Section: Biochemistry

Original Research Article

STUDY OF MICROALBUMINURIA LEVEL AMONG PATIENTS WITH TYPE-2 DIABETES MELLITUS COMPLICATIONS IN TERTIARY CARE HOSPITAL OF MAHARASHTRA

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Abstract

Background: Type II diabetes is linked to a higher risk of heart disease and kidney disease. Level of microalbuminuria (MA) is the best way to predict heart disease related illnesses and deaths. **Materials and Methods:** 90 (ninety) adult patients with type II DM with MA are compared with 90 type II DM patients; having normal MA, BP was recorded. The blood examination included HbA1C, GFR, lipid profile, fasting plasma glucose, serum creatinine, and urine analysis. A calorimetric semi-quantitative urine test strip, pH, and specific gravity were also carried out. **Result:** Clinical and biometric manifestations, BMI, BP, fasting plasma glucose, serum creatinine, and GFR have significant p-values ($p < 0.001$). **Conclusion:** The prevalence of microalbuminuria in type-2 DM patients indicates a longer duration of undiagnosed diabetes and prognosis of type-II DM complications.

INTRODUCTION

Diabetes mellitus is a chronic progressive disease characterized by altered glucose homeostasis, is a significant cause of global morbidity and mortality.[1] Due to chronic hyperglycemia associated metabolic abnormalities, patients with DM are at risk for developing several macro and micro vascular complications,[2] namely retinopathy, nephropathy, and neuropathy.

The term microalbuminuria (MA) originated in 1964 by Professor Harry Keen as he observed a small amount of albumin in type II diabetes.[3] Almost a half century later, the status of Microalbuminuria (MA) is associated with disease outcomes. MA as a risk factor for diabetic kidney disease and cardio-renal risk, MA is a marker of cardiovascular disease; progression in MA is associated with elevation of plasma glucose, lipids, and blood pressure.[4] Hence, an attempt is made to evaluate the level of MA and correlate it with various parameters of kidney disease to study the morbidity and mortality in type 2 diabetes patients.

MATERIALS AND METHODS

90 (ninety) patients aged between 45-60 years admitted to the medicine department of the Vedanta Institute of Medical Sciences hospital in Saswand, Dahanu, Palgarh, and Maharashtra-401606 were studied.

Inclusion Criteria

Patients with known type II diabetes with microalbuminuria aged between 45 to 60 years. The patients who gave their consent in writing were selected for the study.

Exclusion Criteria

Known type I diabetes, terminally ill diabetes patients, urinary tract infections with Pus cells in urine ≥ 5/high power field, pregnant women, febrile patients. Obstructive uropathy and nephrolithiasis were excluded from the study.

Method

90 (ninety) type-II DM patients with microalbuminuria were compared with 90 healthy type-II DM patients with normal microalbuminuria (controlled group).

Every patient was interviewed with related clinical details, i.e., onset of diabetes, history of hospitalization, history of hypertension, family history of diabetes, complications of retinopathy, cardiovascular disease, and cerebrovascular disease were recorded.

Complete physical examination was carried out, body mass index (BMI), waist and hip circumferences were measured by the standard technique recommended by WHO. Blood pressure was recorded in the sitting position in the right arm using a mercury sphygmomanometer. The latest fasting

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plasma glucose, glycated hemoglobin (HbA1C), blood urea, and serum creatinine values were noted and compared with controlled group patients. Random spot urine was collected, and a calorimetric semi-quantitative urine test strip (Combur 10 (1), Roche Diagnostics Mannhein, Germany) was used to test for pH, specific gravity, leukocytes, nitrite, blood hemoglobin, and protein. If pH and specific gravity were normal and other parameters were negative, then microalbumin was tested using a dipstick (Micral II (2), Roche diagnostics).

