



ISSN - 2320-6039 (Print) ● ISSN - 2320-608X (Electronic)

Volume 4 / Number 1 / January-June 2016

INTERNATIONAL JOURNAL OF PHYSIOLOGY

Website: www.ijop.net

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International Journal of Physiology is a double blind peer reviewed international journal which has commenced its publication from January 2013. The journal is half yearly in frequency. The journal covers all aspects of physiology. The journal has been assigned ISSN 2320-6039 (Print Version) and ISSN 2320-608X (Online Version). The journal is covered by Index Copernicus, Poland and many other international data bases. **All rights reserved.** The views expressed by authors in journal are not necessarily views of International Journal of Physiology. The advertisements are purely commercial in nature and journal does not guarantee their efficacy.

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Print-ISSN: 2320-6039 Electronic-ISSN: 2320-608X Frequency: Six Monthly

Website: www.ijop.net

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4th Floor, Statesman House Building, Barakhamba Road,
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Printed, published and owned by

Dr. R.K. Sharma

Institute of Medico-legal Publications
4th Floor, Statesman House Building, Barakhamba Road,
Connaught Place, New Delhi-110 001

Published at

Institute of Medico-legal Publications

4th Floor, Statesman House Building, Barakhamba Road,
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CONTENTS

Volume 4, Number 1

January-June 2016

1. Study of Changes in Blood Pressure and Lipid Profile in Professional Bus Drivers 01
as an Impact of Stress
D Balanaga Nandhini, R Shanthini, M Tamilkodi
2. Gender Differences in Autonomic Nervous System Activity of Children 05
Mona Bedi, Shilpa Khullar, V P Varshney
3. Comparative Study of Pulmonary Function Tests during Third Trimester of 11
Pregnancy and Non- Pregnant Women in Bihar
Tarun Kumar, Poonam, Sunita, J R Keshari, Jhillmill Kumari, Anju Kumari, Bandana Kumari, Ashok Sharan, Manish Kumar
4. Metabolic Syndrome in COPD; A Case Control Study from Himachal Pradesh 15
Sandip Chindhi, Surinder Thakur, Malay Sarkar, Prakash C Negi, Venkappa S Mantur
5. Pre-hypertension and Hypertension : Prevalence and its Awareness among 21
Personnel of Administrative Class in Agra
Manju Lata Arya, Pallavi Anand
6. Effects of a Single Session of Yogic Relaxation on Cardiovascular Parameters in a 27
Transgender Population
Ananda Balayogi Bhavanani, Meena Ramanathan, Madanmohan Trakroo, Senthil Thirusangu
7. Electro-retinogram Changes in Adult Individuals with Myopia 32
L K Sumitra, A Santa Kumari, Ch. N Rajkumari
8. A Descriptive Study of Physical Activity Level in I Year Medical Students of 39
Bangalore Medical College and Research Institute- A Pilot Project
Sowmya R
9. Comparison of Serum Magnesium Levels with Body Mass Index and duration of Diabetes 44
A Gayatri, D Uma Devi, A Rasheeda
10. Introducing Role Play in I MBBS- A Feedback 47
Sangeetha P, Venkata Subramanian S, Sarayu K
11. Impact of Cigarette Smoking on Lipid Peroxidation and Atherogenic Indices in 51
Asymptomatic Young Adults
Hemamalini RV, Arpita Priyadarshini, Prabhavathi K, Saravanan A

