1. Introduction

1.1. Introduction:

Smoking is the most important public health problem. Many studies performed have proved its deleterious effects on many organ systems mainly respiratory, reticuloendothelial system and cardiovascular systems (Oztuna *et al; 2004*).

Tobaccos smoking mainly have two forms, either in form of cigar or in Narghile (Shisha) style.

Shisha, water pipe, Narghile is known by a number of different names, including argileh, goza, hookah, hubblebubble, and shisha. Its origin is often traced to India, although there are theories that it was first used in South Africa, Persia, Ethiopia, or other countries (Akl*et al; 2010*). The narghile pipe is usually decorated with floral or other motifs and has served as an artistic medium for craftsmen in many countries. Its use declined considerably during the past century and had principally become the habit of elderly and retired men who spent their time in bazaar cafes, particularly in poor areas. There has been a resurgence of water pipe smoking (WPS) in the past several years. This phenomenon has been attributed to the perception that WPS is less dangerous than cigarette smoking, its easy availability, its low cost, and a number of other factors. It is usually a social activity, engaged in by peer groups and families, and often practiced in special cafes. The expanding influence of Eastern and Arab cultures in the United States and Europe places WPS as a potentially important public health issue for adolescents in these countries (Al-Azzawy*et al; 2011*). It has been claimed that >100 million people worldwide smoke water pipes daily. It is a common practice in the Arabian Peninsula, Turkey, India, Pakistan, Bangladesh, and some regions of China (Grant*et al; 2013*). In some areas, WPS is more prevalent than cigarette smoking. Among Arab women in many countries, there is less of a stigma associated with narghile

than with cigarette smoking and therefore less of a gender differential (Maziak, *et al; 2004).*

Tobacco use leads most commonly to diseases affecting the heart and lungs, with smoking being a major risk factor for heart attacks, strokes, chronic obstructive pulmonary disease (COPD), emphysema, and cancer (particularly lung cancer, cancers of the larynx and mouth, and pancreatic cancer). It also causes peripheral vascular disease and hypertension, all developed due to the exposure time and the level of dosage of tobacco. Cigarettes sold in developing nations tend to have higher tar content, and are less likely to be filtered, potentially increasing vulnerability to tobacco-related disease in these regions.

Despite its widespread use, few studies to date have documented the adverse health consequences of WPS. This lack of data results from that WPS is mostly a non-Western habit, the high prevalence of smoking is a relatively recent phenomenon, lack of standardization of narghile content, and the difficulty in studying the isolated effects of narghile because most of the smokers are also current or past cigarette smokers.

Hookah smokers are at risk for the same kinds of diseases caused by cigarette smoking. These include oral cancer, lung cancer, stomach cancer, cancer of the esophagus, reduced lung function, and decreased fertility. Relative to a single cigarette, completed in about 5 minutes, a single waterpipe use episode typically lasts for about 1 hour (Brelan*det al; 2006*).

1.2. Literature review

1.2.1.Prevalence of smoking:

In 2000, smoking was practiced by 1.22 billion people, predicted to rise to 1.5 to 1.9 billion by 2025. As 2002, about twenty percent of young teens (Lowe *et al;*1992) and(Korenman, 2004) smoke worldwide, with 80,000 to 100,000 children taking up the habit every day—roughly half of whom live in Asia. Half of those who begin smoking in adolescent years are projected to go on to smoke for 15 to 20 years.The WHO states that "Much of the disease burden and premature mortality attributable to tobacco use disproportionately affect the poor". Of the 1.22 billion smokers, 1 billion of them live in developing or transitional nations. Rates of smoking have leveled off or declined in the developed world. In the developing world, however, tobacco consumption is rising by 3.4% per year as of 2002.

The WHO in 2004 projected 58.8 million deaths to occur globally, from which 5.4 million are tobacco-attributed, and 4.9 million as of 2007. As of 2002, 70% of the deaths are in developing countries, as of 2002, Approximately 5.5 trillion cigarettes are produced globally each year and are smoked by over 1.1 billion people or greater than one-sixth of the world population. While smoking rates have leveled off or declined in developed nations, they continue to rise in developing parts of the world. Smoking rates in the United States have dropped by half from 1965 to 2006 falling from 42% to 20.8% of adults. In the developing world, tobacco consumption is rising by 3.4% per year.

1.2.2. Cigarette smoking:

Tobacco use leads most commonly to diseases affecting the heart and lungs, with smoking being a major risk factor for heart attacks, strokes, chronic obstructive pulmonary disease (COPD), emphysema, and cancer (particularly lung cancer, cancers

of the larynx and mouth, and pancreatic cancer) (shihadeh, 2003). It also causes peripheral vascular disease and hypertension, all developed due to the exposure time and the level of dosage of tobacco. Furthermore, the earlier and the higher level of tar content in the tobacco filled cigarettes causes the greater risk of these diseases (McAuley*et al;* 2012). Cigarettes sold in developing nations tend to have higher tar content, and are less likely to be filtered, potentially increasing vulnerability to tobacco-related disease in these regions. Many studies performed have proved its deleterious effects on many organ systems mainly respiratory, reticuloendothelial system and cardiovascular systems. With 6000 chemical substance it contains, it exerts pharmacological, mutagenic, cancerogenic, toxic, and inflammatory effects. (Oztuna *et al; 2004*) Nowadays, it is responsible for every six cases of death (Kumar, 2000). Cigarette contains carcinogens (polycyclic aromatic hydrocarbons etc.), irritant substances, nicotine, carbon monoxide, and other gases. Cigarette smoke contains many oxidants and free radicals which can harm lipids, proteins, DNA, carbohydrates, and other biomolecules (Al-Azzawy*et al; 2011*). The effects of smoking on various metabolic and biological processes, hormone secretion, and hematopoietic system have been demonstrated. In many studies, among acute effects of smoking on hematological system, increases in WBC, eosinophil, and platelet (PLT) counts have been shown (Oztuna *et al; 2004*). A correlation was established between smoking, and WBC counts. Relatively higher WBC counts were detected in smokers (de Heens*et al; 2009*). Smoking has been suggested to increase the levels of hematological parameters as hemoglobin (Hb) concentration, red blood cell (RBC), neutrophil, eosinophil, monocyte, and platelet counts. Smoking cessation studies have demonstrated that some of these changes are reversible, and transitory in case of cessation of smoking. The result of scientific studies done in neonatal rats seems to indicate that exposure to cigarette smoke in the womb may reduce the fetal brain's ability to recognize hypoxic conditions, thus increasing the chance of accidental asphyxiation (Guindon *et al; 2003*). Incidence of

impotence is approximately 85 percent higher in male smokers compared to non-smokers (Korenman, 2004), and is a key factor causing erectile dysfunction (ED) (Hunter *et al; 2001*). The World Health Organization (WHO) estimates that tobacco caused 5.4 million deaths in 2004 and 100 million deaths over the course of the 20th century. Similarly, the United States Prevention describes tobacco use as "the single most important preventable risk to human health in developed countries and an important cause of premature death worldwide. Smoke contains several carcinogenic pyrolytic products that bind to DNA and cause many genetic mutations. There are over 19 known chemical carcinogens in cigarette smoke. Tobacco also contains nicotine, which is a highly addictive psychoactive chemical. When tobacco is smoked, nicotine causes physical and psychological dependency. Tobacco use is a significant factor in miscarriages among pregnant smokers, it contributes to a number of other threats to the health of the fetus such as premature births and low birth weight and increases by 1.4 to 3 times the chance for Sudden Infant Death Syndrome (SIDS)(Pendlebury*et al; 2008*).

1.2.3. Shisha water pipe smoking:

Most of the epidemiologic studies of water pipe use have been conducted among adults; only a few reports have been published about university students and only 1 of middle and high school students. The nicotine content of water-pipe tobacco has been reported to be 2% to 4%, in comparison with 1% to 3% for cigarettes. Only a small number of studies have examined the composition of narghile smoke. A study of carbon monoxide in water-pipe and cigarette smoke found carbon monoxide concentrations of 0.34% to 1.40% for water-pipe smoke and 0.41% for cigarette smoke. The carbon monoxide concentration in water-pipe smoke was significantly greater for smaller water-pipe size and for commercial as opposed to domestic charcoal. Another study found elevated end-expiratory carbon monoxide levels in a group of 18 healthy Jordanian water-pipe smokers .In a separate report, the carboxyhemoglobin concentration was measured in

1832 healthy Saudi Arabian male volunteers after smoking for 10 to 40 minutes. The mean carboxyhemoglobin concentrations were higher among water-pipe smokers (10.1%) than among cigarette smokers (6.5%) or nonsmokers (1.6%), and a linear relationship was found between smoking intensity and carboxyhemoglobin concentration (Shafagojand mohammed, 2002). Recent technical innovations confirm this duration and also provide a more detailed analysis of waterpipe tobacco smoking episodes. Data collected from actual waterpipe tobacco smokers in natural settings show that a waterpipe use episode typically involves almost 200 puffs, with an average puff volume exceeding 500 ml. Thus, compared to a cigarette, which involves inhalation of approximately 500–600 ml of smoke (ie, 10–13 puffs of about 50 ml, on average) (Djordjevic*et al*; 2000), a single waterpipe use episode involves inhalation of approximately 90,000 ml of smoke (Shihadeh,2003).

