

# Effect of Opium Addiction on Aspirin Resistance in Stable Angina Pectoris

Afsaneh Forood MD<sup>1</sup>, Reza Malekpour-Afshar MD<sup>2</sup>, Jamshid Sarnevesht MD<sup>3</sup>

## Original Article

### Abstract

**Background:** The rate of cardiovascular diseases in developing countries is approximately 60% and it is still has an increasing trend. The clinical effectiveness of aspirin in preventing cardiovascular events has been well proven. Although aspirin is an effective and inexpensive drug, its consumption is not equally beneficial for all patients. Many factors can be affective on the efficacy of antiplatelet drugs such as aspirin.

**Methods:** This study was carried out on 260 patients who had stable angina pectoris and coronary artery disease was approved by coronary angiography. Based on opium addiction, the patients were divided into two groups. Opium addiction was diagnosed base on Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) criteria. The mid-stream morning urinary sample were collected for measuring the urinary 11-dehydroxy thromboxane B<sub>2</sub> level (UTXB<sub>2</sub>). Urinary level of UTXB<sub>2</sub> was considered as an aspirin resistance index.

**Findings:** The mean age of patients was  $57.3 \pm 8.9$ ; and 44.6% of them were females. The aspirin resistance rate was 41.5%. Significant difference in aspirin resistance was observed between the opium addicts and non-addicts. (51.5% vs. 31.5%) ( $P = 0.001$ ). The effects of confounding variables such as diabetes, hypertension, and hyperlipidemia were eliminated by regression logistic multivariable analysis.

**Conclusion:** The prevalence of aspirin resistance in patients with stable angina pectoris was 41.5%. The prevalence of aspirin resistance in patients with stable angina pectoris who had opium addiction was significantly higher them non-addicts.

**Keywords:** Opium addiction, Aspirin resistance, Stable angina pectoris, Coronary angiography

**Citation:** Forood A, Malekpour-Afshar R, Sarnevesht J. **Effect of Opium Addiction on Aspirin Resistance in Stable Angina Pectoris.** *Addict Health* 2014; 6(1-2): 7-13.

**Received:** 04.08.2013

**Accepted:** 01.11.2013

1- Assistant Professor, Department of Cardiology AND Physiology Research Center, Kerman University of Medical Sciences, Kerman, Iran

2- Associate Professor, Neuroscience Research Center, Kerman University of Medical Sciences, Kerman, Iran

3- Resident, Department of Cardiology, School of Medicine, Kerman University of Medical Sciences, Kerman, Iran

Correspondence to: Afsaneh Forood MD, Email: afsanehforood144@gmail.com

## Introduction

Cardiovascular diseases are one of the leading causes of disablement and death in the world. The global burden of cardiovascular disease in developing countries is over 60% and in.<sup>1</sup> Since the accumulation of platelets is highly effective on causing cardiovascular diseases, the inhibition of this phenomenon can play an important role in preventing cardiovascular diseases.<sup>2</sup> Despite recent developments in new platelet medications in the past decades, aspirin is still one of the most commonly used medication for preventing cardiovascular diseases worldwide.<sup>3-7</sup> The clinical effectiveness of aspirin in preventing cardiovascular events has been well proven. Aspirin interferes with blood's clotting action. Long-term use of aspirin lowers the risk of non-fatal MI by 34%, non-fatal stroke by 25%, and death in patients with coronary occlusion.<sup>6,7</sup> It appears that the antithrombotic potency of aspirin for preventing platelet function through blocking of Eicosanoids is essential.<sup>8</sup>

Despite the fact that aspirin is an effective, inexpensive and safe medication, its consumption is not equally beneficial for all patients. Many modifiable and non-modifiable factors could be effective on the efficacy of antiplatelet drugs. A proposed mechanism for the limitations of the effects of aspirins caused inadequate response to aspirin or even resistance to aspirin. Resistance to aspirin reduces the preventing effects of aspirin against cardiovascular diseases. Some affecting factors on aspirin resistance have been reported such as side effects of smoking that can lead to increased platelet aggregation, the use of non-steroidal anti-inflammatory drugs, activation of the sympathetic nervous system, metabolic abnormalities (dyslipidemia, hyperglycemia), different methods of platelet activation, diabetes, hypertension, the amount and timing of drug use, and the genetic alternations of the population.<sup>9-16</sup>

