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HISTOPATHOLOGICAL ALTERATIONS IN INTERNAL ORGANS RESULTING FROM ALCOHOL INTOXICATION

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Abstract. This article explores the morphological alterations in internal organs resulting from alcohol intoxication, with particular emphasis on thanatogenesis and its underlying pathogenetic mechanisms. It highlights structural changes in the liver, heart, brain, endocrine, and respiratory systems under both chronic and acute ethanol exposure. Key life-threatening conditions that lead to multiple organ dysfunction and death are identified. The study underscores the significance of early diagnosis and a comprehensive understanding of cellular-level morphological progression for improved treatment outcomes and prognosis.

Keywords: alcohol intoxication, morphological changes, thanatogenesis, internal organs

Аннотация. В данной статье рассматриваются морфологические изменения внутренних органов, вызванные алкогольной интоксикацией, с особым акцентом на танатогенез и его патогенетические механизмы. Освещаются структурные изменения в печени, сердце, головном мозге, эндокринной и дыхательной системах при хроническом и остром воздействии этанола. Определены ключевые жизнеугрожающие состояния, приводящие к полиорганной недостаточности и смерти. Подчёркивается важность ранней диагностики и всестороннего понимания последовательности морфологических изменений на клеточном уровне для повышения эффективности лечения и прогноза.

Ключевые слова: алкогольная интоксикация, морфологические изменения, танатогенез, внутренние органы

Annotatsiya. Ushbu maqolada ichki a’zolarda spirtli ichimliklar bilan zaharlanish natijasida yuzaga keladigan morfologik o‘zgarishlar, xususan, tanatogenez va uning patogenetik mexanizmlari tahlil qilinadi. Etanolning surunkali va o‘tkir ta’siri ostida jigar, yurak, miya, endokrin va nafas olish tizimlaridagi tuzilmaviy o‘zgarishlarga alohida e’tibor qaratilgan. Bir nechta a’zolarning yetishmovchiligi va o‘limga olib keluvchi asosiy hayot uchun xavfli holatlar aniqlanadi. Morfologik o‘zgarishlar ketma-ketligini hujayra darajasida erta aniqlash va chuqur anglash samarali davolash va prognoz uchun muhimligi ta’kidlanadi.

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Kalit so‘zlar: alkogolli intoksikatsiya, morfologik o‘zgarishlar, tanatogenez, ichki a’zolar

A terminal condition develops when a pathological factor surpasses the body’s compensatory capacity due to prolonged or intense exposure. This leads to a shift from compensatory to destructive responses and functional breakdown. During the agonal stage, hypoxia causes a switch from aerobic to anaerobic metabolism (glycolysis), a key compensatory mechanism. However, the progression toward death is gradual, and it remains difficult to pinpoint the precise onset of thanatogenesis or generalize its mechanisms throughout the disease course [51]. With timely intervention, terminal states can be reversed—hemodynamics stabilized and homeostasis restored—yet this may trigger new pathological processes termed post-resuscitation disease, which arise from multiple factors. Insufficient resuscitation efforts exacerbate patient conditions and hasten homeostatic disruption [37]. Hypoxia, in various forms, is the predominant factor in terminal pathophysiology, damaging cell membranes and disturbing electrolyte balance, particularly postoperatively, during trauma, bleeding, or late-stage alcoholism.

One consequence is fluid accumulation in serous cavities, cerebral and myocardial edema, glycogen and glucose depletion, and morphological alterations in neurons and cardiomyocytes [7, 13]. Death typically results from organ failure—lungs, heart, brain—representing pulmonary, cardiac, or cerebral types of thanatogenesis [51]. Renal, pancreatic, coagulopathic, and mixed types are also recognized, often accompanying multiple organ dysfunction [5, 7]. The progression of death correlates with diagnostic timeliness and treatment adequacy, highlighting the importance of understanding terminal conditions and prognosticating thanatogenesis [22].

