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www.karger.com/doi/10.1159/000363269

The 49th Congress of the European Society for Surgical Research

May 21-24, 2014, Budapest, Hungary

Abstracts

Congress Organizing Committee
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Walter Brendel

WB 01

Demonstration of the Effects of Portal Vein Ligation on Glucose Metabolism Using in Vivo Multi-modal Pet/Mri Measurements in Healthy Rat Liver

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Background: Portal vein ligation (PVL) results in ipsilateral atrophy and hypertrophy of contralateral liver segments. It is unknown how PVL affects metabolic patterns of hepatic tissues. The aim of this study is to evaluate the effect of PVL on glucose metabolism, using multi-modal PET/MRI imaging in healthy rat liver.

Material and Methods: Male Wistar rats (n=30) underwent PVL. 2-deoxy-2-(18F)fluoro-D-glucose (FDG) PET/MRI imaging and morphological/histological examination were performed before; 1-, 2-, 3-, 7-days after PVL. Dynamic PET data were collected and the standardized uptake values (SUV) for ligated and non-ligated liver lobes were calculated in relation to cardiac left ventricle (SUVVOI/SUVCLV) and mean liver SUV (SUVVOI/SUVLiver).

Result: PVL induced atrophy of ligated lobes, while non-ligated liver tissue showed compensatory hypertrophy. Dynamic PET scan revealed altered FDG kinetics in both ligated and non-ligated liver lobes. SUVVOI/SUVCLV significantly increased in both groups of lobes, with a maximal value at 2nd postoperative day and returned near to the baseline 7 days after the ligation. After PVL, ligated liver lobes showed significantly higher tracer uptake compared to the non-ligated lobes (significantly higher SUVVOI/SUVLiver values were observed at postoperative day 1, 2 and 3). The homogenous tracer biodistribution observed before PVL reappeared by 7th postoperative day.

Conclusion: Our study demonstrated an altered glucose metabolism in both ligated and non-ligated liver lobes. The observed alterations in FDG uptake dynamics should be taken into account during the assessment of PET data until the PVL induced atrophic and regenerative processes are completed.

WB 02

The Effect of Nitric Oxide Synthase Inhibitor(L-NAME) on Ischemia-reperfusion Injury Following Pringle Maneuver of Cirrhotic Liver Induced by Bile Duct Ligation

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Background: Pringle maneuver(PM) has been used widely to control blood loss during liver resection. However, hepatic inflow occlusion can also result in hepatic ischemia-reperfusion injury (IRI), especially in patients with a cirrhotic liver. Nitric oxide(NO) is generated from NO synthase(NOS), and has not only cytoprotective but also cytotoxic effect. The aim of this study was to investigate the effect of the NOS inhibitor(L-NAME) on IRI after PM of cirrhotic livers induced by bile duct ligation(BDL).

Material and Methods: Male Wistar rats were divided into three groups: sham group(non-BDL/no treatment), L-NAME group(BDL with L-NAME treatment) and BDL group(BDL/no treatment). Cirrhosis was induced by BDL. Two weeks after BDL, 50 % partial hepatectomy with PM was performed. The rats of L-NAME group were injected with L-NAME(1.5 mg/kg) 15 minutes before PM. We evaluated the hepatocellular damage, portal venous flow and microcirculation of the liver after reperfusion.

Result: Although there was no significant difference in portal venous flow between L-NAME and BDL group, microcirculation of the liver in L-NAME group tended to be higher than that of BDL group(Table). Histopathologically, the semi-quantitative score for liver damage and apoptotic index were significantly lower in L-NAME group than in BDL group 168 hrs after reperfusion. Ki-67 labeling index of L-NAME group was significantly higher than that of BDL group 24 hrs after reperfusion.

Conclusion: L-NAME attenuated IRI induced by Pringle maneuver, and improved microcirculation of cirrhotic livers.

Microcirculation flow of the liver (AU) (Mean ± S.E.M.)		
Time after reperfusion (hr)	L-NAME group	BDL group
1	226.8 ± 62.2	114.5 ± 7.63
3	202.0 ± 73.4	109.5 ± 18.2
24	166.2 ± 30.5	125.0 ± 2.65
168	161.3 ± 13.7	94.3 ± 15.2