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EFFECT OF CIGARETTE SMOKING AND 3.2% ETHANOL ALONE OR TOGETHER ON RBC AND PLATELET COUNTS IN RATS

M. NAGVEKAR* AND M. E. ABRAHAM

Department of Physiology,
Goa Medical College,
Bambolim, Goa – 403 202

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Abstract : The present study was conducted on 32 rats divided into four groups. Group C served as control, Group S rats were exposed to cigarette smoke alone, Group SE to cigarette smoke and ethanol (3.2%) and Group E to ethanol alone for twelve weeks. The basal RBC and platelet count were determined and compared with the values obtained at the end of 12 weeks. A significant increase in RBC and platelet counts was seen in Groups S ($P<0.001$ and $P<0.01$ respectively) and SE ($P<0.01$ for both counts). The increase in group SE is less than that seen in Group S. Ethanol consumption alone has shown a significant decrease ($P<0.01$) in RBC count and apparent decrease in platelet count as compared to control. This study indicates that cigarette smoke is damaging to health alone or when combined with ethanol.

Key words : ethanol smoking RBC count platelet count

INTRODUCTION

It is known that smokers have a high mortality. The causes of the excess mortality include lung cancer, chronic bronchitis, emphysema and cor-pulmonale. Smoking is also a major cause of atherosclerotic disease and is considered one of the three major risk factors for coronary heart disease along with high blood pressure and cholesterol disorders (1). Smoking has both acute and chronic effect on haemotologic systems. There is increase in platelet activation, agregation adherance, thromboxane release and plasma viscosity (1).

Alcohol consumption has adverse effects on health but alcohol also has a cardioprotective role. Moderate drinkers have a reduced risk of death compared with nondrinkers or heavy drinkers (2). Smoking results in increased lipid and lipoprotein levels except HDL cholesterol which is diminished. Alcohol may be having some beneficial effects on lipoprotein by causing low LDL, high HDL cholesterol and reduced platelet coagulability. Alcohol alters eicosanoid metabolism in favour of increased prostacyclin and decreased thromboxane synthesis (3).

Smoking is injurious to health. Inspite of this warning we see people smoking. Alcoholism and smoking mostly go together.

*Corresponding Author

In order to assess the risk of coronary heart disease in such cases a preliminary study was done on total R.B.C. and platelet count in rats exposed to cigarette smoke, rats on ingestion of ethanol and in both.

METHODS

32 male adult albino rats were used in this study. They were housed in individual polyvinyl cages. RBC count and platelet count were determined in all the rats. They were divided in four groups.

Group C (n = 8) Served as control. Food and water given *ad libitum*.

Group S (n = 8) Food and water *ad libitum*, exposed to cigarette smoke for one hour every day in air tight glass chamber.

Group SE (n = 8) Exposed to smoke as in Group S animals, food *ad libitum*, 3.2% ethanol was fed instead of water.

Group E (n = 8) Food *ad libitum*, 3.2% ethanol ingestion.

After 12 weeks RBC count and platelet count were repeated.

Means of RBC and platelet count in each group were calculated, standard errors of mean were found and the values were compared.

RESULTS

RBC count – RBC count increased significantly in Groups S ($P<0.001$) and SE ($P<0.01$) as compared to Group C and decreased significantly in Group E ($P<0.01$). Increase in group S is more than in group SE and this difference is also statistically significant ($P<0.01$). A significant decrease was seen in group E rats as compared to the other groups ($P<0.01$).

Platelet count – Compared to control group, there was a significant increase in group S ($P<0.01$) and SE ($P<0.01$) rats whereas in group E rats, platelet count is less compared to group C though the decrease was not statistically significant. In group S and SE platelet count is increased though the increase is more in group S as compared to group SE rats. As compared to group S and SE rats in which smoking was a common factor in group E rats platelet count was low and the difference was very significant ($P<0.001$).

TABLE I: RBC count in millions/μl in different groups.

