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UDC 616.89-008.441.33:616.31**FEATURES OF ANESTHESIA PROVISION IN MAXILLO-FACIAL SURGERY IN PATIENTS WITH COMPLICATED MEDICAL HISTORY (LITERATURE REVIEW)**O.V. Kravets¹, V.V. Yekhalov¹, A.V. Samoilenko², I.A. Romaniuta², O.V. Pylypenko¹, O.V. Kovryha¹¹*Dnipro State Medical University, Department of Anesthesiology, Intensive Care and Emergency Medicine, Faculty of Postgraduate Education, Dnipro, Ukraine*²*Dnipro State Medical University, Department of Therapeutic Dentistry and Faculty of Postgraduate Education Dentistry, Dnipro, Ukraine*

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Abstract: Currently, the problem of chronic alcoholism affects all segments of the population in the whole world. Providing dental care to patients suffering from this pathology is a complex task that requires a comprehensive approach. Alcohol and its metabolites have a negative effect on the condition of the oral cavity. The incidence of leukoplakia, erythroplakia, glossitis, caries, periodontitis and carcinoma of the oral cavity is several times higher in drinkers compared to non-drinkers. This is associated with several factors. Most alcoholic beverages have chemical properties which can be damaging to the tooth enamel and mucous membranes. Vomiting can create a destructive acidic environment in the mouth. Metabolites of ethyl alcohol, like acetaldehyde, can also damage the mucous membranes of the oral cavity and affect its microbiome. Acetaldehyde is also a known carcinogen, and it contributes to the development of oral cancer. Acute alcohol intoxication provokes systemic inflammation response, while the immune system is compromised and cannot react adequately to the infection. Moreover, alcoholism impacts personality, which leads to the changes in eating habits and deterioration of personal hygiene – both are the factors which eventually affect oral health. Somatic diseases often occur in alcohol-dependent persons, and can also manifest in lesions of the oral cavity. Functional and morphological disorders of various organs, systems and metabolism in patients create problems not only in the treatment of dental pathology, but also in anesthesia. Alcohol-induced personality changes hinder the development of positive and healthy doctor-patient relationships. Disorders of the cardiovascular and nervous systems need adjustment of the dosage and selection of drugs. Liver dysfunction may lead to hypocoagulation and facilitate serious peri- and postoperative bleeding and hemorrhage. All these changes require appropriate correction in the perioperative period: such patients need careful monitoring and management of delirium, potential withdrawal syndrome, cardiovascular dysfunction and coagulation disorders. Both acute alcohol intoxication and chronic alcoholism significantly alter the metabolism of inhaled and intravenous anesthetics, opioids, and other drugs used for anesthesia. Doses of anesthesia drugs should be reduced in case of acute alcohol intoxication and adjusted during elective procedures, taking into account the fact that in chronic alcoholism, a cross-tolerance forms between alcohol and most of the drugs used for anesthesia. Acetaminophen dose should be adjusted, considering impaired liver function. Tolerance to hypoxia is usually decreased. Also, such patients are at high risk for regurgitation and aspiration, and have an increased acidity of the gastric contents. Regional anesthesia and analgesia in such patients also have their own features, because such patients tend to have a combination of decreased sensitivity to local anesthetic with a compromised cardiovascular function and increased rate of side effects, which makes choosing a correct dose a challenge. Pre-medication plays a significant role in reducing anxiety and intoxication symptoms. Recovery period may be complicated by alcohol withdrawal syndrome. Healthcare professionals' awareness of the pathophysiology of chronic alcoholism, timely treatment and promotion of patients' abstinence from alcohol consumption can reduce the number of complications and mortality in dental patients.

Keywords: chronic alcoholism, oral cavity lesions, dental treatment, anesthesia, complications.

Introduction. More than 2 billion people worldwide consume alcohol, and approximately 80 million of them face oral health issues associated with it [1, 2]. The General Dental Council of the United Kingdom has officially included the study of alcoholism pathophysiology in the educational programs for students and residents specializing in Dentistry [3].