The following definitions were used in the present study—

- Macro proteinuria: a change in color of the dipstick that corresponds to ≥|+ (30 mg/dl) was considered to be macro proteinuria.
- MAU: A change in the color of the Micral II strip that corresponds to > 20 mg/L was considered to be positive for MAU.
- Renal failure: Individuals with serum creatinine >1.5 mg/dl were considered to have renal failure and excluded from the study.
- Hypertension: Subjects with self-reported hypertension on drugs and those who had systolic blood pressure > 140 mm Hg and/or diastolic blood pressure ≥ 90 mm Hg were considered to have hypertension.
- Obesity: As per WHO Body Mass Index (BMI), a BMI of ≥ 25 kg/m² is considered as obesity.
- Estimated glomular filtration rate modification of diet in renal disease (MDRD) formula is used to estimate glomerular filtration rate (GFR).

The duration of the study was from January 2024 to December 2024.

Statistical Analysis

Various clinical and biochemical parameters of microalbuminuria were compared in the controlled group versus the microalbuminuria and macroproteinuria groups using the ANOVA test. Moreover, some parameters were classified with a percentage. The statistical analysis was carried out using SPSS software. The ratio of male and female was 2:1.

RESULTS

- 5.8 (± 1.5) in controlled group, 6.4 (± 2.4) in MAU, 10.2 (± 3.4) Macro protenuria F=5.42 and p<0.001.
- Hypertension: 32 (35.5%) in controlled group, 20 (44.4%) in MAU, 28 (62.2%) in Macro protenuria
- BMI (kg/m2): 26.2 (± 3.8%) in controlled, 26.8 (± 4.8) in MAU, 24.8 (3.92) in Macro protenuria, F=3.06 and p<0.004.
- Waist circumference: 93.4 (± 8.2) in controlled, 93.0 (± 8.4) in MAU, 89.4 (± 7.8) in Macro protenuria, F=3.83 and p<0.002.
- Blood pressure systolic: 134.2 (± 8.2) in controlled, 140.38 (± 9.98) in MAU, 145.4 (± 7.8) in Macro protenuria, F=26.9 and p<0.001.
- Diastolic: 82.06 (± 9.60) in controlled, 88.9 (± 8.80) in MAU, 90.45 (± 10.8) in Macro protenuria, F=5.44 and p<0.001.
- Retinopathy: 8 (± 8.5) in controlled, 10 (± 22.1) in MAU, 20 (± 44.4) in Macro protenuria.
- Coronary artery disease (CAD): 13 (14.4%) in controlled, 9 (20.5%) in MAU group, 6 (13%) in Macro protenuria group.
- Cerebro vascular disease: 2 (2.2%) in controlled, 1 (2.3%) in MAU, 2 (4.4%) in Macro protenuria.
- Fasting plasma (mg/dl): 148 (± 5.34) in controlled, 162 (± 11.9) in MAU group, 178 (± 10.2) in Macro protenuria, F=182.6 and p<0.001.
- HbAIC: 7.62 (± 158) in controlled, 82 (± 1.4) in MAU group, 8.38 (± 1.8) in Macro protenuria, F=4.82 and p>0.009 (p value is insignificant)
- Serum creatinine: 0.95 (± 0.15) in controlled group, 1.008 (± 11.9) in MAU group, F=18.6 and p<0.001.
- GFR (Glumulofilteration rate) (ml/min/1.73 m2): 78.7 (± 8.6) in controlled, 75.30 (± 9.02) in MAU group, 66.58 (± 11.2) in Macro protenuria, F=24.9 and p<0.001.
- Insulin therapy: 14 (15.5%) in controlled group, 13 (28%) in MAU group, 13 (28%) in Macro protenuria.
- ACE inhibitor or Angio tension blocker therapy: 14 (15.5%) in controlled group, 12 (26.6%) in MAU group, 18 (40%) in Macro protenuria.

