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Centre Hospitalier Régional
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Oral Communication List

* Presenting authors

* At the time of printing, the presenting author was not registered to the congress.

Walter Brendel Session

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Kazuhiro Kudoh^{1,*}, Hiroshi Uchinami¹, Yuzo Yamamoto¹
¹Department of Gastroenterological Surgery, Akita University Graduate School of Medicine, Akita, Japan
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Natacha Rousse^{1,*}, Francis Juthier¹, Frederic Mauquet¹, Carlo Banfi¹, Alexandre Ung², Laura Ravasi³, Sophie Susen², Alain Prat¹, Brigitte Jude², Andre Vincentelli¹
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Lisa Rancan^{1,*}, Luis Huerta², Gabriel Cusatti³, Iñaki Erquicia³, Jesus Isea², Victor Garcia¹, Ignacio Garutti³, Carlos Simón², Elena Vara¹
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Mathieu Manassero^{1,2,*}, Véronique Viateau^{1,2}, Fani Anagnostou², Sandra Guerard³, David Mitton³, Véronique Mignonney⁴
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- OP-05 A simple, low risk method to increase liver ischemic-tolerance: Perconditioning**
Zoltan Czigan^{1,*}, Zsolt Turóczi¹, Orsolya Bulhardt¹, Viktor Hegedüs¹, Gabor Lotz², Laszlo Harsanyi¹, Attila Szijarto¹
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Julien Brossier^{1,*}, Faiza Mohand Kaci¹, Jean François Deux², Hicham Kobeiter², Brigitte Gillet³, Pascal Desgranges⁴, Jean Pierre Becquemin⁴, Fabrice Schneider¹, Eric Allaire¹
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Session 1: Stem cells and cell therapy

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Guy Magalon^{*}
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- OP-07 The mobilization and recruitment of bone marrow-derived stem cells contribute the acceleration of wound healing after surgery**
Yoshihiro Takemoto^{1,*}, Tao-sheng Li², Mako Ohshima¹, Hiroshi Kurazumi¹, Masayuki Kubo¹, Kuzuhiko Ueda¹, Tadahiko Enoki¹, Kimikazu Hamano¹
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- OP-08 In-vivo survival assessment of human mesenchymal stem cells in a large femoral bone defect in mice**
Mathieu Manassero^{1,2,*}, Véronique Viateau^{1,2}, Jose Retortillo¹, Mickael Deschepper², Morad Bensidhoum², Delphine Logeart-Avramoglou², Hervé Petite²
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- OP-09 Bone regeneration using a resorbable scaffold: study in sheep**
Mathieu Manassero^{1,2,*}, Véronique Viateau^{1,2}, Adeline Decambon^{1,2}, Karim Oudina², Hervé Petite², Morad Bensidhoum²
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- OP-10 Treatment of chronic liver failure by transplantation of liver cells and bone marrow stem cells: 1 year experience.**
Murat Shagidulin^{1,*}, Nina Onishchenko², Mikhail Krasheninnikov², Elena Volkova¹, Pavel Avramov¹, Igor Iljinsky³, Natalia Mogeiko³, Olga Luneva⁴, Andrei Pulin⁵, Anastasia Gorkun⁵, Irina Saburina⁵, Tatiana Demura⁴, Nadezda Perova⁶, Viktor Sevastjanov⁶, Sergei Gautier⁷
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- OP-11 Esophageal tissue engineering using a hybrid approach. In vitro analysis of cell seeding conditions**
Tigran Poghosyan^{1,*}, Sébastien Gaujoux¹, Rony Sfer², Thomas Domet³, Laurent Michaud⁴, Frederic Gottrand⁴, Patrick Bruneval⁵, Valerie Yanneau³, Jerome Larghero³, Pierre Cottan¹
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Session 2: Liver transplantation

- OP-12 Impact of DonorNet on liver donor characteristics and donor management attitudes: A comparative analysis of liver primary and re-transplantation**
Mehmet Fatih Can^{1,*}, Juan C. Mejia¹, Roberto C. Lopez¹, Mark Sturdevant¹, Christopher B. Hughes¹, Abhinav Humar¹
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OP-05

A simple, low risk method to increase liver ischemic-tolerance: Perconditioning

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Introduction

Liver ischemia-reperfusion (IR) induced injury is a frequent sequel of major liver resections and transplantations caused by long-term exclusions. These conditions resulting in increased risk of postoperative morbidity and mortality. Considering the mentioned facts the importance, as well as clinical relevance of experimental research to mitigate liver IR injury is unequivocal. Perconditioning - which means the application of brief IR cycles in a remote organ parallel with target organ ischemia - might be a reasonable possibility.

Objectives

Hepatoprotective effects of this novel method: ischemic perconditioning (IPer) was investigated in our rodent model of IR injury.

Material/Patients and Methods

Male Wistar rats (n=45) underwent normothermic, 60 minutes long ischemia on two-thirds of the liver. Three groups: sham, IR and IPer (15 animals in each) were divided into three subgroups according to the length of reperfusion times: 1h, 6h, 24h (5 animals in each). 20 minutes after induction of liver ischemia, rats in IPer group received 5 min of lower limb ischemia and 5 min of reperfusion in 4 cycles (40 min in total), by infrarenal aortic cross clamping. Hepatic and lower limb microcirculation was monitored by laser Doppler flowmeter during the first hour of reperfusion. After reperfusion liver samples were taken for routine histologic and redox state assessment. Suzuki score was used for semi-quantitative histological analysis. Serum transaminase (ASAT, ALAT) levels were measured. The total scavenger capacity of liver homogenate was measured, via detecting of chemiluminescence intensity with luminometer (LM). For detailed analysis of antioxidant contents in homogenate, we carried out three further measurements with spectrophotometry. Free SH-groups (SHG) show the protein related reducing power. The H-donating (HD) ability refers to the non-protein-bound antioxidant capacity. Reducing Power (RP) was adopted to determine a more complex, global antioxidant status.

