

Analysis of Some Behavioral Risk Factors in Relation to Acute Coronary Events

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ABSTRACT: The association of acute coronary events and behavioral risk factors is already known. Of these, smoking and alcohol consumption are the behavioral risk factors with the most intense impact in the occurrence of these events. The correct knowledge of the dynamics and their involvement in the evolution of acute coronary events remains of overwhelming importance in the light of current data. To achieve the purpose of this study data from three family medicine practices from the period November 2018 to May 2019 were corroborated. Anonymous questionnaires were applied to the subjects. For this study, questions related to the habit of smoking and consuming alcohol were selected. The study aimed to analyze the associative relationships between acute coronary events and two of the most common behavioral risk factors, smoking and alcohol consumption. The highest prevalence of acute coronary events was observed in current smokers and in former smokers. The period of exposure to smoking showed that this is one of the variables most strongly associated with an increased risk of acute coronary events. Moderate consumption of wine or beer seems to have a weak association with acute coronary events, even weaker than those who do not consume at all suggesting a protective effect.

KEYWORDS: Acute coronary events, smoking, alcohol consumption.

Introduction

In the context of an increased frequency of acute coronary events (ACE) and the pressure exerted by their multidimensional (medical, economic, social) consequences in the short and long term, the correct knowledge of the dynamics of involvement and the evolution of risk factors associated with acute coronary events remains of overwhelming importance [1,2,3,4].

In order to achieve a healthy lifestyle, it is important that coronary patients participate in prevention and control programs of risk factors [2].

The role of smoking in the occurrence of acute coronary events as the leading cause of death in Western industrialized countries has been highlighted by longitudinal and cross-sectional epidemiological studies [5].

There is epidemiological evidence that clearly correlates increased levels of alcohol consumption with increased morbidity and

mortality, and it is important to determine what are the safe levels of consumption [6].

Although efforts to provide solutions in the immediate management of ACE have been remarkable achievements, the understanding of the complexity of the actions and interactions of the multiple risk factors involved is still limited and strategies to combat or limit them are unfortunately affected by low effectiveness or difficult to measure blood pressure control in hypertensive smokers [7].

In order to obtain clinical benefits, the challenge is to achieve a multidisciplinary and multifactorial collaboration that focuses on a management to prevent the occurrence of ACE.

Opportunities to access a prevention and rehabilitation program that addresses lifestyle issues, support for smoking cessation, and a healthy lifestyle should be the most important aspect of their prevention [8].

Methods and Material

To achieve the purpose of this study, data from three family medicine practices from the period November 2018 to May 2019 were corroborated.

Informed consent was obtained from all subjects included in the study.

The study was also approved by the Ethics Committee of the University of Medicine and Pharmacy of Craiova.

Anonymous questionnaires were applied to the subjects.

For this study, questions related to the habit of smoking and consuming alcohol were selected.

This study aimed to analyze the associative relationships between ACE and two of the most common behavioral risk factors, smoking and alcohol consumption [9].

The study included a number of 865 adult participants aged 19-86 years. Subjects included in the study (858 responders) completed a complex questionnaire that included questions related to health status, smoker status, former smoker or non-smoker, alcohol consumption and types of beverages consumed.

In order not to limit the correct identification of the association between the effects of smoking and ACE, they were considered to be smokers and former smoker, representing a number of 507 subjects, comparisons being reported to subjects who had never been exposed to smoking (N=351).

The statistical processing of the selected data from the analyzed sample was performed with the support of statistical tools provided by the MedCalc statistical program, the same program was used for more elaborate statistical analyzes.

The distribution of the data was expressed numerically and as a percentage, being used to validate the statistical significance of the chi-square test.

The risk was calculated by using relative risk, accepting the statistical significance of the association between the variables for p-values, below 0.05.

Results

In the context of a prevalence of ACE in the study population of 6,01%, the incidence of smoking in the subjects of the group was of 40,56%, including both people who smoked daily (30,44%) and those who used to smoke occasionally (9,84%).

