PTU-115 HEPATITIS B IN PREGNANCY: WHAT HAPPENS TO THE INFANTS?

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Introduction Antenatal screening for Hepatitis B (HBV) has been offered to all pregnant women in the UK since 2000. Immunoprophylaxis of infants is essential to reduce the risk of vertical transmission. It is recommended that HBV vaccination (4 serial doses) be given to all infants born to HBV positive mothers. In addition, Hepatitis B immunoglobulin (HBIG) is recommended for infants of Hepatitis B e antigen (HBeAg) positive mothers. Infants should have post-vaccination testing between 9–18 months. Hepatitis B surface antigen (HBsAg) negative infants with anti-HBs levels > 10iu/ml need no further management. If anti-HBs is < 10iu/ml infants should receive a second vaccination series. Our aim was to evaluate the management of infants born to HBV positive mothers.

Methods All HBV positive pregnant women seen in our hospital between January 2008 and November 2011 were identified from an obstetric database. We examined if the infants received the recommended vaccinations, HBIG and post-vaccination testing.

Results From a total of 99 pregnancies data was available for 76 infants. All 15 infants born to HBeAg positive mothers were given HBIG at birth. 7 mothers had a HBV DNA > 10^7 IU/ml. 2 were treated with antiviral therapy during pregnancy. 58 (76.3%) infants received a full vaccination course. Only 35 (54.7%) of the 64 infants who should have had their post-vaccination status checked to date have had this completed. 34 (97.1%) had an adequate response to vaccination. One infant (who received 3 vaccinations) is HBsAg positive with a viral load of 5.4×10^8 iu/ml. The mother was HBeAg negative with a viral load of 1.7×10^8 iu/ml.

Conclusion HBIG was administered appropriately to the infants at highest risk of vertical transmission of HBV. However, completion of the 4 dose HBV vaccination in infants was suboptimal postpartum and post- vaccination testing was inadequate. Efforts to improve this are now in place and include: prospective data collection to improve quality of data; development of a central reminder system to advise family doctors 2 weeks before each vaccination dose is due and ensure this is completed; and introduction of dry blood spot testing of the infants to improve the acceptability of testing.

Disclosure of Interest None Declared

PTU-116 TRANSPLANTATION FOR ALD: LESSONS FROM THE EXPLANT

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Introduction Alcoholic liver disease (ALD) remains one of the commonest indications for liver transplantation in Europe. The histological features of ALD vary, depending on extent and stage of injury, No features are reliably pathognomonic of ALD. We describe the histological spectrum of explants of well characterised cohort of patients undergoing transplantation for ALD.

Methods Consecutive explants (n = 84) of patients transplanted for ALD in our institution between 2002 and 2011 were selected for retrospective histological assessment. Explants were scored blinded by two pathologists using a predetermined pro-forma. Histological assessment including the presence and degree of cirrhosis, steatosis, inflammation, inclusions, siderosis and neoplastic changes were scored semi-quantitatively.

Results Median age was 54 and the majority (70%) were male. All patients had a long history of alcohol excess but reported abstinence for at least 6 months by transplantation. The aetiology was ALD (n = 80) and mixed ALD/HCV (n = 4). The majority (n = 83) had a mixed or macronodular cirrhosis with evidence of re-modelling in a significant number; one had pre-septal cirrhosis. Alpha-1 antitrypsin inclusion bodies were seen in 9 (10.7%); only 4 of these had serum A1AT levels below normal. Parenchymal siderosis was present in 39 (46.4%); in 19 (22.6%) this was grade 3–4. Amongst these, only single mutations of the HFE gene were identified. Induced cell change was seen in 67 (79.8%) and 47 (56%) had the "abstinent cell" phenotype. While 46 (54.8%) had Mallory-denk bodies (MDB), 22 (26.1% of total) patients had both "abstinent cells" and MDB. Ballooning (n = 45, 53.6%) and steatosis (n = 31, 36.9%) were also seen. HCC was present in 14 (16.7%), with dysplastic nodules in 15 (17.9%), small-cell change in 20 (23.8%) and large-cell change 50 (59.5%). Phlebosclerosis and parenchymal extinction were universal findings.

Conclusion We describe a wide spectrum of histological features in a large cohort transplanted for end-stage ALD. We demonstrate that despite abstinence, over half have residual MDB and ballooning. Conversely, over half had the recently described "abstinent cell" phenotype. Therefore, the presence of MDB should not be used as evidence of continued alcohol consumption; the presence of induced or abstinent cells correlates more strongly with reported abstinence.

Disclosure of Interest None Declared

PTU-117 IMAGING HEPATIC NEUTROPHIL MIGRATION WITH INDIUM-111-(111IN)-RADIOLABELLED LEUCOCYTES: A NOVEL NON-INVASIVE DIAGNOSTIC TEST FOR SEVERE ALCOHOLIC HEPATITIS

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Introduction Clinical diagnosis of severe alcoholic hepatitis (SAH) can be unreliable and liver biopsy often problematic. Neutrophil infiltration is a key histological feature of SAH and predicts corticosteroid (CS) response (1). We assessed the use of radiolabelled leucocyte scintigraphy to diagnose SAH through *in vivo* imaging of hepatic neutrophil migration. Liver signals at ^{111}In-leucocyte scintigraphy portray margination at 30min post injection (PI) and destruction at 24h PI, and are normally matched. We hypothesised that both these functions would be impaired in SAH and liver activity would increase between 30min and 24h due to neutrophil migration.

Methods 16 patients with SAH (Discriminant Function 54.8 ± 16.3, 14 biopsied), 14 with inactive alcoholic cirrhosis (Child-Pugh score 5.4 ± 0.9) and 11 controls were recruited. Abdominal gamma camera images were obtained 30min and 24h after IV injection of autologous ^{111}In-labelled leucocytes and change in liver activity expressed as a 24 h:30 min ratio. Biopsies in SAH were stained with the granulocyte marker CD15 and parenchymal neutrophils quantified across 10 high power fields.

Results Liver activity significantly increased in SAH but was static or fell in cirrhotic and normal control groups (24h:30min liver ratio 2.18 (IQR 1.62) versus 0.97 (0.28) and 0.78 (0.15) respectively, $p < 0.001$). Figure 1 shows example gamma camera images. Liver activity ratios in SAH correlated with histological neutrophil infiltration ($\rho = 0.571$, $p = 0.041$) and microautoradiography demonstrated intact intrahepatic radiolabelled leucocytes as the likely source of 24h liver activity. Prominent 30min lung activity was a consistent finding in SAH, providing *in vivo* evidence of neutrophil