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**КЛИНИЧЕСКИЙ СЛУЧАЙ: СОВРЕМЕННАЯ ИНТЕНСИВНАЯ ТЕРАПИЯ В  
ЛЕЧЕНИИ ПОСТРЕАНИМАЦИОННЫХ ОСЛОЖНЕНИЙ, ВЫЗВАННЫХ  
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✓ **Резюме**

*Период после проведения сердечно-лёгочной реанимации (СЛР) остаётся одной из наиболее сложных и недостаточно изученных проблем современной медицины. Согласно мировым статистическим данным, более 61% пациентов, перенёвших СЛР, погибают в течение первых 24 часов. Несмотря на восстановление жизненно важных функций у приблизительно 39% пациентов, у большинства из них развиваются различные осложнения. Полное восстановление наблюдается лишь у 16% больных, тогда как у оставшихся 84% сохраняются неврологические, сердечно-сосудистые и системные нарушения [1–8]. Несмотря на совершенствование алгоритмов сердечно-лёгочно-мозговой реанимации (СЛМР), недостаточный уровень теоретических знаний и практических навыков их применения в клинической практике способствует сохранению высоких показателей летальности и частоты осложнений. В ряде источников отмечается, что даже при позднем начале реанимационных мероприятий возможно полное восстановление жизненно важных функций, что зависит от возраста пациента, температуры окружающей среды, индивидуальной устойчивости к гипоксии, а также качества и своевременности оказания первой помощи немедицинскими лицами на месте происшествия [9–18]. Цель исследования: Изучить современные подходы интенсивной терапии в лечении постреанимационных осложнений, обусловленных сердечными аритмиями.*

*Ключевые слова: Постреанимационный синдром, сердечные аритмии, сердечно-лёгочная реанимация, гипоксия, кислородотерапия, нейропротекция.*

**KLINIK AMALIYOTDAGI HOLAT: YURAK ARITMIYALARI SABABLI YUZAGA  
KELGAN POSTREANIMATSION ASORATLARNI DAVOLASHDA ZAMONAVIY INTENSIV  
TERAPIYA**

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✓ **Rezyume**

Kardiopulmonal reanimatsiyadan (KPR) keyingi davr zamonaviy tibbiyotning eng murakkab va yetarlicha o'rganilmagan muammolaridan biri bo'lib qolmoqda. Jahon statistik ma'lumotlariga ko'ra, KPR o'tkazilgan bemorlarning 61% dan ortig'i dastlabki 24 soat ichida vafot etadi. Bemorlarning taxminan 39% ida hayotiy faoliyat tiklansa-da, turli xil asoratlar kuzatiladi. To'liq sog'ayish faqat 16% bemorlarda qayd etilgan bo'lib, qolgan 84% holatlarda esa turli darajadagi nevrologik, yurak-qon tomir va tizimli asoratlar saqlanib qoladi [1–8]. Kardiopulmonal–serebral reanimatsiya (KPSR) algoritmlarining takomillashuviga qaramasdan, ularni amaliyotda qo'llash bo'yicha yetarli bilim va tajribaning yo'qligi o'lim ko'rsatkichlari hamda asoratlar darajasining yuqoriligicha qolishiga sabab bo'lmoqda. Ayrim adabiyot manbalarida hatto kech boshlangan reanimatsiya holatlarida ham bemorning hayotiy funksiyalari to'liq tiklanishi mumkinligi qayd etilgan bo'lib, bu holat bemorning yoshi, atrof-muhit harorati, gipoksiyaga chidamliligi hamda hodisa joyida tibbiy bo'lmagan shaxslar tomonidan ko'rsatilgan birinchi yordamning sifati va o'z vaqtida bajarilishiga bog'liq ekanligi ta'kidlanadi [9–18]. Tadqiqot maqsadi: Yurak aritmiyalari natijasida rivojlangan postreanimatsion asoratlarni davolashda zamonaviy intensiv terapiya usullarini o'rganish.