Features of dental care in chronic alcoholism. Non-carious dental diseases, including erosive tooth wear (ETW), are partially linked to the presence of polyphenols

in various types of alcoholic beverages. These substances contribute to the removal of proline-rich proteins (precursors of dental calculus) from saliva [4]. The bitterness of certain alcoholic drinks is due to their high concentration of tannins, which can bind mucopolysaccharides to proteins, leading to their precipitation and a loss of oral cavity mucosal moisture and acid protection. Acidic wines increase the sensitivity of the oral cavity and tooth surfaces to mechanical damage during tooth brushing. This also occurs due to vomiting induced by the influence of alcohol

on the esophageal sphincter and reduced salivary secretion (xerostomia) [2, 5, 6, 7]. Morning nausea and vomiting have been termed the "toothbrush elevation syndrome". Alcoholics often resort to taking pills in the morning to alleviate hangover symptoms. The formation of an acidic environment in the oral cavity due to vomiting contributes to the suppression of saliva secretion and its buffering capacity, which increases erosive damage to tooth enamel. Typically, the palatal surfaces of upper teeth and then the occlusal surfaces of lateral teeth are most affected, while lower teeth and upper buccal surfaces are affected the least. Several studies have shown a direct correlation between the duration of alcohol contact with tooth surfaces and the extent of ETW, reaching 50% with prolonged exposure [2]. Acid erosion is considered a professional risk for winemakers and sommeliers [7]. Alcohol-dependent individuals often experience dryness in the mouth at night, consume high-carbohydrate food and drinks, and neglect oral hygiene [8, 9]. The basic trigger factors for ETW are age, gastroesophageal reflux disease lasting more than 1 year, and daily alcohol consumption of ≥ 240 g [4, 10]. The overall prevalence of ETW in alcohol-dependent individuals is 98.6%. Among the studied heavy drinkers, localized erosive enamel lesions were found in every other individual, generalized lesions in 40.1%, and enamel erosions of the upper jaw teeth in 7.2%. Erosive damage occurs less frequently on the palatal, lingual, and buccal surfaces [4, 10].

After the consumption of ethanol, the concentration of acetaldehyde (AA) in saliva increases and exceeds its content in the blood serum. A high concentration of AA persists in the oral cavity for an extended period, leading to damage to mucosal and glandular tissues, along with the suppression of their immune functions and transmucosal microbial migration [5, 11]. Lipodystrophy and depletion of fibrovascular tissue progress in the sublingual salivary gland, but there is no significant reduction in its volume. Regional blood flow, protein levels, and amylase increase, which is attributed to hypertrophy and increased acinar function with lipodystrophy of functional tissues near the parotid salivary gland. Conversely, a decrease in the secretion of total protein, amylase, and salivary flow rate in salivary glands is associated with statistically significant sialadenitis [7]. Systematic alcohol consumption can lead to the development of peripheral neuropathies caused by sialadenosis and swelling of the parotid salivary glands in 30-80% of alcohol-dependent individuals [4, 6, 12].

Acute alcohol intoxication leads to increased levels of cytokines IL-12 and IFN- γ and decreased levels of the anti-inflammatory cytokine IL-10 [4, 13]. This biochemical process is often complicated by inflammation of the tongue (glossitis), gingiva (gingivitis), and sometimes the corners of the mouth (angular cheilitis). Initial signs of glossitis manifest as a painful, smooth tongue with periodically swollen papillae. In the later stages of the pathological process, the tongue takes on an intensely red color with subsequent atrophy of filamentous and fungiform papillae. Angular cheilitis is characterized by painful fissures at the corners of the mouth, while gingivitis results in areas of necrosis on the apical surfaces of interdental papillae [2, 6, 9]. Triggers for these conditions include the effect of harmful chemical substances, alcohol anesthesia, bacterial accumulation, biofilms (toxins are not eliminated with saliva), and xerostomia [9, 14]. Leukoplakia is the most

