

Available Online at http://www.journalajst.com

ASIAN JOURNAL OF SCIENCE AND TECHNOLOGY

Asian Journal of Science and Technology Vol. 6, Issue 02, pp. 1058-1061, February, 2015

RESEARCH ARTICLE

ASSESSMENT OF THE EFFECT OF CIGARETTE SMOKING ON SOME COAGULATION PARAMETERS IN SOKOTO NORTH WESTERN NIGERIA

¹Isah, I. Z., ¹Abdulrahaman, Y., ¹Okwesili, A. N., ²Ikhuenbor, D. B., ²Aghedo, F., ¹Ibrahim, M., ¹Onuigwe, F. U., ¹Buhari, H., ¹Ibrahim, K. and ^{*1}Erhabor, O.

¹Department of Haematology and Blood Transfusion Science, Faculty of Medical Laboratory Science, Usmanu Danfodiyo University, Sokoto State, Nigeria

²Department of Haematology and Blood Transfusion Science, Usmanu Danfodiyo University Teaching Hospital,

Sokoto State, Nigeria

ARTICLE INFO

ABSTRACT

Article History: Received 01st November, 2014 Received in revised form 20th December, 2014 Accepted 11th January, 2015 Published online 28th February, 2015

Key words:

Cigarette smoking, Coagulation parameters, Sokoto, Nigeria.

Objectives: Cigarette smoking is a global public health problem. Worldwide, cigarette smoking has serious health, economic and social implications. There are about 1300 million smokers globally and about 75 of these are in the developing countries. The aim of this present study was to determine the effect of cigarette smoking on some coagulation parameters of smokers in Sokoto, Nigeria. Method: This present study was a case-control study conducted among 100 smokers (subjects) and 100 non-smokers (controls) aged 18-60 years with mean age of 26 ± 6.23 years. Manual method was used for the determination of Prothrombin Time (PT) and Partial Thromboplastin Time with Kaolin (PTTK). Results: The mean PTTK and PT was significantly lower among cigarette smokers compared to nonsmokers (P = 0.001). We observed a negative and significant correlation between PT and PTTK and increased duration of cigarette smoking (r=- 0.58 and - 0.62 respectively, p=0.003). The PTTK of light, moderate and heavy smokers were 34.6, 29.0 and 28.0 seconds respectively while the PT results were 14.8, 11.0 and 8.8 seconds respectively. There was no significant difference in PT and PTTK based on age groups of the subject (P = 0.06 and 0.10 respectively). The effect of duration of smoking on the PT and PTTK indicated that smokers who have smoked for 1-10, 11-20 and \geq 21 years had PT and PTTK values of 12.3 ± 2.9 , 10.3 ± 1.9 and 8.9 ± 0.9 versus 31.7 ± 7.9 , 29.3 ± 6.5 and 26.2 ± 6.2 respectively. Conclusion: Our findings indicates that PT and PTTK of cigarette smokers is significantly higher than those of non-smokers. We observed a negative and significant correlation between PT and PTTK and duration of cigarette smoking. Chronic cigarette smoking was associated with a reduction in the PT and PTTK and may predispose smokers to increased risk of thrombosis. There is need for increased awareness of the negative effects of cigarette smoking on health. We advocate for a tighter tobacco legislation which should also force companies to print graphic health warnings on their cigarette packs to obviate the detrimental consequences of associated atherothrombotic disease, cardiovascular pathology and other smoking-related diseases.

Copyright © 2015 Isah et al. 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

The WHO has estimated that there are about 1300 million smokers globally and about 75 of these are in the developing countries. It is also predicted that if the current pattern of smoking continues, by 2020, there will be 10 million tobaccorelated deaths annually worldwide and seven million (70%) of these deaths will occur in the developing countries (Nwafor et al., 2012). Tobacco use is the single most significant cause of preventable death worldwide. The World Health Organization (WHO) estimates that tobacco caused 5.4 million deaths in

*Corresponding author: Erhabor. O.

