

derives a population of Tregs that is stable and functionally superior compared to freshly isolated Tregs.

Disclosure of Interest

None Declared.

PTU-123 USE OF RITUXIMAB IN RESISTANT AUTOIMMUNE HEPATITIS – BIRMINGHAM EXPERIENCE

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Introduction Autoimmune hepatitis (AIH) is due to breakdown in immunological self-tolerance. Sustained remission in AIH is crucial to prevent the progression to end stage liver disease.¹ Around 9% of the patients are refractory/intolerant to the standard therapy with prednisolone (Pred) ± azathioprine (AZA). High levels of immunoglobulin are typical of AIH and plasma cells are frequently observed in liver histology. Rituximab is an anti-CD20 monoclonal antibody that depletes B-cells and has been used to treat other autoimmune conditions such as systemic lupus erythematosus. However, little has been reported on the role of B cells depletion and its outcome in AIH.

The aim of the study was to evaluate the safety and efficacy of rituximab in the treatment of refractory AIH.

Methods A retrospective case note review of well-defined and biopsy proven type-1 AIH (simplified scoring >6). 5 patients out of 200 who were intolerant/refractory to standard therapy were given Rituximab and the responses were followed up for 72 weeks.

Efficacy was measured by biochemical and immunological parameters (bilirubin, AST, ALT and Immunoglobulin every 12 weeks). The dose of Prednisolone as well as UKELD/MELD score pre and post treatment was also evaluated.

Results All 5 patients were female and mean age was 45 (range 35–66 yrs). The rituximab dose used was 1000 mg and the total number of doses received varied between 2 and 4 (Mean 3.2). Three patients had other concomitant autoimmune conditions (endocrine, rheumatological and renal related autoimmune diseases). The mean dose of prednisolone used pre-rituximab was 19mg (±SD 12.57) and this was reduced to 12.5mg (± SD 5.0) post treatment (statistically not significant=NS). There was a slight improvement of IgG pre and post Rituximab treatment (NS), with no improvement in UKELD score. There was an improvement in biochemical profile but this was not statistically significant throughout the observation period. All five patients were alive and rituximab was well tolerated without any serious adverse events.

Conclusion Rituximab is well-tolerated and safe to use in resistant AIH. It can cause some biochemical and immunological improvement. Current evidence for its use in AIH patients is not well proven. The study numbers are too small to detect the actual outcome of the therapy. A multicenter larger cohort prospective study with longitudinal immunological, biochemical and histological profile assessment is warranted to assess its efficacy in resistant AIH patients.

REFERENCE

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Disclosure of Interest None Declared.

PTU-124 AN EXTERNAL VALIDATION OF THE HEPATOMA ARTERIAL-EMBOLISATION PROGNOSIS (HAP) SCORE: THE LIVERPOOL EXPERIENCE

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Introduction Most Hepatocellular Carcinomas (HCCs) have palliative treatment. Trans-arterial embolisation (TAE) or chemoembolisation (TACE) are used with variable results. The HAP score was recently described to determine patients likely to benefit from TAE or TACE. We report our experience with TAE and TACE to assess whether the HAP score was valid for our cohort of patients.

Methods Retrospective review of cases given TAE or TACE in Liverpool, UK (2006–2013). HAP score [1 point each for albumin < 36 g/dl, AFP > 400 ng/ml, Bilirubin > 17 µmol/l, tumour diameter > 7cm. HAP A = 0 points, B = 1, C = 2, D >2]. Outcome recorded according to HAP score.

Results 137 patients identified having received TAE/TACE with full data to complete HAP score. Mean age 69; 116 (84.7%) male. 78.8% AUH, 21.2% RLUH. HAP score A: 44 (32.1%); B: 40 (29.2%); C: 32 (23.4%); D: 21 (15.3%). Overall median survival 492 days (16 months). Median survival by HAP score, A: 492 days; B: 839 days; C 478 days; D 309 days. Log rank p < 0.001. Survival at 1 year: A 62.8%; B 75%; C59.4%, D 28.6%. Survival at 2 years: A 29.5%, B 52.5%, C 37.5%, D 14.3%. HAP D patients had lower median survival (309 vs. 563 days; p < 0.001) and 1 and 2 year survival (28.6 vs. 65.5%; p = 0.001 and 14.3 vs 39.7%; p = 0.021).

Conclusion Patients with HAP score D due TACE have a relatively poor outcome in this external validation group. This should be considered when planning treatment or further trials.

REFERENCE

Kadalayil et al. *Annals of Oncology* 2013

Disclosure of Interest None Declared.

PTU-125 BIOCHEMICAL PATTERNS OF PRESENTATION IN PRIMARY SCLEROSING CHOLANGITIS: YOUNGER AGE AT ONSET IS ASSOCIATED WITH A LOWER ALP/AST RATIO

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Introduction Autoimmune sclerosing cholangitis is the paediatric term applied to children presenting with features of autoimmune hepatitis and sclerosing cholangitis. We hypothesised that if this inflammatory phenotype was a continuum, young adults with primary sclerosing cholangitis (PSC) would also have a more inflammatory presentation.

Methods We undertook a retrospective case-note review of our patients with an established diagnosis of PSC presenting between 2003–2013 (n = 116). Clinical characteristics and laboratory

parameters were collected, and differences in disease phenotype correlated with age at presentation (SPSSv21).

