standard liver blood tests are inadequate to detect clinically silent disease. We conclude that one-stage testing may be appropriate in populations with high prevalence of underlying disease, such as high-risk alcohol intake, whereas further discrimination is required in cohorts with a lower prevalence.

P069 CREATING A SURROGATE ARTIFICIAL LIVER USING 3D PRINTING AND MECHANICAL CHARACTERISATION OF HEPATIC TISSUES FOR TUMOUR REMOVAL EDUCATION

¹Richard Arm*, ¹Arash Shahidi, ²Chris Clarke. ¹Nottingham Trent University, Nottingham, UK; ²Nottingham University Hospital, Nottingham, UK

10.1136/gutjnl-2021-BASL.78

This research aims to identify suitable synthetic materials and manufacturing methodologies that will enable the production of accurate, patient-specific, anthropomorphic surrogate organs to allow surgeons to rehearse tumour removal using traditional or robotic techniques prior to the live surgery.

Shore hardness (H00) and elastic modulus (MPa) of both porcine tissues and surrogate tissues were tested with the same equipment and methods. We used international test standards (ASTM D 2240-03 and BS 903-A26:1995/ISO 48:1994, BS/ISO 23529:2016, ASTM D412 AND BS/ISO 37:2011). The hardness data was collected using a shore 00 calibrated durometer. Stress versus strain curves were plotted using the data collected from a Zwick/Roell tensile testing machine. Both pieces of equipment were used to characterise and engineer synthetic soft tissues to match the existing data and observations of porcine liver. CT and MRI DICOM data was used to produce multiplanar reconstructions and rigid 3D printed artefacts of liver anatomy (blood vessels, tumour, and parenchyma). The models were transformed into physiological counterparts using multi layered synthetic gels and fibres that mimic variable tissue properties like hardness and elastic modulus based on data of human liver given in literature and our porcine liver observations.

Previously published hardness of live human parenchyma was 15.06 H00 ($\pm$ 2.64), while we found porcine liver parenchyma to be 23 H00 ($\pm$ 0.1). Hardness of the synthetic parenchyma of liver was 10 H00 ($\pm$ 2.3).

Hardness of porcine vascular tissue thickness did not meet requirements for agreed test standard, but suitable bovine aortic tissue was previously investigated and was found to be 41 H00. The hardness of our surrogate vascular tissue was 40 H00. No data exists on the hardness of tumours for comparison.

Literature shows the elastic Young's modulus of healthy adult livers to be between 0.01 – 0.015 MPa. The porcine specimens had a modulus of 0.02–0.08 MPa within 0.2–0.4 strain range. These specimens failed at a force of 7.33 N. The surrogate modulus was 0.006–0.02 MPa within 1–1.7 strain range and failed at 0.67 N.

Surrogate liver tissues had similar hardness and elastic modulus to human and porcine equivalents. We found porcine parenchyma to be more brittle, less extensible and slightly stiffer than both human and synthetic surrogate tissue. Production methodology of surrogate liver and internal vasculature was successful, but better resolution of the smaller vessels (<2mm), refinement of the additive manufacturing process

and methods of multi-layered lamination might improve models.

P070 LIVER STIFFNESS MEASUREMENT PERFORMS POORLY IN HAEMOCHROMATOSIS

¹Alex Hinkson*, ²Guy Gitlin-Leigh, ¹Joanna Moore. ¹Leeds Teaching Hospitals NHS Trust, UK; ²Leeds School of Medicine, University of Leeds, Leeds, UK

10.1136/gutjnl-2021-BASL.79

Background Haemochromatosis can lead to liver fibrosis and cirrhosis. Liver biopsy is the gold standard for assessing fibrosis but is associated with risks to the patient. Non-invasive techniques are increasingly used for staging of fibrosis. Liver Stiffness Measurement (LSM) is commonly used in other disease aetiologies but performance in haemochromatosis is not well characterised. Our aim was to determine whether LSM reliably detects advanced fibrosis or cirrhosis in patients with haemochromatosis.

Methods This study was a retrospective analysis of data from patients being treated for haemochromatosis in Secondary Care who underwent LSM by Fibroscan™ and had biopsy for characterisation and staging of disease. Patients without liver biopsy were excluded. Data regarding genotype, biochemical parameters, biopsy indication, histology and LSM were collected. Patients with cirrhosis on biopsy were assumed to remain cirrhotic. Patients with more than 18 months between biopsy and LSM were excluded. Median LSM values for different patient groups were calculated in Excel. A planned ROC analysis was abandoned due to insufficient patient numbers.

