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Original Article

Assessment of serum myeloperoxidase, apolipoprotein B and glycated hemoglobin levels in type 2 diabetes mellitus patients

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ABSTRACT:
Background: Type 2 diabetes is associated with dyslipidaemia comprising of multiple lipoprotein disorders. The present study was conducted to assess serum myeloperoxidase, apolipoprotein B and glycated hemoglobin levels in type 2 diabetes mellitus. **Materials & Methods:** 46type II DM patients of both genders were enrolled. 2 groups were formed. Group I had type II DM and group II had healthy control. 6ml of fasting venous blood samples is drawn under aseptic precautions in to a sterile bulb from selected subjects. The levels of serum myeloperoxidase (MPO), glycated hemoglobin (HbA1c) and apolipoprotein B was measured. **Results:** We found thatgroup I had 26 males and 20 females and group II had 22 males and 24 females.We found that mean serum Apo B was 236.1 mg/dl in group I and 125.2 mg/dl in group II, HbA1c level was 7.3% in group I and 4.0% in group II, mean MPO level was 21119 pg/ml in group I and 10136 pg/ml in group II. The difference was significant (P< 0.05). **Conclusion:** The level of serum myeloperoxidase (MPO), glycated hemoglobin (HbA1c) and apolipoprotein B was higly elevated in type II DM patients as compared to healthy control.
Key words: glycated hemoglobin, apolipoprotein B, myeloperoxidase

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INTRODUCTION

Type 2diabetes is associated with dyslipidaemia comprising of multiple lipoprotein disorders. The most typical findings are high triglycerides and triglyceride rich lipoproteins, low levels of high-density lipoprotein (HDL) cholesterol, normal or slightly increased low-density lipoprotein (LDL) cholesterol and presence of small dense LDL particles which are cholesterol depleted.[1]

Myeloperoxidase (MPO), an enzyme of the heme peroxidase superfamily in mammals is present within the azurophilic granules of leukocytes. It plays a major role in innate host defence by participating in the oxygen dependent killing of invading pathogens.[2] MPO exhibits its anti-microbicidal property by catalyzing a unique reaction in which hydrogen peroxide reacts with chloride ions. The product formed by this reaction is the potent oxidant hypochlorous acid. In addition, evidence shows that increased enzymatic activity of MPO is seen in many chronic inflammatory conditions. MPO is capable of generating reactive oxygen species that modify lipids and proteins which contributes to atherogenesis.[3] Apolipoprotein B and apolipoprotein A-1 are the main structural proteins of atherogenic lipoproteins and HDL particles, respectively.[4]HbA1c measures chronic glycaemic exposure rather than an acute value, therefore providing a more relevant view of long- term glycaemia and future risk of complications. By measuring the serum levels of MPO, apo B and HbA1c, we can assess the future risk of cardiovascular disease in type 2 diabetic patients.[5] The present study was conducted to assess serum myeloperoxidase, apolipoprotein B and glycatedhemoglobinlevels in type 2 diabetes mellitus.

MATERIALS & METHODS

The present study comprised of 46type II DM patients of both genders. The consent was obtained from all enrolled patients.

Data such as name, age, gender etc. was recorded. 2 groups were formed. Group I had type II DM and

group II had healthy control. 6ml of fasting venous blood samples is drawn under aseptic precautions in to a sterile bulb from selected subjects. The levels of serum myeloperoxidase (MPO), glycatedhemoglobin (HbA1c) and apolipoprotein B was measured.Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Groups	Group I	Group II
Status	Type II DM	Healthy control
M:F	26:20	22:24

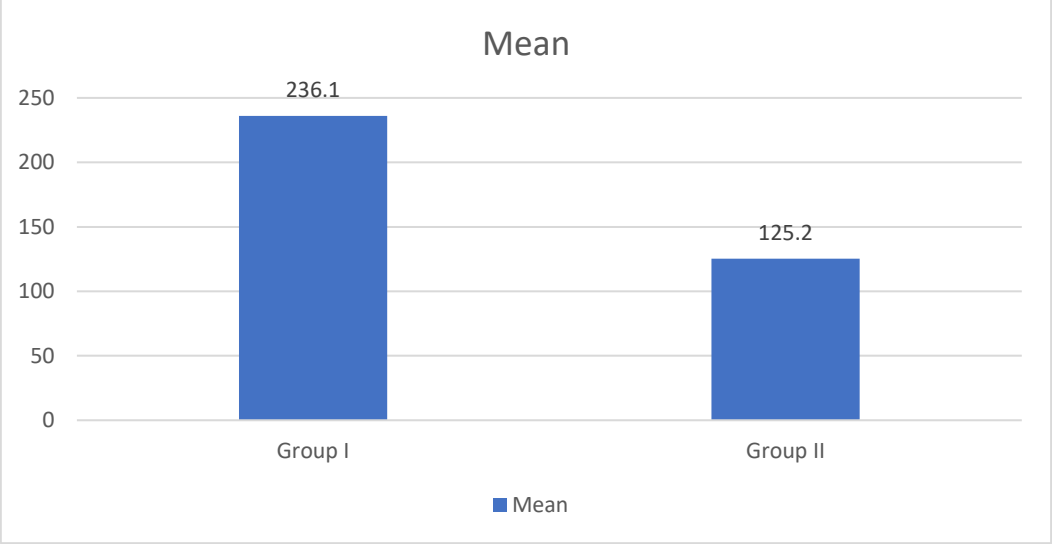
Table I shows thatgroup I had 26 males and 20 females and group II had 22 males and 24 females.