According to the results of a survey published by Mansour et al., miscellaneous factors (including drug interactions, drug, poor compliance of patients, anaphylaxis, etc.) clinical factors (diabetes, heart failure, coronary syndrome, obesity, etc.), and genetic factors can cause aspirin resistance.<sup>17</sup>

In recent years, much attention was given to the changes of platelet suppression in response to

aspirin in patients. Lack of agreement on a standard definition for "aspirin resistance" led to reports of different results on the frequency of aspirin resistance in different studies. The terms "aspirin treatment failure" (used in clinical studies), "aspirin non-responsiveness" and "aspirin resistance" (used in pharmacological studies) have been used in different studies. Generally, the patients who despite receiving aspirin regularly suffered from cardiovascular diseases, or the patients with lab tests indicated platelet suppression, may be considered as aspirin resistant patients.<sup>18</sup> Previous studies reported the experimental frequency of the patients with heart diseases, which had been studied through different methods, as 4% to 83%.<sup>9-16</sup> The results of a survey on 22 articles, which reviewed the aspirin resistance using PFA-100 system, reported the frequency of aspirin resistance to 29%.<sup>9</sup> Although it appears that the therapeutic effects of aspirin are age and sex independent,<sup>5</sup> the study conducted by Christiaens et al. indicated that men are more aspirin resistant than women.<sup>19</sup>

Heart attack is reported the second leading cause of death in Kerman, Iran, among other 30 causes of death (778.9 years of life lost in 100000 people) which puts this province in the fourth place among the other 29 studied provinces, regarding the standardized deaths recorded due to cardiovascular diseases.<sup>20</sup> In a study in Kerman, it was estimated that a daily approximation of 37 people with no heart disease had the history of suffering from heart attacks in Kerman, and 5180 heart attacks happened all over Iran.<sup>21</sup> Previously, the association between platelet dysfunction in methadone users has been investigated,<sup>8</sup> but still, it is unclear how aspirin affects the major platelet receptors in opium-addicted patients with coronary artery diseases.

Due to some incorrect beliefs and myths, recommendations to consume opium as to control or prevent cardiac diseases, are considered as risk factors. Prevalence of opium addiction is different in diverse countries, cultures and jobs. Opium is the most common substance abuse in Iran. Addiction causes great moral and material damages and casualties. Very few studies have been conducted on the effects of opium addiction on body, and the comparison between addicts and non-addicts. In this study, the effects of opium addiction on aspirin resistance in patients with

angina pectoris were investigated. The accepted definition of aspirin resistance, which has been used in several studies and in the current study is "partial (incomplete) suppression of platelet aggression assessed through platelet function analysis which leads to poor biochemical response of platelets to antiplatelet effects of aspirin.

## Methods

In this study, the outpatients were enrolled in the study who were checked in the cardiology clinics of the hospitals affiliated to Kerman University of Medical Sciences, referred to the cardiologists' private clinics, or the Department of Angiography in the second hospital of Kerman University of Medical Sciences, and suffered from stable angina pectoris and were diagnosed with coronary artery diseases through cardiovascular angiography.

This research was conducted as a case-control study. The control group consisted of non-addicted patients and the case group consisted of patients addicted to opium (patients addiction to opium was determined by the patients self-reports or according to the criteria of Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV).

The measurements required for entering into the study were, suffering from stable angina pectoris, which means, typical cardiac chest pains, with a fixed pain severity, time and duration, and daily taking of 80-100 ml aspirin regularly for 3 consecutive months for secondary prevention of cardiovascular events.