	Group C	*Group S*	*Group SE*	*Group E*
Mean	9.76	13.95	11.35	8.58
S.E.	0.2628	0.5455	0.2505	0.5704

Group C – control
Group S – smoking
Group SE – smoking and ethanol
Group E – Ethanol

(i) Group C/S	t = 6.921	P<0.001 HS
(ii) Group C/SE	t = 4.331	P<0.01
(iii) Group C/E	t = 4.014	P<0.01
(iv) Group S/SE	t = 4.331	P<0.01
(v) Group S/E	t = 6.452	P<0.001 HS
(vi) Group SE/E	t = 4.001	P<0.01

TABLE II: Platelet count in lacs/µl in different groups.

	Group C	*Group S*	*Group SE*	*Group E*
Mean	1.49	3.52	3.03	0.94
S.E.	0.3429	0.1682	0.0857	0.1339

Group C – control
Group S – smoking
Group SE – smoking and ethanol
Group E – Ethanol

(i) Group C/S	t = 3.206	P<0.01
(ii) Group C/SE	t = 4.357	P<0.01
(iii) Group C/E	t = 1.494	P<0.01 NS
(iv) Group S/SE	t = 0.9092	P<0.03 NS
(v) Group S/E	t = 4.707	P<0.01
(vi) Group SE/E	t = 13.169	P<0.001 HS

DISCUSSION

Cigarette smoke contains carbonmonoxide. The average COHb found in smokers is 50% which interferes with oxygen transport and utilisation. Chronic mild elevation of COHb is a common cause of mild polycythaemia (4). Cigarette smoke results in numerous pathophysiologic effects including changes in the central and peripheral airways, alveoli, capillaries and the immune system leading to chronic obstructive pulmonary disease (COPD). Smoking reduces tissue oxygen delivery and stimulate erythropoiesis (5).

Platelet activity and survival appear to be adversely affected by chronic smoking. Smoking causes acute and chronic inhibition of cyclooxygenase which inhibits prostacyclin and increase the biosynthesis of thromboxane. Thromboxane is a potent vasoconstrictor and platelet agonist (1). This may be contributing to higher platelet count in smokers. Polycythamia increases in fibrinogen and factor VII level and platelet dysfunction contribute to cardiovascular disease (1).

In our study compared to control group, rats exposed to smoking (group S) showed increase in RBC count from 9.76 to 13.95 million/µl and this rise was highly significant. Platelet count also increased and this rise was also significant.

Group E animals compared to group C showed decrease in RBC and platelet count though platelet reduction was not statistically significant. A fall in RBC count decreased haemotocrit values and haemoglobin levels and leucopenia have been reported following large intake of alcohol (6). Alcohol may interfere with folate metabolism causing folate deficiency. Deficiency may be also due in part to malabsorption (7). Many alcoholics present with mild thrombocytopenia due to a decrease in platelet survival and altered function. Alcohol may decrease platelet agregation and inhibit release of thromboxane A_2 (8). Prolongation of bleeding time was associated with a history of alcohol use (7). During period of abstinance in alcoholics, platelet counts rise, the largest rise occuring in these patients with lowest counts on admission. Bleeding times

returned to normal during abstinance (9). The rebound thrombocythaemia suggest that the bone marrow is no longer suppressed by ethanol and it releases young platelet which are hyperactive. The cardioprotective role of ethanol can be explained by ethanol related changes in high density lipoprotein cholesterol and reduced platelet coagulability (3).

In group SE, animals RBC and platelet counts are increased significantly compared to control. But the rise is less compared to group S. In this group, the effect is due to both smoking and ethanol. The adverse effect of smoking have overcome the effects of ethanol. Rats which are on ethanol consumption show decrease in RBC and platelet count. Determination of bleeding time, clotting time and haemotocrit values would have supported our findings. From this study we can conclude that when ethanol consumption is associated with smoking, beneficial effect of ethanol is overcome and hazardous effects of smoking are seen.

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