, Koushik Chavalla, Muppala Hanvitha. The significance of proteinuria in essential hypertension. IAIM, 2022; 9(2): 1-12.

Abstract

The prevalence of hypertension is reported to be about 29% in India. Proteinuria is often the earliest marker of hypertension mediated renal damage, occurring even before a reduction in glomerular filtration rate (GFR). It can also be used to predict risks of chronic kidney disease (CKD) progression, cardiovascular disease, and all-cause mortality in general population. Thus monitoring proteinuria is a key aspect of assessing disease progression and treatment response in a variety of kidney diseases, including hypertensive renal damage. Target organ damage is common in microalbuminuric patients. They can have high left ventricular mass, a high prevalence of retinopathy , and an increased thickness and presence of plaques in the carotid artery. It is also interpreted as a marker of early intra renal vascular dysfunction in essential hypertension. Microalbuminuria is an independent predictor of cardiovascular morbidity and mortality in patients with essential hypertension. Microalbuminuria seems to constitute a simple and accurate method to detect a hypertensive patient at a high risk for cardiovascular and probably renal damage. The present study is comprised of 50 cases of essential hypertension. Microalbuminuria and Proteinuria was estimated. 27 patients i.e. 54% had microalbuminuria. Microalbuminuria had a positive correlation with severity of Hypertension with p value of 0.047 (< 0.05). Prevalence of microalbuminuria increases with the age and duration and severity of hypertension. Microalbuminuria had a statistically significant correlation with the presence and severity of target organ damage. Screening for microalbuminuria should be performed in hypertensives to start treatment early to minimize morbidity and mortality.

Key words

Proteinuria, Essential Hypertension.

Introduction

Hypertension is one of the leading causes of global burden of disease and was ranked first in worldwide analysis of all risk factors in 2010 and again in 2015 [1].

Because of the associated morbidity and mortality and the cost to society, Hypertension is an important public health challenge. Substantial progress has been made in understanding the epidemiology, pathophysiology, and risk associated with hypertension, and lowering blood pressure (BP) can substantially reduce the premature morbidity and mortality [2 3]. Prevalence of hypertension has been increasing in urban and even in rural Indian population for the last 20 years. Presently the prevalence of hypertension in urban areas is 33.8% and in rural areas it is 27.6%, with an overall prevalence of 29.8% [4, 5].

Hypertension is the most important modifiable risk factor for coronary heart disease, stroke, congestive heart failure, end-stage renal disease, and peripheral vascular disease. Hypertension is the leading antecedent condition for heart failure, approximately 74% of people experiencing an initial hospitalization for heart failure either had or have BP of 140/90 mm Hg or higher [3].

Modifications at cardiac, vascular and renal levels induced by chronic high blood pressure (BP) are defined as target organ damage (TOD) and have been shown to have an adverse prognostic significance independently of BP levels and traditional risk factors. Subclinical signs of organ alterations such as left ventricular hypertrophy (LVH), carotid atherosclerosis and microalbuminuria (MA) are included in the list of hypertensive TOD [6].

Proteinuria is often the earliest marker of hypertension mediated renal damage, occurring even before a reduction in glomerular filtration rate (GFR). It can also be used to predict risks of chronic kidney disease (CKD) progression, cardiovascular disease, and all cause mortality in general population. Thus monitoring proteinuria is a key aspect of assessing disease progression and treatment response in a variety of kidney diseases, including hypertensive renal damage [7].

Although measurement of urinary protein has long been recommended in clinical practice guidelines, recommendations regarding this practice vary substantially [8]. Though the prevalence of hypertension is reported to be about 29% in India [4 5], till now there is little data from India on the prevalence of proteinuria and its association with TOD, among patients with essential hypertension.

