

The Effects of Alcohol Consumption on Recurrent Ischemic Events after Percutaneous Transluminal Coronary Interventions

The possible protective effect of alcohol consumption on recurrent myocardial ischemic events after percutaneous transluminal coronary intervention (PCI) was examined in this study. Patients who underwent PCI in our interventional cardiac catheterization laboratory were identified for possible inclusion in this study. They were queried in detail by telephone and clinic interviews. Medical records were reviewed as needed. Information regarding alcohol consumption after PCI and subsequent follow-up events, such as evidence of recurrent myocardial ischemia after PCI, was collected. Patients were classified by alcohol consumption as nondrinkers, low-intake drinkers, and moderate-/high-intake drinkers. Patients were also identified as having suffered a recurrent myocardial ischemic event in the follow-up period or as being event-free. Of the 247 patients for whom data collection was complete, 155 were nondrinkers, 82 were low-intake drinkers, and eight were moderate-/high-intake drinkers. A total of 25 patients experienced follow-up events—22 nondrinkers and three low-intake drinkers. No moderate-/high-intake drinker experienced a follow-up event. A chi-square test for the independence of alcohol consumption and follow-up events indicates a statistically significant dependence between the two variables ($p < 0.05$). These data indicate a negative association between alcohol consumption and the likelihood that a patient will suffer recurrent myocardial ischemia after PCI. A prospective study is warranted to test these initial observations (CVR&R. 2001;22:83–85,101) ©2001 by Cardiovascular Reviews & Reports, Inc.

Tedd Goldfinger, DO, Gregory Koshkarian, MD, Ann Tunstall, PhD, Christopher McArdle, MS From the Desert Cardiology of Tucson Heart Center; and the Cardiac Rehabilitation Unit, Northwest Medical Center, Tucson, AZ

*Address for correspondence/reprint requests: Tedd Goldfinger, DO, FACC, Desert Cardiology of Tucson Heart Center, 6080 North La Cholla Boulevard, Tucson, AZ 85741
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Background

Myocardial ischemic events following successful percutaneous transluminal coronary interventions (PCI) remain a clinical problem; they are due to early, in situ thrombosis or later proliferative restenosis. The pathophysiologic mechanism of myocardial ischemia after PCI is multifactorial and includes thrombus formation, myoelastic recoil and coronary vasospasm, intimal proliferation, and adverse vascular remodeling. Strategies tested to reduce the likelihood of recurrent ischemic events after PCI have met with variable success and include administration of aspirin and other antiplatelet drugs, lipid-lowering agents, vasodilating agents, fish oils, and antioxidants. A recent study¹ demonstrated reduced restenosis following administration of a unique antioxidant, probucol. In studying the nature of this agent and its properties of decreasing low-density lipoprotein (LDL) cholesterol levels, reducing serum LDL oxidation, and suppressing intimal proliferation, our interest turned to the similar beneficial effects of alcohol consumption after PCI.

Alcoholic beverages, particularly red wine, offer clinical effects that may confer a benefit in preventing thrombotic events and proliferative restenosis following PCI. Consuming wine, beer, or spirits has been shown to inhibit platelet adhesion and enhance the antiplatelet effects of oral aspirin.² Alcohol consumption has been associated with an increased prostacyclin/thromboxane ratio,^{3,4} increased endogenous tissue-type plasminogen activator,⁵ and decreased fibrinogen levels.⁶ High-density lipoprotein subfractions are increased,⁷ and an improved lipid phenotype may improve dysfunctional endothelial physiology in patients with atherosclerotic vascular disease.⁸ Phenolic compounds in red wine increase serum antioxidant capacity and inhibit oxidation of LDL.^{9–11} Polyphenolic tannins, particularly

prevalent in red wines, exert salutary vasodilator properties that may favorably affect the incidence of myocardial ischemia.

This retrospective study was undertaken to ascertain whether or not alcohol consumption after PCI affects the likelihood of recurrent myocardial ischemic events, and whether embarking upon a more difficult prospective study would be of value.

