as a salvage procedure after failed portal vein embolization and portal vein ligation respectively.

Results Median increase of FLR volume was 139.25% (range 40.00%–157.78%). Median hospital stay was 28.5 days (range 23–36). Histology report revealed two complete (R0) and two incomplete (R1) resections. Background liver histology revealed steatosis on two occasions and fibrosis on another. 90 day mortality was zero. Two patients developed grade II complications as per Clavien-Dindo classification, one grade IIIa and one IIIb. One patient is disease free after 36 months. One patient died two years later from viral infection, and two had recurrence in liver and lymph nodes and were treated with microwave ablation and lymphadenectomy respectively.

Conclusions ALPPS procedure allows performing major liver resections for high volume neuroendocrine metastases while minimising the risk of post-operative liver failure.

PTH-102 CIRRHOTIC PATIENTS WITH VITAMIN D DEFICIENCY FAIL TO RESPOND TO ORAL REPLACEMENT THERAPY

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10.1136/gutjnl-2018-BSGAbstracts.258

Introduction Vitamin D deficiency and reduced BMD are highly prevalent in patients with advanced chronic liver disease. For bisphosphonate treatment for osteoporosis to be effective vitamin D levels must be replete. Moreover, vitamin D deficiency has been associated with an increased risk of infections and increased rejection rates following liver transplantation. The optimal dose and route of vitamin D replacement in cirrhosis is unknown. BSG guidance currently recommends 800 IU/day orally for all patients with cirrhosis/cholestatic liver disease.

Methods Retrospective review of 218 cirrhotic patients undergoing evaluation for liver transplant between 2016 and 2017. Vitamin D 'severe deficiency' was defined as <25 ng/ml, 'deficiency' 25–50 ng/ml and normal >50 ng/ml. Response to oral vitamin D therapy was recorded.

Results Out of 218 patients, 128/218 (59%) had low Vitamin D levels with 25% (n=55) 'severely deficient' and 33% (n=73) 'deficient'. Overall 33 patients with levels <50 ng/ml (52%), and 31 patients (48%) with levels >50 ng/ml received replacement therapy. (p=0.86)

Median daily dose of Vitamin D replacement was 2800 units/day (IQR 800–2800) in <25 ng/ml group, 2860 units/day (IQR 800–2800) in <50 ng/ml group and 800 units/day (IQR 800–2000) in >50 ng/dl group. No significant difference in dosing between these groups (p=0.12).

Data on vitamin D levels pre and post 3 months of treatment with Vitamin D therapy were available in 58 patients. Patients received either 400IU/day (n=6), 800–1600IU/day (n=28) or >1600 IU/Day (n=24). Median delta change in vitamins D levels in the 3 groups were –3 ng/ml, –1 ng/ml and 12 ng/ml over the 3 month treatment period. An average daily dose of >1600 IU/day Resulted in a significantly greater increase in Vitamin D levels when compared to doses <1600 IU/day (p=0.01), albeit still sub optimal with only a median increase of 12 ng/ml.

When those patients with Vitamin D levels of <50 ng/ml were reviewed in isolation (n=29), 82% failed to augment

vitamin D levels to within the normal range >50 ng/dl and no significant difference was found between dosages of vitamin D administered.

Conclusion Vitamin D deficiency is prevalent, affecting over 50% of patients with advanced cirrhosis. Oral vitamin D replacement therapy is ineffective in cirrhotics at repleting stores over a 3 month period irrespective of dose given.

Future evaluation of efficacy of IM administration in this unique cohort of patients is urgently needed to evaluate if this allows normalisation of Vitamin D levels.

PTH-103 EPIDEMIOLOGY OF VITAMIN D DEFICIENCY AND BONE MINERAL DENSITY IN PATIENTS WITH CHRONIC LIVER DISEASE

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10.1136/gutjnl-2018-BSGAbstracts.259

Introduction Patients with chronic liver disease have increased risk of bone disease (BD) with reported prevalence of osteoporosis (OP) between 12%–55% and a high prevalence of Vitamin D deficiency. The aetiology is poorly understood with a complex interplay between endocrine, metabolic, nutritional and physical abnormalities. We aim to evaluate the influence of epidemiological parameters on bone mineral density and vitamin D levels in cirrhotics.

Methods Retrospective study of cirrhotic patients from 2016–2017. Data were collected on aetiology of cirrhosis, severity (UKELD/MELD score), bone mineral density (BMD), vitamin D, body mass index (BMI) and hand grip strength. OP was defined as per WHO classification and Vitamin D deficiency as a Vitamin D level <50 nmol/L with severe deficiency <25 nmol/L.

