Conclusions In these PLWH and elevated ALT, ~20% had CSHF and ~60% had HS. Lower HDL and diabetes were independent predictor of CSHF. Hazardous drinking or MS were identified in most patients with CSHF, however no risk factors were identified in almost 20%. This raises the intriguing possibility that CSHF may be caused directly by the HIV infection.

Introduction Decompensated cirrhosis is a complex disorder with a high mortality rate and as a result re-admissions to hospital are common following discharge. Our aim was to evaluate the impact of implementation of a 'Decompensated Cirrhosis Discharge Bundle (DCDB)' and determine whether this improves the provision of evidence-based care and reduces preventable readmissions.

Methods A baseline review of the management of consecutive patients discharged with a diagnosis of decompensated cirrhosis was conducted in 2017 to assess the management of complications including ascites, encephalopathy, varices and alcohol misuse, and to determine readmission rates. Subsequently the DCDB was developed and implemented. Two cycles of evaluation of the impact of the bundle were conducted, the first using a paper version (Nov 2018-Oct 2019) and the second an electronic version (Nov 2019-Mar 2020).

Results Overall, 225 patients (63% male; median age 55; median MELD 17; 72% alcohol-related) were reviewed. Clinical and demographic features were similar in the 3 review periods. The overall 30 day readmission rate was 30% (12% preventable) in baseline review (n=61) and areas for improvement were identified. In the first review following implementation of the DCDB (n=86) only 23 (27%) had a readmission. This increased to 69% (31/45) in the second review following implementation of the electronic DCDB. A comparison between patients with and without a DCDB is shown in the table 1. Overall, use of the bundle was associated with improved care across all domains assessed.

Abstract PWE-35 Table 1

|                      | DCDB     | No DCDB   | P value |
|----------------------|----------|-----------|---------|
| Alcohol misuse       | 63%      | 64%       | p=0.917 |
| Alcohol team review  | 85%      | 66%       | p=0.044 |
| Thiamine prescribed  | 91%      | 85%       | p=0.552 |
| Community alcohol plan| 62%   | 39%       | p=0.026 |
| HE related admission | 30%      | 42%       | p=0.138 |
| Lactulose prescribed | 94%      | 91%       | p=1.0   |
| Rifaximin prescribed  | 94%      | 84%       | p=0.679 |
| Ascites present      | 70%      | 69%       | p=0.886 |
| Discharge creatinine documented | 66% | 6%        | p<0.001 |
| Plan for U&Es check after discharge | 61% | 36%       | p=0.012 |
| Variceal bleed       | 15%      | 11%       | p=1.0   |
| Beta-blockers, repeat OGD planned or TIPSS | 100% | 89%       | p=0.526 |
| Readmissions within 30 days | 31% | 25%       | p=0.470 |
| Potentially preventable liver related 30-day readmission | 4% | 7%        | p=0.407 |

Conclusion Implementation of a care bundle for patients with decompensated cirrhosis improved provision of evidence-based care at discharge. However, uptake of use of the bundle was slow but increased with an electronic version.

Introduction A metaplasia-dysplasia–carcinoma sequence is the most plausible carcinogenic pathway for gallbladder cancer and although the incidence of gallbladder carcinoma is increasing, little is known about its precancerous lesions. The aim of this study was to determine temporal changes in the prevalence of low-grade dysplasia (LGD), high-grade dysplasia (HGD) and gallbladder adenocarcinoma and associated risk factors.

Methods We retrospectively identified consecutive patients who underwent cholecystectomy between January 2011 and March 2020. Patients were grouped according to histology: no dysplasia; LGD; HGD; and adenocarcinoma. Fitted linear models estimated temporal trends in prevalence and mean age for all histological outcomes. Logistic regression estimated associated risk factors.

Results A total of 5 835 patients were included in the analysis. The prevalence of LGD was 1.47%, HGD 0.17% and adenocarcinoma 0.19%. Prevalence for all diseases increased over time, and mean age at diagnoses decreased over time. In a multivariate logistic regression model, with no dysplasia as the reference group, female sex increased the odds of LGD (OR 4.57, 95% CI 3.07-10.10, p<0.0001). BMI was not associated with disease risk.
Conclusions Our data suggests the prevalence of precancerous gallbladder lesions are increasing in younger patients. Although a risk factor for cholelithiasis, BMI was not associated with disease progression. If occurring in a dysplasia-carcinoma sequence, mean age of diagnoses suggests a progression period of 20 years. Further research is required to explain the significant sex disparity and environmental risk factors for gallbladder dysplasia.

**Introduction** Chronic hepatitis B (CHB), as well as metabolic syndrome (MetS) and its associated risk factors, cause liver inflammation, fibrosis and cirrhosis which may subsequently lead to hepatocellular carcinoma (HCC) \(^1\); \(^2\). The percentage of patients with the concomitant chronic hepatitis B and metabolic syndrome/non-alcoholic fatty liver disease (NAFLD) have significantly increased according to the latest reports, they stated that the prevalence of NAFLD in hepatitis B patients varies from 13.6% to 59.3% \(^3\); \(^4\). However, the ramification of combined diseases on treated chronic hepatitis B patients is yet to be thoroughly explored.

**Methods** With the high number of chronic hepatitis B patients on treatment in our cohort; many have concomitant metabolic risk factors that may increase their risk of NAFLD, liver fibrosis, cirrhosis and subsequently hepatocellular carcinoma as well as cardiovascular risks. We aim to evaluate the extent of metabolic risk factors in our cohort of chronic hepatitis B patients and their relation to liver inflammation, fibrosis as well as renal impairment.

Our main objectives are to describe a demographic of a large cohort of patients who are on treatment for chronic hepatitis B, focusing on metabolic risk factors, to check for correlation between metabolic risk factors and liver inflammation and/or fibrosis, and to understand the effect of clinical practice on those patients.

We conducted a retrospective, descriptive, clinical-based study at Barts Health NHS Trust, London, UK. Patients who are followed for chronic hepatitis B and currently on antiviral treatment were considered for this study as part of a service evaluation. We included patients with positive HBsAg who are on antiviral treatment with undetected HBV DNA viral load. We excluded patients who have other comorbidities that may influence the overall results. For those who met inclusion criteria and on viral suppression, data were extracted from Barts health electronic patient records by SNOMED code with relevant demographic and clinical data including latest hepatic enzymes (ALT, AST), platelet count, Hemoglobin A1C (HBA1C), cholesterol, high density lipoprotein (HDL), transient elastography (TE) results, Biopsies and renal function including Glomerular filtration rate (eGFR) and Serum Creatinine levels. We used IBM SPSS software package v.24.0 for statistical analysis. The number of values (n), median (x̃), as well as Interquartile Range (IQR), were used for describing the data. Association between metabolic risk factors and risk of liver inflammation was assessed by correlation and regression analysis techniques using both Pearson’s correlation (r) and Spearman’s rank correlation along with univariate and multivariate regression analysis.

**Results** Eight hundred and eighty-six patients were identified as chronic hepatitis B patient on Antiviral Treatment. However, fifteen patients were excluded as they were only on Prophylactic Antiviral Treatment due to Positive Hepatitis B Core antibodies. Fifteen percent (n=135) were excluded due to detectable viral replication, and fourteen percent (n=126) were excluded due to other chronic conditions that may interfere with the overall results.

It was recognised in this study that nearly half of included patients were of the middle-aged group with male predominance. Given the marked gender difference in our study population, we would highlight that other gender-related results may get affected by this difference. Another pronounced result was the ethnic distribution in our study population; most of included patients were of African/Other Black/Caribbean, Asian or South Asian descents. This result can reflect the worldwide