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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Short-term alcohol consumption may have detrimental effect on fibrinolysis and endothelial function: preliminary report of prospective randomised study

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Abstract

Background and aim: This study was designed to clarify the impact of the short-term consumption of different types of alcoholic beverages on haemostatic factors, C-reactive protein (hsCRP) and endothelin-1 (E-1) plasma levels.

Methods: The study group consisted of 57 healthy male volunteers, aged 20–29 years. Subjects were randomised to consume 300 mL of red wine, white wine, 12% ethanol, blackcurrant juice or water for five days. Blood samples were collected for CRP, tissue type plasminogen activator antigen (t-PA:Ag), plasminogen activator inhibitor antigen (PAI-1:Ag) and E-1 at baseline, on day 2, and on day 6.

Results: A significant increase in PAI-1:Ag concentration was observed in the red wine drinking group (day 1: 44.98; day 2: 56.86; day 6: 47.44 ng/mL; $p = 0.05$). A similar increase of E-1 level was found in the 12% ethanol group (day 1: 0.53; day 2: 1.65; day 6: 1.11 fmol/mL; $p = 0.01$). Dividing the whole study group according to ethanol content of consumed beverages revealed significant changes in tPA:Ag, PAI-1:Ag and E-1 levels. In the alcohol drinking group, significant increases of PAI-1:Ag (day 1: 44.75; day 2: 54.07; day 6: 44.80 ng/mL; $p < 0.05$); tPA:Ag level (day 1: 3.65; day 2: 4.17; day 6: 5.03 ng/mL; $p < 0.02$) and E-1 (day 1: 0.42; day 2: 1.01; day 6: 0.97 fmol/mL; $p < 0.002$) were observed.

Conclusions: Short-term alcohol consumption increases tPA:Ag, PAI:Ag and E-1 plasma levels. This effect may have an unfavourable impact on the fibrinolytic system and endothelial function.

Key words: alcohol consumption, endothelin, fibrinolysis, inflammation

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INTRODUCTION

Alcohol was first mentioned in relation to ischaemic heart disease in 1772. Heberden [1], then, described a relief in anginal pain after alcohol ingestion. Numerous epidemiological studies carried out since then have indicated lower all-cause mortality rates in populations where alcohol intake was moderate, compared to non-drinkers and alcohol abusers (U-shaped curve) [2–4]. Most analyses attribute this fact to the reduced morbidity and mortality of coronary artery disease. Some authors also indicate the advantage that the moderate consumption of red wine confers over drinking other types of alcohol. The cause underlying this phenomenon, known as 'the French paradox', might be the biologically active polyphenolic content of red wines.

Numerous hypotheses on the mechanisms behind the influence that alcohol and polyphenols exert on the pathophysiology of myocardial ischaemia have been proposed on the basis of both randomised controlled trials and reports from basic sciences. Nevertheless, some questions remain unanswered.

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The present study was designed in order to evaluate the influence of ethanol on the plasma fibrinolytic system and, at the same time, to evaluate the connection between alcohol consumption and plasma levels of endothelin-1 — a potent vasoconstrictor crucial for the pathogenesis of acute coronary syndromes. Furthermore, an attempt was made to investigate the short-term influence of alcohol consumption on high sensitivity C-reactive protein (hsCRP), the inflammatory marker most frequently used in cardiology. Red wine maturing in oak barrels and blackcurrant juice used in the course of the intervention phase of the study shall be treated as a response to reports suggesting that polyphenolic substances have a positive effect on the cardiovascular system.

METHODS

Participants

The study group consisted of 57 male volunteers, theological college seminarians, living in a dormitory. The participants had their meals according to the same schedule and menu, both during the study and for the three months prior to the experiment. All group members were non-smokers and lifelong non-drinkers. During the experiment, and for the period of two weeks before it started, the participants had abstained from polyphenolic-rich foods. The mean age of the study group was 24 years (23.6 ± 2.2). Informed consent was obtained from each participant. Local Research Ethical Committee approval for the study was obtained.

The exclusion criteria were: hypertension, diabetes, hypolipemic therapy and oral anticoagulants, positive anamnesis for cancer, chronic inflammatory conditions, gastric and duodenal ulcers, gastroesophageal reflux, liver diseases and other chronic illnesses demanding pharmacotherapy. Persons with significantly elevated lipid parameters and abnormal levels of alanine aminotransferase were also excluded. During the experiment (i.e. six days) all participants were obliged not to leave the premises.