Results Data from 25 patients with liver biopsy were collected. All these patients had LSM. Median LSM for all patients in our cohort was 8.5kPa. Only 6 patients had LSM within 18 months of biopsy, of whom 2 had bridging fibrosis and 5 had peri-portal or no fibrosis. Median LSM for these patients was 9.8kPa and mean was 11.1kPa. Six patients had histological evidence of cirrhosis. Median LSM for this group was 9.1kPa and mean was 9.8kPa.

Conclusion Our data suggest that LSM may not be a reliable tool for quantifying fibrosis in patients with haemochromatosis, as those with low levels of fibrosis often had LSM values higher than patients with known cirrhosis. These results may

Abstract P070 Table 1

Patient number	LSM/Biopsy interval	Fibrosis Stage	Median LSM (kPa)
1	1 months	No bridging	10
2	12 years	Cirrhosis	22.5
3	3 months	Bridging fibrosis	9.6
4	2 months	Bridging Fibrosis	7.6
5	10 years	Cirrhosis	10.3
6	6 weeks	No bridging	12.4
7	13 days	No bridging	3.7
8	4 years	Cirrhosis	7.1
9	3 months	No bridging	2.8
10	14 years	Cirrhosis	6.4
11	10 years	Cirrhosis	9.1
12	10 years	Cirrhosis	9.1

be influenced by relatively small sample size and the early detection of haemochromatosis with the aid of genetic analysis, allowing initiation of venesection before development of advanced disease. Further studies using prospective recruitment of patients for LSM at the point of diagnosis alongside biopsy to stage fibrosis would allow a more complete evaluation of LSM performance.

P071

SILYMARIN-CHOLINE COMBINATION VERSUS URSODEOXYCHOLIC ACID IN NON-ALCOHOLIC FATTY LIVER DISEASE: A RANDOMISED DOUBLE-BLIND CLINICAL TRIAL

¹Purba Chakrabarty*, ²Saubhik Ghosh, ³Mayukh Mukherjee, ⁴Rajasee Adhikary, ⁵Shatavisa Mukherjee, ⁶Souvik Majumder, ⁴Dipak Kumar Sarkar, ⁶Arunansu Talukdar. ¹Walsh, SDH, Serampore, India, Serampore, Hooghly, India; ²Department of Medical Gastroenterology, Medical College and Hospital, Kolkata, Kolkata, India; ³Department of Pharmacology, R. G. Kar Medical College and Hospital, Kolkata, Kolkata, India; ⁴Department of Pharmacology, Medical College and Hospital, Kolkata, Kolkata, India; ⁵ADR Monitoring Centre, School of Tropical Medicine, Kolkata, Kolkata, India; ⁶Department of Medicine, Medical College and Hospital, Kolkata, Kolkata, India

10.1136/gutjnl-2021-BASL.80

Introduction Prevalence of Non-Alcoholic Fatty Liver Disease (NAFLD) is 20%–30% and 5%–18% in Western countries and Asia respectively.¹ Currently there is no evidence-based standard of care. Since an oxidative stress and dietary deficiency of choline have been implicated in the pathophysiology of hepatic insult, the use of natural compounds like Silymarin, Choline and Ursodeoxycholic acid (UDCA) represents an extremely popular therapeutic option for the treatment of NAFLD.²

Objective To compare the efficacy, safety profile and adherence of Silymarin-Choline combination vis – a – vis UDCA in patients with NAFLD

Method The study was a double blind parallel arm trial where 88 NAFLD diagnosed patients, abiding by the inclusion and exclusion criteria, were randomised to receive either Tablet Silymarin (140 mg) - Choline bitartrate (450 mg) 1 tablet thrice daily or UDCA (300 mg) 1 tablet twice daily for 6 months. Lifestyle modification was advised. Participants were monitored for weight, liver function test, lipid profile parameters, HOMA-IR, liver stiffness measurement and liver biopsy at baseline and 6 months of medication. Monitoring of associated adverse events and adherence were done. Results were tabulated and statistically analyzed for any significant inter or intra group differences using standard statistical software.