12.	A Study on Gender Variation of Pain Thresholds	57
	<i>V Prabha, S Waheeda, K N Maruthy</i>	
13.	A Prospective Outlook of Yogic Training on Lung Functions and Antioxidants Status	61
	<i>Vimal Singh Gusain, Anant Narayan Sinha, Desh Deepak</i>	
14.	Cognitive Function Across the Menstrual Cycle of Young Healthy Women	65
	<i>Jhillmill Kumari, Sunita, Manish kumar, Tarun Kumar, Ashok Sharan</i>	
15.	A Comparative Study of Arterial Stiffness Indices between Diabetics & Non-diabetics	71
	<i>V Prabha</i>	
16.	Health Status of Elderly Women Residing in a Hospice in Pondicherry	76
	<i>Meena Ramanathan, Ananda Balayogi Bhavanani</i>	
17.	Study on Relationship between Anemia and Academic Performance of Adolescent Girls	81
	<i>Tarun Kumar, Sanjeet Kumar Singh, Manish Kumar, Sunita, Ashok Sharan</i>	
18.	Estimating Cardiovascular Prognosis by Exercise Treadmill Test: A 5 Year Follow-up Study	87
	<i>Laxmikanta Say, Arati Mohanty, Arpita Priyadarshini, Sunil Kumar Sharma</i>	
19.	To Study the Effects of duration of Yoga Training on Post Exercise Recovery Time	93
	<i>M T Jiwode, Mukesh Mahajan</i>	
20.	Prevalence of Microalbuminuria - An Early Detector of Diabetic Nephropathy	98
	<i>Prabhavathi K, Kirthana Kunikullaya, Hemamalini R V, Jaisri G, Saravanan A</i>	
21.	Median Nerve Conduction Velocity as a Tool to Detect Subclinical Neuropathy in Type II Diabetes Mellitus	103
	<i>R Sunandini, Shashikant G Somani</i>	
22.	Heart Rate Variability(HRV) in Normotensive Subjects with Family History of Hypertension	110
	<i>Mohammed Farhan Ahmed, K Indira, Ch N Rajkumari</i>	
23.	To Correlate the Difference between Slow Vital Capacity and Forced Vital Capacity with the Severity of Chronic Obstructive Pulmonary Disease	116
	<i>Venkata Subramanian S, Sangeetha Partha Sarathy</i>	
24.	Cardiovascular Autonomic Responses to Exercise in Normotensive Healthy Adults with Parental History of Hypertension	120
	<i>Swetha Matta, Ch N Raj Kumari</i>	
25.	Auditory Transmission in Iron Deficiency Anaemia	125
	<i>Vinodha R, Shanmugapriya C</i>	
26.	A Cross-sectional Study of Perceived Stress and Sources of Stress among First Year Medical Students of Gauhati Medical College, Guwahati	129
	<i>Chinmoyee Baruah, Anindita Mahanta</i>	
27.	A Cross-sectional Study of Prevalence of Asymptomatic Coronary Artery Disease in Type2 Diabetic Patients of Kamrup District in Assam by Treadmill Test	135
	<i>Barnali Kalita, Jyotismita Deka</i>	

28.	Muscular Performance in Euglycemic off Springs of Diabetic Parents 140 Using Mosso's Ergograph <i>Amruta S Bernal, Varsha Chavan, R H Taklikar, Ananth Taklikar</i>	140
29.	A Study of Critical Flickering Fusion Frequency Rate in Media Players 144 <i>Chiranjeevi Kumar Endukuru, K N Maruthy, T S Deepthi</i>	144
30.	Pre-donor Haemovigilance: Evaluation of Community Limiting Factors for Blood 149 Donation to Frame Better Transfusion Policies to Increase Blood Availability <i>Mohd Amir, Mohammed Imran, Humaira Naqvi, Pawan Singh, Muzaffar Zaman, A Mushtaque Shah</i>	149
31.	Assessment of Pulmonary Function and Functional Capacity in Overweight 155 Young Adults: Correlation with Visceral Fat <i>P Vijetha, P Apoorva, Harikrishna, T Jeevaratnam</i>	155
32.	Comparative Study of Bone Mineral Density in Premenopausal and 160 Postmenopausal Women <i>K Indira, Mohammed Farhan Ahmed</i>	160
33.	Effect of Isometric Hand Grip (IHG) Exercise Training on Cardiovascular and 166 Echocardiographic Parameters among Pre-hypertensive Young Males <i>Shalini Gandhi</i>	166
34.	PR Interval in Males and Females of Same Age Groups 172 <i>Vijayanath, Swati Jangam Veena H C</i>	172
35.	Comparison of Intraocular Pressure in Normal & Obese Post-menopausal Women 177 <i>S Syed Liyakath Ali, Prince Johnson Samuel, Neelamegam U</i>	177
36.	A Comparative Study of Amplitude of Nerve Impulse and Conduction Velocity of 182 Median and Common Peroneal Nerve among Medical Students with or without Family History of Diabetes <i>Laxmikanta Say, Madhuchhanda Pattanaik</i>	182
37.	Problem Solving Counseling is One of the Best Tools for Stress Reduction in 188 Medical Students <i>Basu Dev Singh, Surendra Kumar Dhruv, Pradeep Kumar</i>	188