The participants were randomly assigned to five groups; for five consecutive days before breakfast, on an empty stomach, each one drank 300 mL of either an alcoholic drink, or blackcurrant juice, or water: group 1 — 12% white wine; group 2 — blackcurrant juice; group 3 — 12% red wine; group 4 — 12% v/v aqueous ethanol solution; group 5 — still mineral water.

Material

The drinks used for the purpose of the study were: white Argentinian Chardonnay (Trivento) and red Chilean Cabernet Sauvignon matured in oak barrels (Porta Select Reserve), aqueous 12% alcohol solution, and 100% blackcurrant juice made from natural blackcurrant concentrate.

Methods

In order to determine levels of the following parameters: alanine aminotransferase activity, concentrations of CRP, fibrinogen, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, tissue-type plasminogen activator antigen (tPA:Ag), plasminogen activator inhibitor type 1 antigen (PAI-1:Ag) and endothelin, blood samples were taken on the first day before the intervention, and on the second and sixth days. Fasting blood samples were always collected between 7 and 8 a.m., after 15 min rest, in the supine position; the venopuncture was done in the antecubital region. The samples were collected into polyethylene Vacutainer test tubes containing anticoagulant — 3.2% sodium citrate (for tPA:Ag, PAI-1:Ag and fibrinogen), or 1.5% potassium versenate (for endothelin). The blood samples were then centrifuged at 3,000 × g at 4°C; thus the obtained low-platelet concentration plasma was sampled into Eppendorf test tubes and frozen at –80°C for a period of max. three months before the measurement was taken.

Plasma levels of tPA:Ag and PAI-1:Ag were determined with the ELISA method using the Imubind test by American Diagnostica. Endothelin was measured with the use of the ELISA method as well, using the Endothelin (1-21) test by Biomedica. Spectrophotometric measurements were made at the wavelength of 450 nm with the referential filter 620 (Labsystem Genesis; Multiscan RC V1.5-0). hsCRP concentration was determined using the immunoturbimetric method with the CRP OSR6199 Highly Sensitive Application test by Olympus. The absorbance was determined using the Olympus AU400.

Statistical analysis

The analysed statistical samples were tested for compatibility with the normal distribution using the Shapiro-Wilk test. Most of the parameters were not derived from normally distributed populations, and to compare two samples the U Mann-Whitney test was applied. When comparing many independent samples from abnormally distributed populations, the Kruskal-Wallis ANOVA test was applied. When comparing many dependent samples from a normally distributed population, the Friedman's ANOVA test was used. Additionally, the individual results of consecutive measurements were compared in pairs by means of the sign test — a non-parametric alternative of the t-Student test for dependent variables. The results were worked out using Statsoft's Statistica 6.0.

RESULTS

The study enrolled 57 male volunteers, randomly assigned to the following groups: the white wine group (n = 11), the red wine group (n = 12), the equivalent ethanol solution (n = 11), the blackcurrant juice group (n = 12) and the mineral water group (n = 11). The initial concentrations of hsCRP, endothelin, tPA:Ag and PAI-1:Ag did not display any statistically significant differences in either individual subgroups or in the entire population under examination (Table 1).

Consecutive measurements performed on days 2 and 6 in the white wine group, the blackcurrant juice group and the mineral water group did not reveal any significant changes in the investigated parameters compared to the initial values (Tables 2–4).

Table 1. Baseline laboratory characteristics of the study group (medians and standard deviations)

Parameter	Groups					P
	White wine	Juice	Red wine	Ethanol 12%	Water	
Cholesterol [mg/dL]	162 ± 35	159 ± 42	174 ± 33	192 ± 40	198 ± 33	NS
Low density lipoprotein [mg/dL]	92 ± 27	89 ± 32	96 ± 22	98 ± 29	98 ± 29	NS
High density lipoprotein [mg/dL]	49 ± 5	53 ± 5	51 ± 10	45 ± 8	55 ± 8	NS
Triglycerides [mg/dL]	100 ± 38	89 ± 29	90 ± 50	92 ± 59	102 ± 64	NS
Fibrinogen [mg/dL]	237 ± 44	229 ± 71	249 ± 42	238 ± 28	243 ± 39	NS
Alanine aminotranferase [U/L]	23 ± 8	20 ± 12	24 ± 18	21 ± 10	25 ± 27	NS

Table 2. Medians and standard deviations of the plasma concentration of the measured parameters in the water group (n = 11)

