

A STUDY OF PLASMA LIPID PEROXIDATION, LIPIDS AND BLOOD SUGAR LEVEL IN OPIUM ADDICTS COMPARED WITH CONTROL GROUP

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Abstract

INTRODUCTION: Addiction to opioids is a major health challenge in the world today. A few studies have addressed the effects of these substances and the wrong beliefs surrounding their use. This study was designed to compare plasma lipid peroxidation levels, blood lipids, fasting blood sugar (FBS), and glycosylated hemoglobin (HbA1C) in opium addicts and non-addict control subjects.

METHODS: This case-control study was conducted on a sample consisting of 64 men. The control group comprised 32 cigarette smokers who were studied by urine morphine strip test. The case group included 32 opium addicts with a history of vaporizing addiction 1 g/daily for at least 3 years. Data were analyzed by T-test using SPSS and EP16 statistical software.

RESULTS: This study showed no significant difference in FBS, HbA1C, triglyceride, total cholesterol, HDL-C, LDL-C, and lipid peroxidation between case and control groups.

DISCUSSION: The results of this study show that opium addiction has no effects on blood sugar or other CVD risk factors and increases the level of malondialdehyde, an important CVD risk factor.

Keywords • Malondialdehyde • Triglyceride • Cholesterol • Glycosylated hemoglobin

Fasting blood sugar • Addiction, Opium

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Introduction

Addiction to narcotics places a great burden on human societies. Opium is derived from poppy plant and contains more than 20 kinds of alkaloids. The prevalence of addiction varies between different countries, cultures and jobs.

In the United States, despite strict anti-narcotics laws legislated in 1914, the use of narcotics especially heroin has increased in recent years.

About 2.5 millions heroin addicts have been reported in the US.⁸ Despite nationwide anti-drug campaigns, addiction to narcotics has increased in Iran in recent years.

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Addiction is rooted in ethnic, hereditary, biological, physical, psychological, social, cultural, familial, occupational and economic factors. Studies show that heroin addiction causes hyperkalemia and morphine use can result in calcium inhibition, hypercholesterolemia¹² and inhibition of vascular endothelial growth factor (VEGF) expression in myocardial ischemia.¹³ Central administration of opiates and opioid peptides may act indirectly via the sympathetic nervous system to cause hyperglycemia and impaired insulin secretion, while peripheral administration tends to stimulate insulin and glucagons secretion.¹⁴ Thus, heroin addicts, like patients with non-insulin-dependent diabetes, do not respond appropriately to glucose signals.¹⁴

The increase in blood lipids, lipid peroxidation and blood sugar are important CVD risk factors, hence this study intended to investigate the effect of opium on these biochemical factors.

TABLE 1. Comparison of biochemical factors (mean values) between the case and control groups

| | Case group (n=32) | Control group (n=32) | P value |
|---------------------|----------------------------------|-----------------------------|----------------|
| Biochemical factors | FBS (mg/dl) | 84.03±26.8 | 0.46 |
| | HbA1C (%) | 8.04±0.91 | 0.15 |
| | HDL-Ch (mg/dl) | 40.17±8.99 | 0.32 |
| | LDL-Ch (mg/dl) | 127±41.48 | 0.42 |
| | Total-Ch (mg/dl) | 208±52.5 | 0.22 |
| | TG (mg/dl) | 204±157 | 0.43 |
| | Malondialdehyde (μ mol/lit) | 1.15±0.2 | 0.76 |

Materials and methods

In this case-control study, 32 male opium addicts were selected as the case group and 32 male non-addict cigarette smokers as the control group. Urine analysis with morphine strip test was performed to rule out addiction in control group. As the majority of opium addicts were also cigarette smokers, the control group in this study was selected among cigarette smokers to eliminate the effect of cigarettes. The group of addicts comprised 25-40 year-old men who had used 1 g opium daily for at least 3 years via inhalation. The control group consisted of 25-40 year-old non-addict cigarette smoking men. Patients with diabetes, hypertension and hypercholesterolemia were excluded from both groups. No one in this study was on a special medication or diet.

Information was obtained by questionnaires. Questionnaires collected information about duration of opium addiction and concomitant cigarette smoking, history of specific disease and physical examination data. Blood samples in both groups were obtained from veins and all of the samples were examined at the laboratory of Isfahan Cardiovascular Research Center. Fasting Blood Sugar (FBS), Total Cholesterol (TC), High-Density Lipoprotein (HDL) and Triglyceride (TG) were measured by Pars Azmoon kit and Elan autoanalyzer and Low-Density Lipoprotein (LDL) was calculated according to formula. Glycosylated hemoglobin (HbA₁C) was measured using the colorimetric method and malondialdehyde (MDA) was measured by Shimadz spectrophotometer.^{2,3,5} Data were analyzed by t-test using SPSS and EPI6 statistical software.

