

*Short Communication***ASPIRIN BRINGS ABOUT ITS BENEFICIAL EFFECTS BY MODIFICATION OF PLASMA CHOLESTEROL CONCENTRATIONS****Dr. Ayed Mizher Habeeb Al-Shammari^{1*}, Dr Ian Naylor², Professor Robert Naylor²**

1. Al-Jahra center Kuwait.
2. School of Pharmacy University of Bradford, Bradford, West Yorkshire, United Kingdom

ABSTRACT

The active component of smoke which is nicotine plays its major role of for deposition of cholesterol in the inner wall of arteries as acting as centre of accumulation of fats. This cholesterol which is considered one of the major factors of high blood pressure and heart attack, which deposited on inner walls of arteries due to involvement of nicotine and shorting the passage of blood which finally develop a high blood pressure. It has found that aspirin effectively dissolve the cholesterol and normalize the blood pressure and acts as dilator [dilating the arteries for normal flow of blood]. The concentrations of cholesterol in milli mole were treated with aspirin and it has been found that aspirin effectively treated against higher concentration of cholesterol in blood.

Key Words: Smoker, cholesterol, aspirin, cardiac risk

Corresponding Author: Ayed Mizher Habeeb Al-Shammari (B-Pharm, M. Pharm, PhD Clinical Pharmacy) P.O.Box 2611 code 01028 Al- Jahra center Kuwait. Tel.: +96599620544, Email: d.rr@hotmail.com

INTRODUCTION

Nicotine is absorbed from the respiratory tract, and through mouth tissue and skin. Approximately 80% to 90% of nicotine is metabolized in the liver, kidneys and lungs. The lungs metabolize a major portion of inhaled nicotine. The major metabolites of nicotine are cotinine and nicotine-N-oxide. The half-life of nicotine after inhalation or injection administration is about 2 hours. The kidney eliminates both nicotine and its by-products. [1]

The brain requires high levels of oxygen and if the arteries in the brain are clogged with plaque or clot, atherosclerotic disease advances rapidly with the possibility of clot formation which can lead to a possibility of stroke and death. With more smoking, the level of nicotine in blood will increase, and chances of high blood pressure increases with the increase in concentration of nicotine in blood. [2]

Correlation of anti-enzyme activity of aspirin with its Aspirin inhibits cyclooxygenase (prostaglandin synthetase) thereby reducing the synthesis of prostaglandins and thromboxanes. These effects are thought to be how aspirin produces analgesia, antipyrexia, and reduces platelet aggregation and inflammation. Mast cells can synthesize new cyclooxygenase, but platelets

cannot. Therefore, aspirin causes an irreversible effect on platelet aggregation. Aspirin has been shown to decrease the clinical symptoms of experimentally induced anaphylaxis in calves and ponies. [3]

Aspirin is a weak organic acid, with a pKa of 3.5. Taken orally, it is absorbed in the stomach and the upper small intestine, mainly in the non-ionized form due to the acidic conditions in the stomach. Once absorbed, it is metabolized to acetic acid and salicylate by esterases in tissue and blood. The free salicylate is then excreted unchanged, or converted to other water-soluble compounds that are then excreted by the kidney. Salicylate and its metabolites are rapidly excreted by the kidneys by both filtration and renal tubular secretion. Significant tubular reabsorption occurs which is highly pH dependent. Salicylate excretion can be significantly increased by raising urine pH to 5-8. Salicylate and metabolites may be removed using peritoneal dialysis or more rapidly using hemodialysis. [4]

METHODOLOGY

The aim of this study to determine the effectiveness or otherwise of aspirin treatment the 186 patients were divided into smokers and non smokers and then divided into three groups made on the basis of their original plasma cholesterol concentration. The patients were placed into three groups namely: a) ≥ 5.9 , b) 5.9-6.7 and c) ≥ 6.7 mmol/L. This resulted in the number of patients who smoked in each of these groups being, 48, 39 and 58 respectively. The total number of patients used in this part of study was 186 (figure 1) of whom 145 were smokers.