2004 and 100 million deaths over the course of the 20th century (Ezzati and Lopez, 2004). An estimated five million people died from tobacco use in 2000, representing about 12% of adult deaths. Of these, about 30% (1.42 million deaths) resulted from cancer alone (Thun et al., 2000). Tobacco smoking is a growing public health problem in Nigeria (Fawibe and Shittu, 2011). Cigarette smoking poses a major challenge not just to the health of Nigerians, but also to socioeconomic development and environmental sustainability (Ibeh and Ele, 2003 and Omokhodion and Faseru, 2007). International Agency for Research on Cancer (IARC) has identified and listed about 4000 chemical agents as potential carcinogens present in cigarette (Hoffmann et al., 2001). Smoke also contains several carcinogenic pyloric products that bind to DNA and cause many genetic mutations. It also

Department of Haematology and Blood Transfusion Science, Faculty of Medical Laboratory Science, Usmanu Danfodiyo University, Sokoto State, Nigeria

contains nicotine, which is a highly addictive psychoactive drug and it contributes significantly to cardiovascular-related morbidity and mortality (Ambrose and Barua, 2004). The thrombotic risks associated with cigarette use has been reported in a previous study (Bazzano *et al.*, 2003). While many people are aware of the negative effect of cigarette smoking on the lungs, less consideration is given to its effect on haemostasis. There appear to be a paucity of information on the effect of cigarette smoking on the coagulation parameters of smokers in the Sokoto, Nigeria. Previous report in the area has focussed on the socio-economic effect of cigarette smoking. The aim of this study was to determine the effect of cigarette smoking on some coagulation parameters among cigarette smoker in Sokoto, North Western Nigeria.

Study area

September).

The present study was carried out in the Usmanu Danfodiyo University Teaching Hospital Sokoto (UDUTH) in North Western Nigeria. The hospital was established in the year 1975 in Sokoto metropolis. It is committed to the provision of a continually improving quality tertiary health care services to the entire North-Western region of Nigeria and the neighbouring border country - Niger Republic. The metropolitan city of Sokoto covers an area of 28,232.37sq kilometre. It is bordered in the North by Niger republic, in the East by Zamfara state and Kebbi state to the South and West. Sokoto is one of the hottest cities in the world with an annual average temperature of 28.3C. The warmest months are February to April (temperatures exceed 45C) while the rainy season lasts from June to October and Harmmattan season starts from late October to February. There are two main seasons in Sokoto, the wet (October to April) and dry (May to

The main occupation of the people is grain production and animal husbandry. More than 80 percent of its indigenes practice agriculture. Crops produced include millet, beans, onions, tomatoes, rice, maize, guinea corn, wheat and cotton. Other occupations commonly practiced are dyeing, blacksmithing, weaving, carving, trading and cobbling. Sokoto ranks second in livestock production in Nigeria. Report from the 2007 National Population Commission indicated that the state had a population of 3.6 million people (NPC, 2007). Sokoto is made up of predominantly Hausa and Fulani majority and a minority of Zabarmawa and Tuareg. The major language in this state is Hausa and Fulfulde among the Fulani. They have two major festivals Eid - el - Fitri and Eid-el-Kabir. Socio cultural characteristics is homogenous as majority of its indigenes are Muslims, therefore the Muslim religion provides the code of conduct and behavioural characteristics. Common practices include; consanguity, child marriage, polygamy, multiple births and male dominance.

Inclusion Criteria

Inclusion criteria included; age ≥ 18 years, non-alcoholics, male gender, absence of diseases thought to have a significant effect on coagulation, non-long term medication use and willingness to offer informed consent to participate in this study.

Exclusion Criteria

Exclusion criteria included; age less than 18 years, female gender, presence of diseases thought to have a significant effect on coagulation, history of alcoholism, long- term medication use, smoking of marijuana and failure to offer informed consent to participate in this study.

MATERIALS AND METHODS

A total of one hundred adult male subjects aged 18 - 60 years and resident in Sokoto metropolis were recruited as subjects for this case-control study. Subjects were categorised into 3 groups; heavy smokers (39), moderate smokers (33) and light smokers (28). One hundred gender and age matched noncigarette smokers serving as control. Semi-structured questionnaires were used in the selection of suitable subjects. For the purpose of this study, a heavy smokers (H) is one that smokes at least 10sticks of cigarette per day, moderate (M) smokers is one who smokes 5- 10sticks of cigarettes per day and light smokers is one who smokes 1-5 sticks of cigarette per day (Loprinzi et al., 2013). Three millilitres (3mls) of blood was collected under aseptic condition by venepuncture into a test tube containing 0.5mls of 32g/l sodium citrate. The sample collected was properly mixed with the anticoagulant to prevent clotting. Sample was centrifuged at 1000rpm for 5 minutes. Citrated plasma was separated and used for the manual testing using PT and PTTK reagents (Diagnostic Reagents Ltd, UK). The manufacturer's standard operating procedure was followed strictly in the performance of the test.