**Kalit so'zlar:** Postreanimatsion sindrom, yurak aritmiyalari, kardiopulmonal reanimatsiya, gipoksiya, kislorod terapiyasi, neyroproteksiya.

**CASE IN CLINICAL PRACTICE: MODERN INTENSIVE CARE IN THE TREATMENT OF POST-RESUSCITATION COMPLICATIONS CAUSED BY CARDIAC ARRHYTHMIAS.**

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✓ **Resume**

The situation after cardiopulmonary resuscitation (CPR) is one of the most complex and unresolved medical problems, with world statistics showing that more than 61% of patients who undergo CPR die within the first 24 hours. Although 39% of patients survived, various complications were observed, of which only 16% of patients fully recovered, and the remaining 84% of patients retained various complications [1-8]. Despite the improvement of the pulmonary-cardiac-cerebral resuscitation (PCCR) algorithm, the lack of knowledge and practical experience in conducting PCCR to patients has led to a high mortality rate of patients and a high level of complications. In some sources it is known that in cases of late onset of pulmonary heart resuscitation, the patient's vital functions are fully restored, depending on the patient's age, ambient temperature, resistance to hypoxia, first aid provided by non-medical personnel around the patient [9-18]. Objective of the study: To study modern intensive care in the treatment of post-resuscitation complications resulting from cardiac arrhythmias.

**Keywords:** Post-resuscitation disease, cardiac arrhythmias, cardiopulmonary resuscitation, hypoxia, oxygen therapy, neuroprotection.

## Introduction

**Case study:** Patient A., 30 years old, was brought to the Bukhara branch of the Republican Scientific Center of Emergency Medicine (BBRSCM) on December 26, 2021 at 23:10 in a road car in case of clinical death.

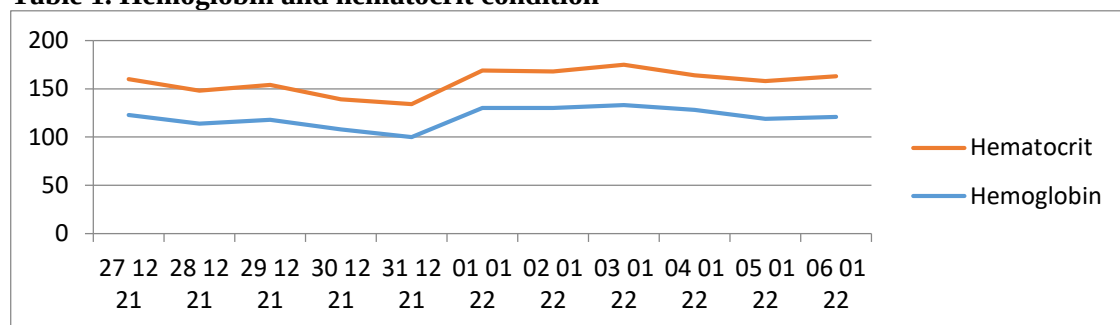
From the anamnesis, the patient has been drinking alcohol and energy drinks regularly during the last 4-5 days while attending wedding ceremonies. 20 minutes before arriving at the hospital, he came out of the kitchen and collapsed and fainted while talking to his colleague. The patient was transported by colleagues to the BBRSCM in his car on 26.12.2021 at 23:10 and was taken to the shock ward of the admission department. Pulmonary cardiopulmonary resuscitation was started immediately due to the absence of pulsation in the central carotid arteries in the shock chamber, shortness of breath, asystole in the cardiomonitor. The patient's heart function was restored in 20 minutes. The patient's hemodynamics was transferred to the Department of Surgical Resuscitation at 01:30 on 27.12.2021 in case of maintenance under the influence of high-dose vasopressor drugs.

From the anamnesis of life, the patient denies hereditary diseases, which he grew up in satisfactory conditions. Does not consume regular medications.