Results

In the IPer group in comparison with IR group, the serum transaminase levels were significantly ($p < 0.05$) lower after 1, 24 h of reperfusion, but not after 6 h of reperfusion (1h ALAT: $p = 0.042$ // 24 h ASAT: $p = 0.027$; ALAT: $p = 0.014$). Severity of histologic damage was reduced in IPer group compared to the IR group at every examined reperfusion period. Significant improvement has been observed in partial parameters of the redox-homeostasis (IPer vs. IR) at each examined reperfusion time points (SHG: 1 h $p = 0.017$; 6 h $p = 0.049$; 24 h $p = 0.048$ // HD: 1 h $p = 0.031$; 6 h $p = 0.038$; 24 h $p = 0.029$), as well as in global parameters after 6 h and 24 h of reperfusion (LM: 6 h $p = 0.034$; 24 h $p = 0.034$ // RP: 6 h $p = 0.024$; 24 h $p = 0.028$). The integral of the liver reperfusion curve (RA) and the plateau maximum average (PM) of the flow graph showed a significant improvement (RA: IPer vs. IR: $55.8 \pm 22.7\%$ vs. $32.7 \pm 13.1\%$, $p = 0.007$ // PM: IPer vs. IR: $70.6 \pm 14.7\%$ vs. $39.9 \pm 14.9\%$, $p = 0.001$), in parallel with the favorable changes of lower limb microcirculation (RA: IPer vs. IR, $79.55 \pm 29.1\%$ vs. $60.95 \pm 21.6\%$, $p = 0.101$ // PM: IPer vs. IR, $92.21 \pm 26.9\%$ vs. $55.62 \pm 25.1\%$, $p = 0.038$).

Conclusion

Perconditioning is a feasible method, with notable practical significance in those situations when we are forced to apply long-term liver exclusions, thus the possibility of severe IR injury should be considered.

Conflict of interest : None

OP-06

Modeling and endovascular cell therapy of aortic isthmus saccular aneurysms in pigs

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Introduction

Aortic isthmus saccular aneurysms (AISA) are an evolution of traumatic ruptures of the isthmus, exposing patients to death by late rupture. Endovascular treatment requires supra aortic trunks coverage and the use of rigid stents in a mobile part of the aorta. Our hypothesis was that the endoluminal space of AISA can be filled with mesenchymal stem cells (MSC) incorporated in a matrix, maintained by a stent, allowing us to cover the origin of the supra aortic trunks (SAT) without transposing them.

Objectives

Our goals were: 1 to create a model of AISA in pigs, 2 to inject MSC incorporated in a hyaluronan gel, maintained by a stent in AISA, 3 to evaluate the healing capacity of the MSC.

Material/Patients and Methods

An AISA was created in 33 pigs, and the bone marrow was collected in 9 pigs for MSC culture. A characterization of the MSC was conducted after 3 weeks of culture. Pigs were separated into 4 groups: Group 0 AISA only; Group 1 AISA+stent; Group 2 AISA+stent+gel; Group 3 AISA+stent+gel+MSC. At postoperative day (POD) 21, the sac patency was assessed. A stent was deployed in the aorta, at the level of the aneurysm. 5.10^6 MSC, labelled with PKH26, were incorporated in the hyaluronan gel. The gel and the MSC were injected into the AISA by a catheter. At each sacrifice made on POD21+7 (n=3) or POD21+28, the patency of the aneurysm and SAT was assessed. Aortic arches were harvested for histological and immunohistochemical (IHC) studies. Quantitative variables were expressed as mean. Comparisons between two groups were made by non-parametric Mann and Whitney U test. A difference was considered significant when $p \leq 0.05$.

Results

33 AISA were created with a mortality of 9%. At POD21, all AISA were patent on angiography. In Group 0 (n=3), 2 aneurysm sacs were patent at POD21+28. In Group 1 (n=6), the patency of the aneurysmal sac was 100% at POD21+7 and 67% at POD21+28. In Group 2 (n=10) gel injection was not more lethal, the patency was 67% at POD21+7, and 67% at POD21+28. In these groups, microscopic studies showed: cellularization of the AISA, partial disruption of collagen and elastin fibres in the aneurysmal wall, and absence of endothelium.

After 3 weeks, the MSC collected in pigs were able to differentiate into osteoblasts and adipocytes. Further, the study of surface markers showed strong expression of CD90 associated with lower expression CD105, characteristic of the MSC.

In the Group 3 (n=11), only 8 AISA could be studied histologically. The patency was 33% at POD21+7 and 20% at POD21+28. We have highlighted in this group a fluorescence corresponding to the PKH26, intraluminally and in the wall. We have also highlighted the presence of intimal hyperplasia largely filling the aneurysmal sac at POD21+28 (n=5) ($p < 0.05$). In addition, in this group, a better organization of the collagen and elastin fibres was found.

Conclusion

We have developed a model of AISA remaining patent to 7 weeks. Our results show a persistent location of MSC at the aortic level. For the first time in vivo, our results demonstrate the MSC's capacity to induce an endovascular healing of the AISA. These results can lead to a non-mechanical treatment of AISA avoiding exposing young subjects to a conflict between the aorta and stents.

Conflict of interest : None