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18FDG PET SCANNER AS A SELECTION CRITERION IN LIVER TRANSPLANTATION FOR HEPATO-CARCINOMA. C. Lambrecht (1), M. Vandermeulen (1), M. Delbouille (1), J. Monard (1), A. Warmoes (1), C. Amicone (2), O. Warling (2), A. Lamproye (2), J. Delwaide (2), N. Meurisse (1), P. Honore (1), R. Hustinx (3), P. Lovinfosse (3), O. Detry (4) / [1] CHU of Liège, Belgium, Abdominal Surgery and Transplantation, [2] CHU of Liège, Belgium, Hepatogastroenterology, [3] CHU of Liège, Belgium, Nuclear Medicine, [4] Centre Hospitalier Universitaire de Liège, Liège, Belgium, Abdominal Surgery and Transplantation.

Introduction: For years, Milan criteria have been the standard for selection criteria for liver transplantation (LT) of patients with hepatocellular carcinoma (HCC). Previous retrospective studies suggested that 18 FDG positron tomography (18FDG-PET) could be an effective tool to select HCC patients beyond Milan criteria for LT if their tumour is not FDG avid. A prospective national study evaluating the potential role of 18FDG-PET in LT for HCC was initiated by BeLIAC and financed by the National Cancer Foundation for the years 2019-2022. Complete results of this study with 2-year follow-up will be available by the end of 2024.

Aim: In this report the authors aimed to present the preliminary results of this study in the CHU Liege LT centre.

Methods: This study is a prospective national study accepted by all Belgian transplant centres and BeLIAC. All patients signed an informed consent form. Between January 2019 and October 2022, 51 patients (43 males, mean age 63y) were transplanted with HCC, and 44 had PET CT before LT or neoadjuvant therapy. Amongst these 44 HCC patients with PET, 28 were Milan-in, 1 Milan-in after successful downstaging, and 13 were Milan-out (11 Milan-out Up-to-seven-in and 2 Milan-out up-to-seven-out) at time of listing. Two Milan-in patients were PET positive, and all others were PET negative. **Results:** Three patients died during the follow-up, without recurrence. No patients developed recurrence at 1- and 2-year follow-up. Three patients developed recurrence between 2- and 3-year follow-up and all were Milan-in. Importantly, no Milan-out PET negative patients developed recurrence at October 2022 follow-up.

Conclusions: Conclusions: PET FDG could be an important tool to offer potential LT opportunity to HCC patients outside Milan criteria. A longer follow-up and complete Belgian data are important before drawing further conclusions on this important matter.

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HEPATIC STELLATE CELL SINGLE CELL ATLAS REVEALS A HIGHLY SIMILAR ACTIVATION PROCESS ACROSS LIVER DISEASE AETIOLOGIES. V. Merens (1), S. Verhulst (1), L. van Grunsven (1) / [1] Vrije Universiteit Brussel (VUB), Jette, Belgium, Basic (Bio)Medical Sciences.

Introduction: Chronic liver disease (CLD) is a major cause of morbidity and mortality worldwide. The progression of CLD is characterized by excessive extracellular matrix deposition, thereby disrupting hepatic architecture and function. Hepatic stellate cells (HSCs) are the major source of this excessive collagen deposition in all underlying aetiologies of CLD. Upon liver injury, HSCs lose their ability to store vitamin A, differentiate towards a myofibroblast phenotype and become migratory, inflammatory, proliferative and fibrogenic. Several animal models such as common bile duct ligation, CCl4 intoxication and Western diets are used to model the different aetiologies of CLD, but to date, a comprehensive comparison between the mechanisms of activation of HSCs in different aetiologies has not been made.

Aim: In this study, we aimed to identify transcription factors responsible for the differentiation of HSCs towards myofibroblasts and to determine whether the transcriptional program differs between mouse liver injury models and human liver disease.

Methods: Mesenchymal cells, based on Pdgfrb expression, were selected from 7 single-cell RNA-Sequencing datasets describing 10 distinct mouse liver injuries. HSCs were selected from the merged mesenchymal atlas based on Lrat and Reln expression and were subjected to batch-effect removal, multidimensional reduction (UMAP) and Louvain clustering. The same approach was used to construct a human HSC atlas from 3 single-cell RNA-Sequencing datasets describing both healthy and cirrhotic livers. Transcription factor (TF) activity was calculated using pySCENIC. Pseudotime was calculated by trajectory inference using Slingshot on the TF activity matrix.

Results: First, a liver mesenchymal cell atlas was constructed to facilitate the identification of HSCs in all studies. HSCs could be clearly divided into 3 categories: Quiescent HSCs, which show a high expression of Lrat, Reln and Rgs5, Initiatory HSCs representing the first events of HSC activation characterized by high expression of YAP downstream targets Ankrd1 and Thbs1 and chemoattractant cytokines Ccl2 and Ccl7, while fully activated myofibroblasts show a high expression of collagens 1a1, 1a2, 3a1, 5a2 as well as classic HSC activation markers Lox11 and Mmp2. HSCs isolated from mice without liver injury were primarily identified as quiescent HSCs, while HSCs isolated after acute and chronic liver injury were predominantly identified as initiatory HSCs and myofibroblasts respectively. Surprisingly, there are proportionally more quiescent HSCs after chronic liver injury than after acute liver injury, suggesting that HSCs