Results 248 patients were included, 180 male, 58 female, median age 57 years (IQR 49–63) and median BMI of 27. Underlying aetiology was ALD (n=78), Viral (n=56), PBC/PSC (n=46), NAFLD (n=23) and AIH (n=18). Median UKELD and MELD scores overall were 53 (IQR 49–57) and 14 (IQR 10–19). At the time of evaluation 141 (56.8%) patients were either osteoporotic (n=52) or osteopenic (n=99). The prevalence of BD was significantly higher in cholestatic diseases (71.7%, mean T score –1.86 +/–1.22) and lower in NAFLD (37%, mean T score –0.45 +/–1.50) when compared to other aetiologies (ANOVA p=0.0005). 120 (55.4%) patients were vitamin D deficient with 51 (25%) patients having severe deficiency. Mean vitamin D level was highest in cholestatic disease (75.5 ng/ml +/–59.6) when compared to other aetiologies (ANOVA p=0.003).

Liver severity scores (UKELD/MELD respectively) did not correlate with the presence of BD (p=0.32/p=0.53) but patients with higher MELD scores had lower vitamin D levels (p=0.04). Reduced BMI correlated with the presence of BD (p<0.01) but not vitamin D level. Increased Hand Grip Strength (HGS) was associated with higher vitamin D levels (p=0.049) and higher lumbar T scores (p=0.014). Vitamin D levels did not correlate with BMD (p=0.77).

Conclusion Bone disease and vitamin D deficiency are prevalent in patients with cirrhosis, with cholestatic aetiologies having the highest prevalence of OP and NAFLD the lowest.

Interestingly disease severity does not correlate with BD whereas more functional markers of frailty such as HGS appear to positively correlate. Increased disease severity (MELD) significantly correlates with decreasing vitamin D levels, which raises the question of whether vitamin D could be impacting on progression of cirrhosis, or vice versa. Further prospective research is needed to look at the role of vitamin D in cirrhosis.

PTH-104 THE INCREASING BURDEN OF DIABETES ON THE SCOTTISH LIVER TRANSPLANTATION SERVICE

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10.1136/gutjnl-2018-BSGAbstracts.260

Introduction In Scotland 65% of adults are overweight and 29% obese¹. Among the health implications is a rising prevalence of diabetes within the population. Diabetes is known to contribute to liver damage, including carcinogenesis, and this study seeks to establish the impact of this damage on liver disease, hepatocellular carcinoma (HCC) and need for transplantation within Scotland. Diabetes, as a major risk factor for non-alcoholic fatty liver disease (NAFLD) could also be considered a surrogate marker for the NAFLD component of liver damage in other primary aetiologies.

Methods Prospectively held patient data from all transplant assessments undertaken at the Scottish liver transplant unit (SLTU) since 1992 were retrospectively analysed for the presence of diabetes, HCC and the underlying aetiology of their liver disease. Data were collected through VitalData (©Vital-pulse Ltd) and exported into Microsoft Excel (©Microsoft) for analysis.

Results In 1993 there were 45 assessments, which rose to 214 assessments in 2015, giving a total of 3098 during this time frame (figure 1).

The incidence of diabetes in patients referred for liver transplant remained persistently below 5% until 2004, from which point it started to increase to 24% in 2016 (figure 2).

Figure 3 shows the percentages of patients who are referred for transplant assessment with HCC who were diabetic at the time of referral.

12.0% of the 3098 patients referred had diabetes, this varied depending on their underlying aetiology- NAFLD (41.9%), ALD (13.5%), cryptogenic cirrhosis (9.6%), HCV (9.2%) and PBC (2.6%). The percentage of patients with diabetes and HCC referred was 18.5% (of 574 patients)- NAFLD (45.7%), haemochromatosis (37.2%), ALD (19.1%), PSC (16.7%), HBV (12.5%), HCV (9.9%) and others (0%) (figure 4).

Conclusions The number of assessments has been increasing since the beginning of transplantation at the SLTU in 1992 with an increasing proportion diabetic. The impact is most clear through the trend in NAFLD reaching transplantation but the prevalence in other aetiologies of liver damage suggests a compound effect.

With the exception of PBC, each of the most common aetiologies seen showed a higher prevalence of diabetes in the patients referred with HCC. This suggests that diabetes promotes carcinogenesis, with the more modest impact in HCV mirroring previous studies into the carcinogenicity of diabetes².

Abstract PTH-104 Figure 1 Total number of transplant assessments undertaken at SLTU

Abstract PTH-104 Figure 2 Percentage of patients assessed for transplant who have diabetes

Abstract PTH-104 Figure 3 Percentage of patients referred for transplant assessment with HCC who were diabetic (by year)

Abstract PTH-104 Figure 4 Percentages of referrals with HCC versus all referrals for each aetiology for transplant assessment

The increasing incidence of diabetes in patients being referred for transplant assessment with decompensated liver disease or HCC suggests that the national burden of diabetes and NAFLD³ will contribute to an increasing burden of liver damage of almost any aetiology.