A significant number of subjects (N=159) did not currently smoke but had been smokers, representing 18,4%.

Thus, the category of smokers and former smokers represented 59,09% (N=507), and only 351 subjects (40,56%) had never smoked. (Table 1)

The highest prevalence of ACE was observed in current smokers (N=24; 9,13%) and former smokers (N=14; 8,81%).

Apparently surprisingly, ACE were the least common in occasional smokers (N=2; 2,35%), thus even less than in never smokers (N=12; 3,42%).

The prevalence of ACE in all categories of comparative smokers was of 7,89% (N=40) and in never smokers of 3,42%, outlining a cumulative risk of smoking of 2,3 times higher than in never smokers (RR=2,27; 95% CI 1,21-4,26; p=0.011).

Table 1. Risk and prevalence and differences (compared to never smokers).

	Type of smoking	ACE prevalence	N	Risk	95%IC	P
All	Never	3.42%	12			
	All smokers	7.89%	40	2.67	1.36 - 5.2386	P = 0.0043
	Dailly	9.13%	24	2.58	1.2191 - 5.4411	P = 0.0132
	Ocassionaly	2.35%	2	0.32	0.0715 - 1.398	P = 0.1290
Male	Former	8.81%	14	2.27	1.2068 - 4.2604	P = 0.0110
	Never	2.59%	10			
	All smokers	6.44%	17	2.49	0.7441 - 8.3317	P = 0.1388
	Dailly	6.67%	10	2.58	0.7259 - 9.154	P = 0.1430
Female	Ocassionaly	0.00%	0	0.45	0.0239 - 8.5458	P = 0.5963
	Former	8.97%	7	3.47	0.9254 - 13.0125	P = 0.0650
	Never	4.64%	11			
	All smokers	7.17%	17	1.55	0.7398 - 3.2286	P = 0.2468
477	Dailly	8.33%	9	1.61	0.6671 - 3.8901	P = 0.2892
	Ocassionaly	4.08%	2	0.88	0.2012 - 3.8437	P = 0.8644
	Former	7.50%	6	1.62	0.6175 - 4.2283	P = 0.3282

The risk compared to never smokers was 2,67 times higher for current daily smokers ($p=0.004$), 2,57 times for former smokers ($p=0.013$) and 0,32 times for occasional smokers ($p=0.129$).

Acute myocardial infarction (AMI) was identified in the history of 1,51% ($N=13$) of subjects, most subjects with acute myocardial infarction there were current smokers or former smokers ($N=11$; 84,62%).

The prevalence of AMI in smokers was of 2,21% ($N=11$) and of 0,83% ($N=3$) in non-smokers ($p=0.11$).

The risk of AMI in current smokers and ex-smokers compared to never smokers a former over 2,5 times higher ($RR=2,65$; 95% CI 0,75-9,43; $p=0.13$).

Of all the categories of smokers, the most affected by AMI was that of former smokers ($N=7$; 4,4%) and daily smokers ($N=4$; 1,57%).

The prevalence of ACE has steadily increased with the size of the period of exposure to smoking.

The prevalence is maximum in subjects with an exposure over 20 years ($N=10$; 8,62%) and minimum for those with exposures below 5 years ($N=1$; 1,61%).

For smokers with intermediate exposures the prevalence was below 5%, for those exposed to smoking 5-10 years of 3,64% ($N=4$) and 4,92% ($N=9$) for those with an exposure of 10-20 years.

The evaluation of alcohol consumption did not allow the identification of a significant association ($p=0.19$) of ACE risk ($RR=0,69$; 95% CI 0,4-1,21; $p=0.19$) in alcohol consumers ($N=32$; 5,32%) compared to non-consumers ($N=19$; 7,6%), being noted rather a tendency for the prevalence of ACE to be higher in abstainers, especially among non-consuming women ($N=17$; 8,53%) than consumers ($N=14$; 5%).