Prevalence of Microalbuminuria - An Early Detector of Diabetic Nephropathy

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ABSTRACT

Background: Microalbuminuria is the presence of albumin in urine above the normal level but below the detectable range of conventional methods. Its presence is an early marker of diabetic nephropathy.

Aim & Objectives: 1. Detect prevalence of microalbuminuria in type 2 diabetic patients.

2. Correlate microalbuminuria with age, sex and duration of disease.

3. Correlate microalbuminuria with blood sugar (fasting and post- prandial), serum creatinine and Blood urea nitrogen (BUN).

4. Compare microalbuminuria in controlled (HbA1c<7) and uncontrolled (HbA1c> 7) type II diabetics.

Method: Around 100 cases of diabetic patients of age group 30-80 years, with duration of diabetes more than one year were examined for fasting and postprandial blood sugar(FBS and PPBS), HbA1c, BUN and serum creatinine. Also 24 hour urine sample was analysed for microalbuminuria by turbidometric method.

Results: The prevalence of microalbuminuria was 48% with p value 0.004. Also a significant correlation of microalbuminuria was seen with FBS, PPBS, BUN, and serum creatinine. However no correlation of microalbuminuria was established with HbA1c and duration of disease. Further microalbuminuria was increased in uncontrolled diabetics than controlled diabetics though it is not statistically significant.

Conclusion: Our study concludes that microalbuminuria is essential for early detection of diabetic nephropathy. Therefore it is recommended that microalbuminuria test should be done at regular intervals for type II diabetes mellitus.

Keyword: Type II diabetics, microalbuminuria, HbA1c, diabetic nephropathy.

INTRODUCTION

Diabetes mellitus is a worldwide public health concern and is associated with reduced mortality and significant morbidity. It leads to specific diabetes related macro vascular complications like myocardial infarction, stroke, renal failure, blindness and amputations. According to the World Health Organization (2004), diabetes affects more than 170 million people worldwide, and this number will rise to 370 million by 2030. About one third of type 2 diabetics will eventually have progressive deterioration of renal function¹. Microalbuminuria

is an abnormal elevation of albumin levels in the urine that cannot be detected using conventional dipstick method². It is defined as urinary albumin excretion between 20-200 µg/min or upto 15 mg/L over a 24 hour period³. It represents very early stages of diabetes mellitus (DM) when glomerular filtration rate (GFR) is normal. Thus it is an early marker of the subsequent development of proteinuria and diabetic nephropathy a leading cause of end stage renal disease and premature cause of death worldwide. Progression to established diabetic nephropathy occurs through several stages and microalbuminuria predicts future development of overt nephropathy⁴.

Diabetic nephropathy occurs in about 30% of patients with type 1 diabetes mellitus & 25% of patients type 2 diabetes mellitus⁵. The purpose of this study was to evaluate the presence of microalbuminuria in patients with DM and to ascertain its relationship with the age, disease duration, blood sugar levels, HbA1c, BUN and Creatinine.

MATERIAL & METHOD

This is a retrospective study carried out in 100 type 2 diabetes mellitus patients to detect the prevalence of microalbuminuria and to investigate correlation of microalbuminuria with age, sex, duration of the disease, blood sugar levels, HbA1c and markers of renal damage like BUN, serum creatinine. The study was conducted in M.S. Ramaiah Medical College and approved by institutional ethical committee board. Patients from both sexes were included. Fasting blood samples of the patients were analysed for FBS, PPBS, HbA1c, and BUN and serum creatinine. Also 24 hour urine collection was done for the analysis of microalbuminuria by turbidometric method which is considered as more sensitive assays.