common pathology (18.4%), followed by erythroplakia (2.6%), submucous fibrosis (7.9%), and mycoses (2.6%) [8]. Depending on the concentration of alcohol, its consumption within a period of up to 12 days causes epithelial hyperplasia, acanthosis, chronic inflammatory infiltration, and vascularization. In the female group (40%), the most severe changes were observed in the mucous membrane of the cheeks and tongue. 10% of tissue sections demonstrated points of epithelial hyperplasia with acanthosis and hyperkeratosis. 90% of microtomes exhibited points of epithelial atrophy of various degrees of damage. Moderate infiltration with lymphocytes and macrophages was detected in the basal layer of the oral mucous membrane. In the deep mucosal layers, there may be atrophy of muscle fibers with involvement of the neuroregulation system. Cytomorphometric analysis of the oral mucous membrane in individuals over 25 years of age who consumed at least 45 ml of alcohol per day for a minimum of 10 years showed changes in cells, characterized by an increase in the average area of the cytoplasm and nucleus, as well as an increase in the cell-nuclear parameters ratio [9, 14]. Alcohol consumption for more than 12 months is capable of causing leukoplakia-like epithelial dysplasia (dyskeratosis or keratosis), thickening of the basal layer of the epithelium, and some increase in the fraction of mitotic figures. Usually, the most affected area of transformation is the floor of the oral cavity. Initial signs of leukoplakia include erythema, swelling, and thickening of the oral mucous membrane, which later turns white or gray. Its surface may be smooth or wrinkled and may be at the same level as the surrounding tissues or raised above them. In most cases, leukoplakia progresses asymptotically [5]. Active cellular proliferation is the initial stage of carcinogenesis [14]. Erythroplakia is considered a relatively rare oral pathology with a high degree of oncogenicity [15], visualized as a slightly elevated red lesion of the oral mucous membrane that may be associated with leukoplakia. It usually progresses asymptotically. Lesions are mostly located on the floor of the oral cavity, soft palate, tongue base, and cheek mucous membrane. Most specimens demonstrate a high level of epithelial dysplasia or invasive carcinoma [5]. In addition to atrophic changes, glossitis is also responsible for the elongation of filamentous papillae (black or white "hairy" tongue). Recurrent painful aphthous ulcers on the oral mucous membrane have an autoimmune origin [5].

Alcoholism is an underappreciated trigger for precancerous conditions of the oral cavity, primarily due to the formation of acetaldehyde resulting from the oral microbial conversion of ethanol. Acetaldehyde directly damages DNA through the formation of mutagenic adducts and interstrand crosslinks. In 2012, the International Agency for Research on Cancer officially classified alcoholic beverages and acetaldehyde as Group 1 carcinogens for humans. Precancerous conditions of the oral cavity include leukoplakia, erythroplakia, red flat lichen, and submucous fibrosis. The prevalence of these lesions increases with age, and early prevention is crucial for preventing oncogenic transformation [5,7]. Simultaneous alcohol consumption and tobacco smoking increase the concentration of acetaldehyde in saliva to 40 $\mu\text{M/L}$, with an established threshold of 5-10 $\mu\text{M/L}$ for acetaldehyde to promote carcinogenesis. Acetaldehyde is formed from alcohol, binds to nuclear DNA and proteins in cells, damaging the genetic

code and leading to folate destruction, which results in increased cell proliferation. Practically all oral cancers are being diagnosed at advanced stages, worsening the prognosis [14, 16]. Histological examination of the buccal mucosa reveals signs of pyknosis, karyorrhexis, and karyolysis with keratinization [14, 17]. Morphologically altered cell nuclei indicate a high degree of oncogenicity [16]. Dietary deficiency reduces the activity of the antioxidant system in alcohol consumers, which should prevent tissue oncogenic transformation [2, 16].

All types of alcohol, including hard liquors, wine, and beer, are associated with oral cavity cancer [11]. Consuming ethanol four or more times a day increases the risk of oral cavity cancer by 400% compared to non-drinkers. Carcinoma is more common in the 50-70 age group, but oncogenic risk is more associated with the quantity of alcohol consumed than with age [5, 17]. In the French population, the population risks of oral cavity cancer reach 80.7% with simultaneous use of tobacco and alcohol [14]. The most common malignant neoplasm of the oral cavity is squamous cell carcinoma, which results in high mortality because it is often diagnosed at advanced stages [15]. The initial symptoms of oral cavity cancer include a burning pain accompanied by difficulty in swallowing, teeth loosening, and constant bleeding. These signs typically indicate the progression of a process already in an advanced stage. Initial lesions appear as clearly defined erythema of the mucous membrane with a velvety or smooth surface, with or without white patches. Later, the carcinoma may transform into an ulcer. Exophytic growth without ulceration, with signs of leukoplakia, is less common. The most frequent site of oral cancer is the so-called retromolar area, which includes the floor of the oral cavity, lateral borders, the ventral surface of the tongue, the retromolar region, and the soft palate. Metastases to the cervical lymph nodes are typical for malignant oral cancer. The diagnosis of cancer is based on clinical examination, biopsy, and radiological studies. Treatment for this condition is almost always surgical, combined with radiation and chemotherapy [5, 12]. The consumption of the most common alcoholic beverages is associated with the highest risk of developing oral cavity cancer [4].