1.2.4.Component of tobacco:

1.2.4.1. Nicotine:

Nicotine is the primary addictive substance in tobacco products. The data indicate that the nicotine levels in e-cigarettes vary considerably. E-cigarette brands and models differ in the efficacy and consistency of nicotine yields and the delivery of nicotine is not uniform either from puff-to-puff or across products of the same brand. Furthermore, the level of nicotine listed on the labels of e-cigarette cartridges and refill solutions is often significantly different from measured values and labelling may not adequately convey the amount or concentration of nicotine (Shihadeh and Saleh, 2005).For example, Goniewicz et al conducted a quantitative analysis of nicotine in aerosols generated from 15 e-cigarette brands (16 products) that were selected based on their market popularity. They found that total nicotine in aerosol varied by brand from 0.5 to 15.4 mg per 300 puffs (20 series of 15 puffs, 70 mL/puff, triplicate tests of each product) and that the

nicotine in aerosol varied from 21% to 85% of the nicotine present in the cartridge (Goniewicz*et al;* 2013).The nicotine content of water-pipe tobacco has been reported to be 2% to 4%, in comparison with 1% to 3% for cigarettes (Benowitz *et al;* 1993).A study of nicotine and cotinine in water-pipe smokers found high values of these substances after smoking. After a single 45-minute smoking session, the mean plasma concentration of nicotine rose from 1.11 to 60.31 ng/mL, and cotinine rose from 0.79 to 51.95 ng/mL. Saliva nicotine concentration rose from 1.05 to 624.74 ng/mL, and cotinine rose from 0.79 to 283.49 ng/mL. The mean amounts of nicotine and cotinine excreted in a 24-hour urine sample after smoking were 73.59 μg and 249 μg, respectively. According to another report, urinary nicotine concentrations were similar for water-pipe smokers (median of 2 pipes per day) and for cigarette smokers (median of 30 cigarettes per day), (Shafagojand mohammed, 2002). These studies provide limited data to suggest that water-pipe smoke is at least as toxic as cigarette smoke. Water-pipe smokers may absorb higher concentrations of these substances because of higher concentrations in the smoke itself or because of the mode of smoking, including frequency of puffing, depth of inhalation, and length of smoking session. Water-pipe smokers may smoke for several hours at a time and may breathe in more deeply because of the less irritating nature of the moisturized smoke (Brelan*det al;* 2006).

1.2.4.2. Carbon mono oxide:

Carbon monoxide concentration, specifically, may also be elevated because of the charcoal used to burn the narghile tobacco. Contrary to popular opinion, the water in the pipe probably filters out only a small portion of the noxious substances. More research is needed to compare the toxic effects of the 2 types of smoking, taking into account the presmoking carbon monoxide levels, frequency and intensity of inhalation, and other factors (Shafagojand mohammed, 2002). Only a small number of studies have examined the composition of narghile smoke. A study of carbon monoxide in water-pipe and

cigarette smoke found carbon monoxide concentrations of 0.34% to 1.40% for water-pipe smoke and 0.41% for cigarette smoke. The carbon monoxide concentration in water-pipe smoke was significantly greater for smaller water-pipe size and for commercial as opposed to domestic charcoal. Another study found elevated end-expiratory carbon monoxide levels in a group of 18 healthy Jordanian water-pipe smokers (Djordjevic*et al;* 2000). In a separate report, the carboxyhemoglobin concentration was measured in 1832 healthy Saudi Arabian male volunteers after smoking for 10 to 40 minutes. The mean carboxyhemoglobin concentrations were higher among water-pipe smokers (10.1%) than among cigarette smokers (6.5%) or non-smokers (1.6%), and a linear relationship was found between smoking intensity and carboxyhemoglobin concentration (Shafagojand mohammed, 2002*).*

1.2.4.3.Other chemical substances:

1.2.4.3.1. Hydrogen cyanide:

The lungs contain tiny hairs (cilia) that help to clean lungs by moving forging substances out.Hydrogen cyanide stops this lung clearance system from working properly. Which means the poisonous chemicals in tobacco smoke can build up inside the lungs. Other chemical in smoke that damage the lungs include hydrocarbons, nitrous oxide, organic acid, phenol, oxiding agents (Guindon *et al;* 2003).

1.2.4.3.2. Oxiding chemicals:

These highly reactive chemicals (include free radicals) can damage the heart muscles and blood vessels. They react with cholesterol, leading to the buildup of fatty material on artery walls. Their actions lead to heart disease, stroke and blood vessel disease(Bjartveitand Tverdal, *2005*).

1.2.4.3.3. Metals:

Tobacco smoke contains dangerous metals including arsenic, cadmium and lead. Several of these metals are carcinogenic (Ezzati*et al; 2005)*.

1.2.4.3.4. Radioactive compounds:

Tobacco smoke contains radioactive compounds that are known to be carcinogenic. Quantitative and qualitative studies have identified a wide variety of chemical components in the cartridges, refill solutions and aerosols of e-cigarettes. Substances identified include tobacco-specific nitrosamines (TSNAs), aldehydes, metals,volatile organic compounds (VOCs), phenolic compounds, polycyclic aromatic hydrocarbons (PAHs), flavourssolvent carriers, tobacco alkaloids,and drugs (amino-tadalafil and rimonabant). These TSNAs, aldehydes, metals, VOCs, phenolic compounds, PAHs and tobacco alkaloids are harmful or potentially harmful constituents released during the smoking of conventional cigarettes, and their public health risks have been the focus of many studies.(Ezzati*et al; 2005*), (Bjartveitand Tverdal, 2005)and (Willett*et al; 1989*).

1.2.5.Alterations in Blood cells:

1.2.5.1. Red blood cells and hemoglobin:

Smoking has been suggested to increase the levels of hematological parameters as hemoglobin (Hb) concentration, red blood cell (RBC). Smoking cessation studies have demonstrated that some of these changes are reversible and transitory in case of cessation of smoking (McAuley*et al*; *2012)*.

1.2.5.2. White blood cells:

The mechanism of action of smoking on WBC is not clear-cut yet. In smokers, lymphocytosis is thought to be mainly associated with an increase in T-cells (Smith *et al; 2003*). Increases in peripheral blood WBC counts, and alterations in WBC function can be the result of direct damage stemming from alterations in epithelial and endothelial surfaces and/or cytokine levels (especiallyIL-6) caused by components of cigarette smoke .Blood contains platelets, red blood cells, and leukocytes suspended in plasma. Plasma in turn contains a variety of coagulation proteins and lipids that also contribute to the clotting process. Smokers tend to develop MI at a lower burden of atheroma than do non-smokers. This finding suggests a greater role for formed elements of blood or for cardiac electrical instability in cardiovascular events in smokers (Zafar*et al;* 2003).

1.2.5.3. Platelets:

Platelets, although only a minor component of the solid phase of blood, are critical to the coagulation process and are important mediators of the impact of smoking on cardiovascular outcomes. Potential sites of actions and mechanisms of effects of smoking on platelets. Smoking decreases nitric oxide NO-mediated inhibition of platelet activation and increases platelet activation through oxidative stress and other mechanisms (Wolfram, 2003).

1.2.6. Coagulation system effects:

The effects of chronic cigarette smoking on the coagulation system were examined in 2964 men aged 50 to 61 years and clinically free of cardiovascular disease. Factor VII activity (VIIc), factor VII antigen (VIIag), prothrombin fragment 1+2 (F 1.2), fibrinopeptide A (FPA) and fibrinogen were measured in all participants, and activated factor VII (VIIa), factor IX activation peptide (IX pep) and factor X activation peptide (X pep) in a large sub-sample. The levels of all indices except FPA differed significantly

between non-smokers, ex-smokers and current smokers (KaturjiMaster's, 2006). After adjustment for other conventional cardiovascular risk factors, mean VIIc was raised slightly by 3% in ex-smokers and current smokers as compared with non-smokers, owing to increases in VIIa and VIIag. Plasma IX pep, X pep, F 1.2 and fibrinogen concentration were highest in current smokers, intermediate in ex-smokers and lowest in non-smokers. These findings accord with the increased risk of arterial thrombosis in smokers (Miller *et al; 1998*). Benowitz and associates found that factor VII coagulant activity was significantly lower during cigarette smoking than during either nicotine or placebo patch conditions (Benowitz *et al; 1993*). Cigarette smoking increased the urinary excretion of 11-dehydro-thromboxane B2 (reflecting thromboxane A2 generation) and increased plasma concentration of the platelet alpha-granule constituents, platelet factor 4 and beta-thromboglobulin, compared with placebo treatment, indicating in vivo platelet activation. Cigarette smoking was also associated with higher levels of fibrinogen in plasma. Transdermal nicotine produced plasma levels of nicotine in the same range as those during smoking but had no effect on thromboxane A2 metabolite excretion, platelet alpha-granule release or plasma fibrinogen, compared with placebo. Excretion of 2, 3-dinor-6-keto-PGF1 alpha (reflecting prostacyclin generation) was not significantly influenced by any treatment. These results suggest that nicotine as such is not responsible for the platelet activation or elevation of plasma fibrinogen seen in smokers. However, we cannot exclude the possibility that intermittent bolus-like dosing of nicotine from cigarettes could have different effects from those produced by continually released transdermal nicotine (Brelan*det al; 2006*). Other findings were that cigarette smoking and transdermal nicotine treatment were both associated with a higher white blood cell count compared with the placebo patch condition, suggesting a direct effect of nicotine to increase circulating white cells. Factor VII coagulant activity (VIIc) was significantly lower during cigarette smoking, than during either nicotine or placebo patchedconditions (Benowitz*et al; 1993*).

