



Effects of Cigarette Smoking on White Blood Cells Count and von Willebrand Factor Levels in Male Smokers in Khartoum State

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Abstract

Background: Tobacco cigarette smoking is one of the major leading causes of death and essential public health challenge in world over. Elevated von Willebrand factor (vWF) concentrations are associated with an increased risk of ischemic heart disease. High total leukocyte count can promote cardiovascular diseases through multiple pathologic mechanisms, and has been considered as an independent predictor of atherosclerosis and cardiovascular disease. **Objective:** To assess the effects of cigarette smoking on White blood cells and von Willebrand factor levels in apparently healthy male smokers. **Materials and Methods:** One hundred subjects were included in this study: 50 were Sudanese male smokers, and the other 50 were controls. All subjects were evaluated to determine the effects of cigarette smoking on White blood cells and von Willebrand factor levels. The TWBCs was determined using Sysmex® Kx21-N Analyzer. vWF level was measured using ELISA method. **Results:** vWF was significantly higher in male smokers compared to non-smokers ($p = 0.000$). Total White Blood cell (TWBC) count was also higher among smoker than nonsmokers ($P = 0.000$). There were significant correlations between TWBC count and vWF levels with cigarette consumption/day and the duration of smoking. **Conclusion:** Our results concluded that smoking increases the white blood cell count and von Willebrand factor levels in males.

Subject Areas

Hematology

Keywords

Sudanese, Smoking, TWBCs, vWF

1. Introduction

Tobacco cigarette smoking is one of the major leading causes of death and essential public health challenge in world over [1] [2]. Smoking has both acute and chronic effect on hematological parameters. There are more than 4000 chemicals found in cigarette smoke [3], and a cigarette smoker is exposed to several harmful substances including nicotine, free radicals, carbon monoxide and other gaseous products [4]. A strong correlation has been found between cigarette smoking and atherosclerosis and cardiovascular disease [5]. Several studies have considered endothelial injury to be a key initiating event in the pathogenesis of cardiovascular disorders [6]. However, the precise pathophysiology of the adverse effects of smoking on endothelium is not very clear. Smoking has also been shown to influence levels of von Willebrand factor protein (vWF). vWF synthesized and stored in endothelial cells and megakaryocytes, has been reported to be a useful marker for endothelial cell damage [7]. This protein, which acts as a carrier and stabilizer for factor VIII, was reported to facilitate platelet aggregation and adhesion to the sub endothelium of an injured vessel wall [8].

vWF is a multimeric glycoprotein that plays an important role in primary haemostasis by promoting platelet adhesion to the sub endothelium at sites of vascular injury under high shear-rate conditions. It is also a carrier of FVIII and this association protects FVIII from rapid proteolysis. vWF is synthesized by endothelial cells, megakaryocytes and platelets. In endothelial cells vWF may be secreted directly into the circulation or stored in Weibel-Palade bodies. The vWF produced in megakaryocytes and platelets is not secreted but stored in alpha-granules. Release of VWF from these stores occurs following activation of endothelial cells or platelets. Some therapeutic products and conditions, may work to stimulate the release of stored vWF [9]. Elevated vWF concentrations are associated with an increased risk of ischemic heart disease [10]. A sufficiently low level of vWF predisposes to bleeding that can be quite serious, and low vWF is a diagnostic feature of von Willebrand disease (VWD) type 1, which is characterized by partial quantitative deficiency of vWF [11]. While leukocytosis may simply be a marker of smoking-induced tissue damage, the high count can promote cardiovascular diseases through multiple pathologic mechanisms that mediate inflammation, plug the microvasculature, induce hypercoagulability and promote infarct expansion [12] [13]. In fact, several studies have shown that WBC count is an independent predictor of atherosclerosis and cardiovascular disease [12] [13]. The aim of this study was to assess the effects of cigarette smoking on White blood cells and von vWF factor levels in apparently healthy male smokers.

2. Materials and Methods

This study is a case-control study, conducted in Khartoum, Sudan. One hundred subjects were included in this study, all of them were males, 50 were smokers and the other 50 were apparently healthy age matched nonsmokers (control

group). Smokers were classified into 3 groups according to the number of cigarettes smoked per day: mild smokers, smoked 1 to 10 cigarette/day; Moderate smokers, smoked 11 to 20 cigarette/day; heavy smokers, smoked more than 20 cigarette/day). Subjects were evaluated to determine the changes in vWF levels and TWBCS.

3 ml of venous blood was collected from each subject. 2 ml in 3.8% trisodium citrate (9:1 vol/vol), kept on ice until centrifugation at 2500 g for 30 minutes at 4°C, plasma samples were immediately frozen and stored at -80°C for subsequent analysis; and 1 ml in EDTA for the ABO blood grouping. Laboratory analysis was performed at the Department of Haematology, Faculty of Medical Laboratory Sciences, Alneelain University.

