



RESEARCH ARTICLE

THE EFFECT OF CIGARETTE SMOKING AND SMOKING CESSATION ON FIBRINOGEN
LEVEL IN SUDAN

¹Hozeifa H. E. Abdalla and ^{2*}Mahdi H. A. Abdalla

¹Department of Hematology and Blood Transfusion, Faculty of Medical Laboratory Sciences,
Al Neelain University, Sudan

²Department of Hematology, Faculty of Health Sciences - Omdurman Ahlia University, Sudan

ARTICLE INFO

Article History:

Received 06th January, 2013
Received in revised form
14th February, 2014
Accepted 26th March, 2014
Published online 23rd April, 2014

Key words:

Cigarette smoking,
Smoking cessation,
Fibrinogen level,
Sudan.

ABSTRACT

Cigarette smoking is one of the major health hazards, and it contributes significantly to cardiovascular morbidity and mortality. The aim of this study was to determine the effect of smoking and smoking cessation on fibrinogen level among Sudanese population. 150 adult men aged 19-54 years old were enrolled (50 cigarette smokers, 50 Ex-smokers and 50 non-smokers). Citrated blood samples were collected, plasma were separated and stored at -80 °C, and fibrinogen levels were measured by Clauss method. Fibrinogen levels were significantly higher among smokers group compared to non-smokers group (P<0.000) and smoking cessation group (p 0.000). Increase fibrinogen levels were significantly associated with the duration of cigarettes smoking (p value 0.000), and the number of cigarette smoked per day (p value 0.000). In conclusion, smoking potentially increases fibrinogen level. Our results suggest that the effect of smoking on fibrinogen level is reversible with smoking cessation. Increased fibrinogen level in the smokers is dose-dependent, that is directly associated with the smoking duration and frequency.

Copyright ©2014 Hozeifa H. E. Abdalla and Mahdi H. A. Abdalla. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

Cigarette smoking is a serious health problem and most important avoidable causes of death in worldwide (Kume *et al.*, 2009; Islam *et al.*, 2007). There are more than 4000 chemicals found in cigarette smoke (Green *et al.*, 1996), and a cigarette smoker is exposed to a number of harmful substances including nicotine, free radicals, carbon monoxide and other gaseous products (Gitte *et al.*, 2011). Smokers are known to be associated with high risk for cardiovascular diseases, hypertension, inflammation, stroke, clotting disorder, and respiratory disease (Abel *et al.*, 2005; Tiel *et al.*, 2002). Cigarette smoking increases the risk of cardiovascular disease through many mechanisms including haemostatic disturbances (Meade *et al.*, 1987; Barua *et al.*, 2002). Inflammation is another possible mechanism for the increased risk of cardiovascular disease in smokers (Frohlich *et al.*, 2003). Fibrinogen is the major plasma protein coagulation factor. Low plasma fibrinogen concentrations are associated with an increased risk of bleeding due to an impaired primary and secondary hemostasis; higher plasma fibrinogen concentrations are associated with cardiovascular diseases (Low *et al.*, 2004). Fibrinogen also considered as an important contributor in the development of coronary, peripheral and cerebral vascular disease due to its involvement in both

atherogenesis and thrombosis (Ernst *et al.*, 1993; Koenig *et al.*, 1992). Moreover it is an acute phase reactant and thus increased plasma fibrinogen concentration may also be a marker of the inflammation associated with the atherosclerotic process (Pearson *et al.*, 2003; Danesh *et al.*, 2004). An association between smoking and inflammatory markers such as C-reactive protein, white cell count, fibrinogen, and albumin has been previously reported (Pradhan *et al.*, 2002). This study aimed to determine the effect of cigarette smoking and smoking cessation on the fibrinogen level in Sudan.

MATERIALS AND METHODS

Following informed consent, one hundred and fifty apparently healthy individuals were enrolled; 50 cigarette smokers; 50 Ex-smokers; and 50 non-smokers as control. Two ml of venous Blood samples were collected in 3.8% trisodium citrate (9:1 vol/vol) and kept on ice until centrifugation at 2500g for 30 minutes at 4°C. Plasma samples were immediately frozen and stored at -80°C for subsequent analysis. Laboratory analysis was performed at the Department of Haematology, Faculty of Medical Laboratory Sciences, Alneelain University, Sudan. Fibrinogen level was measured by Clauss modified method using a test kit produced by (TECHNOCLONE GMBH, AUSTRIA). The method uses a functional assay based upon the time for fibrin clot formation, in brief, Diluted plasma is clotted with a high concentration of thrombin, and the concentration of fibrinogen is determined by comparing the

*Corresponding author: Mahdi H. A. Abdalla, Department of Hematology, Faculty of Health Sciences - Omdurman Ahlia University, Sudan.

plasma clotting time to a calibration curve of a reference plasma with a series of dilutions (1:5 –1:40). 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.