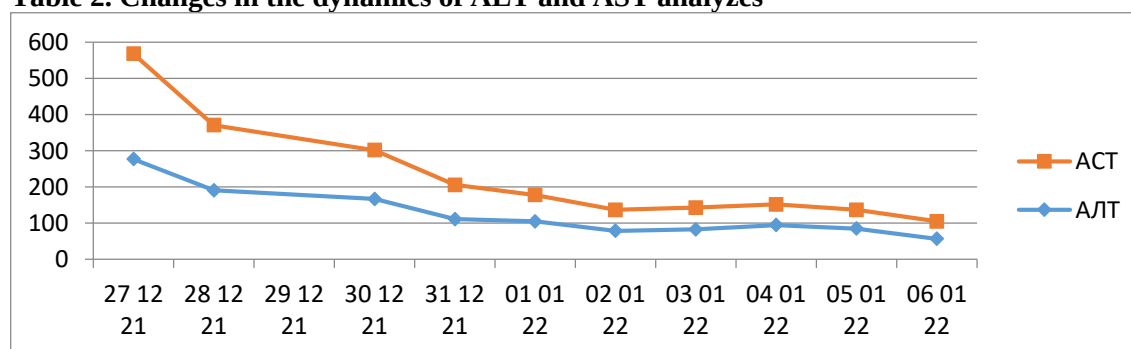
When he arrives, the patient is not conscious. Body structure is normostenic, subcutaneous fat layer is moderately developed. The skin and visible mucous membranes are cyanotic. There are no scars on the body. Deformations in the musculoskeletal system are not detected. Temperature 36.0 OS. Maximum pupil dilation OD = OS, photoreaction is not called. Atonia. Areflexia. Breathing-abs. Saturation - abs. Heart sounds are not audible, no pulse is detected in the central arteries. Asystole is detected in the cardiomonitor. ABP-not detected. The tongue is wet, clean. There are no vomit masses in the oral cavity. The abdomen is soft, the liver and spleen cannot be felt. Involuntary defecation and urinary incontinence are not detected.

In the shock chamber of the reception department of the BBRSCM reception of the patient, there was no pulsation in the central carotid arteries, no breathing, asystole in the cardiomonitor. External cardiac massage was initiated and tracheal intubation was performed using an bu7.5 intubation tube while simultaneously breathing with an Ambu copy. Artificial Lung Ventilation(ALV) apparatus Hamelton C3, A / S Volume mode. DO-600 ml, f-18 times per minute, PSV-14 mambr, PEEP-2, FiO2 -100%. In order to restore cardiac function, r-r Adrenaline-0.18% -1.0, r-r Atropine sulfate-0.1% -1.0 were used 3 times in 5 minutes. For maintenance of blood pressure and vasopressor therapy, r-r Dexamethasone-8 mg every 5 minutes, r-r Dopamine 40 mkg.kg.min., R-r Noradrenaline-2,0 mkg.kg.min. started at. Due to the observed ventricular fibrillation on the cardiomonitor at 18-19 minutes of pulmonary-cardiac resuscitation, 3 defibrillations + external cardiac massage + defibrillation + external cardiac massage + defibrillation were performed with a current of 200-250-300 Dj. At 20 minutes, after 3 defibrillations, normal heart function was restored. The patient's heart function was restored in 20 minutes. As the patient's vital signs began to breathe with resistance to the ALV apparatus, sodium oxybutyrate-4 g, Sibazon-10 mg, morphine hydrochloride-10 mg were administered intravenously, the patient was adapted to the ALV apparatus and transferred to the intensive care unit against vasopressors.

