

Somatic Complications of Alcoholism in the Surgical Patient

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Abstract: *Background. Alcoholism is a public health problem. Chronic ethanol consumption determines physiological and morphological changes, reflected by an abnormal response to regular medication and nutrients, with consequences on the efficiency of anesthesia and the surgical act.*

Material and methods. A retrospective study was conducted on the latest 1750 cases (2016-2018), hospitalized and operated, in which the preoperative identification of alcohol abuse was made by dosing biochemical markers (gamma-glutamyl transferase, poor carbohydrate transferrin and medium erythrocytic corpuscular volume), by standard questionnaires and, of course, by preanesthetic exam.

Results and discussions. The study showed a rate of 8% (140 cases) of alcohol abuse among these surgical patients. The paper reviews the somatic complications of alcoholism: neurological, gastrointestinal, cardiovascular, metabolic, hematological, immunological and hormonal, and discusses the effectiveness of the study method.

Conclusions. It is proposed to implement a preoperative identification protocol for patients with alcoholism to prevent postoperative withdrawal.

Keywords: *alcoholism; surgical patient.*

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Introduction

Alcoholism is a public health problem (Mihók-Géczi & Hatos, 2019). Chronic ethanol consumption determines physiological and morphological changes (Damian et al., 2019), reflected by an abnormal response to regular medication and nutrients, with consequences on the efficiency of anesthesia and the surgical act (Clero, 2018). Fortunately, in some cases, the stoppage of alcohol is followed by physiological restitution, whether or not passing through withdrawal (Osaki et al., 2016; Schuckit, 2009).

Material and Methods

A retrospective study was conducted on the latest 1750 patients (2016-2018), aged between 42 and 75, hospitalized and operated in 1st Surgery Unit of the County Emergency Hospital Braila, in which the preoperative identification of alcohol abuse was made by dosing biochemical markers (gamma-glutamyl transferase – GGT and carbohydrate deficient transferrin - CDT), by standard questionnaires [fig.1] and, of course, by preanesthetic exam (De Meneses-Gaya et al., 2009; Rumpf et al., 2002). Positive value for CDT was considered ≥ 20 U/l, and for GGT was ≥ 45 U/l in females and ≥ 65 U/l in males. As far as the questionnaire is concerned, it was considered positive to a value of at least 8 points.

1. How often do you have a drink containing alcohol?
 Never 2 to 3 times a week
 Monthly 4 or more times a week
 2 to 4 times a month
2. How many drinks containing alcohol do you have on a typical day when you are drinking?
 1 or 2 3 or 4 5 or 6 7 to 9 10 or more
3. How often do you have 6* or more drinks on one occasion?
 Never Weekly
 Less than monthly Daily or almost daily
 Monthly
4. How often during the past year have you found that you were not able to stop drinking once you started?
 Never Weekly
 Less than monthly Daily or almost daily
 Monthly

5. How often during the past year have you failed to do what was normally expected from you because of drinking?
 Never Weekly
 Less than monthly Daily or almost daily
 Monthly
6. How often during the past year have you needed a first drink in the morning to get yourself going after a heavy drinking session?
 Never Weekly
 Less than monthly Daily or almost daily
 Monthly
7. How often during the past year have you had a feeling of guilt or remorse after drinking?
 Never Weekly
 Less than monthly Daily or almost daily
 Monthly
8. How often during the past year have you been unable to remember what happened the night before because you had been drinking?
 Never Weekly
 Less than monthly Daily or almost daily
 Monthly
9. Have you or someone else been injured as a result of your drinking?
 No Yes, but not in the past year Yes, during the past year
10. Has a relative or a friend or a doctor or other health worker been concerned about your drinking or suggested you cut down?
 No Yes, but not in the past year Yes, during the past year

*—4 in women (National Institute on Alcohol Abuse and Alcoholism. National Alcohol Screening Day).

Procedure for scoring:

Questions 1-8 are scored 0, 1, 2, 3, or 4. Questions 9 and 10 are scored 0, 2, or 4. The maximum possible score is 40. A score of 8 or more is suggestive of problem drinking. For women, the cutoff point should be 4 or more.

Fig. 1 Alcohol Use Disorders Identification Test (AUDIT)

Results and discussions

The study showed a rate of 8% (140 cases) of alcohol abuse among these surgical patients, most men (137), average age 53.

All patients (100%) presented various neurological complications in terms of type and intensity, 84 (60%) had gastrointestinal co-morbidities associated with alcohol abuse (in 2 cases, gastric hemorrhagic ulcer was the reason for hospitalization), 69 (50%) were diagnosed from the beginning with neoplasia, 35 (25%) had cardiovascular suffering induced by alcohol

consumption, and in 104 cases (78%) metabolic, nutritional and hematological disorders were reported.