RESULTS

All participants were male. Fifty smokers were enrolled, the median age of smokers was 28 years with minimum age of 19 years and maximum age of 54 years, mean number of cigarettes smoked per day was 16.2 ± 15.6 cigarettes (minimum of 5 and maximum of 30 cigarettes), mean duration of smoking was 10.6 ± 16.6 years (minimum of 1 and maximum of 30 years); fifty age matched Ex-smokers were enrolled, mean duration of smoking cessation was 8.3 ± 15.8 years; and fifty age matched non-smokers with no history of smoking were enrolled as controls. Fibrinogen level was determined in all participants, means fibrinogen levels were as follows: 421 ± 80 mg/dl for the smokers group; 324 ± 95 mg/dl for the smoking cessation group; and 270 ± 83 mg/dl for the non-smokers group (Figure 1).

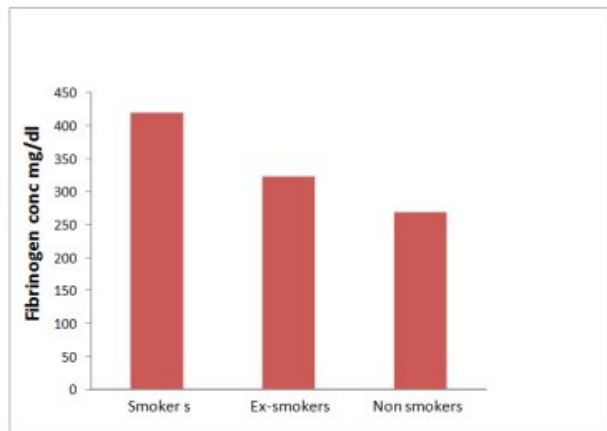


Fig. 1. Fibrinogen levels among smokers, smoking cessation and non-smokers

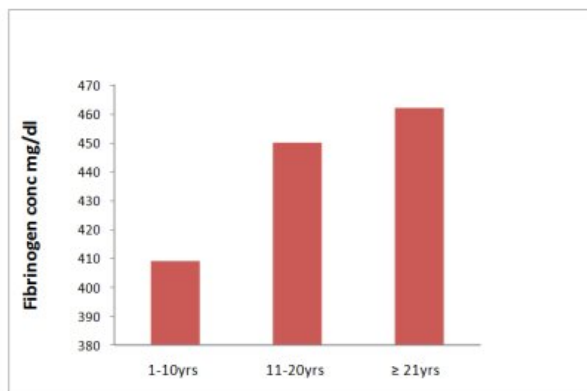


Fig. 2. Association of smoking duration with fibrinogen level

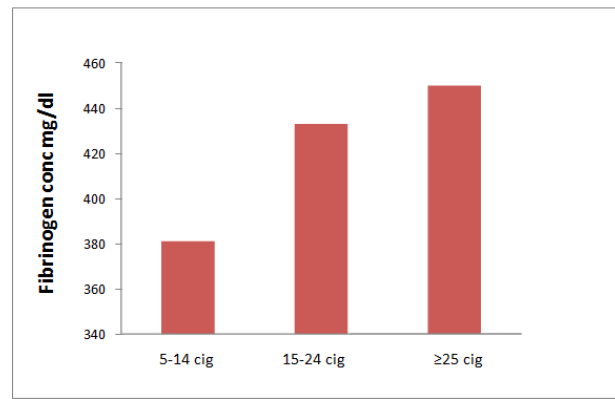


Fig. 3. Association of cigarette number with fibrinogen level

Mean fibrinogen level was significantly higher in smokers group, when compared with non-smokers group (p value 0.000), and smoking cessation group (p value 0.000), it was also higher in Ex-smokers group when compared with non-smokers group (p value 0.000). Increase fibrinogen levels were significantly associated with the duration of the cigarettes smoking (p value 0.000), and also significantly associated with the number of cigarette smoked per day (p value 0.000). Figures 2 and 3 show the association of the fibrinogen levels with the duration of cigarettes smoking (when classified into three groups; 1-10, 11-20, and ≥ 20 years), and with the number of cigarettes smoked per day (when classified into three groups; 5-14, 15-24, and ≥ 25 cigarettes) respectively.

DISCUSSION

Cigarette smoking is one of the major leading causes of death and essential public health problem in world over (Kume *et al.*, 2009; Islam *et al.*, 2007). In this study we utilized a quantitative approach for the determination of fibrinogen level. The study included 50 Sudanese smokers, their fibrinogen levels were measured and compared with 50 age matched Ex-smokers and 50 non-smokers as control. We observed a significant increase in the mean of the fibrinogen level among smokers, when compared with the control group and the smoking cessation group. Similar findings in previous studies have been reported (Subratty *et al.*, 1990; Rashmi *et al.*, 2011) Increased fibrinogen levels among smokers may promote a hypercoagulable state, and may in part explain the association of smoking with high risk for cardiovascular diseases, hypertension, stroke and clotting disorder. The effects of smoking on fibrinogen synthesis may be a part of a generalized inflammatory reaction, as smoking is strongly related to other measures of inflammation (Pearson *et al.*, 2003; Danesh *et al.*, 2004). Our finding confirms that smoking potentially increases fibrinogen level. Fibrinogen level was significantly decreased among smoking cessation group, when compared with smokers group, indicating that the effect of smoking on fibrinogen level is reversible with smoking cessation. Kirsty *et al.* concluded that abstention from smoking for a period of only 2 weeks induces a significant decrease in the rate of fibrinogen synthesis by the liver, with a concomitant reduction in the plasma fibrinogen concentration (Kirsty *et al.*, 2001). Increased fibrinogen 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 level of fibrinogen is a dose-dependent.

