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PC4-Insulin resistance is the leading mechanism of metabolic disorders and pregnancy complications in obese women

Tarasenko KV

Higher State Educational Institution "Ukrainian medical stomatological academy" Poltava, Ukraine. Email: bcp-p@i.ua

Abstract: Normal pregnancy is accompanied by physiological insulin resistance that facilitates fetal development. Progression of insulin resistance in obese women depends on severity of obesity. Intensity of fetal energy metabolism is several orders of magnitude higher than in the maternal organism (Gordyunina, 2008). A decrease of glucose uptake by cells in obese pregnant women is characterised by an increase of the HOMA-IR index that reached maximum values in pregnant women with class I and class II obesity and was two times significantly higher than control values. The conditions initiate autoregulated activation of lipolysis in the adipose tissue causing hyperlipidemia type IV. Macrophagal infiltration of the adipose tissue and increased apoptosis of adypocytes at obesity activate cytokine secretion (Schwartz, 2009). The maximum level of tumor necrosis factor alpha that initiates cytokine imbalance and inhibits activity of insulin receptors was found in pregnant women with class I obesity. It was two times significantly higher than in the control group of pregnant women. Frequency of placental dysfunction and gestoses rises with increased severity of obesity. We found placental dysfunction as a pregnancy complication in 63.6% women with class I obesity and in 71.4% women with class II obesity compared to 28.6% in the group of pregnant women with normal body mass ($p<0.02$ and $p<0.05$, respectively). Frequency of gestoses likewise grew with increase of body mass of pregnant women. It reached 64.3% in women with class II obesity compared to 14.3% in the control group ($p<0.001$). Presence of systemic inflammation in pregnant women with obesity is verified by a rise of C-reactive protein concentration concomitantly with increased severity of obesity. Thus, progression of insulin resistance is a pathophysiologic basis of pregnancy complications in obese women. It is accompanied by dyslipidemia, development of low-intensity systemic inflammation and promotes energy deficiency in the maternal organism.

PC5-Pathophysiological aspects of hypothyroidism in the experiment

Mykhailychenko V, Vasylyanska O

SI "Institute of Urgent and Recovery Surgery n.a.V.K.Gusak, NAMS of Ukraine", Donetsk, Ukraine. Email: pancreas1978@mail.ru

Abstract: In the experiment with the surgical model of hypothyroidism that involved 30 female Wistar type rats, were demonstrated pathophysiological processes presented in the animal immune system and cardiac activity. In rats with hypothyroidism model significantly decreased the number of cardiac beats at rest; the stress imitating loading leads to the ischemic events in the myocardium, disturbances in the heartbeats and significantly lower response of miocardium in comparison with the intact animals. During the ultrasound examination of heart in rats with hypothyroidism is observed the left heart enlargement and decrease of number of myocardium contractile abilities. Hypothyroidism in animals leads to decrease of functional reserves of oxygen-dependent biocide of neutrophils on the background of methabolitic oxidative processes increase (diene conjugation, malonicdialdehyde), accompanied by the decrease of glutathione level. Simultaneously with this advance are developing the processes of endotoxicosis, accompanied by methylsulfonilmethane increase. All the above-listed processes took place on the background of thyroid-stimulating hormone (TSH) concentration increase and decrease of T3, T4. Pathophysiological changes in the body lead to the 100% death rate of animals without treatment most probably basically from the cardiac deficiency.

PC6-The measurement of thyroglobulin autoantibodies in the presence of thyroglobulin

Snezana Zivancevic Simonovic[1], Ljiljana Mijatovic Teodorovic[1,2], Olgica Mihaljevic[1], Irena Kostic[1], Marijana Stanojevic Pirkovic[1]

[1]*Faculty of Medical Science, University of Kragujevac, Serbia.* [2]*Department of Nuclear Medicine, Clinical Center, Kragujevac, Serbia. Email: snezana@medf.kg.ac.rs*