Cross-sectional population-based study of 5029 men and women aged 59–80 years from primary care practices in Great Britain found that active cigarette smokers had lower albumin and higher triglycerides, CRP, IL-6, white cell count, fibrinogen, blood viscosity, factor VIII, VWF and t-PA than non-smokers. Among non-smokers, serum cotinine levels were independently positively associated with CRP, fibrinogen, factor VIII, VWF and t-PA and inversely associated with albumin, after adjustment for age, gender, social and behavioral factors. The differences in CRP, fibrinogen and albumin between cotinine ≤0.05 and >0.7 ng/ml were one-third to one half the size of differences between cotinine ≤0.05 ng/ml and current smokers, but were of similar magnitude for VWF and t-PA (Jefferis *et al; 2010*) and (Jackson *et al; 2008*).

1.2.7. Plasma protein effects:

1.2.7.1. Circulating Proteins:

In addition to its effects on the cellular elements of blood, smoking alters the proteins involved in the coagulation pathway by changing procoagulant factors in the circulation and anticoagulation factors derived from the endothelium (Abu-Hayyeh*et al*; 2001).

1.2.7.2. Fibrinogen:

Plasma fibrinogen is an independent predictor of cardiovascular disease, including coronary heart disease (CHD), stroke and peripheral arterial disease. Its predictive value for CHD is similar to that of classical risk factors, such as smoking habit, blood pressure or serum cholesterol, and adds to risk prediction from these three variables. Plasma fibrinogen levels show a dose dependent increase in smokers; following smoking cessation, levels decrease towards similar values in those who have never smoked. In parallel with the fall in risk of CHD, an early rapid reduction is followed by a slower reduction over 10±15 years. Plasma fibrinogen levels may promote cardiovascular disease through several biological mechanisms, including atherogenesis, thrombogenesis

and increased blood viscosity, which may reduce blood flow. It has been estimated that up to 50% of the increase in CHD risk associated with smoking could be attributed to the effects of smoking on fibrinogen. However, the causal significance of fibrinogen levels in cardiovascular disease remains to be established by large randomized trials of fibrinogen reduction. Increased fibrinogen synthesis in the liver, rather than decreased fibrinogen catabolism, has long been suspected, but there is a lack of published studies addressing this hypothesis(Hunter *et al; 2001)*. In the April issue of Clinical Science Report two studies of fibrinogen synthesis in male smokers (Korenman, 2004). In the first study, current smokers had higher absolute fibrinogen synthesis rates than non-smokers, which were correlated with increased fibrinogen levels. In the second study, 2 weeks ' abstention from smoking in current smokers reduced both absolute synthesis rates and plasma fibrinogen levels. The authors conclude that increased fibrinogen synthesis plays a primary role in the increased plasma fibrinogen level associated with smoking. This is an important step in elucidating the pathways through which smoking increases fibrinogen levels (Hunter*et al; 2001*). Also discuss such pathways, including the possibility that the effects of smoking on fibrinogen synthesis are part of a generalized inflammatory reaction (e.g. to smoking-induced injury to the respiratory tract, blood vessels or other organs). Similarly to fibrinogen, such measures also show a dose-dependent increase in smokers and decrease following smoking cessation. Cytokines, such as interleukin-6, are important mediators of these measures of the inflammatory response, including fibrinogen synthesis (Hunter *et al; 2001*). Future studies of smoking, smoking cessation and plasma fibrinogen are required to define their relationship to other acute-phase markers and inflammatory cytokines. Fibrin degradation produces, such as D-dimer, may also play a role in fibrinogen synthesis and inflammatory reactions; D-dimer is also elevated in smokers and weakly related to plasma levels of fibrinogen and C-reactive protein (Hunter*et al; 2001)*. Also discuss the possible role of catecholamine in smoking-induced hyperfibrinogenaemia. However,

studies to date have shown variable effects of adrenergic-blocking drugs in reducing plasma fibrinogen levels.Study findings indicate that circulating levels of fibrinogen increase in smokers and decrease with smoking cessation. Also, research suggests that elevated fibrinogen values are an independent risk factor for CHD and deep-vein thrombosis. For CVD, the predictive effect was reportedly similar and additive to traditional cardiovascular risk factors (Abu-Hayyeh*et al*; 2001). The effect of fibrinogen on CVD is partly attributable to smoking and seems to be mediated through alterations in rates of synthesis by the liver. Use of snuff is not associated with increased fibrinogen levels (Rothwell*et al;1991*).Nitration of tyrosine residues, a marker of NO-dependent damage, is increased in smokers. Presence of these residues depends strictly on availability of nitrogen dioxide radicals that are in turn derived from $ONOO^-$. Tyrosine nitration modifies a variety of proteins, including fibrinogen. Nitrogenated fibrinogen is more reactive and thrombogenic than is native fibrinogen, a fact that seems attributable to accelerated formation of clots without modification in plasmin-induced thrombolysis. The antioxidants glutathione and vitamin C protect against formation of nitrogenated fibrinogen by interfering with interaction of nitrate radicals with tyrosine. $ONOO^-$ is likely to derive from interaction of NO from cigarette smoke with superoxide radicals from pulmonary macrophages .In studies of both animal models and human volunteers, even brief exposure to tobacco smoke induced prolonged production (>30 minutes) of $ONOO^-$, apparently from pulmonary macrophages (Zafar*et al; 2003*).

1.2.7.3. C-Reactive Protein:

Other measures of inflammation are also predictive of cardiovascular risk, including C-reactive protein, leukocyte counts and low serum albumin, as well as plasma viscosity and the erythrocyte sedimentation rate (which are determined by plasma levels of fibrinogen and other macromolecules) (Jabbour*et al;* 2003).Chronic, low-level inflammation—reflected by elevated levels of C-reactive protein (CRP) and other

biomarkers—is an important risk factor for atherosclerosis. (DeBias*et al;1976*). Investigators reported that levels of CRP, which likely contributes to both oxidative stress and mutagenic and fibrogenic characteristics of atherosclerotic plaque, are higher in smokers than in non-smokers in a dose-dependent manner. This increase persisted even after adjustment for diabetes, lipid profile, and CVD, as well as age, gender, and race. More important, five years after smoking cessation, CRP levels were decreased to levels similar to those in lifetime non-smokers. This finding suggests vascular healing. The timeframe is consistent with that observed in the multinational monitoring of trends and determinants of CVD (MONICA) and in the Northwick Park Heart studies. In those studies, cardiovascular risk was reduced at two to five years after a person stopped smoking (Allred *et al; 1989*).

1.2.8. Alterations in Blood Vessels:

1.2.8.1. Nitric Oxide:

Cigarette smoking has injurious effects on the vascular endothelium .Abnormalities in the release of chemical mediators occur as a consequence of endothelial dysfunction and are likely to contribute to the prothrombotic condition of smokers. Examples include decreases in NO-mediated inhibition of interactions between platelets and the blood vessel wall, in platelet-induced NO, and in inhibition of platelet activation (Burke *et al;* 2003). Blood vessel tone is more sensitive to low NO levels than is platelet function.The importance of NO deficiency mediated by oxidative stress in thrombosis is suggested by familial childhood stroke resulting from deficiency in glutathione peroxidase. This condition decreases NO levels in association with both increased expression of P-selectin in platelets and platelet aggregation and activation (LyKKesFeldt*et al; 2000*).

1.2.8.2. Inflammation:

Studies demonstrate that cigarette smoking results in a chronic inflammatory state, evidenced by increased counts of circulating leukocytes, CRP, and acute-phase reactants such as fibrinogen. Cigarette smoking also activates monocytes and enhances recruitment and adhesion of leukocytes to blood vessel walls, an integral step in vascular inflammation (Park*et al; 2007*). Nicotine may contribute to inflammation by acting as a chemotactic agent for migration of neutrophils. One study indicates that nicotine enhanced leukocyte-endothelium interactions, resulting in greater leukocyte rolling and adhesion in the cerebral microcirculation of mice. Nicotine reportedly acts on human monocyte-derived dendritic cells to stimulate an inflammatory response. Dendritic cellswhich were detected in the walls of arteries and in atherosclerotic lesions, present antigens and are thus required for the start of adaptive immunity. Studies showed that nicotine is a potent inducer of expression of a variety of co-stimulatory molecules and that it increases secretion of the proinflammatory cytokine interleukin-12 in cultured dendritic cells. Nicotine augmented the capacity of dendritic cells to stimulate proliferation of T cells and cytokines. Finally, intravenous injection of nicotine increased the movement of dendritic cells into atherosclerotic lesions in vivo in mice deficient in APO E (Jefferis*et al;* 2010). This line of research suggests that nicotine could contribute to adaptive immunity, which may have a role in atherogenesis. However, switching from smoking to transdermal nicotine resulted in a significant decline in the leukocyte count. In addition, use of smokeless tobacco did not produce higher leukocyte counts or higher CRP levels than are seen in persons who do not use tobacco. These observations suggest that nicotine is not the main determinant of the inflammatory response in smokers (Abu-Hayyeh*et al; 2001*).

1.3. Previous study

Table (1-1):Health Effects of Waterpipe Smoking.

