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ACUTE ETHYL ALCOHOL POISONING (ALCOHOLIC COMA)

Ibragimov Botirjon Inomidinovich

Andijan State Medical Institute, Uzbekistan

Abstract: The article summarizes the authors' long-term experience in the diagnosis and treatment of acute ethyl alcohol poisoning and presents data regarding the epidemiology of ethyl alcohol poisoning, as well as modern terminology and systematization of alcohol poisoning in accordance with the international classification of diseases ICD 10. The pharmacokinetics and pharmacodynamics of ethyl alcohol under its toxic effects were analyzed.

Keywords: Ethyl alcohol, acute poisoning, diagnosis, electroencephalography, treatment.

INTRODUCTION

Acute poisoning with ethyl alcohol (EA) occupies a leading place among household poisonings in our country, reaching 30–40% of patients hospitalized in poisoning centers. In absolute terms, poisoning with alcohol and its substitutes accounts for more than 40% (up to 58%) of all fatal poisonings. The mortality rate for OOA in hospitals ranges from 2.2 to 5.0%, while even in specialized poisoning centers it is 1.8%, which emphasizes the relevance of the problem of diagnosis and emergency medical care for this pathology.

MATERIALS AND METHODS

The basis of the toxic effect of ethanol is its pronounced neurotropic effect on the functions of the central nervous system (CNS), which is manifested by impaired consciousness, mental, autonomic and neurological disorders [1]. An alcohol molecule, penetrating into the lipid bilayer of cell membranes, affects the structure of phospholipids and changes their fluidity [2]. As a result, the intensity of synthetic processes in the neurotransmitter systems of the brain changes, and with acute exposure to ethanol, various mediator and membrane disorders lead to the development of certain manifestations of poisoning [1].

RESULTS AND DISCUSSION

In the clinical picture of alcoholic coma (AC), it is customary to distinguish three degrees, reflecting the dynamics of the toxic process: superficial (I and II degrees) and deep - uncomplicated and complicated variants of the course.

superficial coma of the first degree is characterized by unstable neurological symptoms. The leading symptoms of this stage are associated with disturbances of cortical-subcortical functions: consciousness is completely absent, there is a sharp hypotonia of muscles, a decrease in pain sensitivity, loss of involuntary movements, in particular indicative ones (movement of the head and eyes in the direction of the source of visual irritation), adjustment (attempt to rise, turn the body) and lack of protective reflexes (removing the source of irritation with the hand).

However, at this stage of OOA, there is preservation and at times even disinhibition of proprioceptive, i.e. tendon reflexes with the appearance of hypertonicity of the muscles of the limbs and trismus of the masticatory muscles. Against the background of pronounced hypertonicity, fibrillar twitching of individual muscles of the chest is noted. The dynamics of neurological symptoms described above occurs in response to ongoing therapeutic measures (gastric lavage, injections, etc.). After the cessation of manipulation and warming of the patient, muscle hypotonia and cessation of myofibrillation are again observed. Characterized by the

inconsistency of neurological symptoms: the play of the pupils - miosis with the transition to mydriasis when painful stimuli are applied (medical manipulations) with the appearance of a randomly occurring facial reaction on the face, periodically floating movements of the eyeballs and transient anisocoria. The inhalation effect of ammonia (cotton wool soaked in its 25% solution is brought to the nose) causes an involuntary facial reaction and weak defensive movements. Body temperature remains within normal limits. The skin is somewhat hyperemic. At this stage, breathing disorders may occur—mild bronchorrhea in the form of large bubbling rales over large bronchi during auscultation against the background of moderate tachypnea (up to 26–28 per minute).

Superficial coma of the second degree is manifested by a more severe inhibition of cortical-subcortical functions and disinhibition of brainstem and spinal centers. Consciousness is completely lost, tendon reflexes are suppressed. Corneal and pupillary reflexes, as well as pharyngeal and cough reflexes, are sharply reduced. Pain sensitivity is lost, however, when pressure is applied to the pain points of the trigeminal nerve, a protective reflex reaction appears in the form of shortening movements of the lower extremities, shoulder pulses and forearm pronation, as well as a weak facial reaction. Muscle tone is sharply reduced. Sometimes single fibrillary twitching of individual chest muscles is observed. In contrast to superficial coma of the first degree, in response to painful stimulation, only a weakly expressed, rapidly passing hypertonicity of the upper and lower extremities appears. Fibrillar muscle twitching also occurs in response to painful stimulation and therapeutic measures (gastric lavage, etc.) and quickly disappears after their cessation. Breathing is shallow, weakened. During auscultation along the large bronchi, abundant moist coarse bubbling rales are heard. In some cases, breathing disturbance occurs, which becomes superficial and arrhythmic (stridor) as a result of aspiration of mucus and vomit.

There is a moderate increase in blood pressure - up to 140/90–150/95 mm Hg. and severe tachycardia up to 80–100 beats. in 1 min. On the part of the digestive organs, there is pronounced salivation and repeated vomiting of gastric contents. The alcohol content in the blood at this stage of poisoning ranges from 2.5 to 6.5 g/l, in the urine - from 2.5 to 8.0 g/l. Involuntary urination is characteristic.

CONCLUSION

The patient was sent to the neurosurgical operating room. Clinical diagnosis: severe closed head injury. Acute subdural hematoma of the right fronto-parietal-temporal region. Concomitant diagnosis: Alcohol intoxication.

It is known that in acute intracranial hematomas due to alcohol intoxication, cerebral vessels dilate and venous outflow slows down, which, along with the development of hematoma, leads to an increase in intracranial pressure, an increase in hypoxia of brain tissue and its edema, as well as to disorders of the cerebral and systemic circulation [2]. In acute TBI in combination with alcohol intoxication, the effect of alcohol, changing and hiding (levelling) the picture of classic TBI, requires a more thorough differential diagnosis.

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