

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

¹VJ Appleby*, ²A Khan, ²J Rehman, ³GR Foster, ¹S Moreea. ¹Digestive Diseases Centre, Bradford Royal Infirmary, Duckworth Lane, UK; ²Barkerend Health Centre, Bradford, UK; ³Blizard Institute of Cell Research, London, UK

10.1136/gutjnl-2014-307263.537

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

¹N Noor, ²P Banim, ³R Luben, ⁴N Wareham, ³K-T Khaw, ¹A Hart*. ¹University of East Anglia, Norwich, UK; ²James Paget University Hospital, Great Yarmouth, UK; ³Institute of Public Health, University of Cambridge, Cambridge, UK; ⁴MRC Epidemiology Unit, University of Cambridge, Cambridge, UK

10.1136/gutjnl-2014-307263.538

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

(highest vs. lowest category HR=1.98, 95% CI: 0.94–4.16, $p = 0.07$, trend HR=1.23, 95% CI: 0.96–1.57, $p = 0.10$) and when BMI was included (trend HR=1.21, 95% CI: 0.94–1.55, $p = 0.13$).

Conclusion The association between PA and cancer risk is dependent on the age at which PA is measured. This possibly reflects occupational activity and differences in general medical health with age or residual confounding. The associations were similar when adjusted for BMI, suggesting an independent mechanism of PA. If the inverse association of increased PA in younger participants is causal, one in six cases of pancreatic cancer might be prevented by encouraging more PA. Aetiological studies should measure PA at different ages when investigating pancreatic cancer.

Disclosure of Interest None Declared.

PTH-093 CHROMOGRANIN-A : CAN IT PREDICT RADIOLOGICAL PROGRESSION IN NEUROENDOCRINE TUMOURS?

^{1,2}RE Rossi, ¹J Garcia-Hernandez, ³NG Martin, ¹M Mullan, ⁴T Meyer, ⁴C Thirlwell, ⁵J Watkins, ¹ME Caplin, ¹C Toumpanakis*. ¹Neuroendocrine Tumour Unit, Centre for Gastroenterology, Royal Free Hospital, London, UK; ²Postgraduate School of Gastroenterology, Università degli Studi Di Milano, Milan, Italy; ³Department of Clinical Biochemistry, Royal Free Hospital, London, UK; ⁴Cancer Institute, University College London, Huntley Street, London, UK; ⁵Department of Histopathology, Royal Free Hospital, London, UK

10.1136/gutjnl-2014-307263.539

Introduction Chromogranin A (CgA) is considered as the best general marker for the diagnosis and follow-up of neuroendocrine tumours (NETs) and is also of prognostic value. In literature, there are no available studies which analysed the role of CgA as a predictor of radiological disease progression in all NETs. Present study investigates the prognostic value of CgA as a predictor of radiological disease progression in NET patients.

Methods Patients with metastatic NETs and evidence of Radiological Progression (RP) according to RECIST 1.1 were identified from a NET database. Plasma CgA were measured 6 and 12 months before RP and at the event of RP. CgA was measured with the Supra-regional-Assay-Service radioimmunoassay (Hammersmith Hospital), normal value <60 pmol/L. The tumours were graded according to the 2010 WHO classification, as G1 (Ki67 <2%), G2 (Ki67: 2–20%), G3 (Ki67 >20%).