Among the types of beverages that were most frequently associated with the risk of ACE beverage spirits, especially in the case of daily consumption, but moderate consumption of wine or beer seems to have a weak association with ACE, even weaker than those who do not consume at all suggesting a protective effect (Figure 1).

The prevalence of ACE depending on frequencies of alcohol intake showed a J-shaped relationship between alcohol intake and ACE prevalence (Figures 2-4).

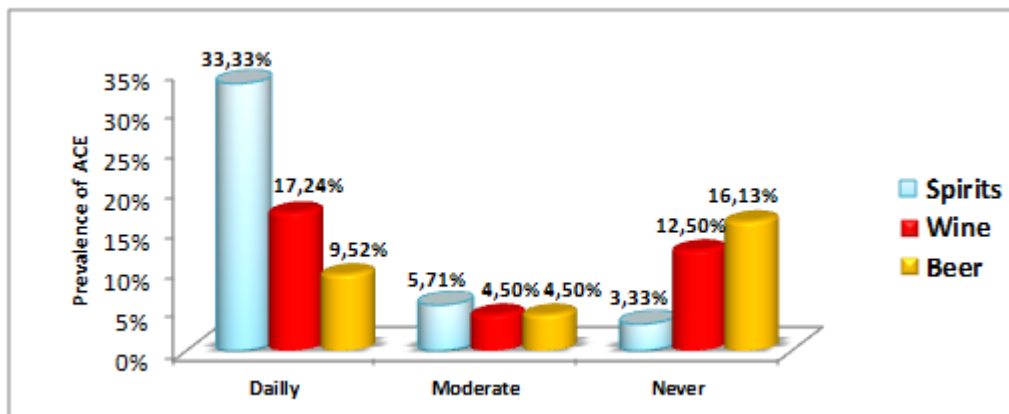


Figure 1. ACE prevalence depending on the type of alcohol consumed.

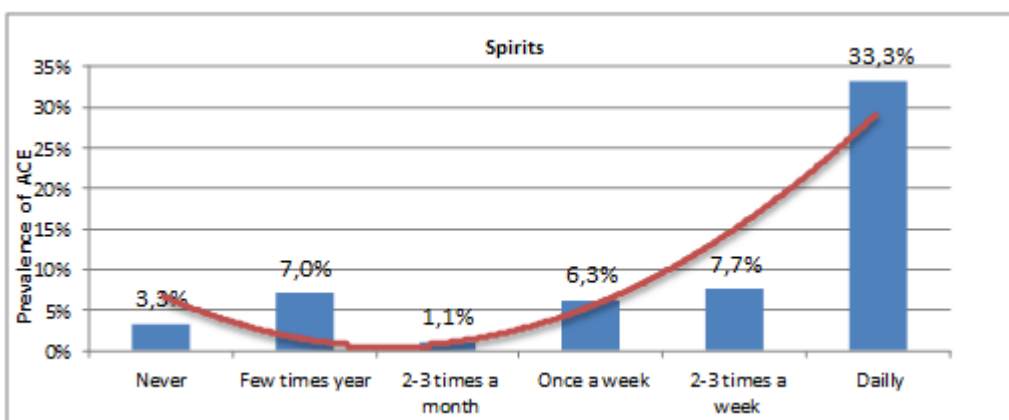


Figure 2. Prevalence of ACE depending on the frequency of consumption of spirits.

