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Рус. Eng.

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ISCHEMIA/REPERFUSION EFFECT ON PANCREATIC VOLUMETRICAL BLOOD FLOW VELOCITY

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Abstract. We investigate blood flow velocity at ischemia/reperfusion early stages. Transient hyperemia absence after ischemia/reperfusion cased by pancreatic edema development at early stages confirmed by Wet/Dry ratio elevation and morphological changes was determined.
Key words: ischemia, reperfusion, blood flow, pancreas.

Ischemia is a trigger and pathogenesis stage in many pathological conditions. Anderson (1961, 1963) was the first who discovered ischemia leading role in acute pancreatitis pathogenesis [5, 6]. This discovery made possible to create vascular model of acute pancreatitis (Hoffmann at all., 1995) later used in domestic and foreign studies [3, 8, 10, 13].

Recent years many authors reveald мetabolic changhes during primary and secondary ischemia are main reasons of pancreatic necrosis formation and spreading even in small primary damage. That was proven by cardiosurgeons found a pancreatitis activation after hypoperfusion during heart surgerys artificial circulation [4, 9, 11, 12, 14].

Thereby pancreatic microvessels blood flow velocity investigation is crucial as it correlates with tissue damage intensity.

Previosely dramatic blood flow decreasing in ductal pancreatitis were proven with minimum at 24 hours folowed by significant stromal changes.

We did't find blood flow investigations at early stages of acute pancreatic damage during blood supply imparement and that was the goal of current research. The latter is of particular importance in assessing the protective effect of ischemic, distant and pharmacologic preconditioning and metabolic effects of drugs [1, 2, 7].

Materials and methods: 30 white (male and female) 280 -300g rates were used. All investiagations were made at the same time under general anesthesia («Zolitel 100» 60 mg/kg with chloral hydrate 125 mg/kg intraperitonealy).

Transient ischemia reproduced by 30 minutes pancreatic arteries compression followed by reperfusion.

Blood flow velocity was measured by Biopaq systems MP150 with TSD144 probe in perfusion units (PU).

Wet/Dry index were measured as ratio between pancreatic weight after section and after drying with weight stabilization at 56^0C.

Results. Average initial pancreatic perfusion level was 418.09±39.11 PU, droping to undetecteble data during ischemia period. 1 reperfision minute charectorised by arrising blood flow up to 87.15±12.83 PU and 3 minute 99.47±10.74 PU. At 7.5 minute pancreatic blood flow recovered up to 103.749±10.47 PU, and 151.35±12.32 PE at 15 reperfusion minute correspondingly. Microcirculation blood flow velocity were seen up to 30 reperfusion minute 333.21±26.32 PU and were changed by up to 266.69±18.79 PU decreasing at 60 reperfusion minute. Revovery of pancreatic bood flow velocity thus comprised 79.69% of initial level at 30 reperfusion minute and followed by dropping to 63.78% level at 60 blood flow restoration minute (Fig.1.).

Recived data contradict with data obtained previously in our laboratory for microcirculatory blood flow velocity in other abdominal organs (liver, small intestine) ischemia and reperfusion so intestinal deep 30 minutes ischemia leads transient hyperperfusion with maximum at 15-20 min exceeding initial level 1.75-2.1 times.
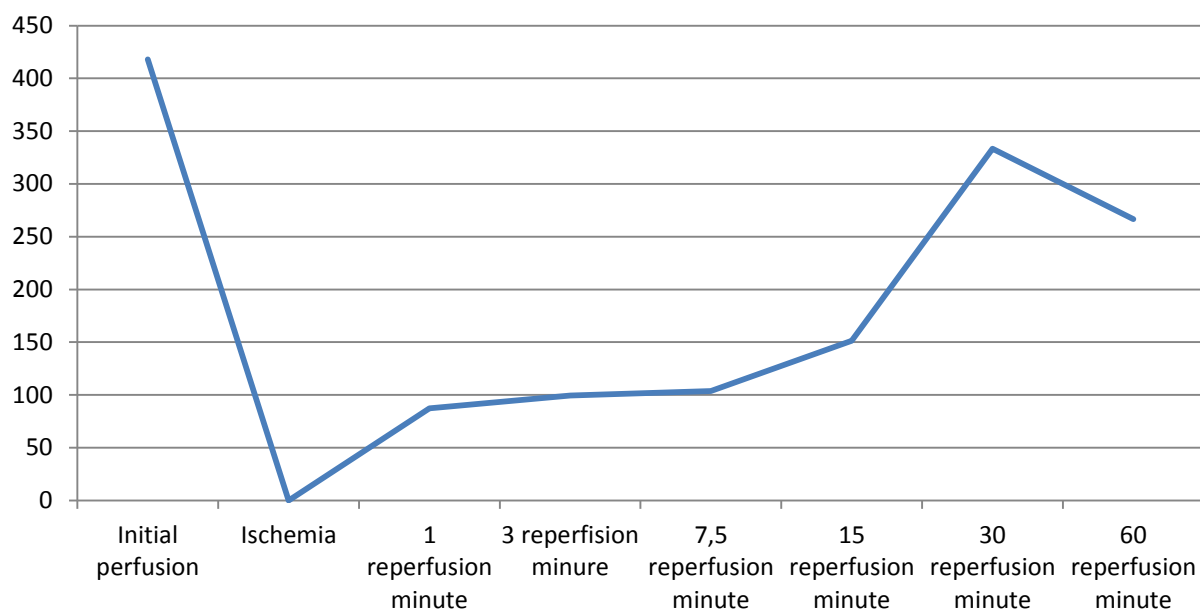
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Figure 1. Dynamic of blood flow velocity data in pancreatic gland during ischemia/reperfusion