Statistics Analysis

Statistical analysis were conducted using SPSS (version 11) software. Comparisons were assessed using mean and chi-square test. A p-value of ≤ 0.05 was considered statistically significant in all statistical comparison. Correlation was compared using a version of linear regression analysis.

RESULTS

The mean PTTK and PT was significantly lower among cigarette smokers compared to non-smokers (P = 0.001) (Table 1). We observed a negative and significant correlation between PT and PTTK and increased duration of cigarette smoking (r=- 0.58, - 0.62 respectively, p=0.003) (Table 2). The PTTK of light, moderate and heavy smokers were 34.6s, 29.0s and 28.0s respectively while the PT were 14.8s, 11.0s and 8.8s respectively.

Table 1. Partial Thromboplastin Time with Kaolin (PTTK) and Prothrombin Time (PT) of cigarette smokers compared with nonsmokers

Parameters	Ν	PTTK (s)	PT (s)
Smokers	100	30.3 ± 7.2	11.3 ± 2.8
Non-smokers	100	36.5 ± 3.7	14.8 ± 1.2
P -value		0.001	0.001
T- value		3.29	1.96

 Table 2. The PTTK and PT values of cigarette smokers based on the number of cigarettes smoked/day

Categories of smokers	Ν	PTTK	PT
LSM	28	34.6 ± 5.9	14.8 ± 2.1
MSM	33	29.0 ± 6.7	11.0 ± 1.5
HSM	39	28.0 ± 7.2	8.8 ± 0.7
P- values		0.002	0.001

Key: HSM - Heavy smokers (smokes at least 10sticks/day), MSM - Moderate smokers (smokes 5- 10sticks/day) and LSM - Light smokers (smokes 1-5 sticks/day).

There was no significant difference in PT and PTTK among age groups of the subject (P = 0.06) (Table 3). The effect of duration of smoking among cigarette smokers on the PT and PTTK indicated that smokers who have smoked for 1-10, 11-20 and ≥ 21 years had PT and PTTK values of 12.3 ± 2.9 , 10.3 ± 1.9 and 8.9 ± 0.9 and 31.7 ± 7.9 , 29.3 ± 6.5 , 26.2 ± 6.2 respectively. Table 4 show the effect of duration of smoking on PTTK and PT values of cigarette smokers.

 Table 3. The PTTK and PT values of cigarette smokers among age groups in years

Age(years)	n	PTTK (s)	PT (s)
18-25	48	34.2 ± 6.0	13.5 ± 2.6
26-40	34	31.7 ± 6.9	12.7 ± 2.7
41-60	18	34.1 ± 6.4	12.6 ± 3.0
P value		0.05	0.108

 Table 4. The effect of duration of smoking on PTTK and PT

 values of cigarette smokers

Duration (Years)	n	PTTK (s)	PT (s)	
1-10	60	31.7 ± 7.9	12.3 ± 2.9	
11-20	26	29.3 ± 6.5	10.3 ± 1.9	
≥ 21	14	26.2 ± 6.2	8.9 ± 0.9	
P – value		0.026	0.001	

DISCUSSION

In this present study, we investigated the effect of cigarette smoking on some coagulation parameters (PT and PTTK) among adult males in Sokoto, North Western Nigeria. We observed that the PT and PTTK of smokers was significantly lower compared with those of non-smokers. This observation is in line with previous report which showed that continuous cigarette smoking has severe adverse effects on coagulation parameters; haemoglobin, haematocrit, TLC count, RBC count and platelet count and that these alterations might be associated with a greater risk for developing atherosclerosis, polycythaemia vera, chronic obstructive pulmonary disease and cardiovascular diseases (Reinders et al., 1986 and Garry and Geraets, 2007). Smoking causes the development of a state of hypercoagulability, potentially leading to thrombosis. The precise mechanisms responsible for the effect of cigarate smoking on the haemostatic function is thought to involve multiple mechanisms; exacerbation of platelet adhesiveness and aggregation, decrease in plasminogen activation and increase in fibrinogen levels (Frayn et al., 1996). Cigarette related alteration of the function of endothelial cells and coagulation factors appears to also play a central role (Barua and Ambrose, 2013 and Csordas and Bernhard, 2013). We observed that increased duration of cigarette smoking was associated with a significant reduction in the PT and PTTK