Table II Assessment of HbA1c, Apo B, MPO in both groups

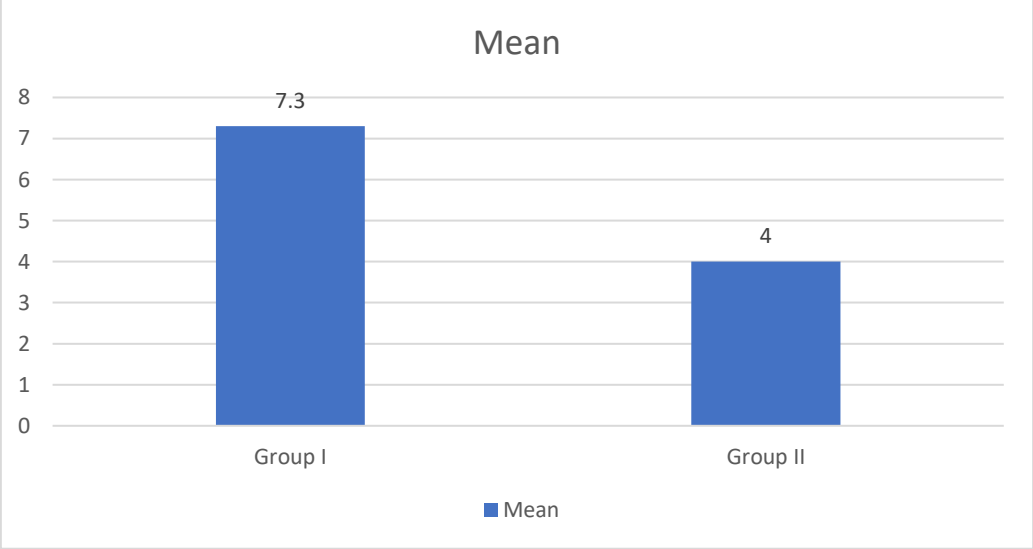
Parameters	Group I	Group II	P value
Serum Apo B (mg/dl)	236.1	125.2	0.01
HbA1c (%)	7.3	4.0	0.02
MPO (pg/ml)	21119	10136	0.01

Table II, graph I a, b, c shows that mean serum Apo B was 236.1 mg/dl in group I and 125.2 mg/dl in group II, HbA1c level was 7.3% in group I and 4.0% in group II, mean MPO level was 21119 pg/ml in group I and 10136 pg/ml in group II. The difference was significant (P< 0.05).

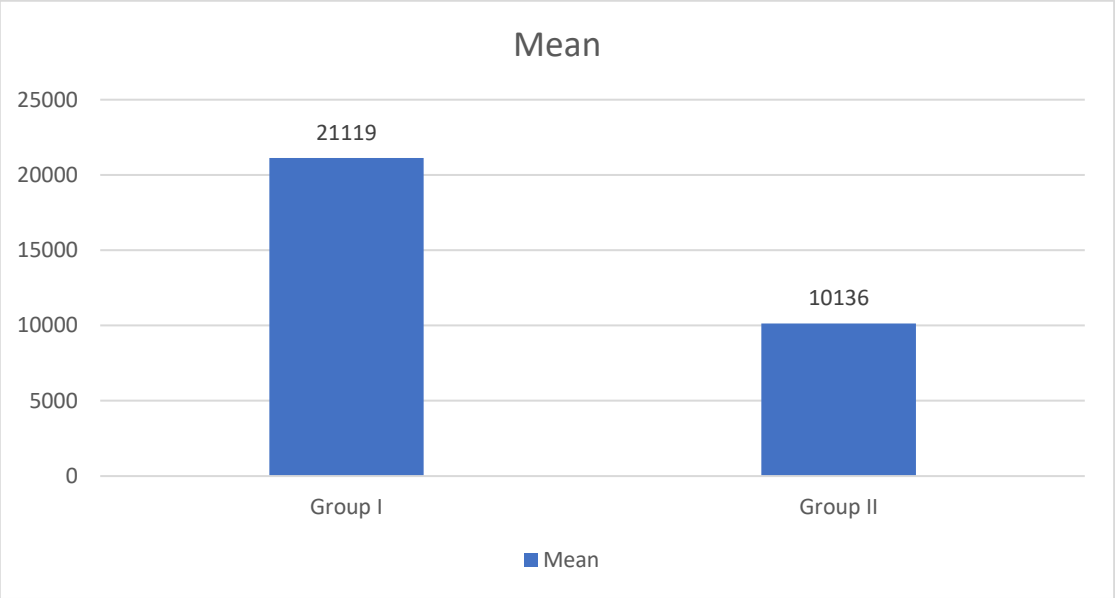
Graph Ia Serum Apo B level in both groups