The cases opt to withdraw from the study were by heart failure, acute coronary syndrome, history of coronary-artery bypass graft (CABG) or PCI within the past 6 months, major surgeries within the past month, having stroke within the past 3 months, active abdominal ulcers, chronic kidney disease requiring dialysis, severe liver diseases, progressive systematic diseases, Myeloproliferative disease, history of hemorrhagic diseases, clotting disorders, chronic infections and inflammation, recent urinary tract infections, recent aspirin intolerance, taking aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) simultaneously, Clopidogrel, Ticlopidine, Dipyridamole, heparin, enoxaparin, warfarin, proton pump inhibitor drugs consumption within the past 10 days before entering into the study, obesity with body mass

index ( $BMI \geq 30 \text{ kg/m}^2$ ), of consumption, consumption of green tea, intake of Omega 3, Vitamin C and E.

Initially, after explaining the objectives and the process of the study to the patients and obtaining a written consent from the patient or an alternate decision-maker, we started the study. Morning midstream specimen of urine were collected from the patients in a calm mental state, and stored in  $30^\circ\text{C}$ , until the time to measure the Urinary 11-dehydroxi Thromboxane B2 (UTXB2) level of the samples. Urine drug tests were taken to ensure the addiction of the subjects in control group. Morning urine specimens were collected before angiography, from the patients who were admitted to Shafa Hospital. Whereas, the angiographic results were positive, the urine specimen would enter into the study; otherwise, it would be disposed of it. UTXB2 measurement was carried out by ELISA, one of the most accurate laboratory methods, and due to the effects of urine concentration on UTXB2, after measuring creatinine levels in urine, UTXB2 level in urine was stated based on the creatinine level of urine. UTXB2 level in urine was considered as an indicator of aspirin resistance<sup>7</sup> through UTXB2 (in pg/ml)  $\times 100 \div$  urine creatinine (mg/dl). UTXB2 levels higher than pg/mg 1500 were considered as aspirin resistance.

The researcher collected the patients' demographic information such as age, sex, height, weight, history of drugs such as beta blockers, statins, ACEI (Angiotensin Converting Enzyme Inhibitors), ARB (Angiotensin Receptor Blockers), cigarette smoking, DM (Diabetes Mellitus), HTN (Hypertension), history of MI (Myocardial Infarction) or CABG or PCI (Percutaneous Coronary Intervention), CBC (Cellular Blood Count) including Hb (Hemoglobin), Platelet count, creatinine serum, duration of opium addiction, its method of use and amount, the type of aspirin consumed (coated or non-coated), the duration of taking, and the time aspirin was taken, by means of a questionnaire.

Due to some confounding factors such as diabetes, smoking, and blood pressure, that can affect aspirin resistance, the information was collected from all the subjects, and was taken into consideration with statistical analysis.

Subsequently, all the data were collected and statistically analyzed by SPSS for Windows

(version 17, SPSS Inc., Chicago, IL, USA) ,and the descriptive analysis of the mean  $\pm$  standard deviation (SD) was used for quantitative data and the relative and absolute frequencies were used for qualitative data. To compare the acetylsalicylic acid (ASA) resistance of both groups, chi-square test was used. To eliminate the confounding effects of some variables from logistic regression test, and to determine the index , and to calculate the risks of becoming aspirin resistant in both groups of addicts and non-addicts, both crude odd ratio (OR) and adjusted odd ratio were used.

## Results

In this study, 260 subjects have participated, which were divided into two groups of case and control, 130 people in each, based on their opium addiction status, i.e. addicts and non-addicts. The general profile of the patients participated in the study are presented in table 1. The majority of the patients were male (55.4%).

**Table 1.** General profiles of the participants

Patients profiles	Data
Age (year)	8.9 $\pm$ 57.3
Sex (female)	116 (44.6%)
Body mass index (25-30)	108 (41.5%)
Diabetes mellitus	59 (22.7%)
Hypertension	88 (33.8%)
Hyperlipidemia	36 (13.8%)
Smoking cigarettes	85 (32.7%)
History of aspirin consumption	85 (32.7%)
Aspirin resistance	108 (41.5%)

The results of this study indicated that of 260 patients, 108 cases (41.5%) were aspirin resistant. The variables are shown in table 2. The effect of confounding variables such as diabetes hypertension, and hyperlipidemia were eliminated by regression logistic multivariable

analysis in table 3.