Alcoholics often exhibit excessive gum inflammation with swelling and a bluish-red color, as well as bleeding upon slight provocation [2, 6, 7, 8, 16]. Changes in oral tissues are also associated with increased oxidative stress, which occurs in alcohol consumers due to elevated oral peroxidase activity, leading to worsening periodontal health [1, 6]. The prevalence of periodontitis is three times higher in alcoholics compared to non-drinkers [8]. Alcohol causes complement deficiency, impairs neutrophil function (reduced adhesion, mobility, and phagocytic activity), and increases the frequency of periodontal infections [8], which are related to gum inflammation, interdental papilla involvement, and deep gum pockets with bone loss. Men are more prone to horizontal bone loss and dental calculus compared to women, and alcoholism is considered a risk factor for osteoporosis. Patients prescribed bisphosphonates for osteoporosis treatment are at risk of medication-related jaw osteonecrosis following tooth extraction [4]. Early research established a link between alcoholism and periodontitis, but most researchers attribute the higher frequency of periodontitis to poor oral hygiene [4, 18]. Alcohol-dependent individuals with periodontitis have a

higher frequency of certain periodontal pathogens, particularly *Prevotella intermedia*, *Eikenella corrodens*, and *Fusobacterium nucleatum* [4].

A direct correlation has been established between regular alcohol consumption and tooth decay (caries) [4]. Alcoholics typically have significantly higher rates of tooth decay, leading to the need for tooth extraction or restoration (filling). Consequently, alcoholics experience three times more loss of permanent teeth than the average population [8, 9]. Comparative analysis has shown significantly fewer preserved teeth and more active carious lesions in alcoholics, who also have a higher number of endodontically treated teeth compared to non-drinkers [2, 6, 8, 9].

Apart from the direct damage to the oral cavity, alcoholics suffer from a range of indirect consequences due to inadequate nutrition [5]. Vitamin A deficiency contributes to the development of leukoplakia, epithelial metaplasia, and salivary duct keratinization leading to xerostomia; vitamin B deficiency results in beriberi, Gayet-Wernicke syndrome, and Moeller-Hunter glossitis; folate deficiency manifests as inflammation, ulcers, and necrotic changes in the gingival papillae, tongue inflammation, and may lead to megaloblastic anemia and delayed wound healing; vitamin K deficiency leads to bleeding after invasive dental procedures due to impaired hepatic production of blood clotting factors; vitamin C deficiency (scurvy) is characterized by the presence of gingivitis with swollen gums and periodontal pockets, further promoting inflammation with petechiae, ecchymosis, and oral ulcerations; iron deficiency is complicated by hypochromic anemia, glossalgia, erosive or aphthous lesions of the gums with the development of Plummer-Vinson syndrome, precancerous conditions, and cancer [4, 5, 13, 19].

Alcohol-related liver disease is associated with oral candidiasis due to immune system suppression, carcinomas of the tongue and floor of the mouth [5], but hematological disorders may manifest even without apparent liver disease [19].

In cases of alcohol-induced cardiac depression and hypertension, there is dilation of capillaries and veins on the ventral surface of the tongue, with varicose changes and telangiectasia [20].

Alcohol and malnutrition are linked to complete atrophy of the papillae on the dorsal surface of the tongue, referred to as "smooth tongue", and dysgeusia (distorted taste perception). Alcohol-induced epilepsy often leads to tooth fractures and lip and tongue injuries. Uncontrolled movements of the chewing muscles and tongue are accompanied by buccal mucosa biting. Such injuries often have complications in the form of secondary infections [5], potential depletion of beneficial commensal bacteria, and increased colonization by potentially pathogenic (including gram-negative) microorganisms with impaired periodontal resistance [1, 21, 22, 23]. Alcohol takes longer to exert its bactericidal action than just the act of swallowing and drinking [2, 4, 5, 18]. In drinkers, the frequency of postoperative infections increases 3-5 times compared to non-drinkers [9, 13, 14]. It is mostly associated with acute pseudomembranous or atrophic candidiasis [5, 18]. Alcoholics exhibit slower wound healing because ethanol, as demonstrated, reduces the mobility and phagocytic ability of leukocytes, theoretically increasing the risk of infection and osteomyelitis after tooth extraction [4, 24].