RESULT AND DISCUSSION

Figure 1. The numbers of subjects in each group and their plasma cholesterol concentration

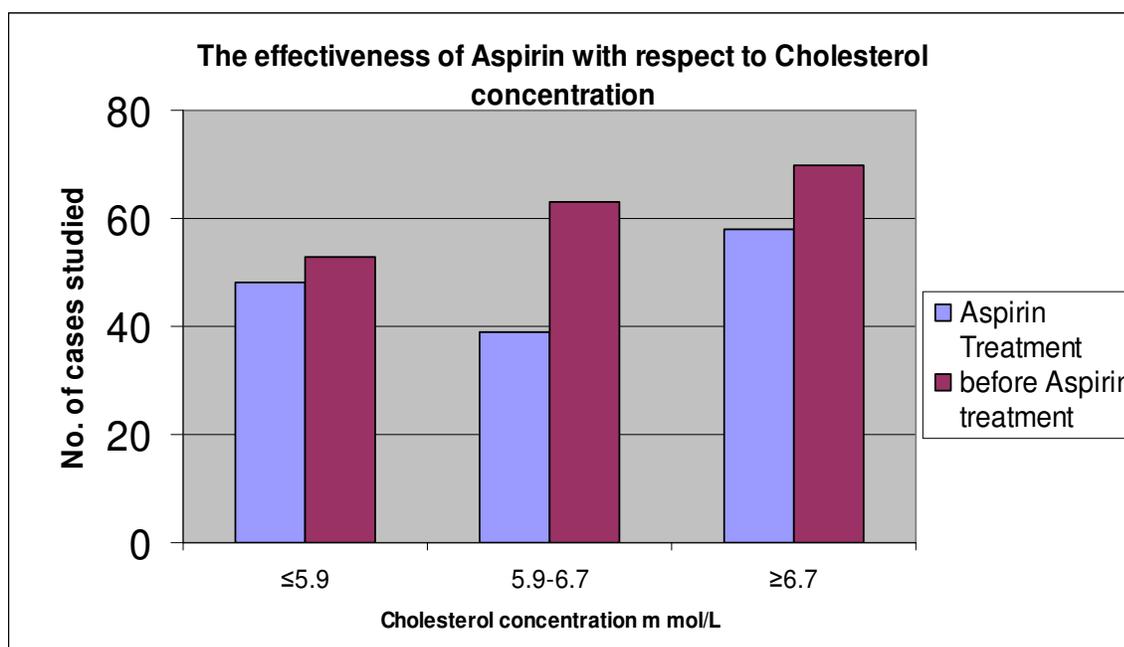
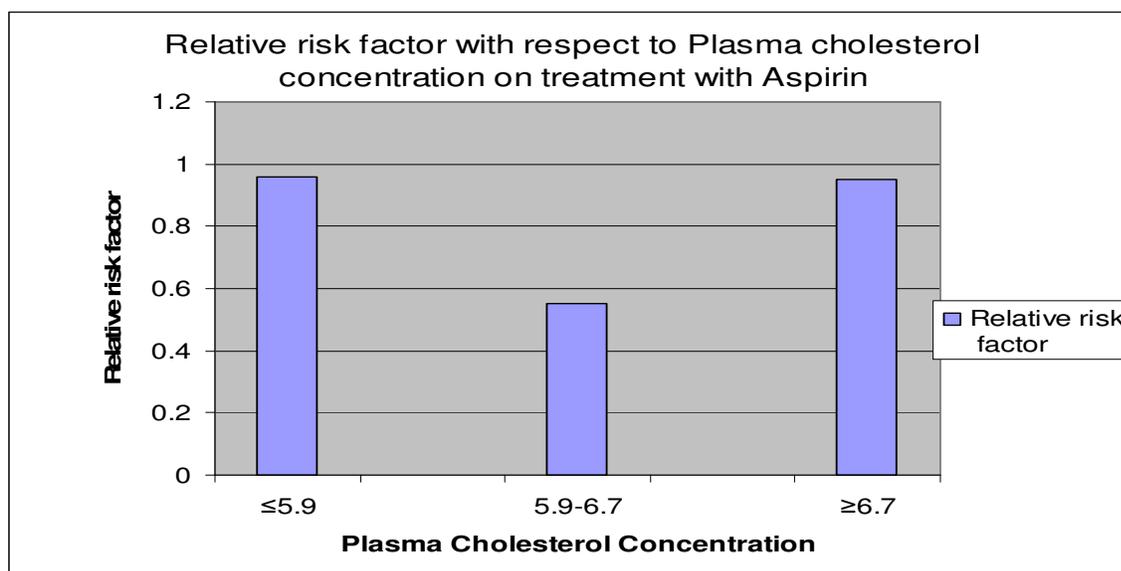


Table 1. The distribution of patients into three 'plasma cholesterol levels' and the numbers treated by aspirin (75mg/day for x months). The difference between the total number of patients and the treated group are those that received aspirin preparations.