73 patients (52%) with positive CDT and GGT values, with or without significant AUDIT testing, were considered at risk of alcohol withdrawal. Prophylaxis used benzodiazepine, their internship in ICU was 3 days on average, and the treatment lasted at least 5 days.

Although all patients had severe postoperative evolutions, with significant associated morbidity and prolonged hospitalization, the immediate surgical outcome was favorable in all cases.

It is considered useful to review the somatic complications of alcoholism encountered in the studied surgical patients.

Neurological complications of alcohol consumption (Sullivan et al., 2010; Noble & Weimer, 2014)

- Alcoholic dementia is produced both by the direct toxic effect of alcohol and by the multiple nutritional and vitamin deficiencies, induced by chronic alcoholism, as well as by repeated cerebral traumas. It is manifested in chronic alcoholics, after many years of degradation and social marginalization.

- Haemorrhagic or thromboembolic stroke occurs due to hypertension, dyslipidemia, atherosclerotic disease and/or coagulopathy. Coagulopathy induced by chronic alcohol consumption may explain the increased incidence of subdural hematomas, in the case of craniocerebral trauma.

- Alcohol-related symptomatic epileptic seizures are usually generalized, they do not have the aura and appear shortly (6-8 hours) after discontinuing their use, and they may have the withdrawal removed. The chronic use of alcohol and its repeated interruptions lower the threshold of epileptic seizures. The risk of crises is proportional to the amount of administered alcohol.

- Cerebellar manifestations (coordination, equilibrium disorders) are also the causes of ethanol intoxication.

- Peripheral alcoholic neuropathy appears to be produced both by direct action on the nerve fiber (including vegetative), and as a consequence of deficient nutrition. Symptomatology may include: paraesthesia, limb pain, hypotonia and muscle cramps, heat intolerance (after exercise), impotence in men, urinary incontinence/dysuria, constipation, diarrhea, nausea, vomiting, etc.

- Alcoholic amblyopia, consisting of visual disturbances, including scotomas, and decreased visual acuity, starting with the central portion of the visual field, is caused by the toxic effect of alcohol on the optic nerve, with the induction of optical neuropathy. Because alcohol generates depletion of the entire body of nutrients, alcoholic amblyopia is clearly linked to thiamine deficiency. Alcoholic amblyopia is reversible if treated with proper diet and multivitamins, especially vitamin B1. Ignoring it leads to irreversible lesions of the optic nerve and cecity.

- Alcohol influences sleep as it is a stimulant that leads to insomnia, as well as fatigue, primarily at the psychological level, during the day. Paradoxically, many people with chronic alcohol use require alcohol to reduce activation of the sympathetic system, coupled with withdrawal, in order to fall asleep. In a middle-aged non-alcoholic person, even moderate use of alcohol at dinner reduces the REM (Rapid Eye Movement) sleep period. After cessation of alcohol consumption, symptoms occur which are manifested by living dreams that usually awaken the person thus forming a vicious circle, by disrupting the REM phase, which ultimately leads to sleep deprivation. The appearance of obstructive apnea during sleep may be caused or aggravated by alcohol, due to the inhibitory effect of alcohol on breathing and the relaxation of the upper respiratory tract.

Gastrointestinal complications of alcohol consumption (Rehm et al., 2010; Engen et al., 2015)

- The effect of alcohol on the liver. Liver cells preferentially use alcohol as fuel in energy metabolism. Alcohol, even in small quantities, disrupts gluconeogenesis and leads to the metabolic shunting of some carbohydrate into lipids. Hepatic impairment begins with liver steatosis (which occurs with the presence of constant levels of alcohol in the blood above 80 mg%), followed by alcoholic hepatitis, fibrosis and, finally, cirrhosis of the liver. Chronic alcohol use exacerbates postnecrotic infectious hepatitis by stimulating viral replication, and implicitly interfering with therapeutic success. The combination of alcoholism and hepatitis C increases the toxic effect on the liver even more than just the additive effect of the two.

- The effect of alcohol on the stomach. Alcohol favors the colonization of the stomach with *Helicobacter pylori*. This bacillus, by the production of ammonia, promotes bacterial multiplication with the occurrence of gastritis and ulcers (a fact particularly found in wine and beer consumers). Alcohol can cause acute hemorrhagic gastritis, but is not

directly responsible for the development of ulcer disease; it acts concurrently with *Helicobacter pylori*, in the genesis, worsening and delaying gastric ulcer healing. Chronic ethanol consumption increases the incidence of gastroesophageal reflux by disrupting normal peristalsis, secondary to autonomic neuropathy. Increased alcoholism leads to a delayed gastric emptying of the solid bowl and to an acceleration in the discharge of the liquid portion of the food bowl.