Methods

Four hundred thirty-seven consecutive cases were reviewed from the interventional cardiac catheterization laboratory at Northwest Medical Center, a community hospital in Tucson, Arizona. A computer database was designed to accept information from a data form. The investigators, with the assistance of nurses and technicians, collected relevant data from this patient group. In some instances hospital records were reviewed at length to confirm information collected from other sources and to fill in deficiencies.

One hundred ninety interventional cases were excluded from the final analysis. Patients requiring coronary artery bypass grafting in addition to PCI during the same hospitalization, and those who died during the reference hospitalization, were excluded. Patients were also excluded due to incomplete information or follow-up data. Incomplete patient information was most often the result of the patient being unreachable by telephone, unwilling to answer questions, or unable to provide a reliable history.

The final study group comprised 247 patients who were contacted by telephone and completed a detailed telephone interview, or who had adequate information from their medical records for inclusion in the final analysis.

Patients who required a second PCI during one hospitalization were included in the study. In this group, the second PCI was recorded as the reference intervention. All confirmed deaths after hospital discharge were included in the final analysis.

Of the 247 patients, 106 (43%) underwent balloon angioplasty alone, and 141 (57%) underwent balloon angioplasty with stent placement.

Alcohol consumption in the form of wine, beer, and spirits was surveyed, as were other historical data. Alcohol consumption was categorized as none (0 g/day), low (0.1–19.9 g/day), moderate (20–39.9 g/day), or high (≥ 40 g/day). The distribution of alcohol consumption in our "drinking" study population is shown in Fig. 1.

Alcohol consumption following PCI was analyzed against follow-up events, defined as a repeat hospitalization with unstable angina, myocardial infarction, death, angiographic restenosis, repeat PCI of the reference lesion, or coronary artery bypass grafting. The

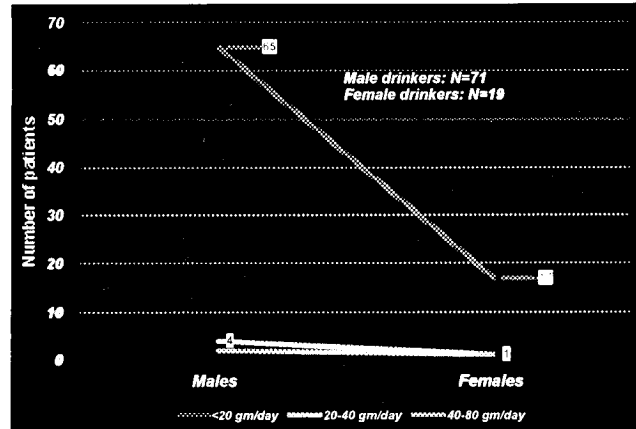


Figure 1. Distribution of alcohol consumption in male and female study participants.

mean follow-up was 68 weeks (range, 42–126 weeks). The presence of confounding variables, such as tobacco use, vitamin and antioxidant use, and others, was recorded.

Raw data were presented to an independent medical statistician for analysis. Chi-square analysis was used to test for the independence of alcohol consumption and the incidence of follow-up events after PCI. The last two levels of alcohol consumption (moderate and high) were merged to meet the requirements for the chi-square test.

Results

All 247 patients were included in the final analysis; 25 were women and 222 were men.

One hundred fifty-seven patients (64%) reported no alcohol use and 90 patients (36%) reported alcohol consumption after PCI.

Nondrinkers experienced 22 follow-up events, whereas only three follow-up events occurred in the patients who reported low alcohol consumption. No events were recorded in the moderate/high alcohol consumption group after PCI (Fig. 2).

Chi-square analysis for the independence of alcohol consumption and follow-up events indicated a statistically significant dependence between the two ($p < 0.05$). The proportion of drinkers who had follow-up events (14%) was significantly lower than the proportion of nondrinkers who had follow-up events (3%) (95% confidence interval). These data indicate an inverse association between alcohol consumption and recurrent ischemic events after PCI.

There was no significant difference between male and female drinkers, or between male and female nondrinkers, with respect to the significance of alcohol consumption after PCI and the occurrence of follow-up events (female drinkers experienced no follow-up events).

