

infarction during surgery. On the other hand, the risk of recurrent stroke during the waiting period elective surgery reaches more than 35%. Improvement of surgical technique, the advent of new modern methods of examination, experience of surgical treatment of chronic cerebral ischemia and experience of neurologists for the treatment of ischemic stroke forced vascular surgeons and neurologists in recent years to reconsider the timing of surgery in patients with ischemic stroke and recommend surgery on the carotid arteries to be administered during the first four weeks after stroke.

Aim of work - to evaluate the results of surgical treatment of patients after reconstructive operations on the carotid arteries, performed in the acute period of ischemic stroke

Materials and methods of investigation - for the period from June 2014 to December 2015 were performed urgent reconstructive operations on carotid arteries for 77 patients who entered the vascular center in Hospital №1 named N.I. Pirogov in Samara city with acute ischemic stroke. The age of patients ranged from 45 to 75 years. Men were 62, women - 15. At admission the number of patients on a Scale Rankin the 1-2 score was 18 patients, 3 points-32 patients, 4 points - 27 patients. All patients underwent CT or magnetic resonance imaging with angio program, color duplex scanning of brachiocephalic vessels, echocardiography. Neurological status was assessed by a neurologist on the stroke scale National Institute of Health (NIHSS), modified Rankin scale (MRS), the index Rivermide before surgery, 24 hours after surgery and in 14 - 24 days to the time of discharge from the hospital. Indications for surgical intervention were determined collectively: the neurologists and vascular surgeons. All operations were performed under endotracheal anesthesia.

Results - carotid endarterectomy was performed for 77 patients, 42 was performed eversion carotid endarterectomy, 9 patients eversion carotid endarterectomy combined with resection of the ICA, in connection with marked tortuosity of the ICA. Resection of the ICA and the elimination of tortuosity was performed on 4 patients. Plastic ECA with resection of the occluded ICA was performed on 17 patients. Carotid endarterectomy with venous patch sewing in CCA-ICA - 5 patients. In the first days of ischemic stroke was operated 5 patients, in the period from 2 to 3 days - 21 patients, in the period from 4 to 7 days - 27 patients, 8 to 18 days 24 patients. Two patients were restored trunk blood flow in the ICA: one patient with thrombosis on the 2nd day of a stroke; another ICA occlusion at day 7 of ischemic stroke. Postoperative regression of neurological symptoms was noted in 66 (86%) patients. The number of patients able to self-care at discharge from hospital was on Rankin Scale: 1-2 points - 59 patients, 3 points - 10 patients. Not capable of self according to the Scale of Rankin 4 points turned out to be 8 people. None of the operated patients was noted not repeated of stroke on the ischemic or hemorrhagic type, during the hospital stay.

In the postoperative period died 2 (3%) patients. One at 24 days after surgery from the increase of respiratory and heart failure, another patient died on day 29 from the kidney and subsequent heart failure. In none of these two cases was not registered repeated violations of cerebral circulation neither clinically nor at autopsy.

Conclusions - surgery on the carotid arteries within the first 14 days from the date of development of cerebrovascular accident is a safe operation in terms of the development of hemorrhagic transformation in a multidisciplinary vascular center.

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ACUTE HYPERGLYCEMIA DETERIORATES CARDIOPROTECTIVE EFFECT OF REMOTE ISCHEMIC PERCONDITIONING IN A RAT MODEL FOR ACUTE MYOCARDIAL INFARCTION

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Introduction: Remote ischemic preconditioning (RIPC), defined as short episodes of ischemia-reperfusion on a distant organ in order to protect myocardium against simultaneous prolonged ischemia, is a promising non-invasive strategy in acute myocardial infarction therapy. However, metabolic diseases, e.g., diabetes mellitus or hyperlipidemia, could interfere with protection mediated by ischemic preconditioning. The detrimental effects of chronic hyperglycemia on cardioprotective signaling pathways are well established, however, it is not known how acute hyperglycemia without preceding diabetes affects RIPC.

Aims: Our objective was to investigate the effects and molecular background of acute hyperglycemia without diabetes on cardioprotection by RIPC in rat model.