These microcirculation differences in different internal organs response to ischemia couldn't be explained by systemic hemodynamic changes.

Visually we recognize pancreatic edema formation till 30 reperfusion minute following deep 30 minutes long ischemia (Fig. 2).
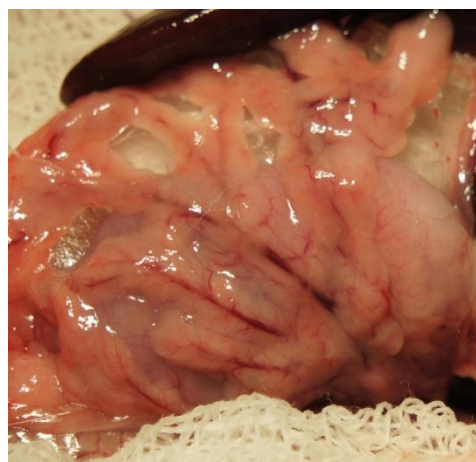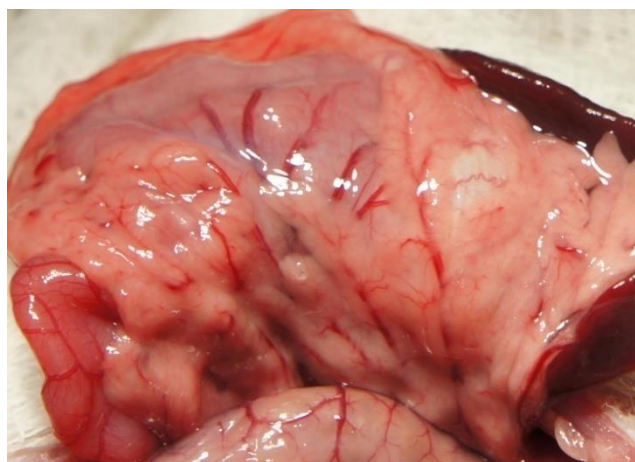

Figure 2. Edema end dispersion of pancreatic lobules at 30 min of reperfusion (right) versus against intact group (left)

Most frequently used methods for edema assessment are Wet/Dry ratio counting and Evans blue dye measuring. Wet/Dry ratio expressed early stages changes versus Evans blue dye extraction used for microvessels permeability assessment for large molecules. That helped us to choose W/D ratio rather than Evans blue dye extraction.

In intact group Wet/Dry ratio was 1.72±0.04 increasing up to 2.1±0.02 till 7.5 reperfusion minute and to 2.52±0.02 till 15 minute. 30 reperfusion

minute characterized by Wet/Dry ratio at 3.06±0.03 level.