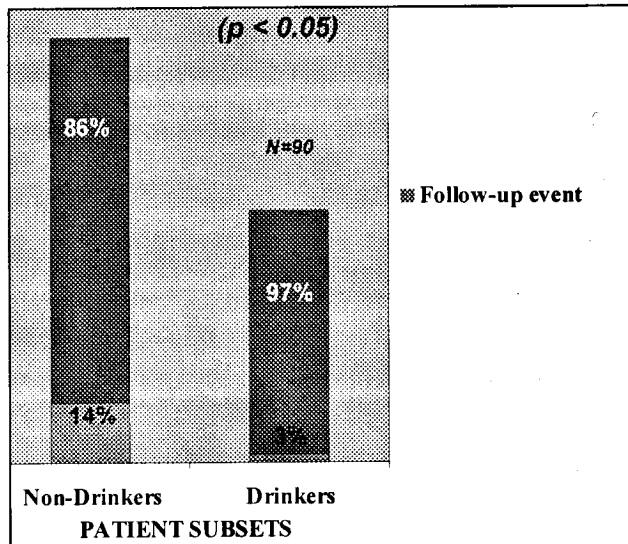


Figure 2. Incidence of recurrent ischemic events after PCI in "non-drinkers" and "drinkers."

Although no patients reported red wine consumption exclusively, 43 patients reported regular red wine consumption, among whom there was one follow-up event. There were not enough data to make a meaningful statement about the exclusive benefit of red wine consumption after PCI, due to the small sample size and the heterogeneity of alcohol consumption among the drinkers.

Discussion

The possible protective effect of alcohol consumption against recurrent ischemic events after PCI was examined in this retrospective study. The data indicate an association between alcohol consumption and a decreased incidence of follow-up events in a cohort of patients with coronary artery disease who underwent PCI. This study supports our interest in further research in this area and justifies the labor and expense of a prospective analysis.

An association between alcohol consumption and a reduced incidence of coronary artery disease and myocardial infarction was reported previously by Klatsky et al.¹² Similarly, Hennikens et al.¹³ reported a reduced incidence of fatal myocardial infarction with light alcohol intake in a large group of cases and controls. Yano et al.¹⁴ found a negative association between moderate alcohol consumption and ischemic heart disease in a sample of Japanese men. Stampfer et al.¹⁵ reported that moderate alcohol consumption decreased the risks of coronary heart disease and ischemic stroke in a large population of middle-aged women. There are reports that conflict with the hypothesis that alcohol has a protective effect against coronary artery disease; however, this hypothesis is

more strongly supported than refuted in the literature. Contemporary studies suggest that the relationship between alcohol consumption and decreased coronary artery disease risk is causal.¹⁶ In most studies, the composite of the relationship is a U-shaped or J-shaped curve in which the heaviest drinkers fare the worst and the lighter drinkers fare the best.¹⁷

St. Leger et al.¹⁸ studied deaths from coronary artery disease in 18 developed countries. The principal finding was a strong and specific association between alcohol consumption and a decrease in coronary death that was wholly attributable to wine consumption. Later, Renaud et al.¹⁹ showed that despite the high dietary intake of saturated fats in France, there was, unexpectedly, lower mortality from coronary artery disease. Stepwise multivariate analysis showed the protective effect of wine consumption and corrected the paradox by moving the expected mortality close to the regression line created by the association of coronary mortality and dietary fat. The implication of the French paradox is that the untoward effects of saturated fats are counteracted by wine.

The present study was unable to distinguish an exclusive benefit from red wine after PCI. The unique mechanisms of coronary protection imparted by the alcohol and non-alcohol components of red wine, however, may offer the greatest protection from thrombosis and restenosis after PCI. These mechanisms include reduction of neointimal hyperplasia after balloon injury,²⁰ inhibition of platelet aggregation,²¹ inhibition of the release of inflammatory mediators by activated polymorphonuclear leukocytes,^{22,23} and enhancement of endothelial-dependent coronary vasodilatation.^{24,25}

This study may be criticized for its retrospective nature and dependence on individual reporting of alcohol consumption, which may be suspect. The sample size was small relative to the initial screening group. Nevertheless, our observations are in accord with others', suggesting that low and moderate alcohol consumption protects against myocardial ischemic events.

Following PCI, it may not be medically appropriate to insist that patients abstain from the use of alcoholic beverages.

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