While the histological and biochemical patterns of acute ethanol poisoning are well described [1, 3, 5, 12, 14, 55], subacute or chronic cases are less covered due to patient admissions in general wards, where diagnoses focus on organ damage and outcomes are often favorable. Alcohol-related deaths may present with cardiovascular or respiratory collapse, CNS depression, reduced pain perception, seizures, tachycardia, dyspnea, and miosis. Interestingly, high blood alcohol levels are not always present; cardiac pathology tends to be more apparent than changes in other organs [39].

Rarely, "true" ethanol poisoning—fatal at non-lethal doses—occurs due to the absence of brain alcohol-metabolizing enzymes, leading to cerebral-type death (alcoholic coma with central respiratory arrest) or aspiration-obturator death from soft palate and laryngeal edema, without significant visceral pathology [35, 39].

In the resorption phase, blood ethanol concentration exceeds urinary levels, lasting 30–180 minutes. Elimination begins after 90–98% absorption. Alcohol metabolism occurs at 100–240 mg/kg/h, with blood alcohol reducing by 10–20 mg/%/h—about 10–12 g oxidized per hour. Damage to the heart and liver correlates with alcohol and acetaldehyde dehydrogenase activity [2, 38, 62, 66].

In alcoholic thanatogenesis, a "catabolic explosion" denotes excessive, inefficient metabolic activity and disruption across systems. Ethanol acts both as a substrate and a

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hormone, with complex effects on bioenergetics. Multiple organs become involved, creating a vicious cycle [42, 45]. Water-electrolyte imbalance ensues, increasing osmolarity in blood and cerebrospinal fluid. Some patients retain normal carbohydrate levels, while others experience fatal hypoglycemia [1, 12]. Protein synthesis in the heart, liver, and adrenal glands is suppressed, and neurons shift to anaerobic metabolism, with increased glucose-6-phosphate dehydrogenase in the brain—unlike in ischemic heart disease—offering potential for differential diagnosis [11, 29, 50, 54].

Chronic alcohol intoxication and poisoning from alcohol substitutes share common pathological traits. Histological findings include cerebral hyperemia, perivascular edema, increased permeability, and severe neuronal damage—such as dark or ghost cells, and nuclear lysis.

Alcohol substitutes—unique to Russian terminology—are liquids consumed for inebriation instead of legal alcohol. They often include moonshine, counterfeit tinctures, or pharmacy products not meant for oral use [44, 52]. These may contain additives that alter ethanol toxicity, and their membranotropic effects resemble those of alcohol, though sometimes produced by chemically similar compounds [27].

In domestic toxicology, alcohol surrogates are classified into two categories: (1) those containing ethyl alcohol (e.g., hydrolytic, synthetic, or denatured alcohol; colognes, tinctures, polishes, and stains), labeled “true” surrogates; and (2) agents lacking ethanol but mimicking its membranotropic effects, such as methanol, ethylene glycol, and other non-ethyl alcohols—considered “false” surrogates [33]. Technical liquids are often grouped under this classification. However, the term “alcohol surrogate” does not fully capture the underlying pathology and can lead to inappropriate therapeutic strategies, raising both ethical and clinical concerns. Some researchers argue that the toxicological profiles of true surrogates are largely similar, justifying a unified treatment approach [44].

Acute alcohol poisoning is characterized primarily by widespread cerebral edema. Swelling of the intercellular matrix, basement membranes, and villous stroma in the choroid plexus leads to capillary compression, epithelial necrosis, and desquamation [13, 29]. Even with minimal hemodynamic disruption, irreversible damage to nuclei in the rhomboid fossa can occur [4]. Additional findings include mucus accumulation in the airways, hemorrhagic lesions in the stomach and duodenum, cardiomyocyte edema, pulmonary edema, and neuronal necrosis [35].