Parameter	Baseline	Day 2	Day 6	P
hsCRP [mg/L]	0.70 ± 0.71	0.60 ± 0.72	0.70 ± 1.01	NS
E-1 [fmol/mL]	0.31 ± 0.16	0.61 ± 1.06	0.55 ± 1.18	NS
PAI-1:Ag [ng/mL]	44.80 ± 27.28	57.35 ± 23.12	51.63 ± 23.09	NS
tPA:Ag [ng/mL]	4.02 ± 2.86	3.60 ± 3.66	3.35 ± 3.30	NS

hsCRP — C-reactive protein measured by a high sensitivity assay; E-1 — endothelin 1; PAI-1:Ag — plasminogen activator inhibitor type-1 antigen; tPA:Ag — tissue type plasminogen activator antigen

Table 3. Medians and standard deviations of the plasma concentration of the measured parameters in the blackcurrant juice group (n = 12)

Parameter	Baseline	Day 2	Day 6	P
hsCRP [mg/L]	0.55 ± 0.42	0.50 ± 0.66	0.50 ± 0.53	NS
E-1 [fmol/mL]	0.49 ± 0.35	0.77 ± 0.86	0.64 ± 2.21	NS
PAI-1:Ag [ng/mL]	44.70 ± 22.05	27.17 ± 26.56	40.51 ± 26.34	NS
tPA:Ag [ng/mL]	3.03 ± 2.40	4.44 ± 2.59	3.51 ± 4.03	NS

Abbreviations as in Table 2

Table 4. Medians and standard deviations of the plasma concentration of the measured parameters in the white wine group (n = 11)

Parameter	Baseline	Day 2	Day 6	P
hsCRP [mg/L]	0.70 ± 0.84	0.80 ± 0.99	0.80 ± 1.22	NS
E-1 [fmol/mL]	0.25 ± 0.37	0.37 ± 6.29	0.69 ± 1.60	NS
PAI-1:Ag [ng/mL]	44.75 ± 15.54	35.60 ± 23.37	34.44 ± 11.31	NS
tPA:Ag [ng/mL]	3.13 ± 2.39	3.17 ± 2.43	4.61 ± 11.19	NS

Abbreviations as in Table 2

The red wine group revealed a significantly increased concentration of PAI-1:Ag on day 2. Changes observed in the levels of other parameters were not statistically significant (Table 5).

The group ingesting 12% v/v ethyl alcohol solution revealed a considerable increase in plasma endothelin concentration, without significant changes in concentrations of the other parameters (Table 6).

For the second stage of the analysis, the investigated population was grouped according to alcohol content of consumed beverages, thus obtaining two sub-groups: the one consisting of 34 participants who drank white wine, red wine and ethanol solution; the other one with 23 participants who drank mineral water or blackcurrant juice. Both groups were investigated regarding statistically significant differences in the initial parameters.

No significant differences between the sub-groups were reported in the initial levels of tPA:Ag, PAI-1:Ag, endothelin, hsCRP or lipid parameters, fibrinogen and alanine aminotransferase.

Table 5. Medians and standard deviations of the plasma concentration of the measured parameters in the red wine group (n = 12)

Parameter	Baseline	Day 2	Day 6	P
hsCRP [mg/L]	0.75 ± 0.89	0.65 ± 0.51	0.65 ± 0.62	NS
E-1 [fmol/mL]	0.44 ± 0.39	2.68 ± 3.61	3.17 ± 5.21	NS
PAI-1:Ag [ng/mL]	44.98 ± 24.58	56.86 ± 18.19	47.44 ± 26.30	0,05
tPA:Ag [ng/mL]	1.98 ± 3.37	0.81 ± 5.26	2.24 ± 5.64	NS

Abbreviations as in Table 2

Table 6. Medians and standard deviations of the plasma concentration of the measured parameters in the group consuming 12% v/v ethanol solution (n = 11)

Parameter	Baseline	Day 2	Day 6	P
hsCRP [mg/L]	0.50 ± 0.99	0.60 ± 0.58	0.50 ± 0.66	NS
E-1 [fmol/mL]	0.53 ± 0.50	1.65 ± 4.80	1.11 ± 4.86	0.01
PAI-1:Ag [ng/mL]	43.86 ± 21.50	52.45 ± 14.53	53.06 ± 22.68	NS
tPA:Ag [ng/mL]	3.32 ± 2.36	3.85 ± 3.06	5.04 ± 3.55	NS

Abbreviations as in Table 2

Table 7. Medians and standard deviations of the plasma concentration of the measured parameters in the alcohol-free group (n = 23)