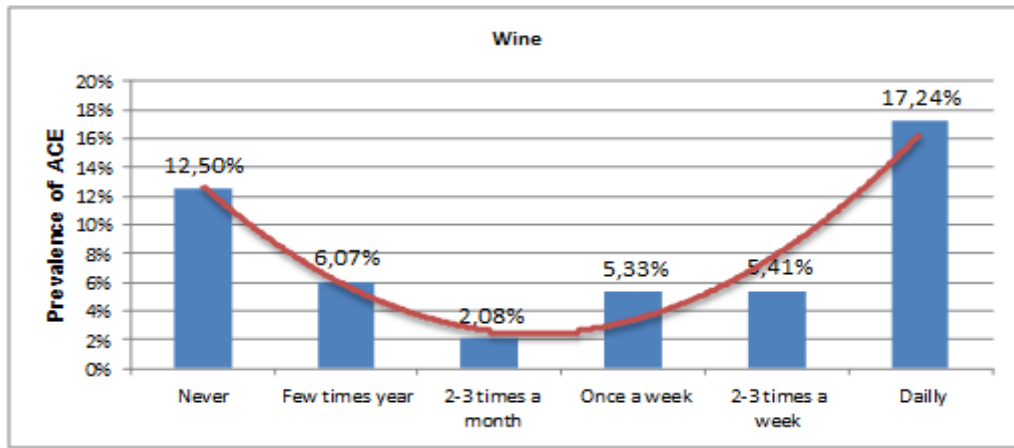


Figure 3. Prevalence of ACE depending on the frequency of consumption of wine.

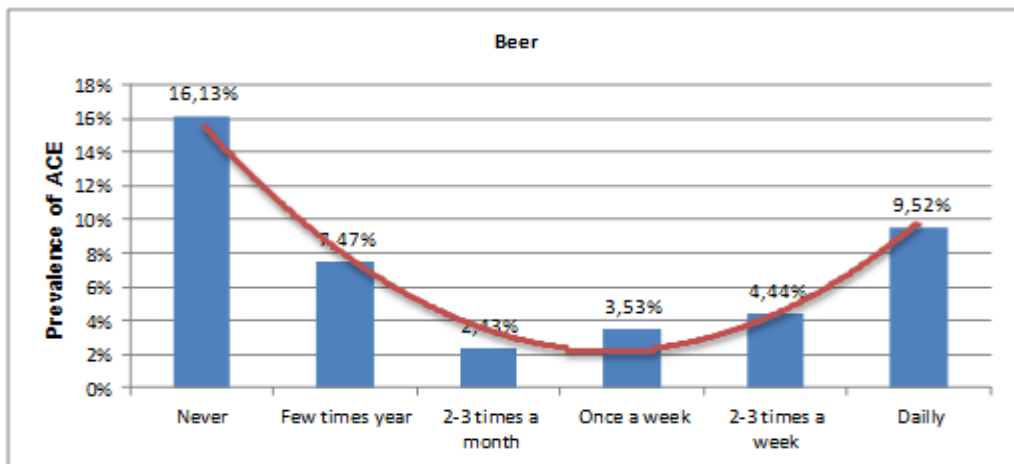


Figure 4. Prevalence of ACE depending on the frequency of consumption of beer.

Discussions

The risk factor that was most frequently identified as being associated with the occurrence of ACE was smoking.

Persistence of smoking is an unfavorable prognostic factor in people who have had a history of ACE, being the most important predictor for the recurrence of these events [3].

The relationship between smoking and the occurrence of ACE is well established, smoking can cause these events through multiple mechanisms: neurohormonal, hemodynamic, metabolic and biochemical [5].

Surprisingly, the category most commonly affected by AMI is found in former smokers, this being explained by the fact that this category often develops a deteriorating state of health.

Usually, these patients have already a deteriorated state of health in the moment of smoking cessation, this being one of the reasons of cessation.

The period of exposure to smoking showed that this is one of the variables most strongly associated with increased risk of ACE.

There are many more issues related to smoking that can act as variables when attempting to calculate the potential risk, for example the age at one begins to smoke, addiction, typed of preferred tobacco, cessation attempts, and addition of passive smoking [10].

In fact it has been showed that passive smoking might have been underestimated in previous studies, and might in fact bring a more important burden to these patients [11].