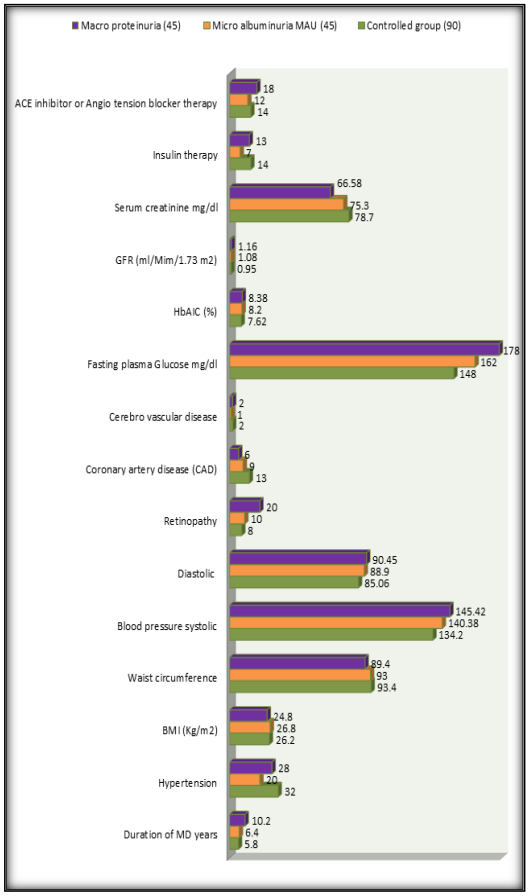

Figure 1: Comparison of clinical and Biochemical Manifestation of Micro albuminuria in type-II DM patients

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Table 1: Comparison of clinical and Biochemical Manifestation of Micro albuminuria in type-II DM patients.

Manifestation	Controlled group (90)	Microalbuminuria (MAU) (45)	Macro proteinuria (45)
Duration of MD years	5.8 (±1.5)	6.4 (± 2.4)	10.2 (± 3.4) *
Hypertension	32 (35.5%)	20 (44.4%)	28 (62.2%) *
BMI (Kg/m2)	26.2 (± 3.8)	26.8 (± 4.36)	24.8(±3.92) *
Waist circumference	93.4 (± 8.2)	93.0 (± 8.4)	89.4 (±7.8) *
Blood pressure systolic	134.2 (±8.2)	140.38 (± 9.98)	145.42 (±7.8) *
Diastolic	85.06 (± 9.60)	88.90 (±8.80)	90.45 (±10.05) *
Retinopathy	8 (8.5%)	10 (22.11)	20 (44.4%)
Coronary artery disease (CAD)	13 (14.4%)	9 (20.5%)	6 (13.3%)
Cerebro vascular disease	2 (2.2%)	1 (2.9%)	2 (4.4%)
Fasting plasma Glucose mg/dl	148 (± 5.34)	162 (± 11.9)	178 (± 10.2) *
HbAIC (%)	7.62 (± 1.58)	8.2 (± 1.4)	8.38 (± 1.8) *
GFR (ml/Mim/1.73 m2)	0.95 (± 0.15)	1.008 (± 0.20)	1.16 (± 0.24) *
Serum creatinine mg/dl	78.7 (± 8.6)	75.30 (± 9.02)	66.58 (± 11.2) *
Insulin therapy	14 (15.5%)	7 (15.5%)	13 (28%) *
ACE inhibitor or Angio tension blocker therapy	14 (15.5%)	12 (26.6%)	18 (40%)

