

(VLCD) help in weight reduction and are known to shrink the liver. Our aim was to assess the role of ARFI in assessing and monitoring the change in liver architecture in a cohort of morbidly obese patients in response to VLCD.

Methods A cohort of non-diabetic morbidly obese patients at risk for NASH was selected for this study (clinical trial no: NCT01950052). Liver volume was estimated with the help of a standardised ultrasound protocol while liver fibrosis was analysed with ARFI. After randomisation, a very low calorie diet (800 kcal) was given to one group while the rest were controls. Four weeks later, ARFI was repeated and all patients underwent a laparoscopic roux-en-y gastric bypass. A liver biopsy was taken during surgery from the same liver segment as the ARFI measurements. The liver histology was evaluated according to the NASH Clinical Research Network Scoring System by two blinded pathologists. Steatosis, fibrosis and NAFLD activity scores were correlated with ARFI scores.

Results Liver volume shrank by 21.5% in the diet arm (n = 10) compared to 2% (p < 0.05) in the control arm (n = 14) in 4 weeks. The ARFI scores were similar in the diet and control group [median 2.92 (1.1–3.8) m/s vs. 3.22 (1.54–3.65) m/s, p = 0.7], p = 0.7] at recruitment and at the time of the biopsy 4 weeks later [2.16 (1.19–3.68) m/s vs. 2.83 (1.5–3.48) m/s, p = 0.3]. ARFI demonstrated a drop in values in the diet group (p = 0.1) but this was not significant. Similarly, liver biopsy at surgery confirmed a trend of lower levels of steatosis in the diet group (27 vs. 42%, p = 0.12). The ARFI scores did not correlate with the steatosis grade (p = 0.8), or NAFLD score (p = 0.48).

Conclusion Low calorie diets shrink liver volumes but ARFI could not detect any change in liver stiffness. ARFI does not appear to correlate with liver steatosis and may not be ideally suited for short term monitoring of successful treatment of NASH. However its role in long term monitoring needs further evaluation.

Disclosure of Interest None Declared.

PTH-089 HEPATITIS B MONOTHERAPY WITH TENOFOVIR OR ENTACAVIR: A UK SINGLE CENTRE EXPERIENCE

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Introduction Tenofovir (TDF) and Entacavir (ETV) are potent oral antiviral medication for chronic hepatitis B¹. Our aim was to record effectiveness of these 2 widely used antivirals in achieving viral suppression, HBeAg seroconversion and HBsAg loss after an initial 12 months of treatment and to further assess long term results (from June 2007 to April 2013) in chronic hepatitis B patients in a single tertiary referral centre.

Methods We retrospectively collected data from hospital record from June 2007 to April 2013. We included chronic hepatitis B patients with high viral load (>2000 IU/ml), treatment naive and treatment experienced and who were on treatment for at least 12 months with either on tenofovir or entacavir. Treatment experienced patients were those who were switched from other antivirals to tenofovir or entacavir with high viral load.

Results 61 patients were treated with TDF monotherapy for a median of 29 months, 25 (41%) were HBeAg positive and 36 (59%) were HBeAg negative. In the HBsAg positive group 17 (68%) achieved virological response in 12 months time while 22 (88%) had achieved it on longer term treatment. 2 (8%) got HBeAg seroconversion within 12 months whilst 4 (16%)

seroconverted on longer term treatment. In the HBeAg negative group 29 (81%) achieved virological response after 12 months treatment whilst 34 (94%) achieved virological response on longer term treatment.

33 patients were treated with ETV monotherapy for a median of 38 months, 14 (42%) were HBeAg positive and 19 (58%) were HBeAg negative. In the HBsAg positive group 7 (50%) achieved virological response after 12 months treatment whilst all 14 (100%) achieved virological response on longer term treatment. 1 (7%) got HBeAg seroconversion in 12 months whilst 4 (29%) seroconverted on longer term treatment. In the HBeAg negative group 15 (79%) achieved virological response after 12 months time whilst all 19 (100%) had achieved it on longer term treatment.

