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Original Research Paper

General Medicine

CORRELATION OF HBA1C AND MEAN PLATELET VOLUME IN TYPE 2 DIABETIC PATIENTS: A CASE CONTROL STUDY

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ABSTRACT Diabetes is collectively characterized by metabolic disorders explained by hyperglycemia due to defects in insulin release, insulin activity, or both. Various organs such as eyes, kidneys, nerves, heart and veins are affected. Digestive irregularities of sugars, fats and proteins in diabetes are caused by inadequate insulin activity in splint tissues. Weakened insulin release and altered insulin activity constantly co-occur in similar patients. It is unknown which abnormality is the main cause of hyperglycemia. Method: This was conducted on patients admitted in medicine wards and ICU and Endocrine ward in Geetanjali medical college & hospital with AMI. Result- Mean FBS among the patients of the group A and group B was 158.65 mg/dL and 82.03 mg/dL respectively. Mean PPBS among the patients of group A and group B was 245.05 mg/dL and 148.29 mg/dL respectively. Mean HbA1c among the patients of group A and group B was 9.76% and 5.79% respectively. Conclusion- Statistical analysis revealed that there is a significant correlation between MPV and HbA1c, FBS, PPBS. However, MPV scores were correlated with the duration of diabetes, but no significant correlation was observed. The mean BMI for patients in Group A and Group B was 26.73 kg / m2 and 24.89 kg / m2, respectively.

KEYWORDS : Diabetes, hyperglycemia, insulin

INTRODUCTION

Diabetes is collectively characterized by metabolic disorders explained by hyperglycemia due to defects in insulin release, insulin activity, or both. Various organs such as eyes, kidneys, nerves, heart and veins are affected. A particularly common sign of diabetes, hyperglycemia, is associated with long-term damage and deterioration of various organs. Digestive irregularities of sugars, fats and proteins in diabetes are caused by inadequate insulin activity in splint tissues. Weakened insulin release and altered insulin activity constantly co-occur in similar patients. It is unknown which abnormality is the main cause of hyperglycemia. The intense and dangerous consequences of uncontrolled diabetes are ketoacidosis or non-ketoacidosis hyperglycemia with hyperosmotic disorders. It provides protection against insulin activity, including autoimmune destruction of pancreatic cells and subsequent insulin deficiency, and even irregularities. Manifestations of stamped hyperglycemia incorporate polyuria, polydipsia, weight reduction, polyphagia, and obscured vision. Hindrance of development and helplessness to specific contaminations might go with constant hyperglycemia.

A few pathogenic cycles are associated with the improvement of diabetes. Retinopathy with plausible vision loss has been one of the long-standing diabetic complexes. Nephropathy that causes renal failure; Marginal neuropathy associated with removal of foot ulcers, and risk of Charcot joints; Autonomic neuropathy exacerbates gastrointestinal, urogenital, and cardiovascular symptoms. Patients with diabetes are more likely to develop marginal vessels, cardiovascular and cerebrovascular infections with atherosclerosis. Lacking insulin activity results from insufficient insulin emission or by decreased tissue reactions to insulin at least one places in the perplexing pathways of chemical activity.

Hypertension and irregularities of lipoprotein digestion are regularly found in patients with diabetes. By far most of instances of diabetes fall into two wide etiopathogenetic classifications. In the principal classification, type 1 diabetes, it is brought about by a flat out lack of insulin emission. People at expanded danger of fostering this sort of diabetes can be recognized by serological proof of an immune system pathologic interaction which happens in the pancreatic islets and by hereditary markers. In the subsequent class, type 2 diabetes, more common classification, it is made by a mix of obstruction insulin activity and a lacking compensatory insulin secretory reaction. A level of hyperglycemia adequate to cause pathologic and useful changes in different objective tissues, without clinical indications, might be available for an extensive stretch of time before diabetes is recognized. During asymptomatic period, it is feasible to delineate an anomaly in carb digestion by estimation of plasma glucose in the fasting state or later a test with an oral glucose load or by A1C.

Patients with type 2 diabetes (DM) are twice as likely to develop coronary artery disease (CHD) horror and mortality, four times more likely to develop congestive cardiovascular failure, and are four times more likely to develop stroke. Is 26 times higher. Additional unfortunate expectations for cardiovascular activity are noted rather than those without diabetes. The risk of cardiovascular mortality is associated with glycemic fixation in cases of type 2 diabetes. Diabetes is called a cerebrovascular risk equivalent to CHD. Diabetic patients with type 2 diabetes show increased platelet responsiveness. Hyperglycemia effects in accelerated platelet reactivity thru the direct outcomes of platelet proteins and innovative glycation. Hypertriglyceridemia for the most part builds platelet reactivity.

Insulin resistance and insulin deficiency boom platelet reactivity. Insulin alienates platelet staging. Therefore, relative or regular insulin deficiency is regular to boom platelet reactivity. Diabetes is likewise related to oxidative stress and exacerbations. The resulting endothelial fragility promotes platelet initiation by reducing the formation of nitric oxide (NO), which weakens the reactivity of platelets. Oxidizing pressure emphasizes this effect by reducing the movement of NO and promoting the initiation of platelets. There is a correlation between irritation and platelet initiation.

Aggravation advances platelet actuation which advances irritation. Further developed metabolic control accomplished with regimens that further develop insulin affectability and protect pancreatic -cell work are factors which decline platelet reactivity and upgrade impacts of antiplatelet specialists. Platelets from patients with diabetes show expanded reactivity (expanded penchant to enact because of an upgrade).