## Discussion

This study was conducted on patients with stable angina pectoris to investigate the association between opium addiction and aspirin resistance. Accordingly, the overall prevalence of aspirin resistance in patients with stable angina pectoris in Kerman was estimated 41.5%. Various studies in diverse populations have estimated the prevalence of aspirin resistance (they were studied based on the measured parameters and the study subjects), between 5.5 to 75.0%.<sup>22-25</sup> However, in some previous studies, aspirin resistance was not observed in normal population and only has been reported in patients with cardiovascular diseases, diabetes and hypertension.<sup>10,26,27</sup> It has been articulated in some previous studies that aspirin resistance increases the risks of MI, cerebrovascular events, and deaths caused by cardiovascular events.<sup>10,26,27</sup> Christiaens et al. showed that 29.0% of the patients with stable angina pectoris were aspirin resistant.<sup>19</sup> Akay et al. reported aspirin resistance in healthy people as 27.0%.<sup>28</sup> Singla et al. reviewed aspirin resistance in patients with type II diabetes and 2.7% were aspirin resistant, and 39.1% were relatively resistant to aspirin.<sup>29</sup>

Zimmermann and Hohlfeld reported in vitro aspirin resistance 60.0%, 70.0% were for stable angina pectoris subgroup, 80.0% in MI group, and 60.0% in stroke and peripheral vascular disease group.<sup>30</sup> Recent studies in this regard reported the resistance causing 70.0% atherosclerotic vascular events.<sup>12</sup> In the study of Ziaie et al. in Isfahan, Iran, the prevalence of aspirin resistance in Isfahan was reported 75.3% that was the highest number reported among previous studies.<sup>25</sup> However, in this study, the number was compatible with the average aspirin resistance reported in other

**Table 2.** Study variables in case and control groups

	Control	Case	P
Age (year)	9.2 $\pm$ 57.8	8.6 $\pm$ 56.8	0.001
Sex (female)	77 males and 53 females	91 males and 39 females	0.386
Body mass index (25-30)	56 (43.1%)	52 (40.0%)	0.615
Diabetes mellitus	23 (17.6%)	36 (27.7%)	0.054
Hypertension	48 (36.9%)	40(30.8%)	0.294
Hyperlipidemia	18 (13.8%)	18 (13.8%)	1.000
Smoking cigarettes	17(13.1%)	40 (30.8%)	0.001
History of aspirin consumption	43 (33.1%)	42 (32.3%)	0.945
Aspirin resistance	41 (31.5%)	67 (51.5%)	0.001

**Table 3.** A multivariable logistic regression analysis with elimination of confounding factors (diabetes, hyperlipidemia, and hypertension)

	95% CI	OR	P
Diabetes	1.50-4.30	2.56	0.001
Hypertension	0.65-2.35	1.24	0.490
Hyperlipidemia	1.58-4.82	2.75	0.001
Opium consumption	0.73-3.49	1.59	0.230

CI: Confidence interval; OR: Odds ratio

studies. It appears that according to having the equivalent sample size and population to other studies conducted in this study, the results of this study were in the same range as the mean result of other studies. The prevalence of aspirin resistance also depends upon the experiment procedure and this can be an acceptable reason for the difference in the result numbers of different studies.<sup>25</sup>

According to the results of this study, aspirin resistance in addition to hypertension, diabetes, and hyperlipidemia can also be caused by chronic abuse of opium or addiction to it. In studies conducted on aspirin resistance, it was shown that any factor that can bind nitric oxide (NO) to two molecules of aspirin, leads to the formation of NO-aspirin molecule, which does not have the properties and efficiencies of aspirin molecules anymore.<sup>31</sup>

Given the role of opium and opiate substances, in oxidizing materials, it causes aspirin resistance

in patients. The limitations of this study were determination of relative and absolute strength of the patients in case and control groups, lack of information on patients' aspirin intake dosage and consumption and duration before the study, and having no accurate information on opium intake dosage.

