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MEAN PLATELET VOLUME IN TYPE II DIABETES MELLITUS, CASE CONTROL STUDY – GEZIRA CENTRAL SUDAN

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Abstract
Aim: Thrombotic disorders are the major complications of diabetes mellitus (DM). Platelets are considered to play a role in these vaso-occlusive complications. The mean platelet volume (MPV) is recently taken as indicator for platelet reactivity. The aim of this study was to show the state of platelet activity in type II diabetes mellitus by indication of MPV.
Methods: This study was done in Wad Madani, Gezira State, Sudan 2007. MPV was measured in 100 adult type II diabetic Sudanese patients and 100 adult healthy control subjects matched for age and gender.
Results: MPV was found to be significantly higher among the diabetic group (mean 9.9 fl) than the control group (mean 9.0 fl) (P value 0.015).
Conclusion: We conclude that elevated MPV may indicate platelet hyperactivity, which may contribute to the vascular complications of type II DM.

Key words: Type II diabetes mellitus, platelets, vascular disorders, Mean platelet volume.

Introduction

Diabetes mellitus (DM) is the most common endocrine disease characterized by metabolic abnormalities, hyperglycemia, and by long term complications. There are two major subgroups of DM, type I insulin dependent (IDDM) and type II (NIDDM).[(1)] Thrombosis, atherosclerosis and other vascular diseases are common complications of diabetes mellitus.[(2)] Most of the morbidity and mortality seen in patients with diabetes mellitus, especially in type II ('non-insulin dependent') diabetes, is the result of these micro- and macro-vascular occlusive diseases in which thrombosis plays an important part.[(3)]

As platelets play a pivotal role in thrombus formation, an increase in platelet reactivity is one potential mechanism that could explain the increased incidence of thrombotic disease seen in the diabetic population.[(4)] Platelets respond to a variety of substances by altering their shape, adhering to material to which they exposed, changing ectomembrane constituents and undergoing the release reaction.[(5)] There is a strong possibility that vascular spasm (for example, of the coronary arteries) may be caused by thromboxane A2, serotonin, or other vaso-active substances released as a consequence of platelet activation. Such spasm may cause ischemic symptoms, particularly if the circulation is already compromised by proximal atheroma.[(6)]

The abnormal metabolic state that accompanies diabetes renders arteries susceptible to

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atherosclerosis by altering the functional properties of multiple cell types, including endothelium and platelets. In particular, an altered platelet metabolism and changes in intraplatelet signaling pathways contribute to platelet hyperactivity.(7) Increased platelet reactivity could also be due to reduced production of factors that inhibit platelet activation, nitric oxide (NO) that has been shown to be reduced in diabetes.(8) One of the consequences of hyperglycemia is oxidative stress that is responsible for enhanced peroxidation of arachidonic acid to form a biologically active and a potent aggregating agent, thromboxane A_2 which cause persistent platelet activation. Increased oxidative stress is responsible for activation of transcription factors and switch of endothelium toward an adhesive, pro-thrombosis condition, initial platelet activation, adhesion and subsequent platelet aggregate formation.(7) The mean platelet volume (MPV) is recently taken as a determinant for platelet function, as it is positively associated with platelet reactivity function.(9) Large platelets contain more dense granules and produce more thromboxane A_2. It can be an indication of platelet turnover because younger platelets tend to be larger.(10) Little is known about effects of various drugs on platelet size. Previous in vitro studies found no effect of aspirin on platelet size.(11) A number of studies was done to compare MPV between diabetic and non diabetic individuals. It was found that MPV is significantly higher in diabetic subjects. (P value < 0.05). (12) (13) (14) (15)

Materials and Methods

This study was done in Wad Madani, Gezira State in Sudan 2007. A total of 200 subjects were included in this study, 100 were type II diabetic Sudanese patients the rest were apparently healthy individuals matched for age and gender. The data were collected by using a direct interviewing questionnaire. Verbal consent was taken from each subject. The MPV was measured by automated cell counter following standard operating procedures. The statistical analysis of the results was performed by using the Statistical Package for Social Sciences (SPSS) for windows version 10. T-test was used for testing difference significance, *P* value < 0.05 considered statistically significant. Microsoft office excel was used to present the result in figures.

Results

The MPV in diabetic patients was higher (mean=9.9fl) than non diabetic subjects (mean=9.0fl) Figure 1. This difference was statistically significant (P= 0.015,). Figure 2 demonstrates that diabetics accumulate on the higher reference values of MPV while non diabetics at lower one.