Results 152 patients were evaluable including 91 midgut NET and 61 pancreatic NETs (PNETs). Of these, 56 were G1 NETs, 65 G2, 10 G3, 21 of unknown histology. 95.4% of the patients had liver metastases, whereas bone and lung metastases were present in a smaller proportion of patients (27.6 and 9.9%, respectively). Median CgA for all NETs 6 months before RP was 213 pmol/L [Interquartile 1 (Q1)=67 and 3 (Q3)=664.5 pmol/L] compared to 166 pmol/L (Q1 52, Q3 535 pmol/L) one year before RP, $T = 598.5$, $p = 0.07$. Significant results were found for PNETs [median CgA 6 months before RP: 100 pmol/L (Q1 53, Q3 286.25 pmol/L) and at 12 months: 52 pmol/L (Q1 36.25, Q3 128 pmol/L), $T=52$, $p = 0.048$], but not for midgut NETs [median CgA 6 months before RP: 389.5 pmol/L (Q1 131.5, Q3 791.5 pmol/L) and at 12 months: 319 pmol/L (Q1 158, Q3 753 pmol/L), $T=191$, $p = .39$]. Both midgut and PNETs CgA values were significantly higher at RP than 12 months before [267 pmol/L (Q1=66, Q3=777) vs. 166 pmol/L (Q1=52, Q3=535), $T = 394.5$, $p = 0.03$]. Overall, G1 tumours had median CgA value at 6 months significantly higher than at 12 months [181(Q1=56.25, Q3=624) vs. 149.5 pmol/L (Q1=44, Q3=247.25), $T=70$, $p = 0.048$].

Conclusion CgA seems to have predictive value 6 months prior to RP for PNETs and G1 tumours, which may be of value to identify specific subgroups of patients who may benefit from a more aggressive follow-up with possible early intervention in case of increased CgA levels. Further prospective studies are needed to enable more definitive conclusions.

REFERENCES

- 1 Oberg K et al. *Pancreas* 2011
- 2 Ter-Minassian M et al. *Endocr Relat Cancer* 2013
- 3 Welin S et al. *Neuroendocrinology* 2009
- 4 Jensen KH et al. *Scand J Gastroenterol* 2013

Disclosure of Interest None Declared.

PTH-094 BENEFIT OF REAL TIME CYTOLOGICAL EXAMINATION IN EUS GUIDED BIOPSY OF SUSPECTED PANCREATIC MALIGNANCY

¹D Lloyd*, ²A Al-Badri, ¹H Gordon. ¹Gastroenterology, Hampshire Hospitals NHS Foundation Trust, Winchester, UK; ²Histopathology, Hampshire Hospitals NHS Foundation Trust, Winchester, UK

10.1136/gutjnl-2014-307263.540

Introduction Endoscopic ultrasound (EUS) guided sampling of advanced malignant pancreatic lesions is increasingly being performed in order to confirm malignancy prior to chemotherapy and/or radiotherapy. The Royal Hampshire County Hospital provides EUS services for central and north Hampshire. Prior to mid-2013 there was no facility for examination of cytological specimens during EUS procedures. In line with national commissioning guidelines, a real time pathology service allowing cytological examination during the EUS procedure was instigated from 1st July 2013. The aim of this study was to assess the impact of real time cytological examination on the yield of EUS guided sampling of suspected malignant pancreatic mass lesions.

Methods All patients with suspected pancreatic malignancy undergoing EUS guided tissue sampling over a 1 year period from 1st January 2013 to 31st December 2013 were prospectively audited. Note was made of whether real time cytological examination was performed by a technician \pm histopathologist. Other data collected included type of needle used, number of passes made with the biopsy needle and total duration of procedure. The diagnostic yield of EUS guided pancreatic sampling was compared with and without real time cytological examination.

Results Twenty-seven procedures were performed over the 12 month period. The majority (25 procedures) were performed using Procore™ fine needle biopsy (FNB) needles. Seventeen procedures were performed without real time cytological examination. Of these, 14 (82%) yielded positive cytology, 1 yielded negative cytology (6%) and there was insufficient tissue in 2 (12%) cases. Ten procedures were performed with real time cytological examination and of these all yielded positive cytology. Median number of passes made with the biopsy needle was 2 (range 2–3) without real time cytological examination versus 2 (range 1–4) with real time cytological examination. Mean procedure duration was 30 (± 12) min without real time cytological examination versus 36 (± 15) min with real time cytological examination.

Conclusion In our centre, the diagnostic yield of EUS guided sampling of suspected malignant pancreatic mass lesions without real time cytological examination was 82% which is in line with published data ¹ However, the addition of real time cytological