Improving outcomes following the temporal changes in the prevalence of gut infection

Abstract PWE-34 Figure 1  Predicting CSHF

(AUROC 0.556 and 0.603 respectively; Abstract PWE-34 Figure 1).

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.

PWE-35 IMPROVED OUTCOMES FOLLOWING THE IMPLEMENTATION OF A DECOMPENSATED CIRRHOSIS DISCHARGE BUNDLE

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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 2020-March 2021).

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% potentially preventable). 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.

Conclusions 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.

PWE-36 TEMPORAL CHANGES IN THE PREVALENCE OF GALLBLADDER DYSPLASIA AND ADENOCARCINOMA IN PATIENTS UNDERGOING CHOLECYSTECTOMY

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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 Acute COVID-19 infection is well-known to cause abnormalities in liver blood tests (LBTs). This study aimed to identify what are the long-term implications of COVID-19 on LBTs.

Methods This is a retrospective cohort study that examined the impact of COVID-19 infection on LBTs both during acute infection and for up to one year following hospital admission in 373 patients. Data analysis was done using Python using the SciPy and NumPy library. R factor was used to identify type of liver injury; hepatocellular, cholestatic or mixed. $\chi^2$ test and Fisher exact was used for statistical analysis with $p<0.05$ being considered significant.

Results During acute infection, 57.5% of patients showed LBT abnormalities with at least one raised liver blood test (ALT, ALP and/or bilirubin). Male patients were significantly more likely to develop LBT abnormalities than were female patients (74.5% versus 25.5%; $p<0.001$). The rate of LBT abnormalities was significantly correlated with severity of COVID-19 infection, such that patients requiring ITU admission were more likely to have abnormal LBTs compared to those treated on a general ward (87% versus 51% respectively; $p<0.001$). During short term follow-up (1-5 months post discharge), LBT abnormalities persisted in 31.3% of patients. LBT abnormalities for up to 12 months in 24.0% of patients. In both the acute setting and long-term follow-up, cholestatic or mixed injury types were most commonly seen (acute; 41.1%, 41.6% respectively, long-term; 50.0%, 44.4% respectively).

Conclusion Our data suggests that up to one in four patients have persistent LBT abnormalities up to one year following COVID-19 infection. Future research is needed to investigate what the clinical significance of this LBT abnormalities is and whether there are interventions, pharmacological or otherwise, that could reduce COVID-19 related liver injury, both in the acute setting, and longer-term.

Keywords COVID-19, coronavirus, hepatology, liver function

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). 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% in HBeAg negative patients. 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 can 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 ($\text{eGFR}$) and Serum Creatinine levels. We used IBM SPSS software package v.24.0 for statistical analysis. The number of values (n), median ($\bar{x}$), and percentage (%), 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 Probablistic 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