Condition	Study Design	Study Population/Setting	Findings	Source
Malignancy				
Lung cancer	Retrospective	25 men with bronchogenic carcinoma	22 of 25 were water-pipe smokers	Nafae*et al* (India, 1973)
	Case-control	107 male tin miners with lung cancer	Increased cancer rates (OR: 1.9, dose response)*	Qiao*et al* (China, 1989)
Gastrointestinal cancer	Retrospective	183 patients with esophageal and gastric carcinoma	Increased cancer rates	Gunaid*et al* (Yemen, 1995)
Bladder cancer	Case-control	151 men with bladder cancer	No difference in cancer rates (OR: 0.8)	Bedwani*etal* (Egypt, 1997)
Lip cancer	Case reports	Oral surgery department	2 cases of squamous cell carcinoma, 1 case of keratoacanthoma	El-Hakim and Uthman (Egypt, 1999)

Pulmonary				
Pulmonary function	Controlled study	595 smokers	Decreased VC, FEV1, FVC; decline with age*	Al-Fayez *et al* (Saudi Arabia, 1988)
	Controlled study	65 exclusive water-pipe smokers	Decreased PEFR*	Bayindir *et al* (Turkey, 1993)
	Controlled study	19 exclusive water-pipe smokers	No difference in PEFR	Altinisik*et al* (Turkey, 1995)
	Controlled study	82 exclusive water-pipe smokers	Decreased PEFR*	Kiter*et al* (Turkey, 2000)
Infectious diseases				
Helicobacter pylori	Retrospective	128 men with Helicobacter pylori	Increased infection rates (OR: 4.1)	El-Barrawy*et al* (Egypt, 1997)
Pulmonary aspergillosis	Case report	Adult leukemia patient	1 case	Szyper-Kravitz*et al* (Israel, 2001)
	Retrospective	45 contacts of 5	Increased	Munckhof*et*

Tuberculosis		men with pulmonary tuberculosis shared a marijuana pipe with the case	risk for a positive tuberculin skin test (OR: 2.22)*	*al* (Australia, 2003)
Potential morbidity			Hepatitis C, HSV, HIV, EBV, respiratory viruses	
Miscellaneous				
Reproductive	Case-control	100 infertile women	Increased infertility rates with WPS husbands (OR: 2.5)*	Inhorn and Buss (Egypt, 1994)
Perinatal	Retrospective	106 pregnant water-pipe smokers delivering in hospitals	Increased low birth weight rate (OR: 2.17)*; increased respiratory distress rate (OR: 3.65)*	Nuwayhid*et al* (Lebanon, 1998)
	Clinical study	18 healthy water-	Elevated	Shafagoj and

Cardiovascular		pipe smokers	heart rates and blood pressures after smoking*	Mohammed (Jordan, 2002)
	Case-control	292 patients with coronary heart disease	Increased rates among ever (OR: 2.2) but not among current (OR: 0.7) water-pipe smokers	Jabbour*et al* (Lebanon, 2003)
Dental: postextraction dry socket	Prospective	100 water-pipe smokers	3 times greater risk for dry socket	Al-Belasy (Egypt, 2004)
Biological effects				
Hematologic	Laboratory	Water-pipe smokers	Increased superoxide anion and leukocyte counts*	Sharma *et al* (India, 1997)
	Laboratory	35 water-pipe	Increased	Yadav and

Chromosomal		smokers	chromosomal abnormalities*	Thakur (India, 2000)
Hematologic	Laboratory	7 subjects	Abnormal platelet function*	Wolfram (Austria, 2003)

Key:

- OR indicates odds ratio; VC, vital capacity; FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; PEFR, peak expiratory flow rate; HSV, herpes simplex virus; EBV, Epstein-Barr virus.
- ↵* Statistically significant.

1.4. Rationale

Smoking is the most important public health problem. Many studies performed have proved its deleterious effects on many organ systems. Shisha smoking is a growing

health concern globally and especially in Sudan. Many Shisha smokers perceive this behavior to be less lethal and addicting than cigarettes. Converging lines of evidence, including Shisha smoke analysis, user toxicant exposure, and health effects research contradict this perception, though more study is required. The study of the effects of Cigarette Smoking and shisha on hematological parameters on the Sudanese population can serve as a significant contribution to answer the question. In spite of that there is no published study in Sudan, so we trying to compare our results with similar studies carried out Sudan.

1.5.Objectives

1.5.1.General Objective:

To evaluate the effects of cigaretteandwater pipe (shisha)smokingon some hematological parameters in Sudanese people.

1.5.2.Specific objectives:

- To measurered blood cells, platelets, white blood cells count and white blood cell differential in shisha and cigarette smokers.
- To estimate hemoglobin level and calculate red cell indices in water pipe (Narghile) and cigarette smokers.
- To compare the white blood cells counts, hemoglobin level and red cell indices on both water pipe and cigarette smokers with the control group.
- To correlate between duration of smoking and CBC in smokers group (cigarette and water pipe).

2. Materials and methods

2.1. Study design:

An analytical case control study approach by measuring red blood cells, white blood cells and platelets count in Sudanese cigarette and shisha smokersin Khartoum State, from February to June 2015.

2.2. Study population:

This study was carried out on Sudanese volunteers, agerange between 18 to 50 years old, from both smoker and non-smoker (control) groups according to the following inclusion and exclusion criteria.

2.2.1. Inclusion Criteria:

Control group are males, age range from 18-50 years and apparently healthy individuals,regular smokers group for both cigarette and shisha group are males, age range from 18-50 years and apparently healthy individuals.

2.2.2. Exclusion Criteria:

Any individual who smoke both cigar and shisha were excluded and irregular shisha smokers, individual with other disease with population under study changes.

2.3.Sample size:

Sample size was 150 samples include 50 cigarette smokers, 50 shisha smokers and 50 non-smokers as control group.

2.4. Method of data collection:

Questioner was used to obtain initial knowledge about the individual age, smoking dose, smoking duration and other diseases related to it.

2.5. Sample processing:

Three ml K_3EDTA anticoagulated venous blood was withdrawn using 5ml disposable syringe, all samples were checked for clots and hemolysis and mixed well before analysis. The samples were then subjected to apparatus analysis,50µl from each sample was sucked by apparatus needle. Immediately the result of each sample was obtained, results were kept until they were statistically analyzed.

2.6. Handling of blood sample:

Three ml (3 ml) venous blood sample was collected in EDTAanticoagulant blood containerin proper way and gently mixed in the hematology mixture and immediately (not more than one hour). The sample was then analyzed by Sysmex KX-21N automated hematology analyzer.

2.7. Methodology:

2.7.1. CBC: (complete blood count)

Evaluations of the blood cell count were performed by Sysmex automated hematological analyzer, which could perform 18 hematological parameters with high accuracy and precision. Principally Sysmex analyzer is based on the electronic resistance (impedance) detection method for counting and sizing recognition of the leukocytes, erythrocytes, and platelet. Through using three preliminary hydraulic systems for WBCs, RBCs, platelet and hemoglobin, and display the mode of the cells blood count results on the liquid crystal displayer (LCD) with histogram and printed out the results in thermal paper (Dacie and lewis, 2006).

2.7.2. Principle of Sysmex KX model 21 hematology analyzer:

Measurement of blood cells (RBCs, WBCs, and platelet). And hemoglobin concentration obtained by aspiration of small volume of well mixed (k2EDTA) blood by sample probe and mixed with isotonic diluents in nebulizer. Diluted aspiration delivered

to RBCs aperture bath for providing information about RBCs and platelet. Other portion of aspirated sample induced in to WBCs bath in which hemolytic reagent (stromatolyser) added to break down (RBCs) and release of hemoglobin which measured in build colorimeter, based in cyanomethemoglobin method (HICN). The through three sensing apertures for each cell type, cell counted and size information generated in triplicate pulses acting to electronic conductively. Mentioned pulses converts in to digital number using in build calculator programmed and designed for RBCs, WBCs counts. Some portion of diluted sample delivered to in build hemoglobin meter at the same time, hence three values directly measured (RBCs, WBCs, and Hb) and displayed on (LCD). Other values of red cell indices, leukocyte differential and absolute count calculated from given information, the result printed out according to the setting mode.On the other hand, platelet count and histogram determined from pulses acting to size of the platelet (Dacie and lewis, 2006).

2.7.3. Quality control of SYSMIX KX 21:

All quality control of the machine done in instructed manner. The daily, weekly and monthly maintenance and calibration used to ensure quality assurance**.** Then before using the apparatus one of the last day samples was reanalyzed for delta check.

2.8 .Ethical consideration:

Before collecting the sample, we describe to our participant what we want to do, we took written consent from our participants, and we used an ideal blood collection procedure in order to safe them.

2.9. Data analysis:

All results were analyzed using Statistical Package for Social Sciences (SPSS, V.15) analysis software program including description statistic of the mean and standard deviation.

2.10. Data presentation:

Data were presented by tables and figures.

3. Results

Thisstudy was based on analytical case control study carried out from February to June 2015, a total of 150 healthy male individuals aged 18-50 years were included in thisstudy. Fifty (50) persons of them were non-smoker as control group. While other hundred participant are smoking cigarette (50) and smoking shisha (50). The criteria of probability sample was concerned, the similarity of age group distribution of all groups was achieved.

The means, standard deviation and P-Value of all groups under study was calculated, the results were shown in the next tables and figures.

Statistical analysis of cigarette smokers group when compare with non-smokers show that, mean of HGB was (14.61±1.11 vs 13.38±1.23), mean of HCT was (45.85%±2.56 vs 42.90%±2.52), mean of RBCs counts was (5.64×10^{6}/µL ±0.56 vs 5.06×10^{6}/µL ±0.45) and MCHC mean was (32.26 g/dl±1.34 vs 30.66 g/dl ±1.00) with P. value equal (0.000) which indicate highly significance difference.