Parameter	Baseline	Day 2	Day 6	P
hsCRP [mg/L]	0.60 ± 0.63	0.50 ± 0.71	0.60 ± 0.52	NS
E-1 [fmol/mL]	0.46 ± 0.57	0.75 ± 1.29	0.60 ± 5.71	NS
PAI-1:Ag [ng/mL]	44.80 ± 24.03	39.73 ± 25.21	42.84 ± 22.59	NS
tPA:Ag [ng/mL]	3.17 ± 2.02	3.77 ± 2.12	3.35 ± 2.65	NS

Abbreviations as in Table 2

Table 8. Medians and standard deviations of the plasma concentration of the measured parameters in the alcohol drinks group (n = 34)

Parameter	Baseline	Day 2	Day 6	P
hsCRP [mg/L]	0.70 ± 0.80	0.70 ± 0.90	0.60 ± 0.48	NS
E-1 [fmol/mL]	0.42 ± 0.45	1.01 ± 1.3	0.97 ± 1.51	0.002
PAI-1:Ag [ng/mL]	44.75 ± 20.37	54.07 ± 19.88	44.80 ± 22.64	0.05
tPA:Ag [ng/mL]	3.65 ± 3.24	4.17 ± 3.96	5.03 ± 4.85	0.02

Abbreviations as in Table 2

The sub-group receiving alcohol-free drinks did not reveal any statistically significant differences in the consecutive measurements of the investigated parameters (Table 7).

Consecutive measurements revealed elevated concentrations of tPA:Ag, PAI-1:Ag and endothelin in the sub-group who received ethanol included in red wine, white wine or equivalent alcohol solution. The concentrations of CRP, measured with the use of a high-sensitivity method, did not undergo any changes (Table 8).

DISCUSSION

The study group examined in the present experiment is a unique one for at least two reasons. First of all, the participants are total lifelong abstainers. Furthermore, their diet was under strict control throughout the experiment and for three months in the lead-up to the study.

C-reactive protein

Our own research did not prove the consumption of any of the beverages to have a statistically significant effect on

the plasma levels of CRP, determined with the use of the high-sensitivity method.

This observation is in concordance with other authors' reports. Mezzano et al. [5] investigating a group of 42 male volunteers drinking wine for 30 consecutive days did not observe any changes in CRP level. Similarly, Retterstol et al. [6] failed to prove the influence of red wine consumption on the plasma hsCRP concentration during a three-week experiment on a group of 87 male volunteers.

Some researchers who have analysed the relation between ethanol consumption and plasma CRP concentration have arrived at an outcome diverging from our observations. Sierksma et al. [7] reported a 35% reduction of CRP related to beer consumption. The crossover trial by Avellone et al. [8] examined a group of 48 volunteers, moderate or occasional alcohol drinkers. Participants consumed 250 mL of red wine a day, with meals, for a period of four weeks. The researchers reported a reduction of CRP serum level. In the study by Estruch et al. [9], the authors also observed hsCRP reduction among wine drinkers.

Our own study does not reveal any statistically significant changes in CRP concentrations in either sub-group. However, the entire experiment accounted for only five days of alcohol consumption, which is actually shorter than any other prospective randomised study quoted above. The study group was also younger than the populations investigated in the majority of the aforementioned reports. Previous drinking habits are also likely to contribute to the reduction of the CRP level observed by some authors.

Endothelin

Our experiment reports a statistically significant increase of endothelin-1 plasma concentrations in the ethanol group, regardless of the kind of alcohol received by the participants. No similar increase was found in the alcohol-free group.

The literature gives only a few accounts of alcohol effect on the levels of endothelin *in vivo*, each revealing changes similar to the results of our own study. Komatsumoto et al. [10] observed, in a group of 24 male volunteers, a statistically significant increase of plasma endothelin-1 level shortly after the consumption of 85 g of alcohol.

The above clinical observations to some extent contradict the conclusions drawn from *in vitro* studies. Corder et al. [11] investigated the inhibited endothelin-1 production in bovine artery endothelial cells in the presence of red wine extract obtained from Cabernet Sauvignon grapes. Those extremely interesting observations largely influenced our own research protocol. Following the observations of Corder et al. [11], in the intervention period of the present study, the participants received Cabernet Sauvignon red wine due to its high content of polyphenolic compounds. The trend toward increased concentrations of endothelin-1 reported in the red wine group implies that, from a clinical point of view, the adverse effect of alcohol included in wine is more significant than the potentially protective function of polyphenolic compounds. The data is nonetheless insufficient to assess the effect that the long-term moderate consumption of any type of alcohol has on endothelin concentrations.

