steatosis grade ($P = 0.001$). By plotting receiver operating characteristic (ROC) curve, the sensitivity and specificity of plasma *MIR17HG* protein in determining the extent of hepatic fibrosis (early vs. advanced) were 92.9% and 93.7% respectively at a cut-off value of 40.3 pg/ml (AUC = 0.978) and were superior to those of APRI (78.6% and 62.5% respectively at a cut-off value of 1.37, AUC = 0.857) and FIB-4 score (85.7% and 81.2% respectively at a cut-off value of 1.61, AUC = 0.902).

Conclusion Plasma *MIR17HG* protein could be a potential simple biomarker for determining the severity of hepatic fibrosis in chronic HCV infection and its decrease in relation to fibrosis regression after direct-acting antivirals needs to be evaluated.

PTU-004 HEPATOCELLULAR CARCINOMA CAN BE MANAGED SAFELY AND EFFECTIVELY IN A DGH-SETTING WITH SUPERIOR SURVEILLANCE-PROGRAMME SURVIVAL

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10.1136/gutjnl-2019-BSGAbstracts.213

Introduction and aims HCC is the second commonest cause of cancer-related death worldwide and strongly associated with liver cirrhosis and with a rising incidence.

Despite screening most HCC cases present at an intermediate or advanced stage unsuitable for curative surgery. The standard of care for most non-curative cases being actively treated remains transarterial chemo-embolisation TACE and/or ablation (RFA.) Both are specialist procedures normally delivered in tertiary centres.

At the Royal Bournemouth Hospital (RBH), a large DGH, specialist HCC treatments are offered to Dorset County following MDT and combined hepatology/IR clinic review. There is an established surveillance programme offered to all suitable at-risk patients.

We sought to assess the outcomes of the service with a focus on the benefits of surveillance and the safety of offering tertiary level services in a DGH setting.

Methods We identified all new HCC cases presented in the pan-Dorset Upper GI MDT from Jan 2017 to Dec 2017. We collected demographic data, whether they had been under a surveillance programme and the treatment outcomes including complications and 1- and 12- month mortality.

Results We identified 35 patients (30 M; 5F.) The aetiology was alcohol in 26% (n=9), NASH 43% (n=15), HCV 17% (n=6) and others 9% (n=3.) Cirrhosis was present in 63% (n=22): Child's A 59% (n=13), Child's B 32% (n=7) & Child's C 9% (n=2.)

Most cases were referred from RBH 77% (n=27), and 23% from the two other referring hospitals in the County. HCC surveillance detected 43% (n=15) of cases with 57% new presentations. Of the surveillance cases, the majority 87% (n=13) were identified at the centre with the most established surveillance programme but as the largest centre RBH also identified most new presentations 70% (n=14.)

More active treatment was offered to the surveillance group at 87% vs 65% of non-surveillance group ($p \leq 0.05$.)

Curative treatment (transplant, surgery or RFA to small HCC) was suitable in only 14.3% (n=5), all identified by surveillance.

TACE was offered to 46% of patients (n=16.) Of the TACE patients, 56% (n= 9) underwent more than 1 procedure. Only 2 patients had decompensation post-TACE, which recovered. Post-TACE survival was 100% at 1 month and 79% at 1 year. These outcomes are comparable to published literature from larger centres.

Overall 1-month and 12-month survival for surveillance cases was better than new presentations at 100% and 73% vs 85% and 50% respectively ($p \leq 0.05$.)

Conclusions Specialist HCC treatment, following combined hepatology/IR review, can be offered safely and effectively in a large DGH setting with mortality and morbidities outcomes comparable to specialist tertiary centres. Our data confirms HCC surveillance allows for earlier cancer detection with more treatment options and improved survival.

PTU-005 REDUNDANCY OF ELASTOGRAPHY IN MONO-INFECTED HCV INDIVIDUALS <30 YEARS OLD- SIMPLIFICATION OF TREATMENT ALGORITHMS

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10.1136/gutjnl-2019-BSGAbstracts.214

Introduction The Hepatitis C Virus (HCV) is a blood-borne, causing chronic infection, defined as the persistence of HCV RNA for at least 6 months after initial infection. Chronic HCV infection predisposes individual patients to liver fibrosis, cirrhosis and hepatocellular carcinoma. Of those with chronic HCV infection; it is estimated that 16–20% of individuals will go on to develop liver cirrhosis within 20 years.

There has been a revolution in the treatment of chronic HCV infection with almost universal cure. The issue is now to minimise the steps required to initiate treatment; especially as we seek to treat younger, more difficult to engage individuals. Currently, fibrosis is assessed before starting treatment. We sought to validate the theory that those individuals who were mono-infected with HCV; young and with no additional hepatic insult were unlikely to have significant fibrosis.

Methods We performed a retrospective analysis of the Tayside Hepatitis C dataset; with collation of relevant basic demographics including age, sex and baseline Fibroscan® measurements pre-treatment. Previous validation of transient elastography (TE) in HCV has suggested that a cut off of less than 11 kPa would exclude significant fibrosis.

Results Our complete cohort consisted of 719 individuals with HCV who had elastography performed before undergoing HCV therapy. We stratified for age across 5 year intervals. The results are summarized in the figure below.

Abstract PTU005 Figure 1

Age Range	Total (n=) (M/F)	Transient Elastography (Mean; Range)	Transient Elastography >9 kPa	Transient Elastography >11 kPa
<30 Years	99 (62/37)	5.71 (3.2–10.6)	1	0
30–35	111 (82/29)	8.9 (2.1–26.6)	6	4

Within this patient cohort we established that no patient under 30 had a TE score > 9 kPa without a co-existent

history of hazardous alcohol consumption; and only a single individual within the 31–35 group had a fibroscan >11 kPa in absence of hazardous alcohol ingestion or co-infectivity with a hepatotropic virus.