DISCUSSION

In our study, we compared the incidence of post-intubation sore throat between intra-cuff alkalinized lignocaine and plain lignocaine. The main finding of this study was that alkalinized lignocaine significantly reduced the incidence of post-intubation sore throat. As the air volume increased during extubation owing to diffusion, the air volume required to inflate the cuff was higher than the liquid volume. No significant difference was observed in the liquid volume used for cuff inflation between groups B and C, although the amount of liquid drawn from the cuff was lower in group C. At extubation, 88% of Group A, 84% of Group B, and 64% of Group C experienced coughing, while Group C showed a significant reduction over time. The incidence of cough declined steadily in all groups, with significant differences noted at extubation and 1, 2, 12, and 24 h ($p<0.001$). Restlessness was observed in 80% of the patients in Group A 72% in Group B, and 48% in Group C at extubation; group C showed no restlessness after 2 h. Significant differences in restlessness were observed at extubation and 1, 2, and 12 h ($p<0.001$, $p<0.001$, and $p=0.02$, respectively), with no significant difference at 24 h ($p=0.078$). Dysphonia was more prevalent in groups A and B at 1 h, with significant differences at 2 h ($p<0.001$); however, no significant differences were observed at 1, 12, or 24 h. Hoarseness was higher in groups A (52%) and B (28%) at 1 h, with significant differences at 1, 2, 12, and 24 h ($p=0.02$, $p<0.001$, $p=0.001$, $p=0.034$). The sore throat was most common in group A at 12 h (68%), with significant differences between the groups at 12 and 24 h ($p=0.001$). In Rao et al.'s study, 90% of intubated patients experience postoperative sore throat, the most frequent complaint following tracheal intubation.1 In the Altintas et al. study, tracheal tube insertion can cause hematomas, mucosal laceration or granulomas, or injury to the cartilage of the arytenoids in the upper respiratory tract.[9] In a study by Seegobin et al., factors such as tube size, tube design, lateral wall pressure, intracuff pressure, tube lubricant use, hypotension, local infection, steroid use, and intubation duration were associated with sore throat. At lateral wall pressures above 30 cm of water (22 mmHg), evidence of mucosal blood flow obstruction was observed. At lateral wall pressures above 37 mmHg, the flow to the mucosa across the tracheal rings and posterior tracheal wall was completely blocked. Large-volume cuffs have been theorised to have a sparing impact on capillary blood flow over cartilaginous rings by draping the intercartilaginous mucosa and exerting pressure on the arterioles in the intercartilaginous submucosa, hence increasing the effective perfusion pressure.[10] Tu et al. concluded that lateral wall pressure, which affects tracheal capillary blood flow, is a significant component of tracheal morbidity, and that tracheal mucosal erosion may be reduced by ongoing monitoring and avoiding high lateral wall pressures. After air inflation, the cuff pressure and volume increased over time. When employing nitrous oxide for anaesthesia, the cuff pressure rises as the cuff's temperature rises and nitrous oxide diffuses into it more quickly than it does so.[11] Sconzo et al. over-inflation of the ETT has been linked to laryngeal nerve palsy and pharyngeal mucosa injury.[12] Ahmad et al. also caused increased receptor stimulation in the tracheal mucosa and thus increased emergence and extubation phenomena and complications were decreased by filling the ETT cuff with liquid. According to Matias, in vitro experiments, lignocaine's diffusion is significantly (63-fold) improved by alkalinization. Estebe et al. studies in vivo and at modest doses (40 mg) have demonstrated a reduction in postoperative side effects. Dollo et al. studies have demonstrated that the dose of lignocaine used between 20 and 40 mg relates to the amount of lignocaine diffusing through the ETT cuff when $NaHCO_3$ is present. Soltani et al. reported that coughing may stop or start as a patient emerges from an intravenous bolus of lignocaine, and the serum level drops. If adequate cough suppression and a fully awake patient are sought at the same time, the ideal timing of treatment during emergence may be challenging because of the limited antitussive window of intravenous lignocaine. Although lignocaine has been used to lubricate ETTs, it

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exacerbated ETT-induced emergence phenomena from anaesthesia whether applied as a spray or jelly. Walmsley et al. found that the incidence of tracheal tube cuff rupture was noted in 30 polyvinyl chloride tracheal tubes lubricated with three different solutions. All cuffs moistened with water were intact after 2 h of cuff inflation, whereas two I0 cuffs lubricated with a 4% lignocaine solution burst. Both had leaked at the site of the cuff attachment to the tube.

CONCLUSION

In the present study, it is confirmed that microalbuminuria is strongly associated with hypertension, poor glycemic control, and other diabetic complications like neuropathy and retinopathy. The present study demands that such a study must be conducted in a large number of patients in hi-tech research centers to confirm the significance of the present study because there is no ideal parameter or device to find undiagnosed diabetes, which leads to multiple irreversible neurovascular complications.

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