and may predispose smokers to increased risk of thrombosis. The long-term cigarette risk may be due to cigarette smoking related increased plasma viscosity and increased synthesis of fibrinogen in plasma (Lee et al., 1996). Our finding is also in agreement with previous reports (Hioki et al., 2001 and Hunter et al., 2001) which indicated that prolonged cigarette in-take causes an increased amount of fibrinogen in plasma which could lead to hastened bleeding arrest, thrombosis or DIC. Previous report indicates that smoking cessation potentially improves fibrin clot architecture which results in the lesser resistance to lysis (Stepień et al., 2011). We observed that the PT and PTTK of chronic smokers was significantly lower compared to moderate and occasional smokers. This is consistent with report by Nascetti and colleagues (Nascetti et al., 2001) who argued that the decrease in PT, INR and PTTK values associated with cigarette smoking is proportional to the number of cigarette smoked per day. Cigarette smoking is associated with an increase of thrombocytes, hyperfibrinolysis and haemoconcentration (Voigt and Voigt, 1990).

Previous report indicates a strong relationship between cigarette smoking, haemostatic parameters and increased risk of cardiovascular events. Smoking influences negatively coagulation-fibrinolysis cascade. Cigarette smoking seems to modify haemostatic parameters via thrombosis with consequently more rate of cardiovascular events (Leone, 2007). It continues to be a major health hazard and contributes significantly to cardiovascular morbidity and mortality. Cigarette smoking has serious implications on all phases of atherosclerosis from endothelial dysfunction to acute clinical events, the latter being largely thrombotic (Ambrose and Barua, 2004). Cigarette smoke exposure promotes vasomotor dysfunction, atherogenesis, and thrombosis (Szyszka et al., 2009). We observed that the highest number of cigarette smokers in this study was in the 18-25 years age range. Our finding is consistent with previous reports in Nigeria which indicated that cigarette smoking was a significant public health problems among Nigerian youths (Nwafor et al., 2012; Thun et al., 2000; Fawibe and Shittu, 2011 and Ibeh and Ele, 2003). The implication of this finding is that cigarette smoking is still predominant among Nigerian youths. There is the need for governmental and non-governmental organizations (NGOs) to develop public enlightenment on the risk of cigarette smoking targeted at Nigerian youths.

Conclusion

This present study indicates that cigarette smoking has a significant effect on some coagulation parameters. Our findings indicates that PT and PTTK of cigarette smokers is significantly lower than those of non-smokers. We observed a negative and significant correlation between PT and PTTK and increased intake of cigarette. Chronic cigarette smoking and increased duration of cigarette smoking was associated with a reduction in the PT and PTTK and may predispose smokers to increased risk of thrombosis. There is need for increased awareness of the negative effects of cigarette smoking on health. There is the urgent need for tighter tobacco legislation which will ensure that tobacco companies print graphic health warnings on their cigarette packs to obviate the detrimental consequences of smoking-related diseases. Evidenced -based data from this study can be used by Federal Ministry of Health to improve national and state programs to prevent and control tobacco use. There is also the need to appropriate more funds for the funding of smoking cessation and prevention programs. We recommend that the Nigerian government should establish and maintain a comprehensive tobacco-control programs to reduce tobacco use among Nigerians. The Nigeria government can discourage the use of tobacco among her citizenry by banning advertisements that encourage smoking among the populace, the presentation of evidence- based data and true life stories to show the harmful effects of smoking, increases of taxes on cigarettes and the implementation of enforceable bans on smoking particularly in public places. There is also the need to encourage smokers to adopt healthier lifestyles and quit smoking so that future smoking –related negative consequences can be avoided.

Acknowledgments

We thank the smokers and non-smokers who participated in this case control study. We are also grateful to the staff of the Haematology Department of Usmanu Danfodiyo University Teaching Hospital Sokoto, Nigeria for their collaboration.

Conflict of Interest

The authors discloses no conflict of interest.