On arrival Total blood test: hemoglobin-123 g / l; erythrocytes-4.0 x 10<sup>12</sup> / l; color index-0.92; hematocrit-37%; blood clotting time: start-2:58, end-3:24; platelet-133.0 x 10<sup>9</sup> / l; leukocyte-5.3 x 10<sup>9</sup> / l; rod nucleus neutrophils-2%; segment nuclear neutrophils-38%; Eosinophils-2%; Lymphocytes-51%; monocytes-7%; erythrocyte sedimentation rate-4 mm / s. Biochemical blood test: Total protein-58 g / l; glucose-7.1 mmol / l; urea-6.0 mmol / l; bilirubin: total-29.0 mmol / l, bound-9.0 mmol / l; ALT-277 Ed / l; AST-291 Ed / l; Calcium-2.3 mmol / l; fibrinogen-4.4 g / l. General urine analysis: color-yellow, clear; relative density-1020; rN-7.0; protein-0.066 g / l; glucose-negative; ketone bodies-positive (+); blood unit detection reaction-positive; bilirubin - negative; epithelial cells in a drying area - flat-0-1; leukocytes -8-9 in a drying area; erythrocytes unchanged in a drying area-6-7. Chest X-ray findings: Bronchitis; Conclusion of abdominal ultrasound examination: Parenchymatous organ injury and free fluid in the abdomen were not detected at the time of examination; Conclusion of cerebral MSKT examination: No organic or traumatic structural changes in brain tissue were detected.

**Table 1. Hemoglobin and hematocrit condition**

As a result of intensive therapy, clinical and laboratory parameters improved in dynamics: In the analysis of whole blood: hemoglobin-121 g / l; erythrocytes- $4.1 \times 10^{12}$  / l; color index-0.9; hematocrit-39%; coagulation time: start-4:10, end-4: 30; platelet- $176.0 \times 10^9$  / l; leukocyte- $8.5 \times 10^9$  / l; rod nucleus neutrophils-2%; segment nuclear neutrophils-70%; Eosinophils-1%; Lymphocytes-25%; monocytes-2%; erythrocyte sedimentation rate-8 mm / s. Biochemical blood test: Total protein-70 g / l; glucose-6.4 mmol / l; urea-6.0 mmol / l; bilirubin: total-11.4 mmol / l, bound-1.8 mmol / l; ALT-95 Ed / l; AST-57 Ed / l; Calcium-2.2 mmol / l; fibrinogen-3.8 g / l. General urine analysis: color-yellow, clear; relative density-1016; protein traces; glucose-negative; ketone bodies-negative (-); blood unit detection reaction-negative; bilirubin - negative; epithelial cells in a drying area - flat-0-1; leukocytes -4-6 in a drying area; erythrocytes unchanged in a drying area-1-2. Chest X-ray findings: Bronchitis; Conclusion of abdominal ultrasound examination: No echopathology was detected in the abdomen at the time of examination; Conclusion of cerebral MSKT examination: No organic or traumatic structural changes in brain tissue were detected. ECG: Sinus rhythm. YuQS-84 times per minute. Mild metabolic changes were detected in the myocardium.

**Table 2. Changes in the dynamics of ALT and AST analyzes**

The patient continued intensive care in the surgical intensive care unit from 01:30 on 27.12.2021. The patient's hemodynamics were captured under the influence of vasopressors. The patient was discontinued vasopressor therapy at 14:00 on 30.12.2021 against the background of a decrease in the dose of vasopressors. The patient's consciousness was completely restored from coma II to 29.12.2021 to coma I, 31.12.2021 to sopor level, 02.01.2022 to mild numbness, 03.01.2022. The patient's consciousness was maintained in constant analgo-sedation until it improved to a mild level of numbness and was awakened from time to time to determine the level of consciousness. The patient underwent tracheostomic tube implant surgery on 29.12.2022 in order to improve the sonication of the tracheobroncheal tree, reduce the volume of the dead cavity, to prevent disruption of the oral microflora. The patient was transferred to independent breathing on 03.01.2022 through a tracheostomy. On 05.01.2022, the tracheostomy tube was removed and the ligament was burned. The patient was voluntarily discharged home on 06.01.2022 without cognitive dysfunction.