## Conclusion

In this study, it was shown that the prevalence of aspirin resistance in patients with cardiovascular diseases was 41.5%, which was in consistent with the global rate of the other studies. Aspirin resistance in patients addicted to opium was higher than patients in control group, and it is recommended that the cardiologists and cardiology specialty clinics use other platelet inhibiting drugs and to avoid prescribing aspirin for addicted patients, and also to encourage these patients to withdraw more strongly.

## Conflict of Interests

The Authors have no conflict of interest.

## Acknowledgements

Special thanks go to all the individuals, organizations and institutions that helped us financially for the study.

## References

1. Gaziano TA. Reducing the growing burden of cardiovascular disease in the developing world. *Health Aff (Millwood)* 2007; 26(1): 13-24.
2. Ruggeri ZM. Platelets in atherothrombosis. *Nature Medicine* 2002; 8: 1227-34.
3. ISIS-2 Collaborative Group (Second International Study of Infarct Survival). Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet* 1988; 2(8607): 349-60.
4. Antiplatelet Trialists' Collaboration. Collaborative overview of randomised trials of antiplatelet therapy--II: Maintenance of vascular graft or arterial patency by antiplatelet therapy. *BMJ* 1994; 308(6922): 159-68.
5. Antiplatelet Trialists' Collaboration. Collaborative overview of randomised trials of antiplatelet therapy--III: Reduction in venous thrombosis and pulmonary embolism by antiplatelet prophylaxis among surgical and medical patients. *BMJ* 1994; 308(6923): 235-46.
6. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ* 2002; 324(7329): 71-86.
7. Lordkipanidze M, Pharand C, Schampaert E, Turgeon J, Palisaitis DA, Diodati JG. A comparison of six major platelet function tests to determine the prevalence of aspirin resistance in patients with stable coronary artery disease. *Eur Heart J* 2007; 28(14): 1702-8.
8. Malinin AI, Callahan KP, Serebruany VL. Paradoxical activation of major platelet receptors in the methadone-maintained patients after single pill of aspirin. *Thromb Res* 2001; 104(4): 297-9.
9. Postula M, Tarchalska-Krynska B, Filipiak KJ, Kosior D, Serafin A, Huczek Z, et al. Factors responsible for "aspirin resistance" - can we identify them? *Kardiol Pol* 2010; 68(4): 403-11.
10. Eikelboom JW, Hirsh J, Weitz JI, Johnston M, Yi