Results The median age of disease presentation in our cohort was 44 years (IQR:25–56). Although there was no significant correlation between patient age and mode of disease presentation, younger age was more commonly associated with lower baseline serum ALP (Spearman's $\rho = 0.239$; $P = 0.011$). Patient age negatively correlated with ALP:AST ratio ($\rho = 0.252$; $P = 0.008$); however, there was no correlation with serum AST, bilirubin, albumin, platelet count, INR, IgG titre or ANA/ASMA status. Using quartile cut-points in order to compare extremes of age, individuals presenting below the age of 25 (Q4; 7.6; 3.2–13.0) ($P = 0.023$). Age <25 at disease presentation was more often associated with an ALP:AST ratio <1.5 (11/25 [44%] vs. 4/25; [16%], $P = 0.017$). There were no significant differences in IBD phenotype, number of patients meeting transplantation or median time to transplant.

Conclusion Younger patients more commonly have a lower ALP/AST ratio at disease presentation, and may indicate a more 'inflammatory' PSC phenotype.

Disclosure of Interest None Declared.

PTU-126 MORTALITY ASSOCIATED WITH HEPATIC ENCEPHALOPATHY IN PATIENTS WITH SEVERE LIVER DISEASE

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Introduction Despite hepatic encephalopathy (HE) being a common complication of severe liver disease, there are comparatively few data describing the epidemiology of the condition. The aim was to characterise mortality risk for patients with HE.

Methods The study was conducted using data from the Clinical Practice Research Datalink (CPRD). Patients with a record of first diagnosis of liver disease were identified between 1998 and 2012. Two Cox Proportional Hazard models were generated. The first followed the whole liver disease cohort with HE modelled as a binary time-dependent variable in quarterly segments. The second compared patients identified with HE to non-HE controls matched at a ratio of 1:1 on age, gender, year of first diagnosis of liver disease, liver disease duration and Baveno IV status.

Results 17,030 patients were identified with a diagnosis of liver disease, of whom 551 (3.2%) had a HE diagnosis. Of patients identified with HE, 304 of 551 (55.2%) died during the follow-up period, compared with 6,693 of 16,479 (40.6%) of those without HE ($p < 0.001$). In the Cox Proportional Hazard model, the hazard ratio of HE modelled as a time-dependent variable was 1.43 (95% CI 1.20–1.70; $p < 0.001$) (Table 1). 389 of the 551 HE patients (70.6%) could be matched to non-HE controls. 226 HE patients (58.1%) died during the follow up period compared with 126 (32.4%) controls. The hazard ratio for time to death was 2.28 (95% CI 1.82–2.87; $p < 0.001$).

Conclusion HE substantially increased mortality risk in patients with chronic liver disease.

Disclosure of Interest C. Morgan Consultant for: Norgine; S. Jenkins-Jones Consultant for: Norgine; A. Radwan Employee of: Norgine; P. Conway Employee of: Norgine; C. Currie Consultant for: Norgine.

PTU-127 RESOURCE USE ASSOCIATED WITH HEPATIC ENCEPHALOPATHY IN PATIENTS WITH SEVERE LIVER DISEASE

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Introduction Overt hepatic encephalopathy (HE) is associated with frequent hospitalisations which are expensive to manage and result in poor quality of life. The aim was to estimate the resource use associated with HE and hospitalisation in the UK.

Methods The Clinical Practice Research Datalink (CPRD) with linked hospital data from Health Episode Statistics (HES) was used to identify patients with a first diagnosis of liver disease between 1998 and 2012 and examine their all-cause hospitalisations. HE patients were matched to controls at a ratio of 1:1 by age, gender, year of diagnosis, duration and severity of liver disease. Hospital admission data (frequency and length of stay) were characterised from HES. Admissions associated with the index diagnosis of HE were excluded.

Results 17,030 patients were identified with an incident diagnosis of liver disease, of whom 551 (3.2%) had a recorded diagnosis of HE. 389 patients (70.6%) could be matched to non-HE controls. Total number of primarily liver-related admissions was greater in the HE group with a crude admission ratio of 3.588 (95% CI 3.085–4.173; $p < 0.001$). In the HE group, a significantly greater proportion of liver-related admissions were through AandE (62.1% vs. 50.0%, $p < 0.001$) and mean length of stay was 8.0 days (sd 11.6) vs 6.8 days (sd 9.5) ($p = 0.148$) in the non-HE group. Following first HE event, patients had

Abstract PTU-126 Table 1 Adjusted hazard ratios associated with hepatic encephalopathy for patients with severe liver disease

	HR	(95% CI)	P
HE defined as time dependent covariate			
HE status (HE +: HE -)	1.430	(1.204–1.699)	0.000
Age	1.040	(1.038–1.042)	0.000
Gender (female : male)	0.828	(0.786–0.871)	0.000
Baveno status (-1)			
2	0.827	(0.702–0.975)	0.023
3	1.566	(1.469–1.669)	0.000
4	1.278	(1.197–1.365)	0.000
Charlson index	1.127	(1.112–1.143)	0.000
Smoking status (never smoked)			
Ex-smoker	1.010	(0.948–1.075)	0.767
Current smoker	1.159	(1.089–1.233)	0.000
HE status compared to matched controls			
HE status (HE +: HE -)	2.281	(1.816–2.866)	0.000
Age	1.018	(1.006–1.029)	0.002
Gender (female : male)	0.804	(0.634–1.021)	0.073
Baveno status (-1)			
2	0.710	(0.365–1.381)	0.313
3	1.139	(0.853–1.52)	0.377
4	0.823	(0.614–1.104)	0.194
Charlson index	1.067	(1.008–1.13)	0.026
Smoking status (never smoked)			
Ex-smoker	0.933	(0.708–1.228)	0.620
Current smoker	0.871	(0.671–1.132)	0.302