Conclusion Going forward we therefore propose that those patients <30 years old do not necessitate elastography prior to treatment. The vast majority of patients between 30–35 years (>95% in our dataset) are also likely to be suitable for treatment without elastography in the absence of other co-existent factors for fibrosis; most notably alcohol in this cohort.

PTU-006 VITAMIN D DEFICIENCY DOES NOT INCREASE THE RISK OF POST-LIVER TRANSPLANT ALLOGRAFT CELLULAR REJECTION (ACR)

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10.1136/gutjnl-2019-BSGAbstracts.215

Introduction Vitamin D deficiency is prevalent in cirrhotic patients, and is increasingly associated with undesirable outcomes including increased rates of infection and higher mortality rates. It has also been documented that, owing to its supposed role in immune regulation, there may be a correlation between vitamin D deficiency pre-liver transplant and an increased risk of graft rejection (independent of other factors such as CMV rates which influence rejection risk). It has been postulated that this correlation may be because vitamin D allows a level of immune tolerance towards the graft. Nevertheless, the evidence is not conclusive. Animal models have demonstrated that calcitriol administration can prevent ACR and improve survival, but this has not yet been trialled in humans. Few retrospective studies have also looked at correlations between vitamin D deficiency and ACR rates- with differing results. We aimed to review outcomes of patients undergoing liver transplant at a tertiary liver centre, and evaluate if any such correlation with vitamin D and graft rejection exists.

Methods This is a retrospective study of patients with chronic liver disease awaiting liver transplantation from 2015–2017. Data collated included: demographic data, vitamin D levels within 6 months pre-liver transplant, use of vitamin D supplementation, post-operative rates of CMV, and episodes of cellular rejection (biopsy-proven).

Results 299 patients were included (72% male). Aetiology was ALD (n=96), viral hepatitis (n=66), PBC/PSC (n=62), NAFLD (n=34), AIH (n=22), and 'other' (n=19). Vitamin D deficiency affected 62% of the cohort. 157/299 patients had so far been transplanted at the time of data collection, and of these 115/157 had vitamin D levels recorded within 6 months pre-transplant. Total rejection rates were 20% (23/115) in the first year post-transplant, with the majority being episodes of early rejection (0–2 months). There was no significant association between vitamin D deficiency pre-transplant and the risk of cellular rejection, with 25% of those with vitamin D levels <25nmol/L pre-transplant experiencing rejection, in comparison to 14% of those with a normal vitamin D (>50nmol/L) (p=0.2). No correlation was seen with vitamin D and CMV rates, or CMV and rejection rates. Prescribing cholecalciferol either pre-transplant or in the first

month post-transplant did not confer any significant reduction in the risk of rejection.

Conclusion Our results are contrary to the limited published data showing increased rates of graft rejection with vitamin D deficiency. There is a pressing need for a prospective study to confirm if a true correlation exists between vitamin D deficiency and risk of cellular rejection, and if vitamin D supplementation would reduce this risk.

PTU-007 THE DEVELOPMENT OF OBESITY AND TYPE 2 DIABETES HARNESSING THE GUT MICROBIOME AND HOST GENETICS

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10.1136/gutjnl-2019-BSGAbstracts.216

Introduction Obesity is increasingly prevalent worldwide and has deleterious consequences on our health, including type 2 diabetes. Risk factors of obesity include genetic factors, diet, lifestyle and the gut microbiome. A preliminary study that combined genetic and environmental manipulations observed that MMP28 gene-knockout (MMP28KO) mice exhibit signs of obesity and type 2 diabetes. Interestingly, the metabolic profile of MMP28KO mice significantly improved upon transferring them from a specific pathogen free (SPF) area into the conventional mouse room for 5 weeks, suggesting the involvement of the gut microbiome. It is hypothesised that the underlying mechanisms are due to modulations in the insulin and Farnesoid X Receptor signalling pathways, which play important roles in regulating glucose and lipid metabolism. Key components within the pathways include Insulin receptor substrate-1 (IRS-1), insulin receptor substrate-2 (IRS-2) and Farnesoid X Receptor (FXR).

Aims This study aims to investigate the underlying mechanisms linking the MMP28 gene knockout effect and the impact of changing environments with changes in metabolic profiles.

Methodology A total of 71 mice were used in this study. Quantitative real time polymerase chain reaction was used to compare the expression levels of the IRS-1, IRS-2 and FXR genes between MMP28KO mice and their wild-type counterparts at 15 and 30 weeks of age. To investigate the impact of changing environments, the change in gene expression levels after the MMP28KO mice were moved from the SPF area into the conventional mouse room for 5 weeks was determined. Parametric data were compared using the unpaired t-test; non-parametric data were compared using the Mann-Whitney U test. A *p* value of <0.05 was considered significant.

Results The MMP28 gene knockout effect resulted in a poor metabolic profile through lowered IRS-2 and FXR gene expressions in the liver and pancreas (p<0.05) in 30-week-old mice. IRS-1 gene expression was also lower in the pancreas (p<0.05). Transferring MMP28KO mice from the SPF area to a conventional mouse room increased IRS-2 gene expression in both the liver and pancreas (p<0.001 at 30 weeks and p<0.05 at 15 weeks, respectively). FXR gene expression only increased in the pancreas (p<0.05) in 15-week-old mice and there were no changes in IRS-1 gene expression in either organ. The gene expression modulations of the two pathways in the liver and pancreas are summarised in figure 1.