Mean of WBCs counts was (5.66×10^{3}/µl±1.50 vs 4.97×10^{3}/µl ±1.00) with P. value equal (0.002) which indicate highly significance differences, the mean of neutrophils percentage was (47.69%±10.75 vs 51.68%±8.15) with P. value equal (0.013) that means there was a significance differences, the mean of lymphocytes percentage was (38.73 %±9.07 vs 38.22%±4.98) with P. value equal (0.045) that indicated significance differences and the mean of MXDs (eosinophils, basophils and monocytes) percentage was (10.80 %±4.40 vs 10.07%±3.41) with P. value equal (0.177) which indicate that no significance difference.

The mean of MCH was (28.34 p.g±1.62 vs 27.86 p.g ±2.13) with P. value equal (0.516) with no significance differences, and the mean of platelets counts was (236.12×10^{3}/µl±67.21 vs 253.04×10^{3}/µl ±66.35) with P. value equal (0.081) and no significance differences.

The result of statistical analysis of shisha smoker group when compare with control group show that, mean of HGB was (15.05g/dl ±0.92 vs 13.38g/dl ±1.23), mean of HCT was (46.51%±2.87vs 42.90%±2.52), mean of RBCs counts was (5.72×10^6/µL ±0.63 vs 5.06×10^6/µL ±0.45) and MCHC mean was (32.37 g/dl±1.51 vs 30.66 g/dl ±1.00) with P. value equal (0.000) which indicate highly significance differences.

The mean of MCH was (28.56 p.g±2.09 vs 27.86 p.g ±2.13) with P. value equal (0.023) which indicate there was a significance differences, and the mean of platelets counts was (267.14×10^3/µl±88.03 vs 253.04×10^3/µl ±66.35) with P. value equal (0.263) and no significance differences.

The mean of WBCs counts was (5.64×10^3 /µl±1.81 vs 4.97×10^3/µl ±1.00) with P. value equal (0.011) that means there was a significance difference, the mean of neutrophils percentage was (52.44 %±10.70 vs 51.68%±8.15), and mean of lymphocytes percentage was (37.39 %±6.85 vs 38.22%±4.98)with the P. value equal (0.477) and (0.391) respectively with no significance differences, the mean of MXDs (eosinophils, basophils and monocytes) percentage was (10.39 %±5.03 vs 10.07%±3.41) with P. value equal (0.649) with no significance differences.

Duration of smoking cigarette and shisha did not significantly (p.value>0.05) affect the hematological parameters under study except hemoglobin concentration.

Table 3.1:

Distributions of age of the study populations.

	10-20 years	21-30 years	31-40 years	41-50 years
Cigarette smokers	**8**	**17**	**13**	**12**
Shisha smokers	**10**	**16**	**10**	**14**
Non smokers	**10**	**26**	**9**	**5**

Table 3.2:

The means±SDand P. value of CBC (complete blood count) of cigarette smokers and non-smokers.

No	Parameter	Mean±SD of Cigarette smokers	Mean±SD of Non smokers	P. Value
50	**HGB**	**14.61±1.11**	**13.38±1.23**	**0.000**
50	**HCT**	**45.85±2.56**	**42.90±2.52**	**0.000**
50	**RBCs**	**5.64±0.56**	**5.06±0.45**	**0.000**
50	**MCV**	**85.55±5.14**	**85.43±4.81**	**0.870**
50	**MCH**	**28.34±1.62**	**27.86±2.13**	**0.516**
50	**MCHC**	**32.26±1.34**	**30.66±1.00**	**0.000**
50	**PLT**	**236.12±67.21**	**253.04±66.35**	**0.081**
50	**WBCs**	**5.66±1.50**	**4.97±1.00**	**0.002**
50	**LYM%**	**38.73±9.07**	**38.22±4.98**	**0.045**
50	**MXD%**	**10.80±5.03**	**10.07±3.41**	**0.177**
50	**NEUT%**	**47.69±10.75**	**51.68±8.15**	**0.013**

Key:

-HGB: hemoglobin, measured by gram per decilitter.

-HCT: hematocrit, expressed by %.

-RBCs: red blood cells, expressed by (cell $\times 10^6/\mu L$).

-MCV: mean cell volume, measured by fimtolitter.

-MCH: mean cell hemoglobin, measured by pictogram.

-MCHC: mean cell hemoglobin concentration, measured by gram per deciliter.

-platelets: expressed by (cell $\times 10^3/\mu L$).

-WBCs: white blood cells, expressed by (cell $\times 10^3/\mu L$).

-NEUT: neutrophil%.

-LYM: lymphocyte%.

-MXDs: mixed%.

-SD: standard deviation.

-P.value: Probability value.

Table 3.3:

The means±SD and P. value of CBC (complete blood count)of shisha smokers and non-smokers.

No	Parameter	Mean±SD of shisha smokers	Mean±SD of Non smokers	P. Value
50	HGB	15.05±0.92	13.38±1.23	0.000
50	HCT	46.51±2.87	42.90±2.52	0.000
50	RBCs	5.72±0.63	5.06±0.45	0.000
50	MCV	86.45±5.21	85.43±4.81	0.171
50	MCH	28.56±2.09	27.86±2.13	0.023
50	MCHC	32.37±1.51	30.66±1.00	0.000
50	PLT	267.14±88.03	253.04±66.35	0.263
50	WBCs	5.64±1.81	4.97±1.00	0.011
50	LYM%	37.39±6.85	38.22±4.98	0.391
50	MXD%	10.39±4.40	10.07±3.41	0.649
50	NEUT%	52.44±8.15	51.68±8.15	0.477

Key:

-HGB: hemoglobin, measured by gram per decilitter.

-HCT: hematocrit, expressed by %.

-RBCs: red blood cells, expressed by (cell $\times 10^6/\mu L$).

-MCV: mean cell volume, measured by fimtolitter.

-MCH: mean cell hemoglobin, measured by pictogram.

-MCHC: mean cell hemoglobin concentration, measured by gram per deciliter.

-platelets: expressed by (cell $\times 10^3/\mu L$).

-WBCs: white blood cells, expressed by (cell $\times 10^3/\mu L$).

-NEUT: neutrophil%.

-LYM: lymphocyte%.

-MXDs: mixed%.

-SD: standard deviation.

-P.value: Probability value.

Table 3.4:

CBC for cigarette smokers group according to duration of smoking

Parameters	less than 10 years	From 10 to 30 years	more than 30 years	P. value
HGB	**13.68±1.01**	**14.65±1.12**	**15.5±0.99**	**0.001**
HCT	**45.85±2.54**	**45.81±2.34**	**45.89±2.21**	**0.176**
RBCs	**5.58±0.50**	**5.63±0.57**	**5.71±0.51**	**0.077**
MCV	**85.55±5.21**	**85.24±5.39**	**85.86±5.15**	**0.772**
MCH	**28.04±1.54**	**28.1±1.62**	**28.9±1.60**	**0.069**
MCHC	**31.3±1.34**	**32.23±1.24**	**33.3±1.15**	**0.216**
PLT	**230.43±76.20**	**234.5±75.98**	**243.4±74.65**	**0.078**
WBCs	**5.1±1.50**	**5.6±1.49**	**6.1±1.44**	**0.098**
LYM%	**37.9±9.12**	**39.4±9.07**	**38.9±9.05**	**0.599**
MXD%	**10.8±5.10**	**10.6±5.04**	**11.0±5.08**	**0.097**
NEUT%	**47.0±10.56**	**52.0±10.49**	**44.0±10.75**	**0.234**

For cigarette smokers group there was significant difference between the duration of smoking and hemoglobin concentrationp.value equal (0.001), and there was no significant difference between the duration of smoking and other hematological parameters under the study p.value > 0.005.

Table 3.5:

CBC for shisha smokers group according to duration of smoking

Parameters	less than 10 years	10 to 30 years	more than 30 years	P. value
HGB	**14.6±0.96**	**15.00±0.98**	**15.57±0.94**	**0.000**
HCT	**45.95±2.74**	**46.57±2.86**	**47.01±2.78**	**0.146**
RBCs	**5.67±0.67**	**5.69±0.70**	**5.81±0.65**	**0.069**
MCV	**85.41±5.23**	**87.69±5.14**	**86.27±5.11**	**0.654**
MCH	**28.31±2.13**	**28.54±2.09**	**28.85±2.10**	**0.061**
MCHC	**31.45±1.57**	**32.11±1.48**	**33.56±1.52**	**0.145**
PLT	**266.32±85.45**	**267.2±87.56**	**267.9±88.09**	**0.254**
WBCs	**5.47±1.01**	**5.59±1.09**	**5.86±1.07**	**0.081**
LYM%	**37.41±6.85**	**36.54±6.71**	**38.22±6.92**	**0.354**
MXD%	**10.51±4.35**	**10.45±4.40**	**10.21±4.28**	**0.082**
NEUT%	**52.75±8.14**	**53.13±8.06**	**51.44±7.98**	**0.321**

For shisha smokers group there was significant difference between the duration of smoking and hemoglobin concentrationp.value equal (0.000), and there was no significant difference between the duration of smoking and other hematological parameters under the study p.value > 0.005.