Results A total of 39 patients received tablet Silymarin - Choline bitartrate (group A) as compared to 40 who received UDCA (group B). Both the groups were comparable at baseline with regard to age (mean±SD) [39.33±9.39 vs 40.63±10.63], weight (mean±SD) [72.80±4.24 vs 72.95±4.22] Kg, BMI (mean±SD) [29.11±1.02 vs 29.32±2.07] Kg/m², LFT (Alanine aminotransferase, median±IQR) [86.00±18.00 vs 87.00±19.00] IU/L, Lipid Parameters, HOMA-IR (mean±SD) [1.42±0.53 vs 1.27±0.71], transient elastography (mean±SD) [7.67±0.97 vs 7.61±0.66] and tolerability. At 2nd visit, significant individual intra-group reduction in weight, liver and lipid parameters, improvement in transient elastography, liver biopsy, HOMA IR score was noted. Notably, independent T test suggests significant decrease (p<0.05) in weight [67.23±5.87 vs 70.70±5.11, p. 0.006], transient elastography [6.32±1.26 vs 6.85±0.98, p. 0.043], HOMA IR

score [0.59±0.28 vs 0.77±0.21, p. 0.002] more in group A compared to group B.

Conclusion NAFLD is an important cause of liver disease burden across the world. The accepted treatment protocol is to treat the associated comorbidities which cannot stop the progression of disease at times. Tablet Silymarin - Choline bitartrate is effective and tolerable as UDCA when combined with lifestyle modification. Tablet Silymarin - Choline bitartrate can be utilized as an alternative to UDCA in the treatment of NAFLD.

REFERENCES

1. Hellerbrand C, Schattenberg JM, Peterburs P, Lechner A, Brignoli R. The potential of silymarin for the treatment of hepatic disorders. *Clin Phytoscience* 2017 Jan 1;2(1):7.
2. Hajiaghahmohammadi AA, Ziaee A, Oveisi S, Masroor H. Effects of metformin, pioglitazone, and silymarin treatment on non-alcoholic fatty liver disease: a randomized controlled pilot study. *Hepat Mon [Internet]* 2012 Aug 3 [cited 2018 Jun 23];12(8). Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3475019/>

P072

SYSTEMATIC REVIEW AND META-ANALYSIS OF THE PREVALENCE OF ALCOHOL RELATED LIVER DISEASE

¹Sachin Amonker*, ¹Aryo Houshmand, ¹Ian Rowe, ²Richard Parker. ¹University of Leeds, Leeds, UK; ²Leeds Liver Unit, Leeds, UK

10.1136/gutjnl-2021-BASL.81

Introduction Alcohol related liver disease (ALD) is a major cause of morbidity and mortality. The aim of this systematic review and meta-analysis was to define the prevalence of ALD.

Methods A systematic review was undertaken using the search terms ((alcoholic liver disease) AND prevalence OR epidemiology)) in PubMed and Embase databases. Reference lists of the included papers and citing literature were also searched for relevant papers. Single-proportion random-effect meta-analysis was done using the ‘meta’ package in R. Planned sub-group analyses were done in populations of persons with alcohol use disorder (AUD), for different methods of diagnosis and by geographical region. For the purpose of this review we considered the overall prevalence of all forms of ALD, and the prevalence of alcohol-associated fatty liver (AFL) and alcohol-associated cirrhosis (AC).

Results The literature search identified 7040 studies. After review, 37 papers were included. ALD was defined by biochemistry in 9 studies, by biopsy in 5 studies and by ultrasound in 13 studies. Ten studies used coding from clinical records to define disease.

The overall prevalence of any form of ALD in unselected populations was 4.5% (95% confidence interval 3.1 – 6.3%). The prevalence of AFL was 6.3% (3.2 – 12.1), and prevalence of AC was 0.2% (0.1 – 0.3%). In populations with AUD, the prevalence of ALD, AFL and AC were 65% (53.3 – 75.8%), 17.4% (15.3 – 19.7%) and 14% (5.9 – 29.9%) respectively. Studies using ultrasound reported higher prevalence of disease. European cohorts tended to have higher prevalence of disease. **Conclusion** This systematic review estimates prevalence of ALD at approximately 1 in 20 people, and prevalence of AC as 1 person in 500. Prevalence varies by geography and by diagnostic modality. The higher prevalence of ALD amongst cohorts with AUD suggests that methods for early detection of liver disease are likely to be better deployed in high risk groups.