Elevated lead levels in the blood of alcoholics result in "lead" stomatitis with a blue-black line on the gums, parotitis, basophilic granularity of erythrocytes, poikilocytosis, and leukocytosis. In case of alcoholism, lead levels typically do not reach critical values [2, 25, 26].

Cephalosporins, metronidazole, ketoconazole, and alcohol can interact, causing a disulfiram-like reaction [2, 4, 6].

Features of anesthesia provision in dental patients with chronic alcoholism

The treatment of patients with alcohol dependence of dental profile presents many challenges for both the dentist and the anesthesiologist. Patients (victims) with signs of acute alcohol intoxication are often prone to anti-social, and sometimes violent behavior that can be difficult for healthcare professionals to handle, hindering the development of positive and healthy doctor-patient relationships [4]. Elective care for such patients should be postponed, and in cases of urgent life-threatening indications, medical assistance is provided exclusively in a hospital setting.

Examinations should focus on the cardiovascular system (hypertension, arrhythmias, and signs of heart failure) and the nervous system (visual impairment, coordination or cognitive function impairments, or signs of autonomic or peripheral neuropathy). Specific symptoms of liver disease (risk of hypocoagulation) should also be monitored [19]. The influence of alcohol on platelets, blood coagulation factors, and the fibrinolytic system can lead to serious peri- and postoperative bleeding and hemorrhage [13].

Acute alcohol intoxication can cause perioperative complications [27]. If the concentration of ethanol in the blood is elevated, competitive inhibition of metabolic enzymes can increase sensitivity to anesthetic drugs [19].

The presence of alcohol in the body can decrease and slow down the action of local anesthetics depending on the dosage. Alcohol reduces blood pH, leading to metabolic acidosis, which counteracts the dissociation of local anesthetic molecules. The corresponding acidity for their effectiveness is approximately 7.35-7.4, corresponding to the normal pH of human blood [28].

Patients with heart diseases are more sensitive to local anesthetics, and the patient may experience a heart attack. The anesthetic effect and duration of action of lidocaine are limited by chronic alcohol consumption. The dose of the local anesthetic in alcohol-dependent patients to achieve the necessary analgesic effect is increased by 15-20%, but the cardiotoxicity of the drug increases proportionally to the dose [28, 29].

When providing anesthesia for emergency dental interventions in individuals in the acute phase of alcohol intoxication, it should be noted that these patients are less tolerant to hypoxia [30, 31].

Alcohol, regardless of the dose, increases gastric acidity and volume, delays gastric emptying, and suppresses laryngeal reflexes, thereby reducing the ability to protect the airway [32, 33]. Due to the significant risk of vomiting and aspiration (even after gastric tube emptying), it is recommended to use rapid sequence induction and rapid tracheal intubation to prevent aspiration pneumonia [13, 19, 33].

Pain sensitivity varies widely depending on the level of alcohol consumption, hence the risk of underdosing or overdosing the required anesthetic dose [34, 35, 36].

Ethanol is a central nervous system (CNS) depressant that alters the function of ion channels in several receptor areas, including N-methyl-D-aspartate, serotonin 5-hydroxytryptamine, glycine, and γ -aminobutyric acid receptors [33]. During acute ethanol intoxication, anesthetic doses are reduced due to additional CNS depression, even in patients with chronic alcohol use disorder [13, 33].

Intraoperative management should focus on three main areas: combating intoxication (if the patient is still in a state of alcohol intoxication, especially during emergency surgery), preventing or treating withdrawal, and achieving adequate recovery and effective pain relief. For the latter, multimodal analgesia and/or regional anesthesia are recommended [37].

Volatile anesthetics compete with ethanol for binding to neuronal receptors of γ -aminobutyric acid and glycine [19, 38]. Ethanol significantly reduces the minimum alveolar concentration (MAC) of sevoflurane in patients and laboratory animals [39]. When maintaining anesthesia, volatile agents are carefully titrated since MAC is usually lower in patients with alcohol intoxication [13, 33].

Alcohol enhances and prolongs the effects of benzodiazepines (midazolam, diazepam, alprazolam, etc.) and potentiates the effect of opioids [4, 13, 35]. In the acute phase, the doses of propofol and thiopental should be reduced, as their metabolism involves cytochrome P450, which is non-specifically inhibited by alcohol [13, 29, 33, 35].