E-cigarette smoking has also been showed to be not risks free, as compared to classical cigarette smoking, as opposite to previous studies [12].

Also, important in the actual pandemic context, a very recent meta-analysis involving 11.590 COVID-19 patients has showed that smoking almost doubled the chance of having a worse evolution of the disease [13].

Daily consumption of spirits is the strongest association with the occurrence of ACE.

It has been highlighted by several epidemiological studies that alcoholic beverages, ie the consumption of beer and wine protect against cardiovascular disease [14,15].

This paradox may be assigned in part to high wine consumption [16].

There are epidemiological studies that prove the beneficial effect of reduced alcohol consumption [17,18].

Although there is evidence to support and demonstrate the beneficial effect of reduced alcohol consumption, in reality the evaluation of its role as an aggressor or as a protective factor must be more nuanced [19,20].

The indirect pathophysiological protective role of moderate alcohol consumption is explained by changes in a favorable sense of several cardiovascular biomarkers on the occurrence of coronary heart disease [21].

Thus, moderate alcohol intake seems to decrease inflammatory cytokines, decreases fibrinogen and fibrin D-dimer levels, increases the levels of high density lipoprotein cholesterol levels, but again, it is difficult to define moderate intake as quantity and frequency is usually not consistent over time in every consumer [22].

The J-shaped aspect in terms of alcohol consumption is a controversial issue, the path obtained should include the causal mechanisms underlying the trajectory of this curve [23,24].

The role of moderate alcohol consumption, such as beer and wine, as part of the Mediterranean diet, suggests rather a protective role, an aspect highlighted by other studies [25].

Conclusions

The highest prevalence of ACE was observed in current and in former smokers.

Apparently surprisingly, ACE were the least common in occasional smokers.

The period of exposure to smoking showed that this is one of the variables most strongly associated with an increased risk of ACE.

Moderate consumption of wine or beer seems to have a weak association with ACE, even weaker than those who do not consume at all suggesting a protective effect.

Abbreviations

ACE - Acute coronary events

AMI - Acute myocardial infarction

COVID-19 - Coronavirus disease

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Conflict of interests

None to declare.

References

1. Howe M, Leidal A, Montgomery D, Jackson E. Role of cigarette smoking and gender in acute coronary syndrome events. *Am J Cardiol*, 2011, 108(10):1382-1386.
2. Kotseva K, Wood D, Dirk De Bacquer D, De Backer G, Rydén L et al. A European Society of Cardiology survey on the lifestyle, risk factor and therapeutic management of coronary patients from 24 European countries. *Eur J Prev Cardiol*, 2016, 23(6):636-648.
3. Rallidis LS, Lekakis J, Panagiotakos D, Fountoulaki K, Komporozos C, Apostolou T, Rizos I, Kremastinos DT. Long-term prognostic factors of young patients. *Eur J Cardiovasc Prev Rehabil*, 2008, 15(5):567-571.
4. Arbel Y, FitzGerald G, Yan AT, Tan MK, Fox KAA, Gore JM, Steg PG, Eagle KA, Brieger D, Montalescot G, Budaj A, Lopez-Sendon J, Avezum A, Granger CB, Goodman SG. Temporal trends in all-cause mortality according to smoking status: Insights from the Global Registry of Acute Coronary Event. *Int J Cardiol*, 2016, 218:291-297.
5. Taylor BV, Oudit GY, Kalman PG, Liu P. Clinical and pathophysiological effects of active and passive smoking on the cardiovascular system. *Can J Cardiol*, 1998, 14(9):1129-1139.
6. Bradley KA, Donovan DM, Larson EB. How much is too much? Advising patients about safe levels of alcohol consumption. *Arch Intern Med*, 1993, 153(24):2734-2740.
7. Scarpa J, Bruzelius E, Doupe P, Matthew Le, Faghmous J, Baum A. Assessment of Risk of Harm Associated With Intensive Blood Pressure Management Among Patients With Hypertension Who Smoke: A Secondary Analysis of the Systolic Blood Pressure Intervention Trial. *JAMA Netw Open*, 2019, 2(3):e190005.
8. Giannuzzi P. Getting patients to target-implementing the guidelines. *Curr Vasc Pharmacol*, 2012, 10(6):715-717.
9. Gyárfás I, Keltai M, Salim Y. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries in a case-control study based on the INTERHEART study. *Orv Hetil*, 2006, 147(15):675-686.
10. West R. Tobacco smoking: Health impact, prevalence, correlates and interventions. *Psychol Health*, 2017, 32(8):1018-1036.
11. Whincup PH, Gilg JA, Emberson JR, Jarvis MJ, Feyerabend C, Bryant A, Walker M, Cook DG. Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement. *BMJ*, 2004, 329(7459):200-205.

