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Management of Cerebral Ischemia in Habitual Alcohol Consumers: The Role of Ethanol

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The harm/benefit ratio for moderate alcohol consumption is a matter of debate. The impact on cardiovascular health is believed to be biphasic: low-to-moderate intake may be protective, but excessive or binge drinking causes harm. Heavy drinking contributes to arrhythmia, atrial fibrillation and hypertension. Furthermore, excessive alcohol intake may accelerate coronary artery disease and type 2 diabetes mellitus through dyslipidemia, vascular inflammation, and insulin resistance, raising risks of stroke, heart failure, and myocardial infarction [1]. According to a recent statement of the American Heart Association, available evidence suggests no risk to possible risk reduction when alcohol is consumed in low amounts in regard to coronary artery disease, stroke, sudden death, and possibly heart failure [2]. Reportedly, moderate intake is associated with a risk reduction of cardiovascular diseases and neuroprotection in Parkinson's disease, Alzheimer's and other dementia [3,4]. Favorable cardiovascular effects may be counterbalanced by health-related and social risks. According to a meta-analysis, alcohol influences the incidence and mortality of stroke according to a J-shaped relationship [5]. Another meta-analysis found that mild to moderate alcohol consumption was associated with a reduced risk only of ischemic stroke, while heavy alcohol use caused increased risk of all stroke types, with a stronger association for hemorrhagic strokes [2,6]. The risk of intracerebral and subarachnoid hemorrhagic stroke may increase with every drink [7]. Review of 11850 adults, including stroke patients, showed that a detectable blood alcohol concentration at hospitalization was associated with significantly decreased odds of the 30-day mortality after critical care [8]. Alcohol use disorder was found to be associated with a lower risk of in-hospital mortality after type A aortic dissection repair [9]. Certain data are conflicting: some studies have found that alcohol is a risk factor for, others - that it has protective role against venous thromboembolism [10]. The pre-stroke alcohol consumption had no significant influence on the stroke severity, short- and long-term outcomes [11].

In experiments, low-to-moderate alcohol consumption has been protective against ischemia/reperfusion (I/R) injury. However, heavy consumption can worsen I/R injury by promoting inflammation [12]. There is considerable experimental evidence of ethanol's favorable action in cerebral ischemia [4]. Physiological explanations for cardiac and neurological benefits of moderate alcohol consumption have raised the concept of ethanol preconditioning, which refers to a phenomenon that heart and brain tissues are protected from harmful effects of I/R by preceding administration of ethanol; potential mechanisms have been reviewed [4]. Simplistically, the beneficial action in cerebral ischemia is conceivable as ethanol is a small nutritive molecule, readily available to neural cells. Analogous hypothesis has been discussed for other substances having nutritive value [13]. As for glucose, the difference is that hyperglycemia may occur in stroke, being associated with adverse effects, so that its blood level should be corrected if indicated, which is beyond the scope of this letter.

Considering the above, ethanol can be used as a first aid for cerebral ischemia especially in aged alcohol-dependent people with no significant blood pressure elevation. In regard to myocardial ischemia and patients without alcohol dependence, further review of literature and well-aimed research is needed. If the patient is conscious, it is a matter of informed consent. If consciousness is impaired, the case history should be taken into account: in a habitual alcohol consumer, the administration of ethanol is generally indicated and may save life. Some aged alcohol-dependent persons confirm that moderate alcohol doses are helpful against dizziness, light-headedness, dullness and other symptoms compatible with transitory or chronic cerebral ischemia. We know a patient with symptoms of vertebrobasilar insufficiency (after an injury of cervical spine), whose working ability increases after a moderate dose [14]. As for the doses, gender and body weight should be taken into account. Clinical research is needed to define the doses; as a rough guess for a male alcohol consumer aged up to 65, having symptoms of cerebral ischemia, 40 ml of ethanol i.e. 100-150 ml of vodka can be tried. This is compatible with recommendations of the National Institute of Alcohol Abuse and Alcoholism (NIAAA) guidelines for acceptable upper limits of alcohol intake for men aged 21-65 years: 14 standard drinks (one drink being equivalent to 14 ml of pure ethanol) per week and four drinks on any given day. For women in the same age bracket and men over 65, the recommended upper limits are seven standard drinks per week and three drinks on any given day [15]. In patients with atrophic gastritis or oesophagitis, equivalent doses of beer or wine may be preferable. It is important, especially for older adults with hypertension, stroke or diabetes, to avoid excessive alcohol consumption in order to mitigate the risk of cognitive decline as alcohol is a known neurotoxin [16]. Poor quality beverages may be outstandingly toxic and should be avoided [17,18]. Future studies should further explore the dose-related effects of alcohol in cerebral ischemia [12].

Conflict of interest statement

The author declares no conflict of interest.

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