The most pronounced increase in endothelin-1 levels was found in the group receiving 12% ethanol solution, which to some extent overlaps with the findings of experiments on animals. Nanji et al. [12] observed elevated plasma endothelin levels in rats receiving alcohol. Similar results were reported by Tsuji et al. [13] on the basis of *in vitro* studies on human umbilical vein endothelial cells, where incubation with alcohol caused a pronounced increase in endothelin-1 and endothelin-2 release to the growth medium.

In the light of the above quoted experiments, and bearing in mind the results of our own research, two clinical cases of alcohol-induced angina pectoris presented by Kaku et al. [14] seem especially interesting. Those authors reported the medical history of two male patients with chest pain appearing 5–7 h after alcohol consumption. The ECG revealed ST segment elevation during pain in both patients, but coronary angiography did not prove coronary stenosis. Both patients underwent the 'alcohol test' — they were given ethanol in an amount normally causing anginal symptoms. In the effect, plasma endothelin-1 levels were significantly increased in both patients. After six months of alcohol abstinence, the patients' endothelin-1 levels returned to a normal range [14]. The outcome of the experiment by Kaku et al. [14] contributes to the discussion confirming that the elevated levels of plasma endothelin-1 concentrations are preserved long after alcohol consumption. This may suggest that alcohol affects not only endothelin discharge, but also the long-term control of endothelin-1 gene expression.

So far, the long-term alcohol effect on plasma endothelin has not been adequately described. It is also difficult to evaluate the clinical effect of ethanol-induced increase of endothelin levels. The phenomenon, reflecting endothelial damage, may be related to the pathogenesis of adverse effects of heavy drinking. Nevertheless, further research, involving the clinical implications of endothelin activity, should be undertaken to verify the hypothesis.

PAI-1:Ag and tPA:Ag

In the course of the present study, the statistically significant increase in PAI-1:Ag concentration was found only in the red wine group. However, when all the participants were re-arranged into two groups, the one exposed to ethanol, the other receiving alcohol-free drinks, the studied parameter changes turned out to be statistically significant. In the ethanol group, the tPA:Ag levels increased, the values being highest at the third measurement (day 6). The increased levels of PAI-1:Ag found in the same group were statistically significant only at the second measurement (day 2). No such changes in the studied parameters were found in the ethanol-free group.

The outcome of the present experiment confirms the results of the so far published randomised prospective studies, as well as of the epidemiological investigations. Similarly to the present study, Hendriks et al. [15], in an intervention study on a group of eight healthy middle-aged male volunteers, found increased tPA levels after alcohol consumption. Another study compliant with the results of the present research is a crossover experiment on 55 male volunteers, conducted by Dimmitt et al. [16]. The authors reported increasing levels of tPA and PAI-1 antigens related to increased alcohol consumption.

On the other hand, Pellegrini et al. [17] did not prove any impact of 28 days of alcohol consumption on tPA:Ag concentrations. Similarly, El-Sayed et al. [18] did not find any significant alcohol-induced changes in the concentration of tPA:Ag and PAI-1:Ag in healthy male volunteers.

The majority of studies, observational and interventional alike, confirm our own observations concerning ethanol-related increase in tPA:Ag and PAI-1:Ag levels. Still, in reference to the plasminogen activator inhibitor, the studies strikingly contradict the findings of experimental research.

Venkov et al. [19], in a study on cultured human umbilical vein endothelial cells, observed an ethanol-induced increase in tPA gene expression, together with a simultaneous precipitous drop in PAI-1 gene expression. Both changes were found on the transcriptional level, with their intensification being linearly proportionate to the alcohol concentration in the growth medium. The authors claim that the discrepancy between *in vitro* and *in vivo* observations might result from the fact that the experimental model apparently did not take into account complex systemic mechanisms activated as a response to alcohol intake. A similar outcome was reported by Grenett et al. [20] who cultured human umbilical vein endothelial cells with ethanol solution, and observed reduced PAI-1 expression on the transcriptional level. What the authors stress is that it is difficult to predict the effect of ethanol on possible post-translational modifications capable of regulating final serum PAI-1:Ag concentrations induced by alcohol.