Organ systems show extensive damage. The liver is notably affected, with reduced oxidative metabolism potentially progressing to toxic hepatic dystrophy, steatosis, or cirrhosis [43]. Sometimes, death results not from liver failure per se, but from secondary cerebral edema. Histologically, astroglial nuclei hypertrophy is evident [41, 65]. Endocrine disruption, particularly in the hypothalamic-pituitary-adrenal axis, impairs systemic adaptation [34, 47]. Hypoglycemic coma is also common: hepatic carbohydrate stores may drop to zero (normal range: 11.15–37.62 mmol per 100g liver), alongside decreased blood glucose. Fatal events often occur during sleep [12], possibly from acute heart failure due to brainstem injury.

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Gastrointestinal hemorrhage may result from esophagogastric varices or Mallory-Weiss syndrome, frequently preceded by hepatic dysfunction [18, 59, 63]. Pulmonary complications may aggravate alcoholic cardiomyopathy, leading to death via cardiorespiratory failure, brainstem hemorrhage, or respiratory center depression [26, 32]. Nosocomial infections may contribute [56].

Pancreatic involvement includes hemorrhagic necrosis or acute pancreatitis, which in early stages coincide with heart and lung failure, and in advanced stages with peritonitis, ARDS, and renal failure [8]. Clinical signs vary with the functional status of each ventricle. Multiple authors have explored this pathology [24, 51]. Some attribute acute cardiovascular collapse to medullary centers' dysfunction, which induces hypoxia, hemodynamic instability, and neuronal changes in the hippocampus and hypothalamus. In these instances, hepatic carbohydrate levels remain normal, but hepatocytes exhibit poor architectural organization. Pancreatic autolysis is commonly observed.

In cases with hypoglycemia, hepatocytes lose glycogen, assuming a rounded shape; the liver appears architecturally preserved, the pancreas remains structured, but cerebral edema, blood element aggregation, and cardiomyocyte homogenization are evident [24, 54].

Left ventricular failure manifests as decreased myocardial contractility, tachycardia, tachypnea, and dyspnea. Pulse amplitude and rhythm are disrupted, and pallor results from microcirculatory compromise. Pulmonary signs include wheezing and frothy sputum. A sudden drop in blood pressure may occur. Histology shows myocardial edema, loss of cross-striation, cytoplasmic granularity, glycogen depletion, and potassium loss.

Right ventricular failure, although less common, is associated with overload from rapid infusions or pulmonary embolism. The terminal state includes diminished right ventricular contractility, tachycardia, cyanosis, jugular vein distension, and tachypnea. Histological findings include myocardial edema and structural homogenization in both ventricles. Sudden cardiac failure is usually the direct cause of death, often linked to brainstem dysfunction. Marked cardiac pathology supports a cardiac cause of death in chronic alcohol intoxication.

Evidence suggests alcoholic cardiomyopathy may result from chronic ethanol exposure or acute toxicity, contributing to primary cardiac arrest or ischemic heart disease manifestations [50, 51]. Notably, fatal cardiomyopathy may develop even with low blood alcohol levels (0.6–0.9‰), the concentration being influenced by the postmortem interval [40]. Macroscopically, enlarged hearts (620–640g) are common. Chronic users may develop high-grade ventricular arrhythmias, such as tachycardia or atrial fibrillation, often leading to sudden death. Patients diagnosed with chronic alcoholism rarely survive long enough to develop cardiomyopathy [49, 58].

Alcohol toxicity also impacts microvasculature: vascular tone and flow decrease, and hypercoagulability of plasma proteins is observed. Microangiopathy manifests as endothelial swelling and proliferation, culminating in sclerosis, hyalinosis, and increased permeability [30]. Alcohol-related ischemic heart disease complicates determination of death causes. Although

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moderate alcohol intake may reduce thrombotic risks in major vessels [8, 25, 54], its protective role doesn't extend to smaller cardiac or cerebral vessels [8]. Post-binge reactive thrombocytosis emerges only after 10–14 days [48].