Conclusion

This study confirmed that smoking potentially increases fibrinogen level; the effect of smoking on fibrinogen level is reversible with smoking cessation. Increased fibrinogen level in the smokers is dose-dependent, that is directly associated with the smoking duration and frequency.

Authors contributions

H.H.E. Abdalla and M.H.A. Abdalla conceived the idea of the study, collected and analyzed samples and data and wrote the manuscript.

Acknowledgement

Special thanks to the Staff of Haematology Department, Faculty of Medical Laboratory Sciences, Alneelain University.

REFERENCES

- Abel GA, Hays JT, Decker PA, Croghan GA, Kuter D J, Rigotti NA. 2005. Effects of biochemically confirmed smoking cessation on white blood cell count. *Mayo Clin Proc.*, 80:1022-1028.
- Daice J.V and Lewis S.M. 2001. Practical Haematology, 9th edition, p 350.
- Danesh J, Wheeler JG, Hirschfield GM, Eda S, *et al.* 2004. C-reactive protein and other circulating markers of inflammation in the prediction of coronary heart disease. *N Engl J Me.*, 350: 1387–1397.
- Ernst, E. 1993. The role of fibrinogen as a cardiovascular risk factor. *Atherosclerosis.*, 100: 1-12.
- Fogari R, Zoppi A, Tettamanti F, Malamani GD, *et al.* 1994. Fibrinogen levels in normotensive and hypertensive men: a cross-sectional study. *Source Journal of Cardiovascular Risk.*, 1: 149-53.
- Folsom A.R, Johnson K.M, Lando H.A, McGovern PG, *et al.* 1993. Plasma fibrinogen and other cardiovascular risk factors in urban American Indian smokers. *Ethnicity Disease.*, 3: 344-50.
- Gitte RN. 2011. Effect of cigarette smoking on plasma fibrinogen and platelet count. *Asian Journal of Medical Sciences.*, 2:181-184.
- Green CR, Rodgman A. 1996. The tobacco chemists' research conference: a half century forum for advances in analytical methodology of tobacco and its products. *Recent Adv Tobacco Sci.*, 22:131–304.
- Islam M.M. Amin M.R., Begum S, Akther D, Rahman A. 2007. Total count of white blood cells in adult male smokers. *J Bangladesh Soc Physiol.*, 2:49-53.
- Kirsty A. H, Peter J. G, Iain B, Susan E. and Margaret A. M. 2001. Effects of smoking and abstention from smoking on fibrinogen synthesis in humans. *Clinical Science.*, 100, 459–465.
- Kume A, Kume T, Masuda K, Shibuya F, Yamzaki H. 2009. Dose-dependent effect of cigarette smoke on blood biomarkers in healthy volunteers: Observations from smoking and non-smoking. *Journal of Health Sciences.*, 55:259-264.
- Low GD, Rumley A, Mackie IJ. 2004. Plasma fibrinogen. *Ann Clin Biochem.* 4:430-40.
- Maat, M.P.M. de (Moniek). 1995. Regulation and modulation of the plasma fibrinogen level. Erasmus MC: *University Medical Center Rotterdam.*, pp.11-12.
- Paola P, Emanuela F, Sunjai G, Michael G, *et al.* 2001. Association Between Smoking and Blood Pressure: Evidence From the Health Survey for England. *American Heart Association.*, 37:187-193.
- Pearson TA, Mensah GA, Alexander RW, *et al.* 2003. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation.*, 107:499–511.
- Scholes D, Daling J R , and Stergachis AS. 1992. Current cigarette smoking and risk of acute pelvic inflammatory disease. *Am J Public Health.*, 82: 1352–1355.
- Seyyed H.S, MD; Ali A, *et al.* 2004. A Comparative Study of Plasma Fibrinogen among Hookah Smokers, Cigarette Smokers and Non-Smokers. *Iranian Heart Journal.*, 5: 48-54.
- Takahashi S, Imaki M, Yoshida Y, Ogawa Y, Tanada S. 2000. A cross-sectional study on the relationship of plasma fibrinogen concentration and age, physical fitness, lifestyle and health examinations of healthy Japanese males. *Japanese Journal of Hygiene.*, 55: 508-15.
- Van Tiel E, Peeters PH, Smit HA, Naqelkerke NJ, *et al.* 2002. Quitting smoking may restore hematological characteristics within five years. *Ann Epidemiol.*, 12:378-388.