- The effect of alcohol on the small intestine. The small intestine is exposed to high alcohol concentrations during alcohol consumption. This leads to changes in the cellular structure, metabolism and vascularization of the small intestine. Alcohol predisposes to hemorrhagic erosion of intestinal villi and the appearance of duodenitis. Combined with accelerated motility, this prevents the absorption of important nutrients, which over time may be the cause of a variety of medical sufferings.

- The effect of alcohol on the colon. It is reduced to the appearance of hemorrhoids, secondary to portal hypertension, due to liver dysfunction. Alcohol also lowers the non-propulsive activity of the colon, along with the increase in propulsive motility, which exacerbates hemorrhoidal disease.

- The effect of alcohol on the pancreas. Alcohol interferes with the exocrine secretion of the pancreas by fragilizing cellular storage structures; thus, there is inflammation of the ductal wall, which blocks the pancreatic duct and leads to the stimulation of enzymatic production. The combination of these processes leads to the occurrence of chronic and/or acute pancreatitis. By recurrent episodes of pancreatitis, manifested by exocrine and endocrine insufficiency, diabetes mellitus and hypovitaminosis can be developed by poor absorption (A, D, E, K, F).

Cardiovascular complications of alcohol consumption (Gardner & Mouton, 2015; Ren & Wold, 2008)

- Alcohol-related cardiac changes. Chronic alcoholic patients may develop a form of cardiovascular disease. Pathogenesis is primarily due to the toxicity of alcohol on the striated muscle fiber that leads to cardiac inflammation, cardiomyopathy, arrhythmias and left ventricular abnormalities. In addition to the direct toxic effect, there is secondary damage, through alcohol induced arterial hypertension and dyslipidemia, which contributes significantly to cardiovascular morbidity. Cardiac response to alcohol reflects physical individuality, consumption rate and quantity of ingested alcohol. In patients with left ventricular dysfunction, the effects of alcohol inhibitors, with decreasing the ejection fraction, prevail. Alcohol also

alters the loco-regional circulation of blood, increasing the cutaneous, splanchnic and myocardial flow, reducing the cerebral, pancreatic and striated muscular ones.

- Alcoholic cardiomyopathy involves myocardial damage. Alcohol dependence can lead to two types of cardiac dysfunction: thiamine-dependent (alcoholic beriberi disease) and thiamine-independent (alcoholic cardiomyopathy). The pathophysiology of alcoholic cardiomyopathy is still unclear. Those with chronic alcohol use (usually more than 10 years of consumption) frequently experience myocardial hypertrophy and varying degrees of myocardial and perivascular fibrosis. Echocardiography reveals dilated cardiomyopathy, and electrocardiographic changes are not specific. Alcoholic cardiomyopathy is generally irreversible, although an improvement in symptoms can be achieved by abstinence. Beriberi disease is a rare cause of heart disease in alcoholics; it is characterized by cardiomegaly, hyperdynamic circulation, circulatory congestion and thiamine reversibility.

- Alcohol-related cardiac toxic syndrome consists of disturbing the rhythm or cardiac conduction (especially supra-ventricular tachycardias), associated with massive alcohol consumption in people without pre-existing cardiac diseases. The most common arrhythmia is atrial fibrillation that spontaneously converts to sinus rhythm within a 24-hour period. The syndrome is recurrent but its evolution is benign, and anti-arrhythmic treatment is usually not necessary. The mechanism of cardiac toxic syndrome is not fully elucidated, but hypotheses include excessive secretion of epinephrine and nor-epinephrine, increased levels of fatty acid blood and indirect action of acetaldehyde (the primary metabolite of alcohol).

- Hypertension often occurs in withdrawal, even in patients without a history of hypertension, and may lead to serious complications. The type of alcoholic beverage consumed (beer, wine, whiskey) and age (over 50 years) can influence the impact of alcohol consumption on blood pressure.

Metabolic complications due to alcohol consumption

- The effect of alcohol on cholesterol. Hyperlipidemia is the result of lipogenesis, accentuated by alcohol oxidation. The liver cells preferentially use alcohol instead of lipids, which leads to the accumulation of lipids in the liver and their subsequent secretion in circulation. As alcohol use leads to hepatic impairment, secretor dysfunction of the liver can lead to the opposite effect, respectively malnutrition and cachexy.