Figure 1

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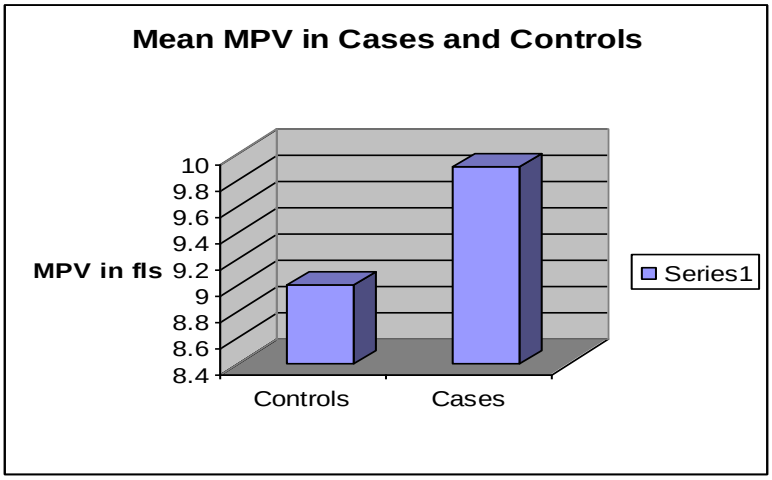
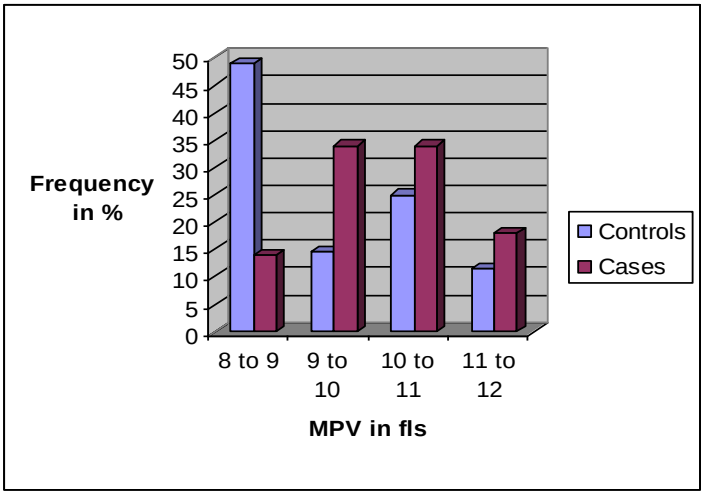


Figure 2



Frequency Distribution of Controls and Cases on Normal Ranges of MPV

Discussion

The burden of cardiovascular morbidity and mortality associated with diabetes is substantial and is likely to steadily increase, because the prevalence of diabetes is estimated to double during the first quarter of the 21st century.[16] Despite control for other risk factors, individuals with diabetes have a 3 to 5 fold greater risk of developing coronary artery disease than non-diabetics.[17] Several experimental and clinical studies have demonstrated that platelet size and function correlate since large platelets are hemostatically more reactive than platelets of normal size.[18] In vitro and ex vivo studies have demonstrated that diabetic patients have increased platelet aggregation.[19] Diabetics have larger platelets, an increased number of GP IIb/IIIa receptors on each platelet and an increased population of activated circulating platelets.[12] Large platelets express among other substances, P-selectin and thrombospondin.[23] These adhesion molecules mediate platelet-leukocyte interactions and therefore are potential triggers of inflammatory response and thrombosis.[13] The widespread availability of particle counters in the clinical laboratory now permits the accurate measurements of MPV on a routine practice.[20]

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There is strong evidence indicating that MPV is an important biological variable and that large platelets have a higher thrombotic potential.[21] Large platelets are metabolically and enzymatically more active than small platelets as assessed by ex vivo aggregometry. Eldor et al found that patients with hemorrhage and thrombocytopenia associated with a high MPV have a lower frequency of bleeding episodes than patients with both thrombocytopenia and a low MPV.[22]

Our study compared the MPV in type II diabetic patients with that of non diabetic subjects. The results revealed that the MPV was significantly raised among diabetic patients. This is in agreement with the literature reports. [12] [13] [14] [15]

Conclusion The elevation of MPV in type II diabetes mellitus may reflect the state of platelet hyperactivity that may play an important role in the vaso-occlusive complications of type II DM.

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