PTH-091 NON-INVASIVE SCREENING REVEALS HIGH RATES OF FIBROSIS IN DIABETIC/OBESE PATIENTS WITH NAFLD AND NORMAL BIOCHEMISTRY

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Introduction The association between non-alcoholic fatty liver disease (NAFLD) and Type 2 diabetes (T2DM) and obesity is well established yet current guidelines in the United Kingdom do not recommend screening for NAFLD these groups. Moreover, metabolic clinics have little hepatology support and few formalised NAFLD management pathways. Resulting in missed opportunities to diagnose, stage and treat NAFLD. We sought to determine the scale of clinically significant NAFLD in our T2DM and obese populations.

Method T2DM or obese patients attending metabolic clinic over a 3 month period were included. Fibrosis risk was assessed via a 2-step pathway. First, NAFLD fibrosis score (NFS) was calculated, followed by Fibroscan for those with indeterminate/high ('abnormal') NFS scores; Fibroscan readings of >8 kPa were considered abnormal.

Results 89 people were screened. We excluded those with both normal liver function (LFT) and ultrasound (n=11), previously diagnosed liver disease (n=3) or insufficient data to calculate the NFS score (n=43), leaving 32 patients (20 T2DM; 12 obese) of whom the majority were middle-aged males with median age 53 [28–75] and BMI 38 [22.1–68].

Most of those with NAFLD had normal LFT (25/32; 78.2%; $p<0.0001$) including median ALT 26 [7–129] and AST 20 [12–88]. Median NFS was -0.381 , with the majority having abnormal scores (84.4%; $p<0.0001$). A higher proportion of T2DM than obese patients had abnormal scores, but this did not reach significance ($p=0.26$). The vast majority of those with abnormal NFS had normal LFT (22/32 81.5%; $p=0.005$).

The 27/32 patients with abnormal score were invited for Fibroscan of whom 70.4% attended. Median Result was 7.05 kPa [2.8–26.3], with a non-significant trend to higher readings in obesity vs. T2DM (7.7 vs 6.6 kPa; $p=0.29$). 36.84% (7/19) had abnormal Fibroscan Result. ALT was significantly higher in those with abnormal Fibroscan (mean 66.9 vs. 18.83; $p=0.43$; 95% CI 33.76–62.32) but importantly, 42.9% (3/7) of those with abnormal Fibroscan had completely normal LFT.

Conclusion NAFLD was common in the cohort, usually undiagnosed and frequently associated with abnormal NFS and Fibroscan despite normal LFT, suggesting there is a sizeable population in metabolic services with potentially significant liver disease. We only included those with proven steatosis and sufficient data to calculate NFS, therefore the true prevalence of significant fibrosis is likely to be greater. Although biopsy has not yet been performed, abnormal 2-step non-invasive assessment alone mandates specialist input and as such active NAFLD screening in these groups should be considered.

PTH-092 N-ACETYL CYSTEINE AND PREDNISOLONE COMBINATION THERAPY IN SEVERE ALCOHOLIC HEPATITIS, AN NHS TEACHING HOSPITAL PILOT STUDY

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Introduction Severe Alcoholic Hepatitis (AH) (defined as a Maddray score >32) is a life-threatening condition with a recently reported 1 month mortality of 14% and 1 year mortality of 57%. The current mainstay of treatment is corticosteroid therapy, but previous studies suggest adding N-acetylcysteine (NAC) improves short-term mortality. We assess the effect on mortality of NAC as additional treatment for severe AH.

Methods We collected data using a standard proforma for patients admitted and diagnosed with severe AH (Maddray score of >32). Patients were treated with prednisolone (40 mg/day) and 5 days of IV NAC (at a dose of 150, 50, and 100 mg per kilogram of body weight in 250, 500, and 1000 ml of 5% glucose solution over a period of 30 mins, 4 hours, and 16 hours, respectively) and on days 2 through 5 (100 mg per kilogram per day in 1000 ml of 5% glucose solution). Patients were collected between 1st of May and 30th of October. We calculated the Lille score on day 7 of treatment and continued prednisolone in responders. Analysis was on a per-protocol basis. Mortality was assessed at 1, 3 and 6 months.

Results 10 patients were included. Mortality is shown in table 1 and compared to previous trials. The mean age of patients included was 51.6 ± 11.06 . Participants baseline characteristics were consistent with previous publications Maddray score 61 ± 27.9 , Bilirubin 233.6 ± 119.3 , Prothrombin time (PT) 21.1 ± 5.7 . Over the six months, there were 4 episodes of infection (40%) and 0 episodes of hepatorenal syndrome. After treatment the mean Lille score was 0.433 ± 0.31 . Of those patients that died there was a significant difference compared to patients who survived in initial Maddray score at 30 days (Alive: 53.4 ± 23.2 , Dead: 95.5 ± 16.3 ($p=0.041$)) and at 6

months (Alive 38.6 ± 16.1 , Dead 83.4 ± 15.4 ($p=0.002$)). There was no significant difference in Lille scores in patients that died at 1 or 6 months.

Abstract PTH-092 Table 1 Mortality summary table of recent publications compared to the pilot data for this study

	STOPAH 2015 -Prednisolone, 266 patients	Nguyen-Khac et al 2011 -Prednisolone, 89 patients	Nguyen-Khac et al 2011 - Prednisolone and NAC, 85 patients	Sheffield pilot 2018 – Prednisolone and NAC, 10 patients
30 day Mortality	14%	24%	8%	20%
3 months Mortality	30%	34%	22%	40%
6 month Mortality	-	38%	27%	50%
12 months Mortality	57%	-	-	-

Conclusions Treatment with combination of NAC and corticosteroids demonstrated slightly worse outcomes compared with recent trials although our numbers are too small to be certain. This study suggests significantly elevated Maddray scores are associated with an increased risk of mortality in severe AH. We feel a larger study to validate previous data and fully assess the effect of NAC in the treatment of AH on short term mortality is needed.