None of the patients on either on ETV or TDF lost HBsAg.

Conclusion ETV and TDF are potent nucleos (t)ide analogues as first-line monotherapies for chronic hepatitis B. Due to the fact that ETV was licensed before TDF treatment durations are longer with this agent which is likely to explain the numerically superior long term results with ETV. It will require more patients and longer duration of treatment to allow a meaningful comparison of the two agents and to determine if HBsAg loss as described in the registration trials can be replicated in clinical practice.

REFERENCE

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PTH-090 NATURAL HISTORY OF NAFLD: A STUDY OF 108 PATIENTS WITH PAIRED LIVER BIOPSIES

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Introduction Non-alcoholic fatty liver disease (NAFLD) is the most common cause of liver disease in many countries. There remains considerable uncertainty about natural history and prognosis. Few studies, totalling <400 patients, have examined the evolution of steatosis/steatohepatitis and fibrosis of NAFLD in patients with paired biopsies. In general it is thought that fibrosis progression in patients with "NAFL" (steatosis +/- mild inflammation) is uncommon, whereas non-alcoholic steatohepatitis (NASH; steatosis + hepatocyte ballooning and inflammation) more frequently progresses. Our aim was to assess the histological severity of NAFLD in a cohort with serial liver biopsy data and to determine clinical factors that predict fibrosis progression.

Methods Patients with 2 liver biopsies >1 year apart were identified from the Newcastle Hospitals NAFLD clinic. Clinical and laboratory data were collected from the time of liver biopsy.

Results 108 patients (mean age 48 ± 12 years; 66% male; 48% diabetic) were identified with ≥2 liver biopsies (median interval 6.6 years, range 1.3–22.6). 81 (75%) patients had NASH and 27 patients with NAFL. Overall 45 (42%) patients had progression of fibrosis, 43 (40%) had no change in fibrosis, while 20 (18%) had fibrosis regression. The mean rate of fibrosis was 0.08 ± 0.25 stages/year overall, increasing to 0.29 ± 0.24 stages/year in

progressors. Importantly, no significant difference in the proportion exhibiting fibrosis progression was found between those with NAFL or NASH at index biopsy (10/27 (37%) vs. 36/83 (43%) $p = 0.65$). 12/27 (44%) with NAFL at baseline progressed to NASH at follow-up biopsy, whereas 6/75 (8%) with NASH regressed to NAFL. Weight change was a significant factor associated with inter-biopsy change in disease activity measured by NAFLD activity score ($r_s=0.23$ $p = 0.026$). Of 10 patients with NAFL who had fibrosis progression, 3 progressed by 1 stage, 5 by 2 stages and 2 by 3 stages; all had NASH on the follow-up biopsy. Of concern, 6 of 27 (22%) patients with baseline NAFL had reached stage 3 fibrosis at the follow up biopsy, but none were cirrhotic. Among the patients with NAFL, 80% of those who had fibrosis progression were diabetic at the time of follow-up liver biopsy compared with 25% of non-progressors ($p = 0.005$). The FIB-4 score was the only significant baseline factor that predicted fibrosis progression (OR 2.1 [95% CI: 1.1–3.9], $p = 0.02$). However, the AUROC was only 0.63 ($p = 0.04$).

Conclusion Contrary to current dogma, this study suggests that NAFL is not entirely benign and has the potential to progress to NASH and clinically significant fibrosis, particularly if patients develop diabetes.

Disclosure of Interest None Declared.

PTH-091 TESTING FOR HEPATITIS C IN HIGH RISK IMMIGRANTS – FINDINGS FROM A SINGLE PRACTICE WITH A LARGE IMMIGRANT POPULATION

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Introduction Chronic Hepatitis C virus (HCV) is a major cause of liver cirrhosis with a worldwide prevalence of 3%. The UK prevalence is between 0.4–1.0% however pockets of higher prevalence will exist in areas with large immigrant populations. Chronic liver disease in HCV infected patients is costly to the NHS. The Hepatitis C Action plan recommends all ethnic minorities from countries where HCV is endemic be offered screening; however testing of this group remains haphazard. Our aims were to determine the number of high risk individuals tested for HCV by interrogation of a Primary Care database in a single GP surgery located in an area with a large immigrant population. Secondary aims include establishing the reason for testing, prevalence of HCV in the tested population and treatment outcomes.