Abstract: It is well known that thyroglobulin antibodies (TgAb) interference is the most serious problem affecting the thyroglobulin (Tg) measurement. The aim of this study was to explore the influence of Tg on TgAb measurement. We have used standard Tg concentrations (53, 200 and 510 ng/ml) and 10 patient's sera samples with elevated TgAb concentrations (100-4245 IU/ml) and undetectable Tg. Patient's sera with known TgAb concentrations were incubated with standard Tg concentrations (53, 200 and 510 ng/ml), volume ratio 1:1, during 30 min. After that, TgAb concentrations were measured by a competitive "one-step" radioimmunoassay. In the same samples Tg concentrations were determined by an immunoradiometric assay. The TgAb values measured in presence of standard Tg concentrations were compared with TgAb values measured without Tg. In nine of ten patient's sera the TgAb concentrations measured in the presence of Tg were unvaried, while in one sera the TgAb concentration was decreased. Decrease of TgAb concentration was obtained after incubation with high Tg concentrations (200 and 510 ng/ml) and it was dose-dependent. When we measured the concentration of Tg in the same samples, the standard Tg concentrations were decreased in the presence of 2 patients sera, one of these had low and another very high concentration of TgAb, but these samples are different from sera in which interference of Tg with TgAb measurement was shown. In conclusion we could say that Tg in some patients' sera might interfere with TgAb measurement, but it is not necessary that in the same sera samples TgAb interference with Tg measurement exist.

Keywords: thyroglobulin antibodies, thyroglobulin, measurement, interference

PC7-Pretreatment with quercetin prevents exhaustion of protease inhibitors in liver tissue in hindlimb ischemia/reperfusion injury

Aliev LL, Kharchenko VZ, Kubyshkin AV, Zhukova AA

Crimean State Medical University, Simferopol, Crimea, 295006. Russia.
Email: Aliev.L@patfiz-csmu.net

Abstract: One of the leading pathogenetic mechanisms of ischemia/reperfusion injury (I/R) is activation of proteases, caused by exocytosis of neutrophilic granulocytes and peroxidative damage of lysosomal membranes. The systemic propagation of active nonspecific proteases accompanied by the exhaustion of antiproteases causes injuries of remote organs, such as liver. Male Wistar rats were undergoing bilateral hindlimb ischemia during 6 hours with 6 hours of following reperfusion period. A watersoluble form of quercetin (10 mg/kg of body weight) was given before reperfusion. Trypsin-like activity (TLA), elastase-like activity (ELA), levels of alfa-1-antitrypsin (A-1-AT) and acid-stable inhibitors (ASI) of liver tissue homogenates were measured by spectrophotometric methods based on registration of enzymal hydrolysis speed of synthetic substrates. Ischemia/reperfusion-induced increase of the ELA level (15,4% ($p<0,05$) compared to control) in liver tissue was not significantly inhibited in animals pretreated with quercetin. I/R-model increased TLA level 109,1% ($P<0,001$). In rats pretreated with quercetin TLA level was 23,3 % ($P<0,05$) lower. The decrease of A-1-AT in I/R-model was 31,6 % ($p<0,001$) (compared to control). In rats with I/R-model on a background of quercetin use change of A-1-AT in comparison to control wasn't statistically-valid. Level of ASI in liver tissue reduced 47,6 % ($P<0,001$) after I/R-injury. In rats pretreated with quercetin ASI level was 19,3 % ($P<0,05$) higher. Both bilateral hindlimb ischemia and reperfusion led to increase of nonspecific proteolytic activity and exhaustion of antiproteases in liver tissue. This study showed that pretreatment with quercetin reduces the increased TLA activity and prevents exhaustion of antiproteolytic capacity.

PC8-LPS-induced microcirculation impairment after partial hepatectomy contributes to hepatic dysfunction in a rat model

Haoshu Fang[1, 2], Anding Liu[2], Olaf Dirsch[3], Uta Dahmen[2]

[1]Department of Pathophysiology, Anhui Medical University, Hefei 230032, China. [2]Experimental Transplantation Surgery, University Hospital of Jena, Jena, Germany. [3]Institute for Pathology, University Hospital of Jena, Jena, Germany. Email: fang.haoshu@hotmail.com