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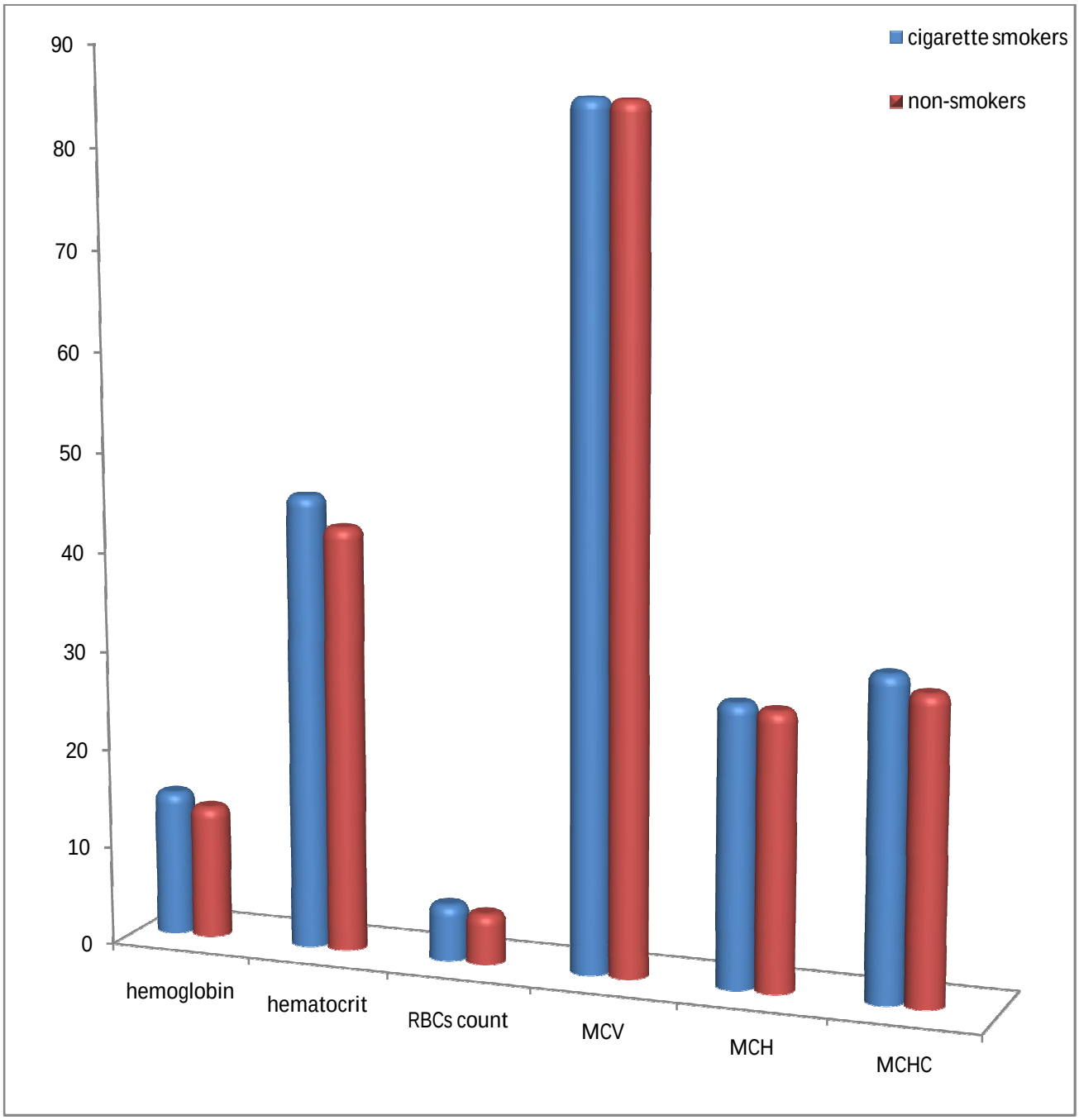

Figure 3.1: hemoglobin, hematocrit, red blood cells counts, MCV, MCH, and MCHC of cigarette smokersand non-smokers.

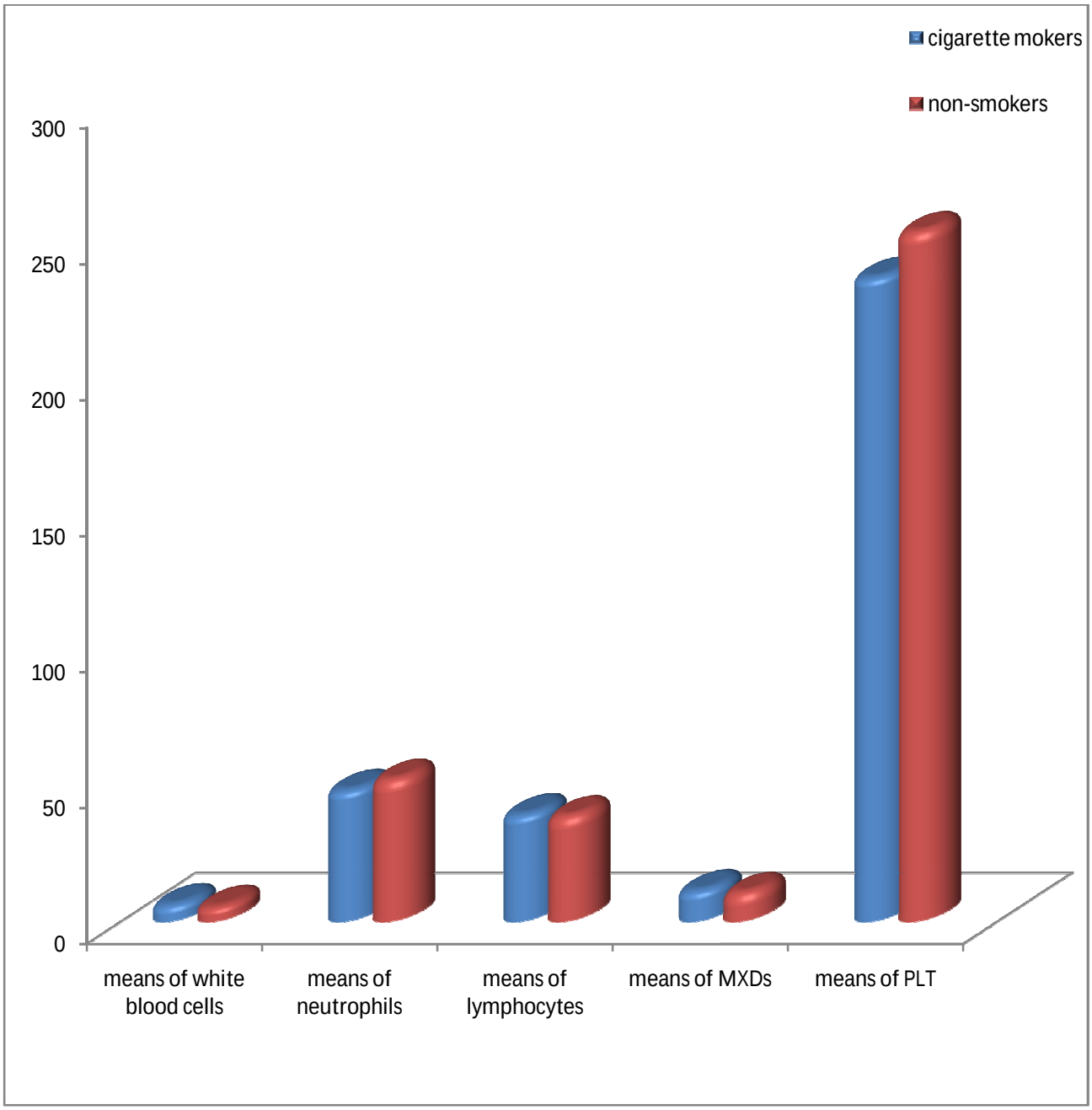

Figure 3.2: means of total white blood cells counts, neutrophils %, lymphocytes % and MXDs % of cigarette smokers and non-smoke