It is advisable to limit the use of acetaminophen (paracetamol) in patients with chronic alcohol use disorder due to the possibility of developing acute liver failure, even with moderate therapeutic doses of acetaminophen [13, 33]. Alcohol enhances and prolongs the action of muscle relaxants with hepatic metabolism, except for atracurium, mivacurium, and doxacurium, which are primarily excreted in the urine [35].

In elective cases, surgical intervention should be postponed until the consequences of acute intoxication have subsided to obtain informed consent and allow time for gastric emptying [33].

Alcohol abuse is typically associated with a tolerance to the effects of central nervous system depressants [32]. Anesthesiologists should be aware of cross-tolerance between alcohol and most volatile anesthetics, as their alveolar concentration decreases [35, 40]. The distribution and metabolism of anesthetics are distorted by hypoalbuminemia and hepatobiliary dysfunction [19, 38]. In alcohol-related liver dysfunction, the clearance of halothane is reduced [13]. Regular alcohol use distorts the effects of isoflurane and nitrous oxide [29].

There is a need to increase anesthetic doses in patients with chronic alcoholism. This can be a consequence of the induction of the enzyme cytochrome P-450 2E1 and is associated with the development of cross-tolerance. Enzyme induction due to chronic alcoholism enhances detoxification pathways' efficiency and increases the inactivation of alcohol, sedatives, and narcotics. This can lead to a decreased clinical response and the need to increase effective doses of propofol, thiopental, opioids, etc. [19, 33, 41].

Pre-medication with benzodiazepines allows an alcoholic to manage anxiety and trauma and also protects medical personnel from their aggressive behavior [24, 42]. However, it's important to remember that anxiety can be an early sign of alcohol withdrawal syndrome [19]. Metadoxil improves ethanol metabolism by affecting the liver's enzymatic system. Additionally, it increases adenosine triphosphate levels, acetylcholine release, and gamma-aminobutyric acid in the brain [42]. Prior to various invasive dental procedures, parenteral administration of B-group vitamins, primarily thiamine, is indicated for the prevention of Wernicke's syndrome. Thiamine deficiency can lead to cardiovascular insufficiency ("wet beriberi") due to vasodilation and reduced overall vascular resistance. Vitamin K, blood coagulation factors, fresh frozen plasma, or platelets may also be required for coagulopathy correction [13, 19, 24, 36, 40].

In the anesthetic management of dental procedures for individuals with chronic alcoholism, outside acute intoxication, regional anesthesia can be performed quite safely. However, it is necessary to take into account liver function disorders, hypoalbuminemia, and heart failure when using it [32]. In chronic alcoholism, cytochrome P450 levels increase, leading to enhanced microsomal (inductive) enzyme activity, necessitating higher doses of local anesthetics. The anesthetic effect and duration of action of lidocaine and ropivacaine are limited by chronic alcohol consumption, which induces metabolic acidosis and hinders the diffusion of anesthetic molecules across neuronal membranes [13, 29]. In regional anesthesia, alcohol withdrawal syndrome is one of the most severe complications that can lead to fatal outcomes without intensive care [43].

Sedative drug doses (benzodiazepines, propofol, thiopental) should be increased, as supported by research results in animal models [13, 29, 33, 38, 40].

The reaction of alcohol-dependent patients to ketamine differs from that of healthy individuals and may manifest as psychotic reactions in the postoperative period [29].

Effective doses of opioids, such as alfentanil, increase. These common anesthesia requirements can pose a risk of cardiovascular instability in patients who may suffer from cardiomyopathy, heart failure, or dehydration [19, 38]. Caution should be used when re-administering opioids (morphine, meperidine, pethidine, fentanyl, etc.), as their metabolism is suppressed in alcohol-related liver disease. An exception is remifentanyl, which is metabolized and eliminated without passing through the liver. Therefore, dosing requirements for analgesic drugs should be adapted [13, 35, 40].

Muscle relaxants are characterized by organ-independent metabolism. Depolarizing and non-depolarizing neuromuscular blockers may have a prolonged duration of action. In alcoholic liver disease, their pharmacokinetics are altered with an increase in distribution volume and a decrease in protein binding [13, 19, 33]. Neuromuscular blocker doses should be titrated to achieve the desired effect, guided by peripheral nerve stimulator monitoring [33].