Enlistment of hyperglycemia and hyperinsulinemia in sound patients without diabetes will in general expand platelet reactivity. Improved glycemic manage has been associated with faded platelet reactivity. Hyperglycemia can boom platelet reactivity via way of means of activating non-enzymatic glycation of proteins with inside the outer layer of platelets. Glycation diminishes layer smoothness and it efficiently expands the inclination of platelets. Osmotic effect of glucose is the second one element via way of means of which hyperglycemia can increase platelet reactivity. It was tracked down that concise openness of platelets in vitro to hyperglycemia or a comparable centralization of mannitol adept expanded their reactivity. Actuation of protein kinase C is a third system by which hyperglycemia can expand platelet reactivity. A fundamental middle person of platelet enactment is Protein Kinase C.

AIM

To determine the correlation of hba1c and mean platelet volume in type 2 diabetic patients.

OBJECTIVES

- To Correlate Mean Platelet Volume with Hba1c by Using Pearson Co-Efficient
- To Correlate Mean Platelet Volume with Fasting Blood Glucose.
- To Correlate Mean Platelet Volume with BMI
- To Correlate Mean Platelet Volume with Duration of Diabetic.

CASE STUDY

This is case control study which will be conducted on 100 patients in which 50 patients are diabetic and 50 are non-diabetic. Detailed history and thorough physical examination along with detailed clinical examination was done. The result was statistically performed.

RESULT

TABLE - Distribution With Aging

Age group (years)	Group A		Group B	
	Number of patients	%	Number of patients	%
Less than 30	5	10	4	8
31 to 40	8	22	7	14
41 to 50	10	24	11	22
51 to 60	19	30	18	36
More than 60	8	14	10	20
Total	50	100	50	100
Mean ± SD	49.04 ± 11.90		49.58 ± 11.87	

Table 2: Gender-wise distribution

Gender	Group A		Group B	
	Number of patients	%	Number of patients	%
Males	31	62	33	66
Females	19	38	17	34
Total	50	100	50	100

Table 3: Duration Of Diabetes

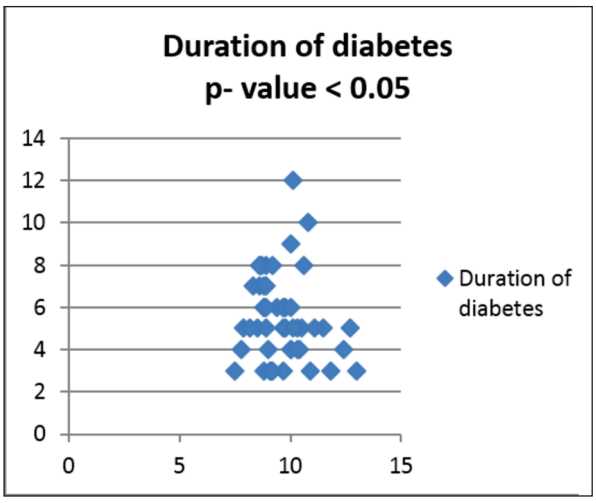
Duration of diabetes	Group A	Group B
Mean	5.46	-
SD	2.04	-

Table 4: Bmi

BMI (Kg/m^2)	Group A	Group B
Mean	26.73	24.89
SD	4.52	2.45

Table 5: Glycemic Profile

Glycaemic profile	Group A		Group B		p- value
	Mean	SD	Mean	SD	
FBS (mg/dL)	158.65	12.78	82.03	13.04	0.000*
PPBS (mg/dL)	245.05	13.65	148.29	13.15	0.001*
HbA1c (%)	9.76	1.24	5.79	0.58	0.012*



Graph 1: Correlation of MPV with duration of diabetes among the patients of group A (Diabetic group)

Table 7: Correlation Of Mpv And Glycemic Variables In Group A (diabetic) Patients

Variables		r- value	p- value
MPV	Duration of diabetes	-0.029	0.328
MPV	HbA1c	0.152	0.000*
MPV	FBS	0.328	0.001*
MPV	PPBS	0.191	0.000*

30 percent of the patients of the group A and 36 percent of the patients of group B belonged to the age group of 51 to 60 years. 24 percent of the patients of the group A and 22 percent of the patients of group B belonged to the age group of 41 to 40 years. Mean age of the patients of the group A and group B was found to be 49.04 years and 49.58 years respectively.

62 percent of the patients of the group A and 66 percent of the patients of the group B were males while the remaining were females. Our results were in concordance with the results obtained by previous authors who also reported male preponderance in their respective studies.

CONCLUSIONS

In diabetes mellitus, platelets are more reactive and more likely to aggregate, increasing their average volume (MPV). Increased platelet size may be a factor in the increased risk of atherosclerosis associated with diabetes mellitus and related vascular complications. Therefore, MPV will be a useful prognostic marker for cardiovascular complications in diabetes.

Since HbA1c is an effective indicator of glycemic control and is linearly associated with diabetic vascular complications, we conclude that MPV can be considered as an indicator of glycemic status and diabetic vascular complications in addition to HbA1c. Attached, but this requires more extensive prospective research.

Therefore, MPV proposes that it can be used as a simple and inexpensive tool for monitoring the progression and control of DM and its cardiovascular complications.

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