12. Lee HW, Park SH, Weng MW, Wang HT, Huang WC, Lepor H, Wu XR, Chen LC, Tang MS. E-cigarette smoke damages DNA and reduces repair activity in mouse lung, heart, and bladder as well as in human lung and bladder cells. *Proc Natl Acad Sci USA*, 2018, 115(7):E1560-E1569.
13. Patanavanich R, Glantz SA. Smoking Is Associated With COVID-19 Progression: A Meta-analysis. *Nicotine & Tobacco Research*, 2020, 22(9):1653-1656.
14. Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary heart disease: a meta-analysis. *Addiction*, 2000, 95(10):1505-1523.
15. Di Castelnuovo A, Rotondo S, Iacoviello L, Donati MB, De Gaetano G. Meta-analysis of wine and beer consumption in relation to vascular risk. *Circulation*, 2002, 105(24):2836-2844.
16. Renaud S, de Lorgeril M. Wine, alcohol, platelets, and the French paradox for coronary heart disease. *Lancet*. 1992, 339(8808):1523-1526.
17. Wannamethee SG, Shaper AG. Type of alcoholic drink and risk of major coronary heart disease events and all-cause mortality. *Am J Public Health*, 1999, 89(5):685-690.
18. Roerecke M, Rehm J. Alcohol consumption, drinking patterns, and ischemic heart disease: a narrative review of meta-analyses and a systematic review and meta-analysis of the impact of heavy drinking occasions on risk for moderate drinkers. *BMC Med*, 2014, 12:182.
19. Roerecke M, Rehm J. Ischemic heart disease mortality and morbidity in former drinkers: a meta-analysis. *Am J Epidemiol*, 2011, 73:245-258.
20. Gheorman V, Dinescu VC, Criciotoiu O, Diana S, Calborean V, Mita A, Miscoci A, Davitoiu DV, Baleanu VD, Nedelcuta RM, Dinescu SN, Dijmarescu AL, Voiculescu DI, Udristoiu I. Clinical and Biochemical Changes Induced by Alcohol at the Patients with Mental Illness. *Rev. Chim*, 2019, 70(4):1406-1410.
21. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. *BMJ*, 2011, 342:d636.
22. Chiva-Blanch G, Badimon L. Benefits and Risks of Moderate Alcohol Consumption on Cardiovascular Disease: Current Findings and Controversies. *Nutrients*, 2020, 12(1):108.
23. Chokshi DA, El-Sayed AM, Stine NW. J-Shaped Curves and Public Health. *JAMA*. 2015, 314(13):1339-1340.
24. Emberson JR, Bennett DA. Effect of Alcohol on Risk of Coronary Heart Disease and Stroke Causality, Bias, or a Bit of Both? *Vasc Health Risk Manag*, 2006, 2(3):239-249.
25. Costanzo S, Di Castelnuovo A, Donati MA, Iacoviello L, de Gaetano G. Cardiovascular and Overall Mortality Risk in Relation to Alcohol Consumption in Patients With Cardiovascular Disease. *Circulation*, 2010, 121(17):1951-1959.

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