Identification of higher blood pressure as a risk factor

The oldest reference to the arterial disease occurs in Exodus VII [9]. Waffer (1655) recognized apoplexy caused by hemorrhage in a monk aged 45 years. Laennac (1819) noted the existence of left ventricular hypertrophy. The relation of hypertension to coronary artery disease (Kreul 1740) and angina pectoris (Fathergill 1776) was recognised in 18th century. Richard Bright (1827) described 3 varieties of cases in relation to hypertension:

- Edema associated with albuminuria
- Degenerative glomerulonephritis and
- Chronic glomerulonephritis with secondary constriction of the kidney.

He observed the changes of hypertension on the cardiovascular system in patients with chronic renal disease. George Johnson in 1868 postulated that the cause of left ventricular hypertrophy (LVH) in Bright disease was the presence of muscular hypertrophy in the smaller arteries throughout the body. Frederick Mahomed was one of the first physicians to systematically incorporate blood pressure measurement as a part of clinical evaluation. Though the estimation of blood pressure by tension and character of the pulse was known by that time the development of sphygmomanometer by Harrison in 1830 gave an useful tool for recognition of blood pressure.

Later, Vonbasch established the normal standards of blood pressure and found the existence of cardiac hypertrophy and hardening of arteries in the hypertension. The recognition of primary (essential) hypertension is credited to the work of Huchard, Vonbasch, and Albutt. Observations of Janeway and Walhard led to the recognition of target organ damage, which branded hypertension as the "silent killer".

Mechanism of hypertension

In most patients, no single or specific cause is known, and the condition is referred to as primary or essential. Since persistent hypertension can develop only in response to an increase in cardiac output or a rise in peripheral resistance, defects may be present in one or more of the multiple factors that affect these two forces.

The interplay of various derangements in factors affecting cardiac output and peripheral resistance may precipitate the disease, and these abnormalities may differ both in type and degree in different patients.

Pathologic consequences of hypertension

Hypertension if left untreated, about 50% of patients succumb to Coronary Heart Disease or Congestive Failure, about 33% to Stroke, and 10 to 15% to renal failure. In those with rapidly accelerating hypertension mortality is frequently due to renal failure, once proteinuria or other evidence of nephropathy develops.

Microalbuminuria and essential hypertension

Clinically, macroalbuminuria (a random urine albumin/ creatinine ratio >300 mg/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 cardiovascular disease [10]. It was C.E Mogensen, who documented and popularised microalbuminuria as an important early marker, not only for early diabetic renal disease, but also for early vascular complications including early mortality in essential hypertension [11]. Recently, clear treatment strategies based upon many intervention trials were proposed and these are now well accepted by the medical community. Therefore screening for microalbuminuria and follow up of patients is now general practice in some countries.

Microalbuminuria is defined as Urine Albumin Excretion Rate (UAER) of approximately 30 - 300 mg/d (24 hour urine albumin excretion rate) in at least two of three consecutive samples of non ketotic sterile urine. Ideal is the first voided, mid stream morning sample (5 ml sample). ADA and NKF (American Diabetes Association and National Kidney Foundation) define microalbuminuria as an albumin creatinine ratio between 30-300 μg/mg in both men and women [12].

Sex specific ACR cut points: > 17μg/mg in men, >25 μg/mg in women. 24 hour urine albumin excretion rate: 30 - 300 mg/d. Overnight urine albumin excretion rates: 20 – 200 μg/hr. **Albumin creatinine ratio:** 2.5 – 25 mg/mmol (for men) or 3.5-25 mg/mmol (for women) 30 – 300 mg/d in both men and women.

Proteinuria in essential hypertension

In 1954 Perera GA, reported incidence of clinically apparent proteinuria in essential hypertension in the absence of therapy was 42% in a group of 500 patients followed until death [13]. It is generally held that proteinuria in benign Nephrosclerosis is almost invariably less than 1.0g/24h, the higher rates of proteinuria, even within nephrotic range, can be found in malignant phase of arterial hypertension [14].