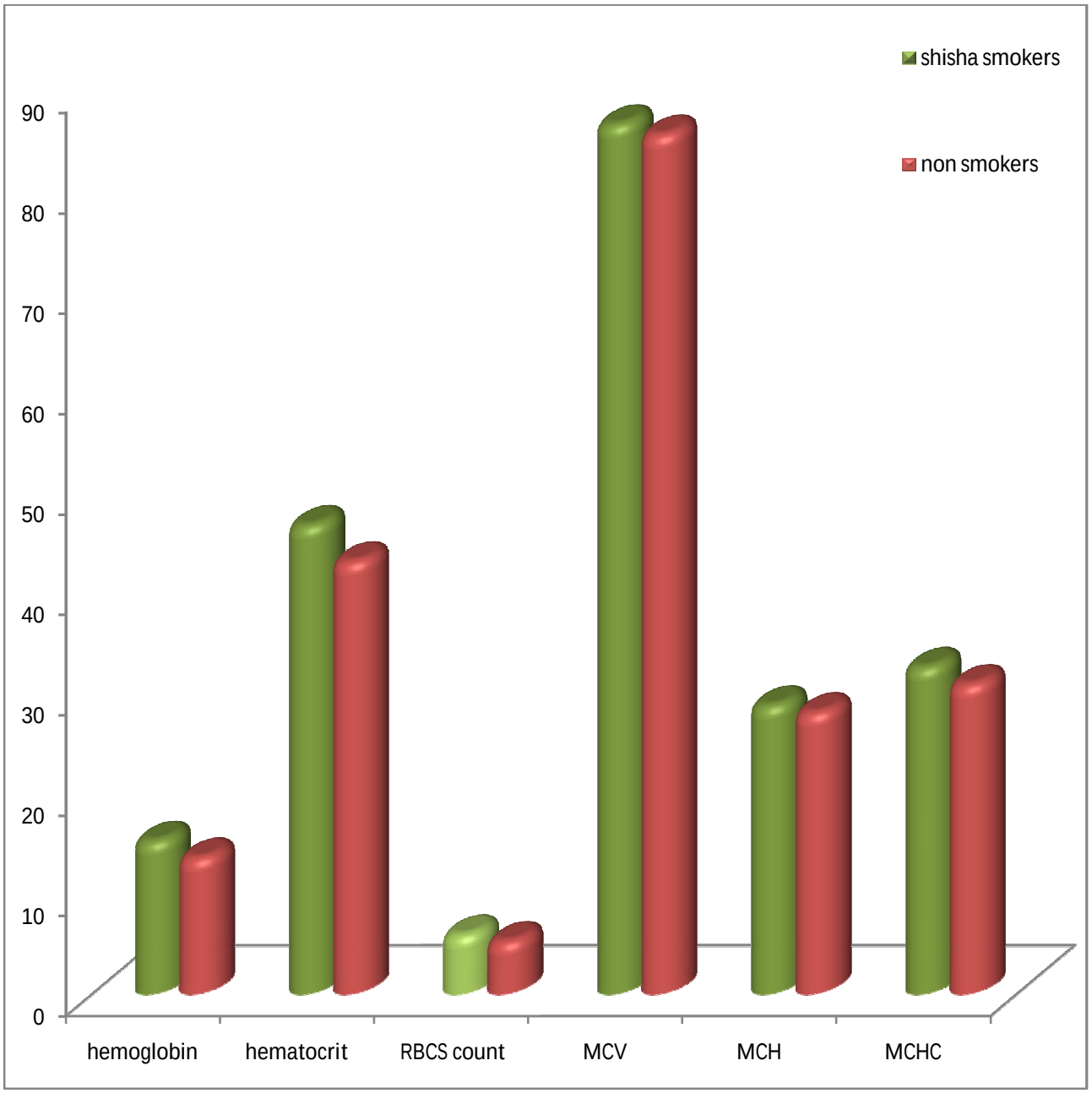

Figure 3.3: hemoglobin, hematocrit, red blood cells counts, MCV, MCH, and MCHC of shisha smokers and non-smokers.

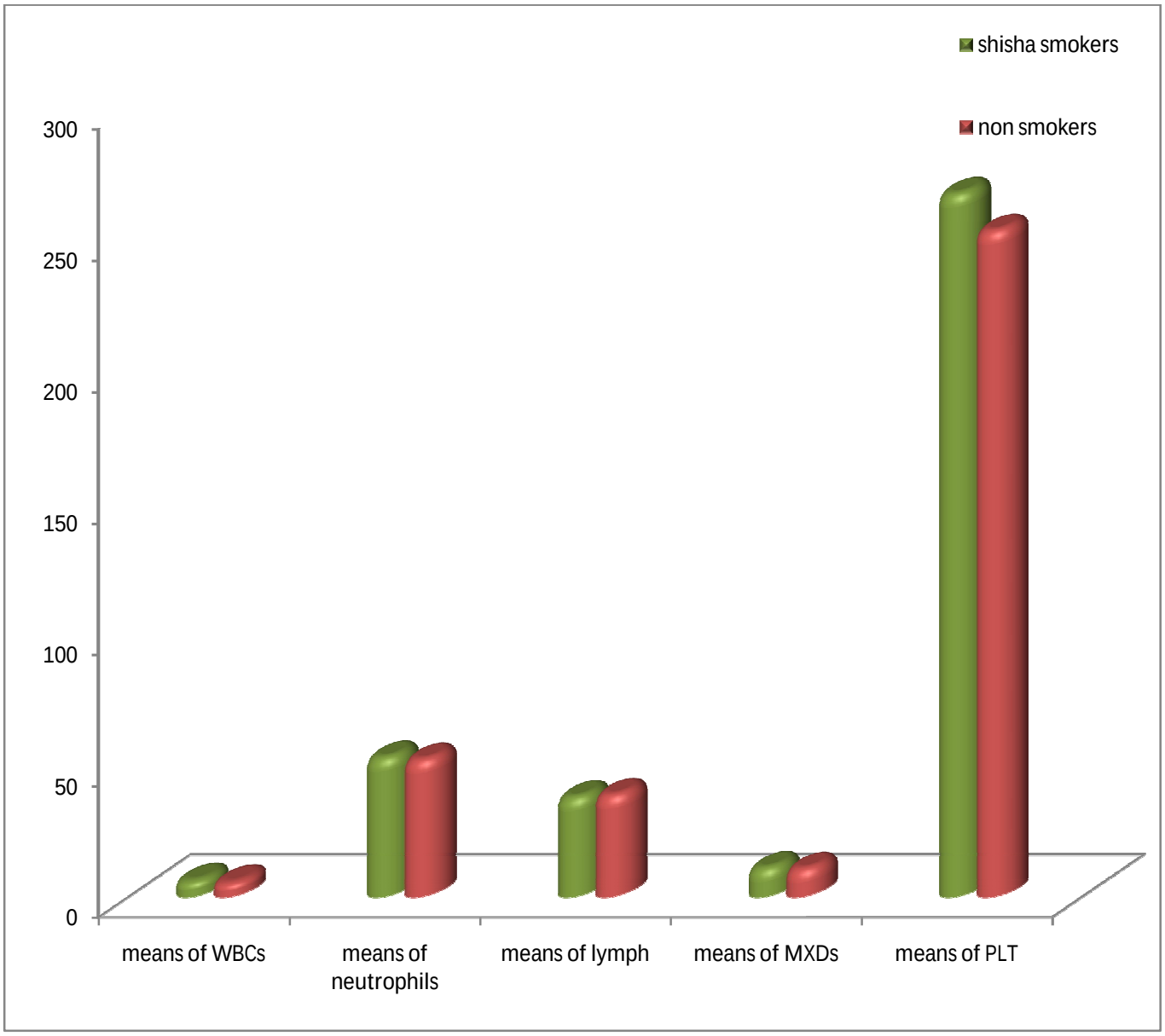

Figure 3.4: means of total white blood cells counts, neutrophils %, lymphocytes % and MXDs % of shisha smokers and non-smokers.

4.1. Discussion

Recently, the rate of cigarette smoking increased among young people due to increase number of new devices concerning multimedia and direct and indirect advertisements. This help in production of new tobacco products other than industrial cigarettes with pleasant odors and many other factors.

The total erythrocytes, hemoglobin concentration, hematocrit and mean cell hemoglobin concentration was increased significantly in cigarette smokers with (p.value 0.000) when compare with non-smokers, however for shisha smokers when compared with non-smokers the total erythrocytes, hemoglobin concentration, hematocrit and mean cell hemoglobin concentration was increased significantly with (p.value 0.000), this indicated to that cigarette and shisha smoking can develop secondary polycythemia in the future as the result from increased levels of carboxyhemoglobin which causes clinically significant hypoxemia and account for the increased erythrocyte masses, this results agree with Muhammad *et al*; (2013) and agree with Pankaj*et al; (2014).*who stated that values for total erythrocytes, hemoglobin concentration, hematocrit and mean cell hemoglobin concentration were significantly higher in the smokers.

Mean cell hemoglobin was increasedsignificantly in shisha smokers when compare with non-smokers with (p. value 0.023), this result agree with Muhammad *et al*; (2013) who stated that shisha smoking cause elevated in mean cell hemoglobin.

There was no significantdifference between shisha smokers and non-smokers in mean cell volume, platelets counts, lymphocyte%, neutrophil% and MXDs% these results disagreed with result Bain; *et al* (2001) who stated that shisha smoking causes elevated in platelets counts, lymphocyte% and neutrophil%.

There was no significant difference between cigarette smokers and non-smokers in mean cell volume, mean cell hemoglobin, platelets counts and MXDs% these results similar to result obtained by Pankaj; *et al* (2014) who stated that was no significant difference between cigarette smokers and non-smokers in platelets counts, mean cell volume, mean cell hemoglobin and MXDs%.

Total leukocyte counts was significantly elevated in cigarette smokers and shisha smokers when compared with non-smokers p.value(0.002) and (0.011) respectively these similar to result obtained by Besime*et al*; (2014) who stated that cigarette and shisha smoker causes elevated in WBC counts, however results obtained by Edward, (2006). Showed that, the smoking is a universally accepted major cardiovascular risk factor, but the mechanisms by which it promotes ischemic vascular disease are not fully understood. It is well documented that smoking increase total white blood cell count and modifies leukocyte function. Neutrophils% and lymphocytes% in cigarette smokers when compare with control group show significant difference (p. value = 0.013) and (p. value = 0.045) respectively which agree with tanasan*et al*; (2012), who stated that smoking cause elevated in Neutrophils% and lymphocytes%.

The result of the present study showed that the effect of the smoking on hemoglobin concentration is directly proportional to the duration of smoking.

4.2.Conclusion

Erythrocyte count, hemoglobin concentration, hematocrit, leukocyte count and mean cell hemoglobin concentration increased in cigarette and shisha smokers compared to control group.

No change in mean cell volume for both cigarette and shisha smokers compared with non-smokers.

No change on Platelets counts and differential leukocyte count was detected in shisha smokers compared to control group.