- Q, Yusuf S. Aspirin-resistant thromboxane biosynthesis and the risk of myocardial infarction, stroke, or cardiovascular death in patients at high risk for cardiovascular events. *Circulation* 2002; 105(14): 1650-5.
11. Krasopoulos G, Brister SJ, Beattie WS, Buchanan MR. Aspirin "resistance" and risk of cardiovascular morbidity: systematic review and meta-analysis. *BMJ* 2008; 336(7637): 195-8.
  12. Gum PA, Kottke-Marchant K, Poggio ED, Gurm H, Welsh PA, Brooks L, et al. Profile and prevalence of aspirin resistance in patients with cardiovascular disease. *Am J Cardiol* 2001; 88(3): 230-5.
  13. Hovens MM, Snoep JD, Eikenboom JC, van der Bom JG, Mertens BJ, Huisman MV. Prevalence of persistent platelet reactivity despite use of aspirin: a systematic review. *Am Heart J* 2007; 153(2): 175-81.
  14. Lordkipanidze M, Pharand C, Palisaitis DA, Diodati JG. Aspirin resistance: truth or dare. *Pharmacol Ther* 2006; 112(3): 733-43.
  15. Postuta M, Kapton-Cieslicka A, Filipiak KJ. Irregular aspirin response – definitions, therapeutic approaches, and known risk factors. *Kardiol Pol* 2008; 10(Suppl 3): 326-31.
  16. Coma-Canella I, Velasco A, Castano S. Prevalence of aspirin resistance measured by PFA-100. *Int J Cardiol* 2005; 101(1): 71-6.
  17. Mansour K, Taher AT, Musallam KM, Alam S. Aspirin resistance. *Adv Hematol* 2009; 2009: 937352.
  18. Szczeklik A, Musial J, Undas A, Sanak M. Aspirin resistance. *J Thromb Haemost* 2005; 3(8): 1655-62.
  19. Christiaens L, Ragot S, Mergy J, Allal J, Macchi L. Major clinical vascular events and aspirin-resistance status as determined by the PFA-100 method among patients with stable coronary artery disease: a prospective study. *Blood Coagul Fibrinolysis* 2008; 19(3): 235-9.
  20. Naghavi M. Death registration in 29 provinces in Iran in 2003. Tehran, Iran: Ministry of Health and Medical Education, Department of Health; 2005. [In Persian].
  21. Talebizadeh N, Haghdoost AA, Mirzazadeh A. Age at natural menopause, An epidemiological model (Markov Chain) of cardiovascular. *Payesh* 2009; 8(2): 163-70. [In Persian].
  22. Ziaaddini H, Ziaaddini MR. The Household Survey of Drug Abuse in Kerman, Iran. *Journal of Applied Science*, 2005; 5(2): 380-2.
  23. Canivano PL, Garcia YC. [Resistance to aspirin: prevalence, mechanisms of action and association with thromboembolic events. A narrative review]. *Farm Hosp* 2010; 34(1): 32-43.
  24. Gasparyan AY, Watson T, Lip GY. The role of aspirin in cardiovascular prevention: implications of aspirin resistance. *J Am Coll Cardiol* 2008; 51(19): 1829-43.
  25. Ziaie N, Sadeghi M, Akhlaghi A, Pirhaji O, Yaran M, et al. Aspirin resistance status as determined by urinary thromboxane B2 (TXB2) level in patients with ischemic heart disease and its relationship with Severity of coronary artery disease. *J Isfahan Med Sch* 2011; 28(116): 1-8. [In Persian].
  26. Grottemeyer KH, Scharafinski HW, Husstedt IW. Two-year follow-up of aspirin responder and aspirin non responder. A pilot-study including 180 post-stroke patients. *Thromb Res* 1993; 71(5): 397-403.
  27. Mueller MR, Salat A, Stangl P, Murabito M, Pulaki S, Boehm D, et al. Variable platelet response to low-dose ASA and the risk of limb deterioration in patients submitted to peripheral arterial angioplasty. *Thromb Haemost* 1997; 78(3): 1003-7.
  28. Akay OM, Canturk Z, Akin E, Bal C, Gulbas Z. Aspirin-resistance frequency: a prospective study in 280 healthy Turkish volunteers. *Clin Appl Thromb Hemost* 2009; 15(1): 98-102.
  29. Singla MK, Lahiri P, Mukhopadhyay P, Pandit K, Chaudhuri U, Chowdhury S. A study of aspirin resistance in type 2 diabetes. *J Indian Med Assoc* 2008; 106(11): 720, 722-3, 740.
  30. Zimmermann N, Hohlfeld T. Clinical implications of aspirin resistance. *Thromb Haemost* 2008; 100(3): 379-90.
  31. Fiorucci S, Mencarelli A, Meneguzzi A, Lechi A, Renga B, Del SP, et al. Co-administration of nitric oxide-aspirin (NCX-4016) and aspirin prevents platelet and monocyte activation and protects against gastric damage induced by aspirin in humans. *J Am Coll Cardiol* 2004; 44(3): 635-41.