White cells count was determined by automated cell counter (Sysmex® Kx21-N). Estimation of vWF was carried out using Enzyme Linked Immunosorbent Assay (ELISA), method with kit obtained from Technoclone GmbH Brunner str 67 1230, Vienna Austria (Lot 3015983-014). Statistical analysis was performed using statistical package for social science (SPSS) software. Evaluation of patient's data was performed using the t-test and Pearson correlation test. Results with P value < 0.05 were considered statistically significant. This study was approved by ethical committee of ministry of health, and informed consent was obtained from each participant before sample collection.

3. Results

In a total of one hundred subjects included in this study, all of them were Sudanese males, 50 were smokers and the other 50 were apparently healthy controls. The distribution of heavy, moderate and mild smokers in smokers group were 10 (20%), 9 (18%) and 31 (62) respectively.

The present study showed that the TWBCs (Mean \pm SD) was found to be 8.24 ± 2.56 and 6.03 ± 2.07 in smokers and nonsmokers respectively. TWBCs was found to be significantly higher in smoker group than nonsmokers ($P = 0.000$). The vWF Mean \pm SD was found to be 0.96 ± 0.40 and 0.64 ± 0.21 in smokers and nonsmokers respectively. vWF was found to be significantly higher in smoker group than nonsmoker groups ($P = 0.000$) as showed in **Table 1**.

Table 2 showed the comparison of mean TWBC count and mean vWF among different smoker groups (mild, moderate and heavy) and non-smoker group.

Table 3 showed that there is strong relationship between TWBC and vWF with cigarette consumption/day and the duration of smoking (Year).

Table 1. Comparing of TWBCs and vWF between smoker and nonsmoker groups.

Parameters	Grouping	N	Mean \pm SD	P-value
T.WBC	Smoker	50	8.24 ± 2.56	0.000
	Non-Smoker	50	6.03 ± 2.07	
vWF	Smoker	50	0.96 ± 0.40	0.000
	Non-Smoker	50	0.64 ± 0.21	

Table 2. Comparison of TWBC count and vWF levels between different groups of smoker and nonsmoker group.

Variables	Group	N (%)	Mean \pm SD	P-value
T.WBC	Heavy smoker	10 (20%)	8.48 \pm 1.75	0.004
	Moderate smoker	9 (18%)	7.28 \pm 2.04	0.033
	Mild smoker	31 (62)	6.15 \pm 2.16	0.994
	Nonsmoker	50	6.03 \pm 2.00	
vWF	Heavy smoker	10 (20%)	0.95 \pm 0.40	0.001
	Moderate smoker	9 (18%)	0.71 \pm 0.13	0.773
	Mild smoker	31 (62)	0.68 \pm 0.25	0.745
	Nonsmoker	50	0.63 \pm 0.19	

Table 3. Correlations between TWBCS, vWF and duration of smoking and cigarette consumption.

Parameters		T.WBC	vWF	Duration of smoking	cigarette consumption/day
T.WBC	R-value		0.293	0.389	0.462
	P-value		0.044	0.005	0.001
vWF	R-value	0.293		0.295	0.387
	P-value	0.044		0.037	0.006
Duration of smoking	R-value	0.389	0.295		-0.276
	P-value	0.005	0.037		0.085
Cigarette consumption/day	R-value	0.462	0.387	-0.276	
	P-value	0.001	0.006	0.085	

4. Discussion

Cigarette smoking is one of the major leading causes of death and essential public health problem in world over. We study the effects of cigarette smoking on the TWBC count and vWF levels. The study included 50 Sudanese smokers; their TWBC count and vWF levels were measured and compared with 50 age matched non-smokers as control. We observed a significant increase in the mean of the TWBC count. Our finding is consistent with other published reports [14] [15] [16]. Several studies have shown that WBC count is an independent predictor of atherosclerosis and cardiovascular disease [12] [13]. The high WBC count in our smoking subjects may suggest that they might be at greater risk for developing cardiovascular diseases than nonsmokers.

Mean vWF was significantly higher in smokers compared to nonsmokers, as previously reported [17]. The high levels observed in smoker group imply injury to the endothelium. It has been proposed that the increased level of vWF among smokers may be due to cytotoxic effects of lipid peroxidase formed by oxygen free radicals, and the effects of nicotine and carbon monoxide [14] [18] [19].

Increased TWBC count and vWF levels were significantly associated with the number of cigarettes smoked per day, and the duration of smoking, indicating that the association of the smoking with the increased TWBC count and vWF

levels is a dose-dependent.

5. Conclusion

This study concluded that smoking potentially increases TWBC count and vWF level. Increased TWBC count and vWF level in the smokers is dose-dependent, which is directly associated with the smoking duration and frequency.

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