In 1974 H.H Parving and C.E Mogensen, et al. reported the finding of elevated Urine Albumin Excretion(UAE) levels in insufficiently treated essential hypertensives, which correlated significantly with blood pressure levels and fell after blood pressure control [15]. This finding has been amply confirmed and it is now recognized that microalbuminuria can be found in up to 40% of untreated hypertensive population.

Prevalence of an elevated UAE increases with age, longer duration and a higher severity of hypertension [16, 17]. Hypertensive target organ damage is more common in microalbuminuric patients. Patients with elevated UAE have higher left ventricular mass [17], a higher prevalence of hypertensive retinopathy [18], and an increased thickness and presence of plaques in the carotid artery [19]. Furthermore, the presence of microalbuminuria in essential hypertensive patients has been interpreted as a marker of early intra renal vascular dysfunction in essential hypertension [19].

In summary, microalbuminuria is an independent predictor of cardiovascular morbidity and mortality in both men and women with essential hypertension. This could, in turn, be facilitated by the frequent association of an elevated urinary albumin excretion to a series of alterations, such as endothelial dysfunction, insulin resistance, altered lipid levels, higher body mass index, and salt-sensitivity. All these alterations could facilitate the accompanying risk for atherosclerosis, and it likely explain such an association between UAE and the risk of coronary heart disease.

In conclusion, microalbuminuria seems to constitute a simple and accurate method to detect a hypertensive patient at a high risk for cardiovascular and probably renal damage.

Mechanisms of association between microalbuminuria and hypertension: Three mechanisms are described

- Increased intraglomerular pressure
- Intrinsic glomerular damage causing changes in glomerular filtration barrier.
- Tubular dysfunction resulting in prevention of normal reabsorption of filtered albumin.

Increased intraglomerular pressure, loss of normal auto-regulatory function is likely to be involved in development of raised intraglomerular pressure leading to protein leakage and renal damage. Possible mechanisms include increased sympathetic nervous system activation, hyperinsulinemia, and decreased production of vasodilator hormones and activation of RAA. ACEI drugs which act by causing relaxation of efferent arteriole is associated with reduction in protein leakage and renal protection in patients with & without diabetes. Systemic hypertension is directly responsible for renal hemodynamic changes facilitating protein leakage. Systolic BP has been particularly strongly correlated with development of microalbuminuria [20, 21].

Changes in glomerular filtration barrier

Permeselectivity of glomerular basement membrane is important in normal glomerular function and may be deranged in association with microalbuminuria.

In patients with diabetes, glycation of long lived tissue proteins may cause loss of anionic charge leading to albumin leakage. This impairment of charge selectivity has recently been shown in individuals with no diabetes. These abnormalities have been associated with, vascular endothelial and vascular permeability factors.

One hypothesis is that microalbuminuria is the renal manifestation of generalized vascular endothelial dysfunction and may have a genetic basis. This could explain the strong link with cardiovascular disease. Several data suggest that interplay with a number of factors such as lipid abnormalities, prothrombotic factors, increased RAA activity and systemic inflammation also have a role in the genesis of endothelial dysfunction seen with microalbuminuria. Supported by all these data, it has been reported that microalbuminuria [20, 21].

- Is associated with reduced size & charge selectivity of glomerular vessel wall in healthy people.
- Is an independent marker of systemic vascular albumin leakage.
- Correlates with Von Willebrand factor antigen, Factor VIII hyperactivity,

fibrinogenand endothelial cell damage.

Microalbuminuria and hypertension - Implications for treatment

Studies in essential hypertension patients with microalbuminuria have shown that ACEI have a greater capacity to decrease microalbuminuria than other group of anti-hypertensives. ARBs may show similar benefits from the point of view of renal microcirculation [22].

Aim and objectives

- To study the prevalence of proteinuria in patients of essential hypertension.
- To study the relationship between proteinuria and target organ damage.