Methods: The in vivo study was conducted on healthy, male Wistar rats. Acute hyperglycemia (15-20 mM, AHG) was induced with continuous dextrose infusion in AHG group, while normoglycemic (NG) rats received isoosmotic solution of mannitol. After 35 min stabilization, the left anterior descending artery was occluded to induce myocardial ischemia for 40 min. RIPC was induced by 3x5 min transient limb ischemia by occlusion of the femoral vessels in both NG and AHG groups. After two hours of reperfusion, hearts were removed for Evan's blue and triphenyl tetrazolium chloride staining to determine infarct size. Electrocardiogram of each animal was recorded for arrhythmia analysis. For in vitro study, left ventricular tissue was collected from NG and AHG animals immediately before myocardial ischemia. To investigate the activity of Akt-mTOR pathway and autophagy, their specific markers were detected by Western blot. The level of oxidative stress was estimated by measuring 3-nitrotyrosine ELISA.

Results: Infarct size was significantly reduced by RIPC in normoglycemia (24.7% vs. 46.3%; NG+RIPC vs. NG+Isch) while not in acute hyperglycemia (52.8% vs. 54.2%; AHG+Isch vs. AHG+RIPC). Acute hyperglycemia increased the incidence and duration of ventricular arrhythmia; however, RIPC did not alter arrhythmia scores of NG or AHG groups. The level of 3-nitrotyrosine was significantly elevated in acute hyperglycemia (0.50 vs. 0.87 µg 3-NT/mg protein; NG vs. AHG). Furthermore, phosphorylation of Akt-mTOR markers was enhanced in acute hyperglycemic myocardium. Decreased ratio of LC3II/LC3I was measured, but other markers of autophagy were not modulated by elevated plasma glucose level.

Conclusion: Cardioprotection by RIPC is abolished by acute hyperglycemia in vivo. Acute hyperglycemia induced oxidative stress and activation of Akt-mTOR pathway may be involved in the background of the deleterious effect of acute hyperglycemia.

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ОСТРЫЙ МИОКАРДИТ С КЛИНИЧЕСКОЙ КАРТИНОЙ КОРОНАРОГЕНННОГО ЗАБОЛЕВАНИЯ СЕРДЦА

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МЕДИЦИНСКИЙ ФАКУЛЬТЕТ, КАФЕДРА ФАКУЛЬТЕТСКОЙ ТЕРАПИИ

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ACUTE MYOKARDITIS WITH THE CLINICAL PICTURE OF A CORONAROGENTIC HEART DISEASE.

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Myocarditis (MT) - is a noncoronary myocardial disease with infiltration, functional decreasing, morphological changes and myocardial fibrosis, necrosis and (or) myocyte degeneration, associated with clinical symptoms of acute heart failure (HF) and sudden cardiac death. The inflammatory process may involve cardiomyocytes, interstitial tissue, blood vessels, cardiac conduction system and pericardium. A possible outcome of MT is dilated cardiomyopathy. The etiological factors of MT are exposure to infectious, toxic, chemical or physical agents, autoimmunity and inflammatory processes of the myocardium in the context of systemic diseases. More than 50% are considered to trigger viruses.

A 53 years old male patient with a long clinic of arterial hypertension (highest numbers of blood pressure 160/100 mmHg) and a body mass index 32.2kg/m² (weight 93 kg, height-170 cm, RT- 110cm) noted a fatigue, weakness, dizziness, sweating and high temperature 38.3C for a five days.

Initial and repair chest radiographs showed a segmental pneumonia on the right. On this occasion he was appointed antibiotics ambulatory - 400mg avekloks ones a day during 7 days. On the 5th day of treatment the body temperature decreased to 35 C, with manifested shortness of breath, unproductive cough and atypical chest discomfort. Respiratory rate 18/min, SpO₂ 98%, BP=130/80 mmHg. HR=74/min. ECG examination demonstrated acute myokardial injury pattern: elevation of ST-segment in V1, V2, V3, a cardiac biomarkers were elevated as well. Basing on of