Social alcohol consumption and low Lp(a) lipoprotein concentrations in middle aged Finnish men: population based study

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Subjects, methods, and results

We performed a population based cross sectional study of 300 men aged 40-60 years selected randomly by age stratification; 259 (86%) participated in the study. Subjects were divided into four groups by alcohol consumption: abstainers (mostly lifetime teetotallers; 37 men) and three groups of drinkers. Drinkers in the lowest third consumed < 39 g alcohol/week (74 men), those in the middle third 39-132 g/week (75), and those in the highest third > 132 g/week (73). Alcohol intake was ascertained from a questionnaire on the amount and quality of alcoholic beverages consumed during the previous two weeks. Plasma Lp(a) lipoprotein concentrations were determined by a two site immunonradiometric assay that showed a close correlation with two different enzyme linked immunosassays (r=0.96). Liver function was assessed by measuring lipid and enzyme concentrations in fasting blood samples using standard techniques. Statistical analysis was carried out with the sas software package. The groups were similar for age and smoking habits. The body mass index, waist to hip ratio, systolic and diastolic blood pressure, and alanine aminotransferase values were highest in subjects in the third drinking > 132 g/week compared with those in the two lower thirds and with non-drinkers (P<0.001 for each parameter, analysis of variance). The mean concentrations of serum γ-glutamyltransferase increased with increasing alcohol intake—that is, 34, 33, 49, and 64 U/l respectively for teetotallers, and the lowest, middle, and highest third of drinkers (P<0.001). Blood glucose and serum insulin values did not differ between the groups. Lp(a) lipoprotein concentrations were higher (median, 206 mg/l) in the teetotallers than in the drinkers. Lp(a) lipoprotein concentrations for the lowest, middle, and highest alcohol thirds were 137, 109, and 94 mg/l (P<0.05, Kruskal-Wallis test) (figure). As noted in other white populations, we observed a highly skewed distribution of Lp(a) lipoprotein concentrations and a wide range within the population. The ranges were similar in the study groups (figure). Lp(a) lipoprotein concentrations showed a weak but significant correlation with body mass index, waist to hip ratio, and insulin concentrations (Pearson’s correlation coefficients −0.15, −0.14, and −0.15 respectively, P<0.025 for each correlation). There were no significant differences in high density lipoprotein (means for the teetotallers, and the lowest, middle, and highest thirds of alcohol consumption 1.19, 1.19, 1.23, and 1.27 mmol/l respectively) or low density lipoprotein cholesterol concentrations between the groups.

Comment

Our report shows that social drinking—that is, <39 g alcohol/week or 1-4 units/week—is associated with low Lp(a) lipoprotein concentrations in middle aged men. No changes were observed in high density lipoprotein cholesterol or low density lipoprotein cholesterol concentrations, blood pressure, or liver enzyme concentrations.

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produce postural hypotension, so treatment with selective antidepressants and monoamine oxidase inhibitors may antidepressant induced postural hypotension. Tricyclic rate of falls and fractures in this age group may relate to position (blood pressure 140/90 mm Hg while lying and 110/90 mm Hg while standing), which resolved on withdrawal of the drug. To our knowledge, the only published report of postural hypotension associated with paroxetine relates to its increasing trimipramine concentrations when prescribed with trimipramine. At the time of writing, 43 cases of postural hypotension associated with paroxetine had been reported to the Committee on Safety of Medicines (personal communication). Other selective serotonin reuptake inhibitors have been reported to exacerbate syncope. Dizziness is cited on the datasheet for paroxetine, though not in relation to postural hypotension.

We suggest that postural hypotension should be considered if dizziness develops. The size of the postural fall in blood pressure seems to be dose related, and the dose should be reduced or drug treatment discontinued.

Other studies of alcohol consumption and Lp(a) lipoprotein cholesterol concentrations have dealt with differences between men and women, analysed alcohol intake qualitatively, and compared heterogeneous groups—that is, non-drinkers together with those who drink regularly on three or less days a week. To our knowledge, ours is the first study to show a relation between moderate alcohol consumption and Lp(a) lipoprotein concentrations. We conclude that low Lp(a) lipoprotein concentrations may be one factor explain-