Materials and methods

The patients who visited to Medicine Department, Malla Reddy Institute of Medical Sciences, Hyderabad, who were diagnosed with Essential Hypertension, were studied considering the inclusion and exclusion criteria during a period starting from January 2020 to October 2021.

Number of study group: One
Sample size: 50
Study design: Prospective Analytic study
Classification of blood pressure is done as per JNC - VII Classification of Blood Pressure for Adults [23].

Statistical method

Chi-square test was used to study the relationship between microalbuminuria and other variables. P value was calculated for all variables. P value of <0.05 is considered significant.

Ethical clearance:

Ethical clearance taken from Malla Reddy Institute of Medical Sciences, Hyderabad.

Study Design:

50 patients selected by random sampling procedure matching the inclusion and exclusion criteria were included in the study. A pretested proforma was used to collect the relevant data.

Inclusion criteria

- Individuals with essential hypertension.
- Age group of 40-60 years.

Exclusion criteria

- Pregnant women.
- Patients with overt proteinuria
- Patients with renal diseases.
- Patients with diabetes mellitus.
- Patients with thyroid disorders.
- Patients with adrenal tumors.
- Patients with ischemic heart diseases.
- Patients on drugs like steroids and oral contraceptive pills.
- Patients who are smokers and alcoholics.
- Patients with auto immune disorders.
- Patients with fever and Urinary Tract Infections.

Microalbuminuria was assessed by one step MAU Rapid Quantitative test. 24 hours proteinuria was assessed by Pyrogallol Red Method.

Results

A total of 50 hypertensive patients were studied during the study period. There were 29 males and 21 females. Age distribution is given in **Table – 1**. Distribution of microalbuminuria among different age groups shown in **Table – 2**.

Table – 1: Age distribution of patients.

Age in years	No. of patients	Percentage
40 to 45	12	24%
46 to 50	7	14%
51 to 55	8	16%
56 to 60	23	46%

Out of 12 patients in 40 to 45 group 5 patients (41%) had microalbuminuria, out of 7 patients in 46 to 50 group 6 patients (85.7%) had microalbuminuria, out of 8 patients in 51 to 55 group 4 (50%) had microalbuminuria, out of 23

patients in 56 to 60 group 46% had microalbuminuria. The correlation between increasing age and microalbuminuria was found to be not significant with P value is 0.3. Out of 50 patients in this study, 29 (58%) are males and 21 (42%) are female. Sub group analysis showed, 71.4% of females had microalbuminuria where as only 41.3% males had microalbuminuria, however no significant correlation was found between gender and microalbuminuria with p value 0.06. The chi square statistic with Yates correction is 3.3 and the p value is 0.06, not significant ($p<0.05$). Out of 50 hypertensive patients, 25 (50%) patients had grade I hypertension where as rest 25 (50%) patients had grade II hypertension.

Table - 2: Distribution of microalbuminuria among different agegroups.

Age inyears	No. of patients	Percentage	Microalbuminuria Present	Percentage
40 to 45	12	24%	5	41.0%
46 to 50	7	14%	6	85.7%
51 to 55	8	16%	4	50.0%
56 to 60	23	46%	12	52.1%

Table – 3: Distribution of microalbuminuria with respect to duration of hypertension.

Duration of hypertension	No. of patients	Microalb	Percentage
Denovo	21	8	38%
1 year and less	9	4	44.4%
1 to 5 years	14	11	78.5%
More than 5 years	6	4	66.66%

Table - 4: Distribution of microalbuminuria with respect to grades of hypertension.

	Total no. of patients	Microalbuminuria Present	Microalbuminuria Absent
Grade 1	25	10 (40%)	15
Grade 2	25	17 (68%)	8

Table - 5: Relationship between Left ventricular hypertrophy and albuminuria.

