95 OKT3 TREATMENT OF STEROID RESISTANT REJECTION IN PEDIATRIC LIVER TRANSPLANT RECIPIENTS. McDiarmid S, Busuttil R, Terasaki P, Vargas J, Ament M. UCLA Medical Center, Los Angeles, CA.

The success of OKT3 in reversing rejection in a pediatric liver transplantation series has not been previously reported. Of a total of 187 consecutive liver grafts in 149 pediatric patients transplanted at UCLA Medical Center, 59 episodes of steroid resistant, biopsy proven, rejection (32% of grafts), were treated with OKT3. OKT3 was given over 10-14 days in a dose of 2.5 mg/day IV (weight < 30kg) or 5 mg/day IV (weight ≥ 30kg). Cyclosporin, azathioprine and low dose steroids were used concurrently. The OKT3 dose was increased if CD3%+ cells in the peripheral blood increased to between 5 - 10%. Outcome was defined as full (normal liver function), partial (reduction in bilirubin and transaminases by 50%), or failure (no improvement). RESULTS: After OKT3 treatment liver function was normal in 40%; improved in 35%; and unchanged in 25%. Of 21 partial responses, 12 episodes eventually resolved to yield an overall full response rate of 59%. The graft survival of 12 grafts failing 15 episodes of OKT3 treatment was 17%. CD3% ≥ 5% occurred during 61% of OKT3 treatment courses. 6 grafts were retreated with OKT3 and in all CD3% could not be maintained < 5%. 5 of 6 grafts requiring repeat OKT3 failed treatment and required re-transplantation. High titre OKT3 antibodies (by ELISA) adversely effected the use of OKT3 in 2 patients. Patients treated with OKT3 after failing more than 2 preceeding steroid bolusses had a significantly increased chance of graft loss (57%, p = 0.01). Graft survival (70%) and patient survival (85%) in OKT3 treated patients was no different from graft and patient survival for all other pediatric liver recipients (64% and 77% respectively). CONCLUSIONS: OKT3 successfully rescued grafts with steroid resistant rejection, and was most effective when given early. Our group of pediatric patients appeared less responsive to OKT3 compared to other reported series combining all age groups, possibly due to a more vigorous immune response in the child. The recommended dose for OKT3 in pediatric patients allowed for the re-emergence of CD3+ cells in the majority. Higher doses of OKT3 may improve the efficacy of OKT3 in pediatric patients.

96 PREDIABETIC INDICES IN CHILDREN WITH GLUTEN SENSITIVITY ENTEROPATHY (GSE)
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This is a prospective study of insulin secretion and immunologic changes in a group of children with GSE on gluten free diet. The purpose of the study is to investigate the possible association of disturbances of insulin secretion with the incidence of insulin dependent diabetes mellitus (IDDM) which is known to be high in GSE. Thirty patients,4 to 18 years old, were examined. They underwent IV glucose tolerance test during which glucose disappearance rate (K) and first phase insulin response ($I_{1'+3'}$) were measured. Typing for HLA A,B,C and DR antigens was performed and sera were analysed for cytoplasmic islet cell antibodies (ICA) on two occasions. Pancreatic isoamylase (PIA) was measured as an index of exocrine pancreatic function. The same procedures were performed on 30 healthy children. The results are as follows: a) In 24% of celiac children $I_{1'+3'}$ and K rate were decreased. b) There was significant correlation between the two parameters ($p<0,01$). c) The incidence of HLA $B_8$ and $DR_3$ was higher in GSE (33% and 60%, respectively) than in healthy individuals (6,6% and 20%, respectively). d) All patients were found to be ICA negative at the time of the study. e) There was no correlation between parameters of endocrine ($I_{1'+3'}$, K rate) and exocrine pancreatic function (PIA).
Conclusions: Although there was no evidence of islet cell immune destruction at the time of the study, a decline of first phase insulin secretion was indeed documented. This decline may be an expression of a prediabetic phase. HLA $B_8$ and $DR_3$ which are detected in celiac patients may indicate a possible common pathogenetic mechanism between GSE and IDDM.

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