Plasma cholesterol concentration (mmol/L)	Total patients (n=186)	Aspirin treatment (n=145)	Relative risk (to show the benefit of aspirin treatment)
≤5.9	53	48	0.96
5.9-6.7	63	39	0.55
≥6.7	70	58	0.95

Figure 1. Bar chart of the numbers of subjects in each group as defined by their plasma cholesterol concentration of i) ≥ 5.9 , ii) 5.9-6.7 and iii) ≥ 6.7 mmol/L. The number of subjects who received aspirin is shown as blue blocks and before aspirin treatment as pink block for each of the three cholesterol plasma level groups which were studied. In this experiment based on the results that aspirin treated groups received 75mg per day for one and half months (45 days)

Of the 48 patients who smoked and had a cholesterol concentration of less than 5.9 mmol/L, the aspirin treatment brought about a very small reduction in the plasma concentration and this was reflected in a relative risk of 0.96 (see Table 1). In the second group, of 39 patients who had a cholesterol concentration between 5.9mmol/L to 6.7mmol/L, aspirin brought about an even greater reduction in plasma cholesterol levels which altered their risk of cardiovascular problems as shown by the relative risk factor of 0.55. In the final group the 58 patients treated with aspirin and whose initial cholesterol concentration was greater than 6.7 mmol/L a similar relative risk to those below 5.9 mmol/ L was recorded.



The total patients admitted high cholesterol concentration were 186 but only 145 who were smoker and also have high cholesterol concentration were benefited by aspirin treatment. To prove the effectiveness of aspirin three groups of 186 patients has been made on the basis of cholesterol concentration. High levels of cholesterol in the blood can damage arteries and are potentially linked to diseases such as those associated with the cardiovascular system (heart

disease). 48 patients who have cholesterol concentration less than 5.9mmol/L has been cured with aspirin treatment where as total admitted patient of this group were 53. The patients which have cholesterol concentration between 5.9mmol/L to 6.7mmol/L have also been treated with aspirin and only 39 patients out of 63 were benefited by this treatment. 58 patients who have heart problem due to cholesterol concentration greater than 6.7 were treated with aspirin and aspirin treatment proved very beneficial for all of them for overcoming their heart problems.

CONCLUSION

Perhaps the last finding indicates that the changes induced by a high plasma cholesterol concentration are such, that reversal of the long lasting effect of cholesterol, in terms of cardiac risk factors, is extremely difficult to overcome simply by the use of aspirin.

Plasma cholesterol levels are considered to be one of the major risk factors along with high blood pressure to make heart attacks and strokes more likely to occur. The finding that aspirin can reduce this level in the group 5.9 – 6.7 mmol/L is potentially of great significance.

ACKNOWLEDGMENT

All aspects of this study were read by and approved by authorities in Kuwait who give ethical approval for such studies to be undertaken. I am extremely grateful for pharmacy and nursing staff in the Ministry of health (Chest hospital) in the state of Kuwait for being patient and kind when I was collecting the relevant data.

REFERENCE

1. Tlisara S.; Elisaf M.; Dimitri P. "Influence of smoking on predictors of vascular disease." "Angiology" Vol.54; No.5 Page 507-530, 2003.
2. Wannamethee S.G.; Sharper A.G.; Whincup P.H.; Walker M. "Smoking cessation and the Risk of stroke in middle-aged men" "JAMA" 274:page.155-160, 1995.
3. Philp B. Gorelick; Ralph L.; Don B.; Mark Alberts.; Lisa M. Alexander.; Dan Rader.; Joycel Ross.; Eric Raps.; Mark Ozer.; Lawrence M.; Mary E.; Sheldon Goldberg.; John Booss.; Daniel F.; James F.; Nancy L.; Green gold.; David . "Prevention of a first stroke" "JMA" 281, (12):page.1112-1120, 1999.
4. Robert G.Hart.; Michael J.G.; Harrison. "The optimal dose of Aspirin to prevent stroke" "American Heart Ass." Vol.27.page.585-587, 1996.