REFERENCES

- Nwafor, C.C., Ibeh, C.C., Aguwa, E.N. and Chukwu, J.N. 2012. Assessment of pattern of cigarette smoking and associated factors among male students in public secondary schools in Anambra State, Nigeria, *Niger J Med*, 21(1), 41-47.
- Ezzati, M. and Lopez, A.D. 2004. Regional, disease specific patterns of smoking-attributable mortality in 2000, *Tob Control*, 13(4), 388-395.
- Thun, M.J., Apicella, L.F. and Henley, S.J. 2000. Smoking vs other risk factors as the cause of smoking-attributable mortality: confounding in the courtroom, *JAMA*, 284, 706–712.
- Fawibe, A.E. and Shittu, A.O. 2011. Prevalence and characteristics of cigarette smokers among undergraduates of the University of Ilorin, Nigeria, *Niger J Clin Pract*, 14(2),201-205.
- Ibeh, C.C. and Ele, P.U. 2003. Prevalence of cigarette smoking in young Nigerian females, *Afr J Med Med Sci*, 32(4), 335-338.
- Omokhodion, F.O. and Faseru, B.O. 2007. Perception of cigarette smoking and advertisement among senior secondary school students in Ibadan, Southwestern Nigeria, *West Afr J Med*, 26(3), 206-209.
- Hoffmann, D., Hoffmann, I. and El-Bayoumy, K. 2001. The less harmful cigarette: A controversial issue. A tribute to Ernst L. Wynder, *Chem Res Toxicol*, 14,767–790.
- Ambrose, J.A. and Barua, R.S. 2004. The pathophysiology of cigarette smoking and cardiovascular disease: an update, J Am Coll Cardiol, 43(10), 1731-1737.

- Bazzano, L.A., He J., Muntner, P., *et al.* 2003. Relationship between cigarette smoking and novel risk factors for cardiovascular disease in the United States, *Ann Intern Med*, 138, 891–897.
- National Population Commission (NPC) 2007. National Census Figures, Abuja, Nigeria.
- Loprinzi, P.D., Walker, J.F., Kane, C. and Cardinal, B.J. 2013. Physical Activity Moderates the Association between Nicotine Dependence and Depression among U.S. Smokers, *Am J Health Promot*, 1539, 95-104.
- Reinders, J.H., Brinkman, H.J., van Mourik, J.A. and de Groot, P.G. 1986. Cigarette smoke impairs endothelial cell prostacyclin production, *Arteriosclerosis*, 6(1), 15-23.
- Garry, J. and Geraets, D.R. 2007. Effect of tobacco on INR, *Am J Health Syst Pharm*, 15, 64(8), 804-805.
- Frayn, K.N., Williams, C.M. and Arner, P. 1996. Are increased plasma non-esterified fatty acid concentrations a risk marker for coronary heart disease and other chronic diseases, *Clin Sci*, 90, 243-253.
- Barua, R.S. and Ambrose, J.A. 2013. Mechanisms of coronary thrombosis in cigarette smoke exposure, *Arterioscler Thromb Vasc Biol*, 33(7), 1460-1467.
- Csordas, A. and Bernhard, D. 2013. The biology behind the atherothrombotic effects of cigarette smoke, *Nat Rev Cardiol*, 10(4), 219-230.
- Lee, A.J., Fowkes, F.G.R., Rattray, A., Rumley, A. and Lowe, G.D.O. 1996. Haemostatic and rheological factors in intermittent claudication: the influence of smoking and extent of arterial disease, *Br J Haematol*, 92, 226–230.
- Hioki, H., Aoki, N., Kawano, K., Homori, M., Hasumura, Y., Yasumura, T., Maki, A., Yoshino, H., Yanagisawa, A. and Ishikawa, K. 2001. Acute effects of cigarette smoking on platelet-dependent thrombin generation, *Eur Heart J*, 22, 56–61.
- Hunter, K.A., Garlick, P.J., Broom, I., Anderson, S.E. and McNurlan, M. 2001. Effects of smoking and abstention from smoking on fibrinogen synthesis in humans, *Clin Sci*, 100, 459–465.
- Stępień, E., Miszalski-Jamka, T., Kapusta, P., Tylko, G. and Pasowicz, M. 2011. Beneficial effect of cigarette smoking cessation on fibrin clot properties, *J Thromb Thrombolysis*, 32(2), 177-182.
- Nascetti, S., Elosua, R., Pena, A., Covas, M.I., Senti, M., Marrugat, J. and REGICOR Investigators. 2001. Variables associated with fibrinogen in a population-based study: interaction between smoking and age on fibrinogen concentration, *Eur J Epidemiol.*, 17(10), 953-958.
- Voigt, T. and Voigt, H. 1990. Effect of smoking on haemostasis and haemorheology in pregnancy, *Zentralbl Gynakol*,112(9),529-533.
- Leone, A. 2007. Smoking, haemostatic factors, and cardiovascular risk, *Curr Pharm Des*, 13(16), 1661-1667.
- Ambrose, J.A. and Barua, R.S. 2004. The pathophysiology of cigarette smoking and cardiovascular disease: an update, J Am Coll Cardiol, 43(10), 1731-1737.
- Szyszka, A., Fałdyga, J. and Religa, L. 2009. Cigarette smoking and cardiovascular disease, *Przegl Lek*, 66(10), 873-874.