LVH Present				LVH Absent			
Total No. of patient	Normal	Micro	Macro	Total no. of patients	Normo	Micro	Macro
16	1 (6.25%)	15 (93.75%)	0	34	22 (64.7%)	12 (35.2%)	0

Of 50 patients in this study, 21 (42%) patients are Denovo (newly detected) detected to have hypertension, 9 (18%) were diagnosed with hypertension within 1 year or less, 14(8%) were hypertensives from 1 to 5 years and 6(12%) were hypertensive from more than 5 years.

In Denovo group 8 patients (38%) had microalbuminuria, in 1 year or less group 9(44.4%) patients had microalbuminuria, in 1 to 5 years group 11(78.5%) had microalbuminuria and in more than 5 years group 4(66.6%) had microalbuminuria as shown in **Table – 3**. The correlation between duration of Hypertension and microalbuminuria was found to be not significant with chi square statistic of 6.2597 and p value of 0.09.

Out of 50 hypertensive patients 27 patients i.e. 54% had microalbuminuria and 23 i.e. 46% had normoalbuminuria.

Left ventricular Hypertrophy (LVH): Out of 50 patients, 16 (32%) had Left Ventricular Hypertrophy (LVH) on Electrocardiography (ECG) and 34(68%) did not have LVH.

Grade of hypertension and microalbuminuria: In this study, out of 25 patients with grade I hypertension, 10 i.e. 40% had microalbuminuria whereas 17 (68%) out of 25 patients with grade II hypertension had microalbuminuria and the correlation between grade of hypertension with microalbuminuria is found to be significant with the chi square statistic of 3.945 and the p value of 0.047 (significant <0.05), as shown in **Table – 4**.

Left ventricular hypertrophy and albuminuria: On sub group analysis between LVH and albuminuria groups, out of 16 patients with LVH, 15 (93.75%) patients had microalbuminuria and 1 patient (6.25%) had normal albuminuria. In patients with no LVH, out of 34, 12 (35.2%) had microalbuminuria and 22 (64.7%) had normal albuminuria as shown in **Table – 5**. A significant correlation was found between LVH and microalbuminuria with the chi square statistic of 14.967 and the p value of 0.0001 (significant <0.05).

Discussion

Hypertension is an "iceberg" disease. It was evident that in most developed countries, only half of the hypertensive patients in general population were aware of the condition, only about half of those aware of the problem were being treated, and only about half of those treated were considered adequately treated. If this is the situation in countries with highly developed medical services, in developing countries, the proportion treated adequately would be far too less [27]. Hypertension is a major public health problem all over the world. The incidence of hypertension in India is 5-15% as compared to 10-12% in the West [24].

Many physicians do not know that the proteinuria occurs in essential hypertension and its importance in assessing morbidity and mortality in relation to cardiovascular and renal complications. The present study is undertaken to generate awareness of importance of proteinuria in essential hyprtension with its added mortality and morbidity. The present study is regarding the prevalence of microalbuminuria in essential hypertension and the relationship of microalbuminuria with different variables and the effects like target organ damage.

Microalbuminuria in Essential Hypertension: Out of 50 hypertensive subjects, 27 i.e. 54% had microalbuminuria in this study.

Prevalence of microalbuminuria in various studies is given in **Table - 6**.

Table - 6: Prevalence of microalbuminuria in various studies.

Study	Microalbuminuria
Present study	54%
Anil Kumar H, et al. [24]	32%
HK Aggarwal, et al. [25]	47%
Hitha, et al. [26]	26.6%
i-SEARCH Global study [27]	58%
Sharma V K, et al. [28]	24%
Sabharwal R K, et al. [29]	33.3%
Stefano Bianchi, et al.[30]	35%
Tsioufis, et al. [31]	47%

Higher prevalence i.e. 54% in this study could be because:

- Study included patients only with abnormal blood pressure.
- Hypertensive patients with adequate control of blood pressure were not Included.
- Small sample size.