Results

There was no significant statistical difference between the case and control groups in respect of intensity of cigarette smoking and mean age. Mean duration of

addiction in the case group was 8.92±5 years. Mean daily opium use was 4.8±1.5 g.

Mean serum FBS level was 84.03±26.8 mg/dl in the case group and 90.03±32.3 ml/dl in the control group. There was no significant statistical difference between the two groups (Table 1).

In this study, mean HDL in the case group was lower than in the control group. There was no significant statistical difference between mean TG levels in the case and control groups. Mean MDA in the current study was higher in the case group than in the control group, but the difference was insignificant.

Discussion

In the current study, there was no significant statistical difference between the case and control groups in respect of FBS, HbA₁C, TG, Total Cholesterol, HDL, LDL and malondialdehyde levels. One study demonstrated that morphine administration increases blood sugar. In another study, morphine was added to the daily diets of rats and led to an increase in their total cholesterol and LDL levels.^{1,10} Studies on rats have demonstrated that morphine administration and a daily diet containing morphine decreases HDL. In heroin addicts, HDL has been reported at lower levels than controls.⁷ In this study, mean HDL in the case group was lower than in the control group, which can be construed as a greater risk of coronary disease in the case group.^{1,7} In some studies morphine administration to rats decreased TG levels. In the percent study, no significant statistical difference was observed between mean TG levels in the case and control groups. It has been confirmed that that lipid peroxidation and oxidative changes of lipoproteins underlie atherosclerosis.

MDA is a measurable index for lipid peroxidation. It has been demonstrated that morphine stimulates MDA production.⁶

In the current study, mean MDA in the case group was higher than in the control group but the difference was insignificant. Increase in sample size may yield a significant difference. Hence it can be argued that increase in peroxidation products like MDA can demonstrate the greater risk for cardiovascular disease.

This study showed that opium use has no beneficial effect on blood sugar or blood lipid levels. Insignificantly lower levels of some biochemical factors in the case group may be accounted for by economic and nutritional challenges affecting opium addicts.

The effect of opium on other important cardiovascular risk factors such as coagulation factors is considerable. One study demonstrated that fibrinogen, a major and independent risk factor in the development and progression of atherosclerosis increases in opium addicts.^{4,9,11}

References

1. Alibeh D. Effects of erythrosine on some antinociceptive and non nociceptive morphine in mice, General pharmacology: 1995;407-9.
2. Edmonds M, Eand. John, P. N. Measurement of stable glycated hemoglobin: Clin chem. 1987;391-402.
3. Esterbaure H, Cheesman KH. Determination of lipid peroxidation product: Malondialdehyde and 4-hydroxynoneal: Method Enzymol. 1990;186-407.
4. Kannel WB. Influence of fibrinogen on cardiovascular disease. Drugs 1997;54 (suppl 3),32-40.
5. Kostner K, Yang P, Neunteufl T, Glogar D, Weidringer F, Maurer G, Huberk. Is oxidative stress causally linked to unstable angina pectoris? A story in 100 CAD patients and matched controls. Cardiovas Res. 1997;330-336.
6. Lurie E, Solviova A, Alybievd T, Koplun A, Panchenk L, Shvest V. Effect of novel aromatic derivative of GABA on lipid peroxidation in chronically morphinized rats. 1995;36(1):13-9.
7. Maccari Z, Zannoni P, Plancher AC. Plasma cholesterol & triglyceride in heroin addicts. 1991; 29 (2):183-3.
8. Same JL. Drug abuse and dependence. In: Goldman, Bennet. Cecil textbook of medicine: Philadelphia. WB Saunders, 2000:54-55.
9. Smith FB, Lee AJ, Fowkes FG, Price JF, Rumley A, Lowe GD. Haemostatic factors as predictors of ischemic heart disease and stroke in the Edinburgh artery study. Arterioscler Thromb Vasc Biol 1997, 17(11), 3321-5.
10. Story M. V. Effects of morphine on plasma and tissue cholesterol. Life Science. 1997;25(2):302-3.
11. Way WL, Fields HL and Schumacher MA. Opioid analgesics and antagonists. In: Katzung BG (Ed). Basic and clinical pharmacology. 8th ed., New York, McGraw-Hill Co. 2001,512-531.
12. Mohs ME, Watson RR, Leonard-Green T. Nutritional effects of marijuana, heroin, cocaine and nicotine. J Am Diet Assoc 1990; 90(9): 1261-7.
13. Roy S, Balasubramanian S, Jinghua Wang, Chandrashekhar Y, Charboneau R, Roderick Brake. Morphine inhibits VEGF expression in myocardial Ischemic surgery, 2003;134(2):336-44.
14. Giugliano D. Morphine, opioid peptides and pancreatic islet function. Diabetes Care 1984;7(1):92-8.