1. FBS, PPBS in blood was estimated by glucose oxidase- peroxidase (GOD- POD) method.
2. Microalbumin in urine was estimated by turbidometric method.
3. Serum Creatinine was estimated by Jaffe's method.

4. BUN by colorimetric method.

STATISTICAL METHOD

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean \pm SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance. Student t test (two tailed, independent) has been used to find the significance of microalbuminuria (mg/Lt) between controlled (HbA1c<7) and uncontrolled (HbA1c>7) type II diabetics. Pearson correlation has been used to find the significance of relationship between microalbuminuria and study parameters in the cases.

RESULTS

In our study the prevalence of the disease is 48% with a significant p value of 0.004 (Table 1). Also the study showed a positive correlation of microalbuminuria with FBS (p = 0.0001) and PPBS (p= 0.005) (Table 2). The study did not show significant correlation with age, disease duration and HbA1c. However a significant correlation of microalbuminuria was observed with serum creatinine (0.0001) (Table 3) and BUN (p= 0.009) (Table 4). Further microalbuminuria was elevated in uncontrolled (HbA1c> 7) type II diabetics than controlled (HbA1c<7) diabetics but it is not statistically significant.

Table 1 - Prevalence for the Present Study

MA						
	N	%		Mean	SD	Independent t Test
Normal	52 [0 - 15]	52.0		7.22	3.89	2.988
Abnormal	48 [> 15]	48.0		426.77	1012.94	

Table 2- Relationship of FBS, PPBS with MA

Correlation between FBS and MA					
	N	Mean	SD	Pearson Correlation [r]	P Value
MA	100	208.606	729.044	0.362 [Moderate +ve Correlation]	0.0001 ***
FBSS	100	158.466	57.686		

Correlation between PPBS and MA					
	N	Mean	SD	Pearson Correlation [r]	P Value
MA	100	208.606	729.044	0.277 [Low +ve Correlation]	0.005 **
PBSS	100	233.929	81.705		

Table 3- Correlation between MA and Serum Creatinine

Correlation between Serum Creatinine and MA					
	N	Mean	SD	Pearson Correlation [r]	P Value
MA	100	208.606	729.044	0.455 [Moderate +ve Correlation]	0.0001 **

Table 4- Correlation between MA and BUN

Correlation between BUN and MA					
	N	Mean	SD	Pearson Correlation [r]	P Value
MA	100	208.606	729.044	0.261 [Low +ve Correlation]	0.009 **
BUN	100	12.759	7.305		

DISCUSSION

Diabetic nephropathy has become the most common single cause of end-stage renal disease (ESRD) all over the world⁶. The first clinical sign of renal dysfunction in patients with diabetes generally is microalbuminuria a sign of endothelial dysfunction that is not necessarily confined to the kidney. Microalbuminuria develops in 2 to 5 percent of patients of type 2 diabetes per year^{7,8}. The present study assessed the prevalence of microalbuminuria in type II diabetes mellitus a strong predictor of nephropathy⁹ and ESRD. As the duration of diabetes progresses in untreated patients it leads to numerous microvascular complication one of them being nephropathy. Thus diabetic nephropathy progresses gradually and accounts for about 40% of ESRD.

Most of the studies reveal that, diabetic patients do not present with well developed clinical manifestation of nephropathy¹⁰. However the same studies have proven significant microalbuminuria in these patients^{5,11}. Thus the magnitude of damage caused by the microvascular complication of diabetes stress needs a sensitive marker for screening nephropathy. And this sensitive marker has known to be the estimation of microalbumin excretion in the urine^{12,13}. According to WHO, the prevalence rate of nephropathy after 15 years of diabetes ranged between 17.7 and 56.6% in men and between 11.1 and 71% in women. Increased protein excretion may be an early clinical manifestation of diabetic nephropathy^{14,15,16}. In other studies the prevalence rate