The wide variability in prevalence of microalbuminuria may be explained by [32]:

- Severity of Hypertension
- Different values used to define microalbuminuria
- Different protocol used to evaluate microalbuminuria.
- Difference in methods of urine collection.
- Characteristics of study population.
- Selection criteria
- Racial differences

Sex distribution of microalbuminuria:

In the present study, there was no significant difference between prevalence of microalbuminuria in men and women (p = 0.06). Sabharwal R K, et al. showed a prevalence of 34% in males and 30.7% in females. In the HUNT study (Norway) [33] a prevalence of 32% in males and 28% in females. In the HUNT study (Norway) a stronger association was observed between microalbuminuria and mortality in men than in women. In this study the interaction between sex and Albumin creatinine Ratio (ACR) was statistically significant (p=0.003) and supported a sex difference, women had more prevalence. They attributed this difference to the higher incidence of asymptomatic UTI in women. They suggested the need for different ACR cut off values in men and women - because men have greater muscle mass and higher creatinine excretion than women although albumin excretion levels are equal (HUNT- Nord Trondelag Health Study). Pontremoli, et al. [34] in the MAGIC study on the prevalence and clinical correlates of microalbuminuria observed that microalbuminuria was more common in males.

Microalbuminuria according to age groups:

In the present study, there was no significant difference between prevalence of microalbuminuria and age .This could be because of small sample size and differences in seeking medical care. Hitha B, et al. study showed a higher incidence of microalbuminuria in older age groups (p<0.001).

This significance could be because if:

- They already have a long duration of hypertension.
- Higher prevalence of atherosclerotic vascular disease and endothelial dysfunction in elderly [35]

Microalbuminuria with duration of Hypertension:

In the present study, microalbuminuria does not have a significant correlation with the duration of Hypertension (P = 0.09). This is in contrary to several studies which found significant positive correlation between microalbuminuria and duration of hypertension. Pontremoli, et al. 1997 in the MAGIC Study have observed that the increase in prevalence of microalbuminuria with increase in duration and severity of hypertension. Cerasola, et al.; Hitha B, et al.; Sharma V K, et al. and Ophsal, et al. had made similar observations. Data from the present study also showed that patients with hypertension of more than 5 years duration were more prone to develop microalbuminuria.

Microalbuminuria with severity of hypertension

In present study, microalbuminuria was found to have a strong positive correlation with severity of hypertension with p value of 0.047 (significant < 0.05). This is in agreement with several studies. Severe uncontrolled hypertension can cause profound and rapid endothelial damage which can be hampered by inadequate dose titration of anti hypertensive drugs.

In HK Aggarwal, et al. [25] 38.82% with stage 1 hypertension and 93.33% with stage 2 hypertension had microalbuminuria. In study done by H.H Parving and C.E Mogensen, et al. [15] urinary albumin excretion rate correlated with systolic, diastolic and mean blood pressure (p <0.001).

Relationship of microalbuminuria with the indices of target organ damage:

Urine albumin excretion and abnormalities in LV structure and function:

It was observed that Left ventricular Hypertrophy (LVH) is found in 16 out of 50 hypertensive patients on ECG. Out of 16 patients with LVH, 15 (93.75%) patients had microalbuminuria and 1 patient (6.25%) had normal albuminuria. In patients with no LVH, out of 34 12 (35.2%) had microalbuminuria and 22 (64.7%) had normal albuminuria. A significant correlation was found between presence of LVH and microalbuminuria with the chi square statistic of 14.967 and the p value of 0.0001 (significant <0.05). Jenson J S, et al. [35] showed

that 9% of them developed IHD and hence confers 3 fold risk. Hence, a strong positive correlation between albumin excretion rate in urine and the presence of IHD and also atherosclerotic risk factors. C. Tsioufis, et al. (2002) in their study observed that 21% of the 249 had LVH. W.Kristian et al in 2002 (LIFE study) observed a higher prevalence of microalbuminuria 30% vs. 9% in patients with concentric hypertrophy on ECHO ($P < 0.0001$) [36]. Pontremoli, et al. in 2002 has observed that patients with microalbuminuria were 21 times more likely to have both LVH ($P < 0.001$) in a study conducted in 279 patients in their institution [37]. C. Stefanadis, et al. made similar observation in 2002 - LVH was significantly higher in microalbuminuric patients compared with normoalbuminuric subjects (32 vs. 5% $p<0.0001$).