The pulmonary form of the terminal state is marked by symptoms such as tachypnea, tachycardia, cyanosis (notably of the nail beds), initial arterial and central venous hypertension followed by hypotension, weakened respiration, and dry fine bubbling rales. In the premortem phase, acute respiratory insufficiency develops, ultimately resulting in cardiopulmonary failure. Morphologically, the lungs exhibit alternating zones of hemodynamic atelectasis and emphysematous alveolar distension, epithelial desquamation, Minakov spots beneath the endocardium, leukostasis, marginal leukocyte aggregation, interstitial and alveolar membrane edema, and accumulation of edematous fluid within the alveolar lumens. These findings reflect diffuse damage to the vascular-tissue barrier, contributing to systemic alteration of the intercellular matrix. Chronic pulmonary inflammation observed in patients with pre-existing conditions should be viewed as a secondary pathology, often arising in conjunction with primary organ damage, particularly of cardiac origin [26, 32].

A possible driver of pulmonary tissue alteration is surfactant deficiency, which reduces lung compliance. This decline is frequently associated with alcohol-induced hepatic dysfunction. Lung damage is especially prominent in acute alcohol poisoning and is often fatal. Immunosuppression is proposed as a contributing factor to this high mortality [40]. While some researchers question the role of pulmonary changes in the fatal outcomes of chronic alcohol intoxication, they still report vascular engorgement, vasoparesis, capillary stasis, hemorrhages, basement membrane swelling, alveolar hemorrhagic edema, and vagus nerve center inhibition leading to hemodynamic instability [36].

The liver, being especially vulnerable to alcohol toxicity, frequently becomes the focus of clinical and forensic evaluations [9, 49]. Alcohol consumption is known to induce hepatic steatosis, hepatitis, and cirrhosis. Even low but regular intake—especially when combined with poor nutrition—can precipitate alcoholic hepatitis and cirrhosis [61]. Liver dysfunction may remain asymptomatic or manifest through decompensation: weakness, gastrointestinal symptoms, jaundice, splenomegaly, ascites, encephalopathy, and renal impairment [49, 63]. Acetaldehyde, formed through the action of alcohol dehydrogenase and microsomal ethanol oxidizing systems, interferes with lipid peroxidation, mitochondrial respiration, DNA repair, and microtubule stability, leading to Mallory body formation (hyaline-droplet dystrophy) and impaired cellular protein export, resulting in cytoplasmic fluid accumulation and edema [54, 62].

High-dose alcohol intake (400–500 ml) may cause acute alcoholic hepatitis with subsequent post-necrotic multilobular cirrhosis. Alcohol also disrupts the phospholipid composition of hepatocyte membranes, disturbing transmembrane transport [41, 60]. Chronic ethanol exposure alters the glycoprotein profile in hepatocyte membranes, promotes cirrhosis, and elevates blood aspartate aminotransferase (AST) levels [45, 40].

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Steatosis is observed in roughly 88.8% of alcohol-related deaths and is defined by triglyceride accumulation in hepatocytes. The liver becomes enlarged, firm, and rounded. Alcohol mobilizes depot fat, intensifies fatty acid synthesis in hepatocytes, and suppresses lipoprotein formation and secretion, resulting in cytoplasmic fat accumulation and cyst formation. Microscopy reveals extensive lipid infiltration. Fat deposition typically reverses following alcohol cessation [46, 65].

Chronic alcohol abuse is often associated with recurring alcoholic hepatitis, though fatty changes may be mild. Around 35% of chronic alcoholics develop hepatitis. Cirrhosis, characterized by hepatocyte necrosis, fibrotic proliferation, and nodular regeneration, may result from chronic alcohol use, viral hepatitis, or genetically increased alcohol dehydrogenase activity. Alcoholic cirrhosis typically presents as a large nodular liver with normal spleen size, signs of steatosis, and early-stage portal hypertension. This triggers the formation of portal-systemic collateral circulation, which interestingly may also be seen in non-alcoholics, including children [40].