## تأثیر اعتیاد به تریاک بر مقاومت آسپرین در بیماران مبتلا به آنژین صدری پایدار

دکتر افسانه فرود<sup>۱</sup>، دکتر رضا ملک پور افشار<sup>۲</sup>، دکتر جمشید سرنوشت<sup>۳</sup>

### مقاله پژوهشی

### چکیده

**مقدمه:** نرخ رشد بیماری‌های قلبی-عروقی در کشورهای در حال توسعه حدود ۶۰ درصد رو به افزایش می‌باشد. تأثیر کلینیکی آسپرین در پیشگیری از حوادث قلبی-عروقی به نحوی ثابت شده است. با وجود این که آسپرین داروی اثربخش و ارزانی است، اما مصرف آن برای همه بیماران به یک میزان سودمند نمی‌باشد. بسیاری از عوامل می‌توانند در میزان اثربخشی داروهای ضد پلاکت از جمله آسپرین مؤثر باشند.

**روش‌ها:** مطالعه حاضر بر روی ۲۶۰ فرد مبتلا به آنژین صدری پایدار که مبتلا به بیماری عروق کرونر در آن‌ها به وسیله آنژیوگرافی ثابت شده بود، انجام گرفت. افراد بر حسب اعتیاد به تریاک یا عدم اعتیاد به آن به دو گروه تقسیم شدند. اعتیاد به تریاک بر اساس معیارهای DSM-IV (Diagnostic and statistical manual of mental disorders, 4<sup>th</sup> Edition) مشخص گردید. نمونه میانی ادرار صبحگاهی بیماران جهت بررسی سطح  $UTXB_2$  (Urinary 11-dehydroxy Thromboxane B<sub>2</sub>) جمع‌آوری شد. سطح ادراری  $UTXB_2$  به نمرات شاخصی از تفاوت به آسپرین بیان شد.

**یافته‌ها:** میانگین سن افراد  $57/3 \pm 8/9$  سال بود و ۴۴/۶ درصد آنان را زنان تشکیل می‌دادند. مقاومت به آسپرین ۴۱/۵ درصد به دست آمد. از نظر مقاومت به آسپرین بین دو گروه معتاد به تریاک و غیر معتاد تفاوت معنی‌داری مشاهده شد (۵۱/۵ درصد در مقابل ۳۱/۵ درصد) ( $P = 0/001$ ). اثرات مخدوش‌کنندگی برخی عوامل مانند دیابت، فشار خون بالا و چربی خون بالا با استفاده از آنالیز رگرسیون چند متغیره حذف گردید.

**نتیجه‌گیری:** شیوع مقاومت به آسپرین در افراد مبتلا به آنژین صدری پایدار ۴۱/۵ درصد می‌باشد. شیوع مقاومت به آسپرین در افراد مبتلا به آنژین صدری پایدار و معتاد به تریاک در موارد واضحی بیشتر از افراد غیر معتاد مبتلا به آنژین صدری پایدار بود.

**واژگان کلیدی:** اعتیاد به تریاک، مقاومت به آسپرین، آنژین صدری پایدار، آنژیوگرافی کرونر

**ارجاع:** فرود افسانه، ملک پور افشار رضا، سرنوشت جمشید. تأثیر اعتیاد به تریاک بر مقاومت آسپرین در بیماران مبتلا به آنژین صدری پایدار. مجله اعتیاد و سلامت ۱۳۹۳؛ ۶(۱-۲): ۷-۱۳.

تاریخ پذیرش: ۹۲/۸/۱۰

تاریخ دریافت: ۹۲/۵/۱۳

- ۱- استادیار، گروه قلب و مرکز تحقیقات فیزیولوژی، دانشگاه علوم پزشکی کرمان، کرمان، ایران
  - ۲- دانشیار، مرکز تحقیقات علوم اعصاب، دانشگاه علوم پزشکی کرمان، کرمان، ایران
  - ۳- دستیار، گروه قلب، دانشکده پزشکی، دانشگاه علوم پزشکی کرمان، کرمان، ایران
- نویسنده مسؤول: دکتر افسانه فرود

Email: afsanehforood144@gmail.com