We think such changes were due to severe permeability increasing in ischemia/reperfusion group. Pancreatic edema leads to acinar vessels compression helping necrotizing process spreads through pancreatic tissue, on the other hand interstitial edema decrease the microvessels quantity and subsequently decreased volumetric velocity.
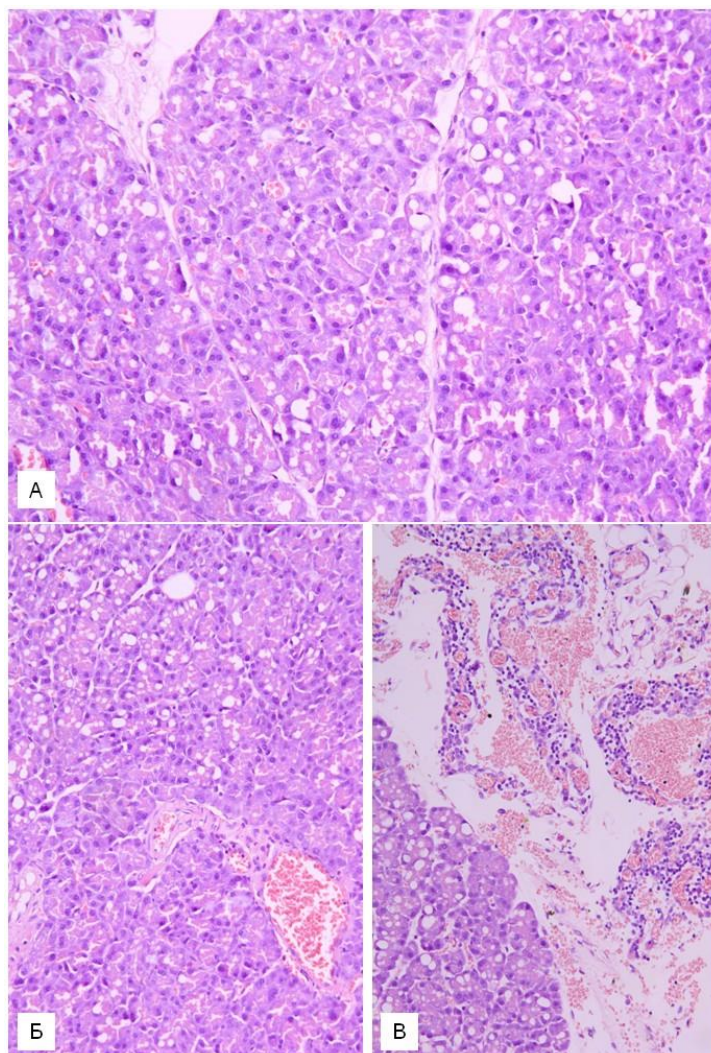
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Figure 3. Morphological changes in pancreatic gland in 30 minutes ischemia group: disorganization of lobular structure, severe diffuse dystrophic acinar cells changes: vacuolization zymogene zones (А), venous hyperemia (Б), haemorrhage into intraacinar stroma. Hematoxylin and eosin dye. Microphoto. Х100

Interstitial edema was proven standard histological examination. disorganization of lobular structure, severe diffuse dystrophic acinar cells changes: vacuolization zymogene zones, venous hyperemia, haemorrhage into intraacinar stroma also were found (Fig. 3).

Reperfusion leads to tissue injury deepening end exhibits as progression of lobular structure disorganization, severe diffuse dystrophic acinar cells changes, vacuolization of cellular apical parts and interstitial edema (Fig. 4).
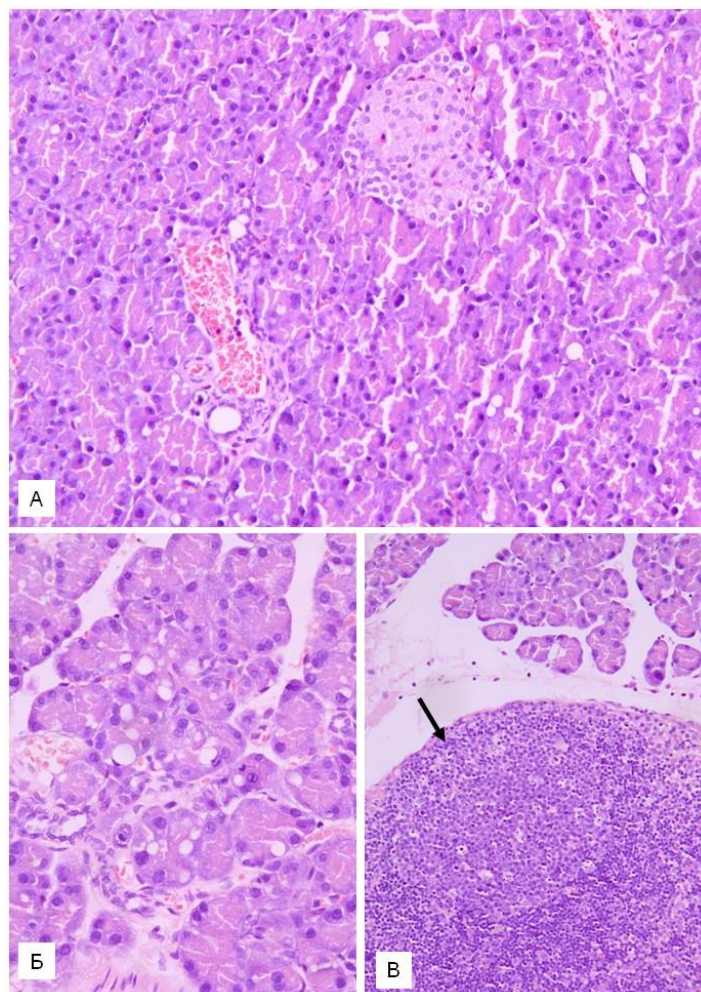
Figure 4. Morphological changes in group with ischemia and 30 minutes of reperfusion: progression of lobular structure disorganization, severe diffuse dystrophic acinar cells changes (А), vacuolization of cellular apical parts (Б), interstitial edema. Hematoxylin and eosin dye. Microphoto. 100 (А, В), x 200 (Б).

Conclusions. The results obtained in the study showed no evidence of reactive hyperemia in the pancreatic tissue during reperfusion. This is probably due to the rising of interstitial edema, evidenced an increase in Wet/Dry factor and dynamics morphological changes

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