Microalbuminuria and Hypertension - Implications for treatment

Studies in essential hypertension patients with microalbuminuria have shown that ACEI have a greater capacity to decrease microalbuminuria than other group of antihypertensives. ARBs may show similar benefits from the point of view of renal microcirculation [22]. The microalbuminuria reflects a specific type of endothelial dysfunction. Treatment favorably affecting endothelial function also contributes to the decrease of UAER in hypertensive subject. Thus a specific renoprotective effect of ACEI and Angiotensin II receptor antagonists has been observed in hypertensive patients with microalbuminuria. This can be attributed to the intrarenal effect of these drugs that selectively attenuates the intraglomerular pressure by mainly promoting vasodilatation of glomerular efferent arterioles.

These classes of drugs have favorable effects on the remodeling regression and the improvement of endothelial function in resistance arterioles particularly in patients with nephrosclerosis.

Relation between microalbuminuria and cardiac function and renal parameters

Microalbuminuria measurement has yet to become routine for hypertensives in the general practice setting. It is a highly sensitive, readily assessed marker of incipient nephropathy and systemic endothelial pathology. It is an independent predictor of either renal failure or cardiovascular morbidity and mortality. Therefore, its measurement should be incorporated within standard management protocols for all patients with hypertension.

Sustained monitoring of renal function should be done regularly. Agents that delay the progression of renal disease are likely to protect against adverse cardiovascular outcomes by protecting against the systemic consequences of renal disease and in addition , may exert beneficial effects on endothelia throughout the vasculature and in the heart..

There is abundant evidence that the occurrence of microalbuminuria in patients with hypertension or diabetes predicts adverse outcome. The LIFE (Losartan Intervention For Endpoint reduction in hypertension) study in high risk hypertensives found that increased urinary albumin excretion is associated with left ventricular hypertrophy, abnormal geometry, and increased ventricular mass. In the Heart Outcomes Prevention Evaluation (HOPE) Study, increase in albumin: creatinine ratio increased the adjusted hazard for major cardiovascular events. Recently, the Prevention of Renal and Vascular End stage Disease (PREVEND) study demonstrated that fosinopril significantly lowered urinary albumin excretion in patients with microalbuminuria and correspondingly was associated with a reduction in cardiovascular events.

Hence, the present study also supports the positive correlation between microalbuminuria and severity of hypertension; the indices of target organ damagei i.e. Left Ventricular Hypertrophy.

Conclusion

- Hypertension is one of the leading health

problems all over the world.

- It is one of the most common risk factor for cardiovascular disorders.
- Hence a thorough assessment is a prerequisite to correctly identify the patients who are at risk.
- The presence of microalbuminuria in essential hypertension points towards the target organ damage.
- The prevalence of microalbuminuria varies in different populations, based on the characteristics of the population and techniques & protocols used for evaluation.
- Microalbuminuria reflects a state of increased renal endothelial permeability and hence considered an early biochemical marker of diffuse endothelial dysfunction.
- Hence, microalbuminuria has become a prognostic marker for cardiovascular disorders and also doubles the risk of renal failure, ischemic and hemorrhage stroke and peripheral vascular disease.
- Prevalence of microalbuminuria increases with the age of the patient and the duration and severity of hypertension.
- Untreated, inadequately treated and non complaint patients have higher prevalence of microalbuminuria.
- Microalbuminuria has proven to have statistically significant correlation with the presence and severity of target organ damage.
- More extensive screening for microalbuminuria should be performed in hypertensive subjects to facilitate better stratification of absolute cardiovascular risk, especially in patients with hypertension of long duration.

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