Abstract IDDF2019-ABS-0199 Figure 1 Cirrhosis with different Child-Pugh class and estimated glomerular filtration rate eGFR cate

years; 1,590 (35.9%), 2,029 (45.8%), 544 (12.3%) and 268 (6.0%) had baseline eGFR ≥ 90 , 60–89, 45–59 and 30–44 mL/min/1.73 m²; 3,217 (72.6%), 1,028 (23.2%) and 186 (4.2%) were in Child-Pugh class A, B and C at baseline (figure 1). At a median (interquartile range) follow-up of 5.3 (2.0–9.7) years, 1,060 (23.9%) patients developed metabolic acidosis. In Child-Pugh class A, the risk of metabolic acidosis elevated in eGFR < 45 mL/min/1.73 m² (adjusted subdistribution hazard ratio [SHR] 4.02, 95% CI 2.42–6.68, $P < 0.001$). The risk of metabolic acidosis increased in Child-Pugh class B and C at any eGFR levels (adjusted SHR ranged from 4.24 to 91.66), particularly in Child-Pugh class C with eGFR < 30 mL/min/1.73 m² (adjusted SHR 91.66, 95% CI 60.69–138.44, $P < 0.001$).

Conclusions The risk of metabolic acidosis increases with renal and liver impairment in diabetic patients with CHB-related cirrhosis.

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FIRST REPORT OF PRIMARY TENOFOVIR RESISTANCE IN A HEPATITIS B VIRAL HEPATITIS PATIENT FROM INDIA WITHOUT HUMAN IMMUNODEFICIENCY VIRUS CO-INFECTION

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Background Tenofovir confers potent and durable HBV-DNA suppression; Levels of Tenofovir resistance in individuals with viral failure ranged from 20% in Europe to more than 50% in sub-Saharan Africa. Here we are reporting a rare case of primary Tenofovir resistance in a patient with HBV hepatitis without HIV co-infection.

Methods Patients were 59 years old female, HBV DNA level 5.49,1000 IU/ml and transaminase high, kPa scores 8.6, Alpha-fetoprotein level 3.79IU/ml and HBeAg level 111.64.

The patient was started on Tenofovir disoproxil fumarate 300 mg OD. One month HBV DNA level 3210 IU/ml, 6 months HBV DNA level was 674000IU/ml. The patient was on regular follow up, regularly purchased medicine from the hospital pharmacy, took medicine regularly every single day, no history of other medicine intakes. Clinical evaluation and laboratory findings excluded the presence of other systemic diseases, there was no past history of exposure to Tenofovir. HIV and HCV were negative.

Results Tablet Tenofovir stopped, Tablet Entecavir 1 mg started. Report of mutation study and genotyping revealed A181T/V mutation with A194T and M204V/I, these mutations are associated with resistance to Lamivudine, Adefovir, Tenofovir and there was no reported resistance to Entecavir and Telbivudine. After one month, 3 months and 6 months treatment with Entecavir 1 mg daily, HBV DNA level decreased to 3600IU/ml, transaminase level normalized on follow up. Possible our patient acquired drug-resistant Hepatitis B virus from patient with HIV-HBV co-infection, the patient taking antiretroviral, or healthcare worker.

Conclusions This is a First report from India of occurrence of Tenofovir mutation A181T/V, A194T and M204V/I in a non HIV infected patient with HBV hepatitis.

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EXCITING RESULTS WITH INJECTION DARBEPOIETIN ALFA AND PEGFILGRASTIM IN PATIENTS WITH DECOMPENSATED LIVER CIRRHOSIS

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Background Dysregulated erythropoietin (EPO) plasma levels may play a role in the pathophysiology of liver cirrhosis. No report of Darbepoetin alpha and Pegylated Filgrastim use in Liver Cirrhosis.