- Glucose. Alcohol leads to abnormalities in regulating carbohydrate metabolism through hepatic and pancreatic dysfunctions. Decreasing tissue

sensitivity to insulin, during alcohol withdrawal, and decreasing insulin response, during acute alcohol consumption, can lead to extreme levels of glucose in the blood. Alcoholic ketoacidosis may occur as a result of disturbing the hydroelectrolytic balance by decreasing Na, K, Mg, Ca and P.

- Proteins. Alcohol leads to cerebral atrophy by decreasing cerebral protein synthesis. Although the effects on hepatic protein synthesis have not been elucidated, alcohol is known to interfere with hepatic secretion of proteins and this phenomenon underlies the hepatocyte “ballooning effect”, seen in cellular necrosis, from alcoholic liver dysfunction.

- Nutrition. Alcohol is a non-nutritional source of calories. Factors contributing to nutritional deficiency are direct toxicity to tissues, disruption of nutrient absorption and metabolism. Lack of thiamine leads to Wernike-Korsakoff Syndrome (WKS), beriberi disease and polyneuropathy. Lack of pyridoxine produces neurological, hematological and dermatological dysfunctions.

Decreasing the amount of folic acid and cyanocobalamin is manifested by megaloblastic anemia and neurological deficits associated with it. Decreased zinc is associated with dry and rough skin, mental lethargy, impaired taste and low appetite. Improved nutrition and abstinence are needed to correct these deficits, as well as supplements with thiamine, folic acid and multivitamins for at least 3 months. Severe cases of malnutrition should be treated by intramuscular administration of thiamine, as its poor absorption is frequently present.

Hematological complications due to alcohol consumption

Hematological complications due to alcohol consumption include anemias (iron deficiency, vitamin B12 deficiency, occult chronic upper gastrointestinal bleeding, chronic inflammation in tissue level), leucopenia and thrombocytopenia.

Immunological complications due to alcohol consumption (Ratna & Mandrekar, 2017; Bagnardi et al., 2015)

Alcohol-induced immune system dysfunction leads to an increased risk of infections and neoplasms.

- Infections. Alcohol use predisposes to infections, through malnutrition, splenic dysfunction, leucopenia, granulocytic dysfunction and impaired pharyngeal reflex. Aspiration pneumonia, empyema, HIV, sexually transmitted diseases, cerebral abscesses, meningitis, spontaneous bacterial

peritonitis and tuberculosis are common in chronic alcohol users and have an increased mortality rate.

- Neoplasms. People with chronic alcohol use are at increased risk of respiratory and digestive cancer (lips, oral cavity, tongue, pharynx, larynx, lung, esophagus, stomach, liver and bile duct, colon), and hepatocellular breast cancer. This risk remains high, even when other factors such as diet, smoking and lifestyle are excluded. Alcohol deteriorates immunological function, potentiates the carcinogenic effect of other factors and causes irritation.

Hormonal complications due to alcohol consumption

Alcohol consumption alters the plasma levels of parathormone, insulin, prolactin, cortisol, somatotrophic hormone and adenocorticotropine. In men, the decrease in testosterone production leads to decreased sperm quality, sexual dysfunction and infertility. In the case of advanced liver dysfunction, with abnormal estrogen secretion, testicular atrophy and gynecomastia occur. Premature menopause, infertility, dysmenorrhea and metrorrhagia, due to malnutrition and coagulopathy, may occur in women.

Conclusions

- The prevalence of chronic alcoholism in surgical patients is significant (8%), especially for men.

- The somatic complications of alcoholism are varied, the most common ones being neurological (100%), metabolic and haematological (78%), gastrointestinal (60%), neoplastic (50%) and cardiovascular (25%). Their pathophysiology must be known for correct, individualized, application of the treatment and therapeutic success (Sandu, 2018).

- Alcohol withdrawal syndrome, which is a serious complication with increased morbidity and mortality, occurred in over 50% of these patients in the absence of appropriate prophylaxis. Benzodiazepine prophylaxis significantly decreases the incidence and intensity of this syndrome, decreases the rate of postoperative complications, and implicitly, the duration of hospitalization (Mirijello et al., 2015; Mo et al., 2018).

- The best way to identify alcohol abusers among surgical patients is the combined use of CDT and GGT biomarkers (Jastrzębska et al., 2016; Andresen-Streichert et al., 2018; Torrente et al., 2019). Questionnaires to identify alcoholism need to be further refined (Kummer et al., 2016; Chitescu et al., 2018).

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