Graph Ib HbA1clevel in both groups



Graph Ic MPO level in both groups



DISCUSSION

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. Type 2 diabetes (T2D) occurs due to a progressive decline in the ability of the pancreas to secrete enough insulin as well as insulin resistance in insulin target tissues.[6] The pathophysiology of T2D is characterized by excessive accumulation of ectopic fat in the liver, pancreas, and skeletal muscles, eventually manifesting as insulin resistance in these tissues and pancreatic beta cell dysfunction that ultimately leads to hyperglycemia.[7] Metabolic abnormalities such as dyslipidemia, hyper insulinemia, or insulin resistance and obesity play key roles in the induction and progression of type 2 diabetes mellitus (T2DM). Apo B identifies high-risk dyslipidaemic phenotypes that are not detected by standard lipid profile intype 2 diabetic patients.[8] The addition of apo B to standardlipid profile could aid in timely introduction of lipid loweringtherapy in these unidentified high-risk patients and thus reducemortality and morbidity due to future cardiovascular complications in them.[9] The present study was conducted to assess serum myeloperoxidase, apolipoprotein B and glycated haemoglobin levels in type 2 diabetes mellitus.

We found that group I had 26 males and 20 females and group II had 22 males and 24 females.Kanani et al[10]analyzed the pattern of dyslipidaemias including apolipoprotein B in type 2 diabetes. A total of 120 diabetics were studied for their lipid profile including serum triglycerides, total cholesterol, HDL cholesterol and LDL cholesterol in fasting state, along with apolipoprotein B levels. Raised apolipoprotein B was the most frequent lipid disorder in type 2 diabetics, occurring in 56.7% of the studied patients. This was followed by high serum triglycerides levels in 55.8% and low HDL cholesterol levels in 55% of patients. Notably, 6% patients had normal triglyceride levels accompanied by raised LDL cholesterol, compared to 20% patients who had normal triglycerides with high apolipoprotien B levels. Overall, 36% of patients had normal LDL cholesterol values but elevated apolipoprotein B.

We found that mean serum Apo B was 236.1 mg/dl in group I and 125.2 mg/dl in group II, HbA1c level was 7.3% in group I and 4.0% in group II, mean MPO level was 21119 pg/ml in group I and 10136 pg/ml in group II. Ley SH et al[11]assessed the association of apo B with incident type 2 diabetes and compared it with traditional lipid variables as a risk predictor in aboriginal Canadians.Of an initial cohort of 606 individuals without diabetes in 1993–1995, 540 were contacted for the 10-year follow-up evaluation in 2003–2005. Fasting and 2-hours post-load glucose concentrations were obtained at baseline and follow-up to determine incident type 2 diabetes. Baseline fasting serum lipids were measured with standard laboratory procedures.Thecumulative 10-year incidence of type 2 diabetes was 17.5%. High concentrations of apo B, triglycerides, and LDL cholesterol, and low concentrations of HDL cholesterol were individually associated with incident type 2 diabetes in univariate analyses. Comparing C statistics of univariate models showed apo B to be a superior determinant of incident diabetes compared with LDL (P = 0.026) or HDL (P = 0.004) cholesterol. With multivariate adjustment including waist circumference, apo B and triglycerides remained associated with incident diabetes, whereas LDL and HDL cholesterol became nonsignificant.

CONCLUSION

Authors found that the level of serum myeloperoxidase (MPO), glycated hemoglobin (HbA1c) and apolipoprotein B was higly elevated in type II DM patients as compared to healthy control.

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