**Table 3. Pharmacotherapy in post-resuscitation disease**

| The name of the drug | Day           |    |    |    |    |    |              |   |   |   |   |   |
|----------------------|---------------|----|----|----|----|----|--------------|---|---|---|---|---|
|                      | December 2021 |    |    |    |    |    | January 2022 |   |   |   |   |   |
|                      | 26            | 27 | 28 | 29 | 30 | 31 | 1            | 2 | 3 | 4 | 5 | 6 |
| Cyticolin            |               |    |    |    |    |    |              |   |   |   |   |   |
| Cytoflavin           |               |    |    |    |    |    |              |   |   |   |   |   |
| Amantadine sulfate   |               |    |    |    |    |    |              |   |   |   |   |   |
| Lidocaine            |               |    |    |    |    |    |              |   |   |   |   |   |
| Dexamethasone        |               |    |    |    |    |    |              |   |   |   |   |   |
| Sodium 7.5% -200.0   |               |    |    |    |    |    |              |   |   |   |   |   |
| Furasemide           |               |    |    |    |    |    |              |   |   |   |   |   |
| Ceftriaxone          |               |    |    |    |    |    |              |   |   |   |   |   |
| Levofloxacin         |               |    |    |    |    |    |              |   |   |   |   |   |
| Reosorbilakt         |               |    |    |    |    |    |              |   |   |   |   |   |
| Heparin              |               |    |    |    |    |    |              |   |   |   |   |   |
| Nebulizer therapy    |               |    |    |    |    |    |              |   |   |   |   |   |

Based on the results of objective examination and laboratory-instrumental examination, the following diagnosis was made: Acute toxic myocarditis of alcoholic etiology? Complications: Ventricular fibrillation. Asystole. Condition after pulmonary heart-brain resuscitation. Postreanimation disease. Keeping the patient in complete hypothermia to the treatment plan; for neuroprotection: citicoline; In order to improve metabolism in organs and tissues: cytoflavin; In order to maintain and improve the activity of brain cells remaining in ischemia: amantadine sulfate; for membrane stabilization: lidocaine, dexamethasone; for dehydration: hypertonic sodium chloride solution, furasemide; For antioxidant therapy: Ascorbic acid; For antibacterial therapy: ceftriaxone, levofloxacin; In order to improve the rheology of the mine: rheosorbilakt, heparin; For construction and power supply: Melkam; For nebulizer therapy: decasan, nebutamol; For analgo-sedation: Sodium oxybutyrate, profol, verzepam, midozalam, metamizole sodium, fentanyl, promedol, morphine hydrochloride; For muscle relaxation: Arduan; For the treatment of hyperthermia: Taken paracetamol.

### Conclusions

1. All over the world, it is accepted that the time norm of clinical death is 3-8 minutes, but due to the achievements of modern medicine, this time can be extended to 10-15 minutes. This could allow thousands of patients in the community to return to social life without cognitive dysfunction, without social death. Intensive therapies performed in 4 patients in our practice in patients who remained in a state of clinical death for 10-15 minutes gave a positive result.
2. Intensive therapy of post-resuscitation disease should be carried out in combination with treatment of post-anoxic encephalopathy and treatment of polyorgan deficiency, which develops in complex forms of post-resuscitation disease.
3. Treatment of postanoxic encephalopathy includes neurotropic therapy: a combination of antihypoxants, antioxidants, sedative drugs, and energy sources.
4. Restoration of brain activity after clinical death depends on maintaining cerebral perfusion in the 1-2 stages of the disease after resuscitation in a slightly higher than normal and early onset of neuroprotective drugs.
5. In patients with pulmonary resuscitation and the restoration of cardiac function, the initiation of cerebral therapy from the first minutes restores the function of nerve cells in hypoxia and ischemia, as well as a sharp decrease in the incidence of cognitive impairment.

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