Methods We used 4 search terms in the primary care database SystmOne to identify our target population: age (>18), ethnic code, place of birth and language spoken. We then applied Read Codes pertaining to HCV to this population to determine the number already tested. The electronic medical records of all individuals tested positive for HCV were reviewed to answer secondary aims.

Results There were 4256 individuals registered age >18. 75% (3210) qualified as the target population, 18% (718) were excluded because of lack of demographic data, 7% (328) originated from low risk countries. We identified 16 read codes pertaining to HCV and these generated 247 ‘hits’ and identified that 6% of the target population had been tested for HCV (115M, 79F). Indications for testing were: isolated raised ALT/bilirubin/ALP/AST 45%, contact testing 12%, mixed raised LFTs

9%, generally unwell 9%, screening pre DMARD therapy 7%, illicit drug use 7%, patient request 3%, other indication 3%, indication unknown 2%, medical intervention overseas 1%, other abnormal bloods 1%. Proactive screening took place in 1%. The prevalence of HCV in the tested population was 7.7% (15/194 M9, F6). 73% (11/15) received treatment, 9/11 (82%) achieved an SVR, 1/11 (9%) was termed ‘responder-relapser’ but achieved SVR on re-treatment, 1/11 (9%) had no response to treatment and the course terminated prematurely, with subsequent spontaneous clearance of the virus. 4/15 had not received treatment: 2 patients were considered high risk for treatment in view of co-morbidities, 1 failed to attend appointments and 1 was recently diagnosed.

Conclusion This study confirms that testing is reactive rather than proactive highlighting the need for a screening programme dedicated to high risk populations. GP work load, prioritisation of chronic diseases forming part of QOF and poor understanding of HCV all exist as possible barriers to screening.

Disclosure of Interest None Declared.

Pancreas

PTH-092 INVESTIGATING THE ROLE OF PHYSICAL ACTIVITY IN PANCREATIC CANCER – THE AGE AT WHICH THIS IS MEASURED IS IMPORTANT IN AETIOLOGICAL STUDIES AND IS INDEPENDENT OF BODY MASS INDEX

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Introduction There are plausible biological mechanisms for how increased physical activity (PA) may prevent pancreatic cancer, although most studies do not report an inverse association. We investigated whether this may be related to the age at which PA is measured, using a validated questionnaire, and whether the effect of PA is independent of body mass index (BMI).

Methods 23,639 participants, aged 40–74 years were recruited into the EPIC-Norfolk cohort study between 1993 and 1997. These participants completed validated questionnaires on both occupational and leisure time PA. From this, four levels of PA index were derived. The cohort was monitored for up to 17 years to identify those participants who developed pancreatic cancer. The hazard ratios (HRs) of developing cancer were estimated using Cox regression and adjusted for covariates (age, gender, cigarette smoking status and type 2 diabetes). Each analysis was first performed in those recruited of all ages and then in those younger and older than 60 years at recruitment.

Results Within 17 years, 88 participants developed pancreatic cancer (55% female, median age of diagnosis 73 years, range 52–89 years). There was no association between PA and risk of pancreatic cancer in the whole cohort (trend HR=1.03, 95% CI: 0.84–1.27). However, in those recruited at younger than 60 years ($n = 29$ cases), higher levels of PA were associated with a decreased risk (highest vs. lowest category HR=0.27, 95% CI: 0.07–0.99, trend HR=0.75, 95% CI: 0.53–1.06, $p = 0.11$). When BMI was included, the associations were similar (highest vs. lowest category HR=0.25, 95% CI: 0.07–0.93, trend HR=0.73, 95% CI: 0.51–1.03, $p = 0.08$). In participants aged greater than 60 years ($n = 59$ cases), higher PA was associated with a non significant, increased risk both when BMI was unaccounted for