Alcohol-induced liver pathology leads to multiple secondary conditions, including impaired surfactant protein synthesis, hepatic encephalopathy, and Mallory-Weiss hemorrhagic syndrome. Alcohol also affects Kupffer cells, stimulating bioamine (notably superoxide anion) release, which accelerates hepatocyte lysis. In cases of gastroesophageal bleeding, including Mallory-Weiss syndrome, histological alterations such as endothelial swelling, basal membrane fragmentation, and mucosal-muscular ruptures in the pyloric region serve as histopathological markers of alcohol intake [57, 63].

The pathogenesis of hepatic encephalopathy primarily involves impaired hepatic clearance of neurotoxic substances originating in the intestine. This results from both hepatocellular insufficiency and portosystemic shunting, where intestinal blood bypasses the liver and enters systemic circulation without detoxification. These processes disrupt cerebral neurotransmitter systems, with ammonia and several mediators—such as serotonin, gamma-aminobutyric acid, glutamate/glutamine, and endogenous benzodiazepines—playing critical roles. Hepatic encephalopathy may rapidly progress into parenchymal coma and often coexists with life-threatening complications such as disseminated intravascular coagulation and sepsis [40, 46].

Chronic alcohol intoxication negatively affects neuroendocrine and immune regulation, manifesting as glandular depletion and dysfunction. Alcohol abusers often exhibit endocrine gland congestion, pituitary shrinkage, adrenal cortical atrophy, and fluid retention in testicular tissues. Experimental studies have demonstrated that thyroid hormones mitigate alcoholic fatty liver degeneration, and their absence increases mortality. Within 24 hours of ethanol intake, mitochondrial iodine uptake drops sharply; total triiodothyronine rises while thyroxine levels fall, although iodine supplementation can reverse this trend [40].

Steroid hormones synergize with alcohol to provoke hepatocyte necrosis [62]. Ethanol toxicity also extends to the kidneys [40], spleen [6, 8], adrenal glands, and reproductive organs [64]. Infrequently, chronic alcoholism causes pancreatic injury, including necrosis or indurative

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pancreatitis, marked by parenchymal atrophy and structural remodeling, sometimes progressing to adenoma. Roughly 2% of alcoholic patients die from hypo- or hyperglycemic coma, associated with fibrotic changes in pancreatic islets and nesidioblastosis. Concurrently, cerebral cortical neurons may undergo acidophilic degeneration. Survivors of hemorrhagic pancreatic necrosis often develop severe diabetes, hyperglycemia, and risk of diabetic coma. This pathology, especially in early stages, is linked with cardiovascular failure, while advanced cases can present with peritonitis or ARDS [8, 40].

Male genital organs in alcoholics commonly exhibit size reduction and fibrosis. Histological analysis reveals fibrotic transformation of cavernous bodies, smooth muscle replacement with sclerotic tissue, testicular atrophy, Leydig cell hyperplasia, brown degeneration, and basal membrane hyalinosis. Pituitary dysfunction in alcoholism leads to decreased testosterone levels and disruption of the estrogen-testosterone balance. Chronic alcohol use impairs spermatogenesis, and advanced liver cirrhosis may result in the "Sertoli cell-only" syndrome—complete absence of mature spermatozoa [28].

Alcohol-induced endocrine and structural damage is also linked to neuropsychiatric disorders. Post-traumatic stress following sexual abuse affects brain function, often causing neuroses and depression. Studies confirm hippocampal volume reduction in both sexes [64]. Animal models reveal that prolonged alcohol exposure leads to hepatic microvesicular steatosis and damage to reproductive glands. In the anterior pituitary, acetophil distribution becomes uneven, while basophils develop central vacuoles and characteristic “ring” formations.

In summary, alcohol-related disease affects nearly all organ systems, yet mortality may stem from dysfunction of a single or multiple organs. While researchers often focus on individual organs, a comprehensive understanding of thanatogenesis—the progression of structural and functional cellular damage—is crucial for predicting disease outcomes and informing therapeutic decisions.

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