of microalbuminuria was 34% and 27.2%^{3,17}. In our study the prevalence rate is 48% which was found to be statistically significant. However the variation in the prevalence range could be due to a difference in the patient selection criteria and different estimation technique. Further proteinuria independently associates with coronary heart disease in patients with diabetics and reflects not only renal impairment but a key pathogenic element of generalised vascular damage. Gene polymorphism of angiotensin converting enzyme has known to be associated with diabetic nephropathy¹⁸. Thus pathogenesis appears to involve complex interactions between genetic and environmental factors¹⁹. The patho-physiologic basis for elevated urinary albumin excretion entails the binding of glucose to proteins resulting in excessive protein glycosylation with the build-up of advanced glycated end products. This leads to deposition of advanced glycated end products on the glomerulus resulting in renal and glomerular hypertrophy, mesangial matrix accumulation and thickening of glomerular basement membrane. This abnormality permits the leakage of low molecular weight proteins [albumin]²⁰. As the diabetic nephropathy progress in early stage no clinical signs and symptoms of glomerular changes are seen. Thus the onset can be diagnosed by screening for microalbuminuria.

High sugar levels for long duration causes microvascular complication like retinal damage leading to loss of vision (retinopathy), sensory nerve damage (neuropathy) and irreversible kidney

damage (nephropathy). Since microalbuminuria is an early indicator of nephropathy, its prevalence rate was assessed in our study and was correlated with other markers of kidney damage like BUN and serum creatinine. Also in our study, microalbuminuria showed a significant positive correlation with both fasting and post prandial hyperglycaemia, which means that as the sugar levels raised the microalbumin excretion in urine also increased. This goes in accordance with previous studies¹⁴. In this study, no positive correlation was seen between microalbuminuria and age when compared to other studies wherein the levels were higher in older age group^{3,6}. However male patients (69%) have been found to be more susceptible to microalbuminuria as compared to female patients (31%) similar to the previous studies⁴. Further no positive correlation between microalbuminuria and duration of disease was established, as compared to other studies where the levels were higher in patients with the longer duration of disease^{21,22}. Also the study did not show positive correlation between microalbuminuria and HbA1c when compared to previous study¹².

Previous studies have shown a significant elevation of microalbuminuria in uncontrolled type II diabetics (HbA1c > 7) than controlled type II diabetics (HbA1c < 7)²³. However in our study the levels were higher in uncontrolled diabetics when compared to normals but it was not statistically significant.

Over the last few years, the association between microalbuminuria and cardiovascular events has been established. A strong relation of microalbuminuria, raised serum creatinine levels and presence of cardiac and vascular hypertrophy explain the excess morbidity and mortality rates observed in patients with renal dysfunction. This supports the role of the kidneys as an integrated sensor of cardiovascular risk²⁴. In this study also a significant positive correlation was seen between microalbuminuria and serum creatinine in accordance with previous study²⁵. Also a linear correlation between BUN levels and prevalence of microalbuminuria was observed in this study. It is interesting to note that increase in urea may increase prevalence of microalbuminuria in diabetic patients.

CONCLUSION

Our study has found a significant prevalence

of microalbuminuria in type II diabetics indicating subclinical damage of microvasculature, which is the predictor of development of diabetic nephropathy. Further as Microalbuminuria showed a positive correlation with FBS, PPBS, Serum creatinine and BUN it is the marker of choice for the detection of renal involvement in patients with diabetics. Hence, it is recommended that microalbuminuria test should be a part of routine check-up for DM. Further this alerts the physician to take precautionary measures for renal damage by timely administration of treatment strategy and correction of risk factor.

Acknowledgement: We would like to express our gratitude to V.Christopher Amalraj, Assistant professor in Biostatistics, Department of Community Medicine, SRM Medical College, Hospital and Research centre, Kattankulathur, Chennai, Tamil Nadu, for helping us with the statistics for this work. The authors disclose that we did not receive any financial or writing assistance.

Conflict of Interest: The authors declare that they have no conflict of interest.

Source of Funding: Nil

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