In the postoperative period, the goals of treatment are patient comfort and safety, which involve ensuring adequate pain management and the continuation of prevention or treatment of withdrawal syndrome. Safety concerns are related to idiosyncrasy (e.g., with opioids), requiring

higher doses with an increased risk of potential side effects [37]. Alcohol is considered a modifying risk factor for negative dental outcomes [27]; in the postoperative period, alcohol-dependent individuals compared to non-drinkers have a higher risk of delayed extubation (30.89% vs. 19.29%), and longer hospital stays [44]. Decreased coagulation factors and thrombocytopenia increase the frequency of postoperative bleeding [19]. Immune deficiency due to leukopenia and depressed phagocytic activity increases the risk of postoperative infectious complications (involving surgical site, respiratory system, or urinary tract) [19, 36].

Recovery may be complicated by alcohol withdrawal syndrome (AWS), the onset of which can be masked by general anesthesia. The syndrome typically develops 6-24 hours after alcohol cessation but can be delayed for up to 5 days, characterized by tremors, gastrointestinal dysfunction, sweating, hypertension, hyperreflexia, anxiety, and agitation progressing to delirium, hallucinations, and seizures [34, 36, 38, 40, 43]. Treatment includes benzodiazepines, neuroleptics, carbamazepines, and sodium valproate [19]. Magnesium sulfate 25% solution is administered from 10 to 40 mL per day, titrated under blood pressure control. Clonidine, dexmedetomidine, baclofen, and ketamine can be used as symptom-oriented adjuncts. Therapeutic administration of ethanol or chlome-thiazole is considered harmful to critically ill patients after the onset of AWS. Underestimating the severity of the condition and delaying intensive care can lead to fatal outcomes [13, 19, 40, 42, 45].

Conclusion. Dentists and anesthesiologists often underestimate the risk of chronic alcoholism during the procedure, analgesia, and the recovery period. Timely diagnosis, optimization of strategies, awareness of pathophysiology, directed treatment, and support to keep patients from consuming alcoholic beverages can reduce the number of complications and mortality in patients of dental profile [13, 46].

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ОСОБЛИВОСТІ АНЕСТЕЗИОЛОГІЧНОГО ЗАБЕЗПЕЧЕННЯ В ЩЕЛЕПНО-ЛИЦЕВІЙ ХІРУРГІЇ У ПАЦІЄНТІВ З ОБТЯЖЕНИМ АНАМНЕЗОМ (огляд літератури)

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Анотація. На сьогодні проблема хронічного алкоголізму стосується всіх верств населення в цілому світі. Надання стоматологічної допомоги пацієнтам, які страждають на цю патологію, становить складну задачу, яка потребує комплексного підходу. Алкоголь і його метаболіти негативно впливають на стан ротової порожнини. Захворюваність потаторів на лейкоплакію, еритроплакію, глосит, карієс, пародонтоз і карциному ротової порожнини в декілька разів вища в порівнянні з непитущими. Це пов'язано з декількома факторами, зокрема з хімічними властивостями алкогольних напоїв, наслідками блювання, дією активних метаболітів етилового спирту, запальними процесами, дисплазією епітелію ротової порожнини, порушеннями метаболізму, зміною харчових звичок і погіршенням особистої гігієни. У алкозалежних часто виникають соматичні захворювання, клінічними проявами яких також бувають ураження ротової порожнини. Функціонально-морфологічні розлади різних органів, систем і обміну речовин у потаторів створюють проблеми не тільки при лікуванні стоматологічної патології, а й при анестезіологічному забезпеченні. Алкоголізм призводить до змін особистості, розладів серцево-судинної та нервової систем, а також печінкової дисфункції, які вимагають відповідної корекції у перопераційному періоді. Як гостре алкогольне сп'яніння, так і хронічний алкоголізм значно змінюють метаболізм інгаляційних і внутрішньовенних анестетиків, опіодів і інших препаратів, котрі застосовують для анестезіологічного забезпечення. Дози препаратів для анестезії доцільно зменшувати при гострому алкогольному сп'янінні та корегувати при планових втручаннях із акцентацією на сформованій толерантності до них. Регіонарна анестезія та аналгезія у таких пацієнтів також мають свої особливості. Обізнаність лікарів у патофізіології хронічного алкоголізму, своєчасне лікування та сприяння утриманню хворих від уживання алкогольних напоїв здатні зменшити кількість ускладнень і летальність у пацієнтів стоматологічного профілю.

Ключові слова: хронічний алкоголізм, ураження ротової порожнини, стоматологічне лікування, анестезія, ускладнення.

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