As with many analyses, the present study revealed elevated PAI-1:Ag and tPA:Ag levels related to alcohol consumption. The statistically significant increase of PAI-1:Ag observed in the red wine group, but not in the white wine and the alcohol solution group, is quite hard to explain. In our opinion, this phenomenon may be caused by the relatively small groups initially observed in the study. Different dynamics of the increase of both parameters reflected by the highest levels of PAI-1:Ag on day 2 and tPA:Ag on day 6 may be related to alternative sources of these substances as well as to a different regulatory mechanism. The unfavourable effect of alcohol consumption on fibrinolytic markers reported by the majority of analyses and by the present study, may serve as the pathophysiological basis in an attempt to interpret the phenomenon of increased mortality relating to heavy drinking, which was described earlier in the present study. Impaired fibrinolytic mechanisms might, in such situations, result in the hypercoagulation state and, in effect, lead to thromboembolic incidents causing sudden death.

CONCLUSIONS

In the presented study, we have demonstrated that short-term consumption of a relatively large amount of alcohol in a previously abstaining population may have an unfavourable effect on plasma fibrinolytic activity. Fibrinolysis impairment, together with other pathophysiologic effects of excessive alcohol ingestion including dehydration, might facilitate thromboembolic complications. The increase of plasma level of endothelin-1, which was distinctly connected with alcohol consumption, may cause vasoconstriction leading to fatal consequences including myocardial infarction, arrhythmias or stroke. Both these phenomena might serve as a partial explanation of the sudden deaths associated with binge drinking frequently observed in Eastern Europe. However, assessment of the clinical implication of endothelin up-regulation associated with alcohol consumption needs further research.

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Conflict of interest: *none declared*

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Krótkotrwała konsumpcja alkoholu może niekorzystnie wpływać na układ fibrynolityczny i funkcję śródbłonka: wstępne doniesienie z prospektywnego badania z randomizacją

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Streszczenie

Wstęp i cel: Badanie zaprojektowano, aby wyjaśnić wpływ krótkoterminowej konsumpcji różnych typów napojów alkoholowych na osoczowe stężenie wybranych czynników fibrynolitycznych, białka C-reaktywnego (hsCRP) i endoteliny-1 (E-1).

Metody: Grupa badana składała się z 57 zdrowych ochotników płci męskiej w wieku 20–29 lat. Uczestnicy badania byli losowo przydzieleni do 5 grup spożywających przez 5 dni po 300 ml, odpowiednio: wody, soku z czarnej porzeczki, białego wina, czerwonego wina i 12-procentowego wodnego roztworu etanolu. Próbki krwi do oznaczenia CRP, tkankowego aktywatora plazminogenu (tPA), inhibitora tkankowego aktywatora plazminogenu typu 1 (PAI-1) oraz E-1 pobierano 1. dnia przed rozpoczęciem fazy interwencyjnej, 2. dnia rano oraz 6. dnia rano.

Wyniki: Znamienny statycznie wzrost osoczowego stężenia PAI-1 zaobserwowano w grupie spożywającej czerwone wino (dzień 1.: 44,98; dzień 2.: 56,86; dzień 6.: 47,44 ng/ml; $p = 0,05$). W grupie otrzymującej wodny 12-procentowy roztwór etanolu odnotowano natomiast wzrost stężenia E-1 (dzień 1.: 0,53; dzień 2.: 1,6; dzień 6.: 1,11 fmol/ml; $p = 0,01$). Po dokonaniu podziału całej grupy badanej na dwie podgrupy: osób spożywających napoje bezalkoholowe (wodę i sok z czarnej porzeczki) oraz uczestników otrzymujących napoje alkoholowe (wino i roztwór etanolu) ujawniono dalsze istotne statystycznie zmiany osoczowego stężenia badanych parametrów. W grupie spożywającej napoje alkoholowe zaobserwowano istotny wzrost stężenia PAI-1 (dzień 1.: 44,75; dzień 2.: 54,07; dzień 6.: 44,80 ng/ml; $p < 0,05$), a także zwiększenie stężenia tPA (dzień 1.: 3,65; dzień 2.: 4,17; dzień 6.: 5.03 ng/ml; $p < 0,02$) oraz E-1 (dzień 1.: 0,42; dzień 2.: 1,01; dzień 6.: 0,97 fmol/ml; $p < 0,002$).

Wnioski: Krótkoterminowa konsumpcja alkoholu powoduje zwiększenie osoczowych stężeń tPa, PAI-1 i E-1. Obserwowane zjawisko może mieć niekorzystny wpływ na funkcje układu fibrynolitycznego osocza i funkcję śródbłonka.

Słowa kluczowe: spożycie alkoholu, endotelina, fibrynoliza, zapalenie

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