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A STUDY OF LIPID PROFILE AND SERUM URIC ACID LEVELS IN SMOKERS AND NON-SMOKERS

Shubhra Chowdhry, Roshan Alam[1], Saba Khan[1] and Jyoti Verma[2]

[1]Department of Biochemistry, Integral Institute of Medical Sciences & Research, Integral University, Lucknow, India.
[2]Department of Medicine, Integral Institute of Medical Sciences & Research, Integral University, Lucknow, India.

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ABSTRACT : Smoking is a demonstrated health hazard. It ranks second as the leading cause of death in the world. Smoking is associated with a more atherogenic lipid parameters. It increases the serum concentration of total Cholesterol, triglycerides, LDL-Cholesterol, VLDL-Cholesterol and declines the level of good Cholesterol *i.e.* HDL-Cholesterol. Whenever, smoking is done oxidative stress is increased. It has also been postulated that the level of uric acid decreases when smoking duration is more than 5 years. The study involved 30 smokers and 30 controls selected from subjects attending the Medicine OPDs at IIMS & R. Lipid profile and serum uric acid levels were estimated. The data was analyzed using SPSS software 20.0. The serum triglycerides, total cholesterol, LDL-C and VLDL-C levels were higher in smokers as compared to controls whereas the HDL-C and serum uric acid levels, were lower in smokers as compared to non smokers. This study clearly reveals a strong relationship between smoking and increase in serum triglycerides, total cholesterol, LDL-C and VLDL-C levels and decrease in HDL-C. This relationship is of great significance since this is atherogenic in nature. The low plasma uric acid level in smokers is a significant source of oxidative stress.

Key words : Smoking, lipid, cholesterol, triglycerides, LDL, VLDL, HDL, serum uric acid.

INTRODUCTION

Smoking is a complex and significant neurochemical and conduct issue disorder under the impact of social, ecological, psychologic and biologic variables (Fisher *et al*, 1990; Fisher *et al*, 1988). Smoking is the act of breathing in smoke, produced by the burning of a component, through the mouth, more often of tobacco in a cigarette, cigar, or pipe. Smoking frequently, particularly cigarettes, which contain numerous lethal substances, for example, nicotine or tar, is a proven health danger [CCM Health (health.ccm.net)].

In India tobacco kills 8–10 lakh individuals every year and major share of these deaths occur exclusively in youngsters. It has been assessed that a normal of five and-a-half minutes of life is lost for every cigarette smoked [(GATS) Fact sheet India, 2009-2010]. In excess of 5 million of those unexpected losses are the consequence of direct tobacco utilization while more than 600,000 are the aftereffect of nonsmokers being presented to second-hand smoke (WHO Tobacco fact sheet, 2016).

It has been seen that smoking is associated with a more atherogenic lipid profile (Gosette *et al*, 2009). It increases the serum concentration of total Cholesterol, triglycerides, LDL-Cholesterol, VLDL-Cholesterol and declines the level of good Cholesterol i.e. HDL-Cholesterol (Adam *et al*, 2012; Kavita *et al*, 2013). Subsequently, smoking is a noteworthy hazard factor for atherosclerosis and coronary artery disease (Fagerstrom, 2002; Vlassis, 2009). The number of cigarettes/ beedis smoked and cardiovascular morbidity and mortality have a direct dose response relationship (Wynder *et al*, 1989).

Generally, overproduction of reactive oxygen species (ROS) or a deficiency in endogenous antioxidant defence system, including enzymatic and non-enzymatic antioxidants has been defined as oxidative stress (Daniela Giustarini *et al*, 2009).

It has been seen that as tobacco smoke contains superoxide and reactive nitrogen species that promptly react with different biomolecules and a reduction of antioxidants, including uric acid, in smokers, show that oxidative stress increases each time a cigarette is smoked (Tsuchiya *et al*, 2002). It has been speculated that a portion of the unfavorable impacts of smoking may come about because of oxidative harm to endothelial cells, which brings about nitric oxide (NO) shortage. (NO) shortage controls vascular tone that quickens inadequacy of coronary artery and vasoconstriction in a wide range of tissues (Benzuly *et al*, 1994).