

MORE THAN MINIMUM CONSCIOUSNESS: APPALLIC SYNDROME

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Abstract

Apallic syndrome (vegetative state or social brain death), which occurs after acute cerebral hypoxia, is a well-known condition that requires immediate intensive care and coordinated work of medical personnel. In acute brain hypoxia, the cortex is predominantly affected. In the traumatic status of apallic syndrome, a significant contribution to the development of apallic syndrome, in addition to edema of the white matter, is made by compression of the midbrain with the ensuing consequences.

Such clinical cases that suddenly arise after acute damage to the cerebral cortex or parts of the brain stem are manifested by various general cerebral symptoms. This article is a review of clinical cases with apallic syndrome.

Keywords: vegetative state, hypoxic encephalopathy, cerebral edema, respiratory instability, pathological reflexes, parenteral nutrition, neuroprotection.

Introduction

Purpose of the study: to identify the priority of therapeutic treatment in the ICU and analyze current and enriched therapy for patients with complications of apallic syndrome.

Materials and methods: materials represent an analysis of existing studies, as well as two clinical cases in the Republican Research Center for Emergency Medicine in the intensive care unit.

According to E. Kretschmer (1940), who described this condition in detail: "... the patient is prostrated, inactive, alert, but silent. The patient's eyes are open, they move senselessly from one corner to another, do not focus on anything, or the patient looks straight. Attempts to attract his attention are in vain. Dialogue with the patient, touching him or visualizing objects to him does not lead to any tangible result. There are no reflex movements of withdrawal or defense. The ability to relax is lost, so the patient is in a randomly adopted position. Some primitive autonomic functions may be preserved. Basic instincts such as grasping, sucking and chewing are observed. The syndrome differs from coma by the appearance of alternation of sleep and wakefulness" [1, 2].

Apallic syndrome (AS) is a symptom complex that combines mental and neurological disorders resulting from the shutdown of the cerebral cortex - decortication [3]. All the most important vital functions (breathing, blood circulation, metabolism) and even sleep and wakefulness are present, but there is no experience, not even elementary sensations of color, light, pain. Patients may react to stimuli, but these reactions are purely automatic and are not associated with any

mental perception of these stimuli. This condition is a polyetiological syndrome, ranging from traumatic brain injury to suicidal hanging. Most often, AS develops in acute brain lesions of both traumatic (road injury, gunshot injury, etc.) and non-traumatic origin (global cerebral ischemia as a result of cardiac arrest, respiratory arrest, asphyxia of various origins, acute cerebrovascular accidents, central nervous system infections, tumors, endogenous and exogenous intoxications) [4]. Calculations based on data from etiological studies in Western European countries show 1-10.5 new cases of traumatic AS per 100,000 population annually. According to some studies, the incidence of AS is 0.7-1.1 per 100,000 population, the prevalence is 2-10 per 100,000 population [8-10].

Studies of patients with AS have shown that this condition is not a final and persistent organic lesion of the central nervous system, but has a cyclical stage [5, 6].

The emergence of such "new" pathological processes as progressive brain atrophy, progressive hydrocephalus, epileptic process, spasticity of the muscles of the limbs. This necessitates a revision of the formed stereotypes in relation to the schematic, standardized, predominantly syndromic drug rehabilitation treatment [7-9].

Study Results:

In our case, patients K.Zh. 16 years old with a diagnosis of strangulation asphyxia, and U.H. 22 years old with a diagnosis of open craniocerebral injury, severe brain contusion with complications of cerebral edema and AS. Upon admission to the ICU, the patients had the following clinical neurostatus: depressed consciousness, first degree coma. Lateral movement of the eyeballs is preserved. Swallowing was preserved. Asymmetrical face, right-sided hemiparesis, hemihypesthesia. Anisoreflexia D>S. Pathological Babinski reflexes and M-Rodovich sign (+) on both sides, meningeal signs are not detected.

Treatment for AS was similar to drug therapy for coma, with some modifications. Citicoline 4.0 ml was used for neuroprotection. Ganglioside was used to stimulate the formation and growth of synapses, restore innervation function, improve nerve conduction, stimulate electrical activity of the brain and restore neurophysiological parameters Mavix 20mg/ml. Sedative therapy Midazolam 15 mg and sodium oxybutyrate 2g. To dehydrate cerebral edema, a 7.5% NaCl solution 200 ml and the diuretic furosemide 1% 2 ml were used. Anticonvulsant Convulex 2ml. To maintain adequate ventilation and oxygenation, a tracheostomy was performed. Central acting muscle relaxant Mydocalm 1ml. To reduce the severity of mental and neurological symptoms and improve cerebral circulation, Cavinton 4 ml was used. Membrane stabilization therapy KCl 4% 30ml + lidocaine 2% 4ml + MgSO₄ 25% 5ml + NaCl 0.9% 200ml. Antibacterial therapy Streptomycin 0.5g. Pyrolytic therapy in the role of Temfulgan 100ml. Anticoagulant therapy Heparin 5000 units. For the purpose of antioxidant therapy, Ascorbic acid 5% 10 ml and Emgops 5% 5 ml were used. To improve blood rheology, Reosorbilact 200 ml. Proton pump inhibitor Omez 20 mg. Maintenance of parenteral nutrition – Neo - Amin 200ml. To prevent bedsores, vibration massage and rubbing were performed. This therapy was carried out for two weeks and the patients were discharged.

Conclusion

Apallic syndrome is a complex condition that requires a comprehensive and individualized approach to treatment. Despite the challenges associated with this condition, modern medicine is committed to finding new methods of rehabilitation and support to improve the lives of patients with apallic syndrome and their families. Thanks to the development of medicine and the discovery of new medications, it is sometimes possible to bring the patient to progressive improvement in development. And in our case, this therapy indicated the effectiveness of the treatment with positive dynamics of development. Apallic syndrome remains a challenge for the medical community, but ongoing efforts and innovations in treatment confirm the positive impact of advanced methods on the recovery process and well-being of patients.

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