

LIVER TRANSPLANTATION (LTx) USING NON-HEART-BEATING DONORS (NHBD) : BELGIAN EXPERIENCE. D. Monbaliu, W. Coosemans, R. Troisi, B. de Hemptinne, J. Lerut, R. Reding, J. de Ville de Goyet, A. De Roover, P. Honoré, O. Detry, V. Donckier, M. Gelin, D. Ysebaert, R. Aerts, J. Pirenne. In name of all Belgian LTx centers and the Belgian Transplantation Society.

Mortality on Liver Transplantation (LTx) waiting lists has increased dramatically. Using Non-Heart-Beating Donors (NHBD) {Death by cardiac arrest} is an accepted practice to enlarge the kidney pool. Until recently, NHBD were not considered suitable for LTx, because NHBD-livers are exposed to warm ischemia prior to procurement and this may cause a prohibiting risk of Primary Graft Non Function (PNF) and biliary strictures postTx. However, 2 factors {(i) recent experimental/clinical evidence that NHBD-LTx is feasible when the period of warm ischemia is short ; and (ii) the "pressure" of the waiting list} led Belgian LTx centers to revisit the option of NHBD-LTx.

Aim/Methods : To characterize the results of NHBD-LTx in Belgium, a survey sponsored by the *Belgian Transplantation Society* was sent to all Belgian LTx centers.

Results : 15 livers originating from NHBD were transplanted in Belgium between January 2003 and November 2005. All were "controlled" Maastricht category III NHBD (e.g. controlled stop medical treatment and awaiting cardiac arrest). Donor age was 50 yo (28-64) ; 9 males/6 females. Irreversible brain damage was due to intracranial bleeding (5), anoxia (2), trauma (2) and other (6). Time interval from stop therapy/ventilation withdrawal to cardiac arrest was 20' (2-45') and from cardiac arrest to liver cold perfusion, 9.1' (2-25'), including an obligatory "no-touch" period of 2'-to-10'. Mean recipient age was 54 (10-69) ; 10males/5females. Indications for LTx were HCC (9), postethyl (2), other (3) ; 66% were child A or B (T3 score) ; 33% were Child C (T2 score). Cold ischemia time was 7h22' (4.22-11.59). No PNF requiring retransplantation was observed. However, postTx peak transaminase (a reflection of ischemic injury) was high 1694IU/L (168-6319). Hospital stay was 30 days (13-98). Biliary complications occurred in 5 patients (33%) requiring reTx in 2 (13%), endoscopic treatment in 2 (13%) and surgical treatment in 1 (7%). Patient and graft survival are 80% and 65%, respectively (follow-up : 15days-22mths).

Conclusions : This Belgian survey shows that patient survival after NHBD-LTx is acceptable and in particular no PNF is seen. However, NHBD-liver grafts suffer a high rate of ischemic injury and biliary complications and therefore should be used very carefully (no additional donor risk factors, low risk recipient, short cold/warm ischemia). *In the future*, modulating ischemic injury (biologically and/or *via* machine perfusion) and assessing graft viability prior to Tx will be pivotal to safely expand the use of NHBD-LTx.

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RELATIONS BETWEEN MUTATIONS OF CYSTIC FIBROSIS GENE AND ALCOHOLIC PANCREATITIS. N. de Suray (1), C. Ars (1), O. Descamps (1), Y. Daumerie (2), A.M. Vandamme (2), P. Jopart (1), J.-M. Ghilain (1). (1) Department of Internal Medicine, Centre Hospitalier Jolimont-Lobbes, Haine Saint-Paul ; (2) Centre de Recherche Médicale de Jolimont, Haine Saint-Paul.

Background : The observation that only few alcoholics develop pancreatic disease has led to search for modifying factors in alcoholic pancreatitis. Amongst these factors, mutations and polymorphisms of the cystic fibrosis transmembrane conductance regulator gene (CFTR) are good potential candidates as they have been strongly associated, at the heterozygous state, with idiopathic or hereditary pancreatitis. In the present study, the aim was to determine whether alcoholic pancreatitis is also strongly associated with CFTR gene mutations and polymorphisms.

Methods : The study was designed to assess an odd ratio superior to 5 for association with the 2 most common mutations of the CFTR gene in Belgium (DF508 and G542X = 90% of cystic fibrosis). Based on the general prevalence of heterozygotes in Belgium (1/30), the minimum size required to detect such OR was 40, for a 2-sided significance level of 0.05 and a statistical power of 80%. We also examined the length variations of the polythymidine sequence (5T, 7T, and 9T alleles) and the TG dinucleotide repeat located in the splice acceptor of exon 9 of the CFTR and known to affect (5T and (TA)12 or (TA)13)the levels of normal CFTR mRNA.

Results : We collected 44 patients with alcoholic acute and/or chronic pancreatitis and compared them with 169 individuals from the same geographical area as well as with 30 patients with alcoholic liver disease (cirrhosis). The DF508 mutation at the heterozygous state was found in 2 patients (4.6%) with alcoholic pancreatitis, and in 2 patients (1.8%) from the general population. The G542X mutation was found in 1 patient (0.6%) from the general population. The odd ratio was 2.63 [95% CI : 0.42 – 16.28 ; p = 0.28]. No mutation was found in 30 cirrhotic patients (NS compared to pancreatitis group or to general population). Preliminary analysis suggested also that there was no significant difference in the prevalence of 5T, (TA)12 or (TA)13 alleles between the pancreatitis group and the general population (20 patients examined in each group).

Conclusions : It is unlikely that a strong association (OR > 5) exists between alcoholic pancreatitis and the most common and relevant mutations and polymorphisms of the CFTR gene. The clinical impact of CFTR gene mutations and polymorphisms may not be as high as in idiopathic or hereditary pancreatitis.