In cigarette smokers Neutrophils % and lymphocytes % when compare with control group show significant difference.

Effect of the smoking on hemoglobin concentration is directly proportional to the duration of smoking.The final outcome of general health in the prolonged smoking is more fetal. The cardiovascular diseases, atherosclerosis, polycythemia Vera, chronic obstructive pulmonary disease is highly incidence in smokers. The very high voice now listens in all medical committee about the relationship between smoking and infertility.

4.3.Recommendation

Awareness about the side effects of cigarette and shisha smoking.

Health education programs emphasizing on this bad habits should be implemented and directed towards target groups in the school children and university students.

More research with larger sample size and more suitable study design (cohort study) should be conducted to give very reliable and applicable result.

More hematological parameters as erythrocyte sedimentation rate(ESR) should be included in the future research.

More advanced technique as Cell markers, follow cytometer and gene study should be used to make the picture more illuminatedin the future research.

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Appendix I

بسم الله الرحمن الرحيم

Questionnaire

Sudan University of Science and Technology

College of Graduate Studies

Effect of Cigarette and Water pipe Smoking on some Hematological Parameters among Sudanese in Khartoum State

-Age:

-Smoker { } - non-smoker { }

-Smoking cigar { } - smoking shisha { }

-No of cigarette per day { } - how many times per day { }

-Duration of smoking { }

-Cardiovascular disease { }

-Respiratory disease { }

-Sport { } -not sport { }

-others:_____

Appendix II

بسم الله الرحمن الرحيم

جامعة السودان للعلوم والتكنلوجيا

كلية الدراسات العليا- برنامج الماجستير- مختبرات طبية

موافـــــــــــقة

الاسم:.....

سوف يتم أخذ عينه دم من الوريد بواسطة حقنة طعن وذلك بعد مسح منطقة العينه بواسطة المطهر وكل الأدوات المستخدمة لأخذ العينة معقمة ومتبع فيها وسائل السلامة المعملية, و ذلك لمعرفة تأثير تدخين الشيشة و السجائر علي الدم و مكوناته.

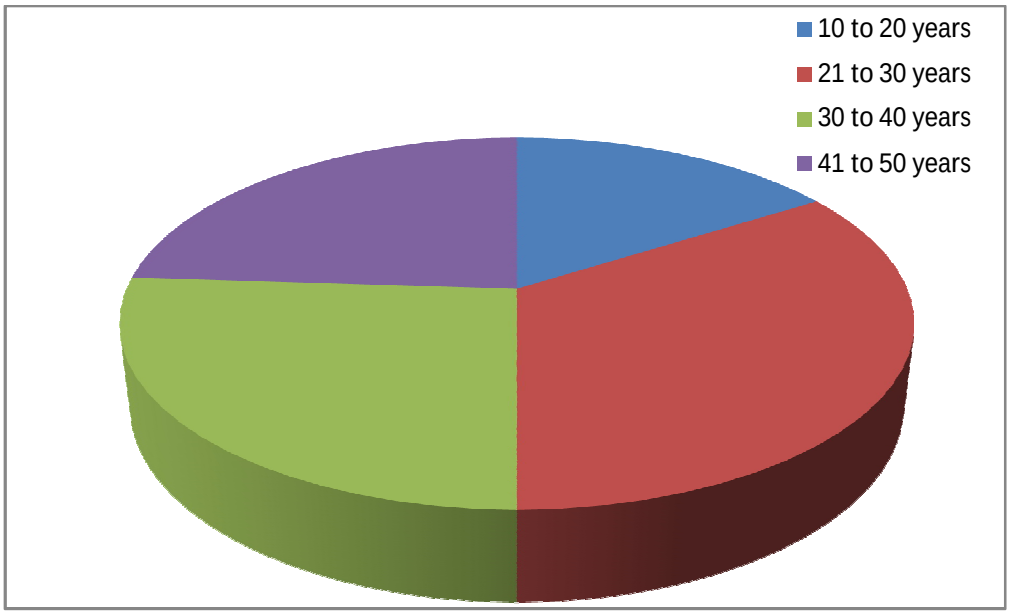
وأنا أقر بأن هده العينات سوف يتم تحليلها فقط لطلب البحث.

أوافق أنا المذكور أعلاه أخذعينه لأجراء الدراسة

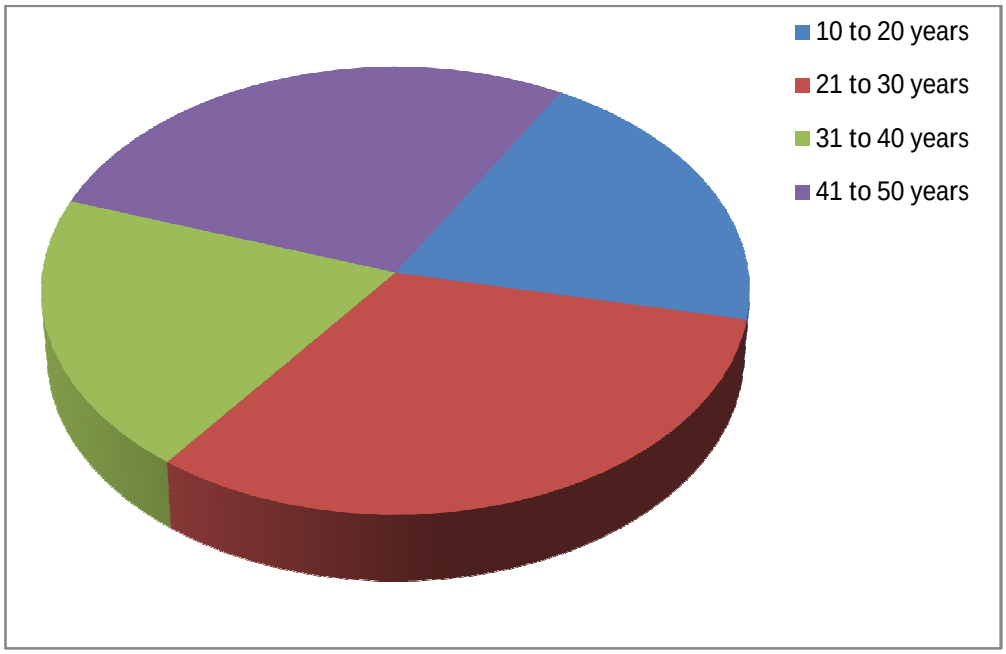
الامضاء: .....

التاريخ: .....

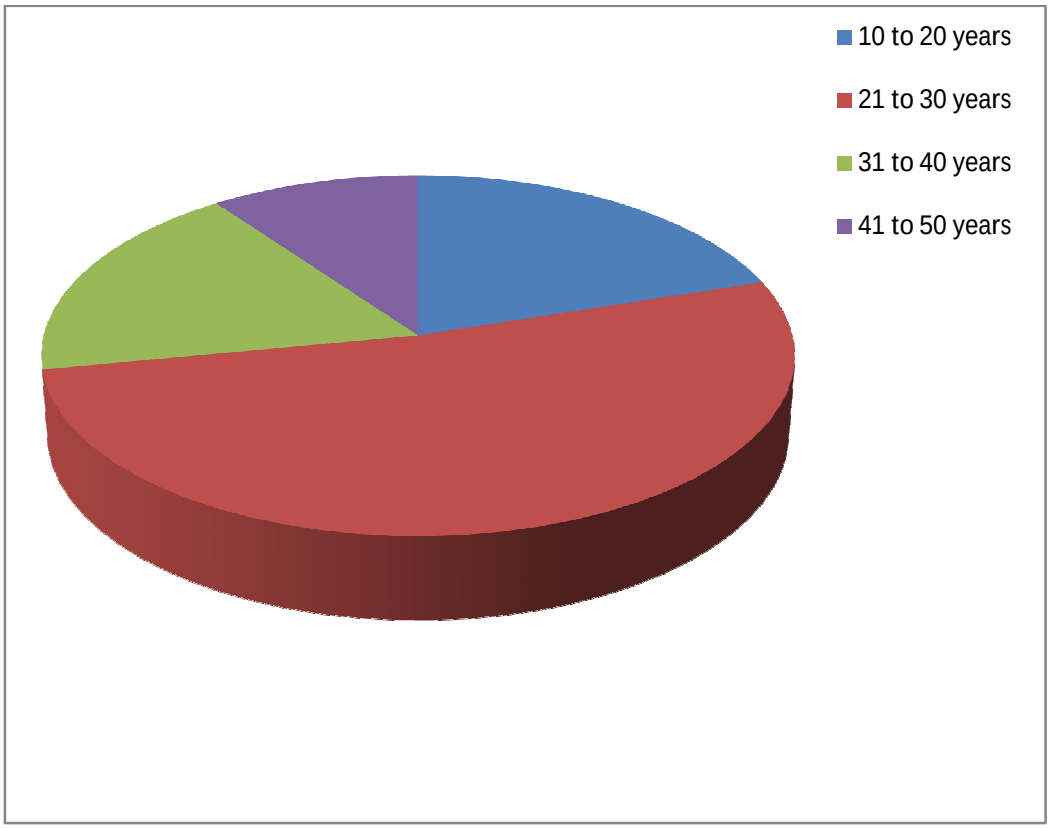
Appendix III



Age distribution of cigarette smokers



Age distribution of shisha smokers



Age distribution of non-smokers (control group).

AppendixIV

SYSMEX KX model 21N hematology analyzer