AZA were included. The incidence of side effects, initial and maximum dose tolerated and discontinuation time was noted.

Blood sample (at-80C) of these patients were retrieved from the biorepository. Age and sex matched group of healthy volunteers served as the control group. DNA was isolated (Qiagen) (n=729 AZA;179 controls). Genotyping of NUDT15C415T(rs116855232;p.R139C) carried out employing Taq-man probes on Real-time PCR(Step one-Life tech).

The association of presence of risk variant of NUDT15 with the development of leucopenia, other side effects and maximum tolerated dosage was evaluated.

**Results** 711/729 patients were included in the analysis (18 inadequate DNA). (IDDF2018-ABS-0269 Table1)

There was no significant differences in the frequency of C/ C, C/T and T/T genotypes of NUDT15p.R139C between AZA group (82.28%; 15.75%–1.97%) and control group (88.27%; 11.73%–0%). 79/711 patients developed leucopenia (26/585 C/C; 42/112C/T; 11/14 T/T). Development of leucopenia was significantly higher in C/T and T/T compared to C/C (OR 15.609; CI 9.198 – 26.490) (table 1). The sensitivity (67.08%) and specificity (88.45%) for predicting AZA-induced leucopenia. C/T group discontinued treatment significantly earlier than C/C genotype.

There was no incidence of hepatitis/pancreatitis in C/T and T/T groups and no significant difference in the mean dose tolerated wild (C/C) vs risk variant (C/T,T/T) carriers(1.33 mg/kg – 1.196 mg/kg;p=0.014).

**Abstract IDDF2018-ABS-0269 Table 1 Base line characteristics of study population results**

<table>
<thead>
<tr>
<th>Age at onset (Median±SD)</th>
<th>28±14.589</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (M/F)</td>
<td>1:54:1</td>
</tr>
<tr>
<td>UC: CD: IBU</td>
<td>358 (50.35%); 323 (45.42%); 304(22%)</td>
</tr>
<tr>
<td>Extent of involvement (UC)</td>
<td>E1:50(Z.97%); E2:197 (55.03%); E3: 111 (31%);</td>
</tr>
<tr>
<td>Disease location (CD)</td>
<td>L1: 117 (36.22%); L2:69 (21.36%); L3:130(40.2%); L4: 7 (2.16%);</td>
</tr>
<tr>
<td>Disease behaviour (CD)</td>
<td>B1: 197 (60.99%); B2:91 (28.17%); B3: 35 (10.8%);</td>
</tr>
<tr>
<td>Smoking</td>
<td>59 (8.29%);</td>
</tr>
<tr>
<td>Age at start of AZA (Median±SD)</td>
<td>35±13</td>
</tr>
<tr>
<td>Duration of AZA therapy in months</td>
<td>21.52±18.20 (1–145)</td>
</tr>
<tr>
<td>Aza induced side effects</td>
<td>22 Gl intolerance; 5 pancreatitis; 79 Leukopenia; 9 skin allergy; 9 Headache; 5 Hepatitis</td>
</tr>
</tbody>
</table>

**Conclusions** NUDT15 risk genotype frequency is around 15% in Indian population and can predict the development of leucopenia. The results suggest thiopurines should be avoided in homozygous T/T and low dose for C/T heterozygous genotypes. This has important implications for clinical practice and is a step towards personalised management of IBD.
study aims to determine the effects of vitamin E taken once a day on the LSM.

Methods NAFLD patients diagnosed by ultrasound who met the inclusion criteria were enrolled in the study. Liver Stiffness Measurement (LSM) measured by FibroScan at baseline and at the end of 3 months. A change in the LSM was the primary objective. Chi-Square analysis was used to measure the change of LSM pre and post-treatment. P value less than 0.05 was considered significant.

Patients were assigned to either the Life style Modification Advice Group (LMAG)–with nutritional counselling and advise to exercise—or the Treatment Group (Vitamin E as Mixed Tocotrienol 100 mg daily for 3 months plus lifestyle modification advise).

Results Fifty-seven percent (38/67) of patients enrolled in both arms of the study improved – with a decrease in their LSM measurements – but 43% (29 of 67 (43%) did not.

Of those who improved, 79% (30/38) were from the Treatment Group (Vitamin E) and 21% (8/38) were from the LMAG.

Twenty-nine (29) patients did not improve; 79% (23/29) from LMAG and only 6/29 (21%) from the Treatment Group. Chi-square analysis showed that treatment with Vitamin E had a significant effect (p≤0.05) on the improvement of LSM.

Conclusions Vitamin E (mixed Tocotrienol) 100 mg daily for 3 months could decrease the LSM among NAFLD patients.

**IDDF2018-ABS-0028**

**INFLUENCE OF HEPATIC STEATOSIS ON THE TREATMENT OUTCOMES OF ENTECAVIR AND TENOFOVIR IN PATIENTS WITH CHRONIC HEPATITIS B**

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10.1136/gutjnl-2018-IDDFabstracts.190

**Background** The influence of hepatic steatosis (HS) on chronic hepatitis B (CHB) is not well-known. We evaluated the influence of HS, assessed using controlled attenuated parameter (CAP) of transient elastography (TE), on the treatment outcomes in CHB patients who initiated antiviral therapy (AVT).

**Methods** Among 1,658 CHB patients who initiated AVT using entecavir or tenofovir between 2007 and 2016, 334 patients with available TE results at the time of initiating AVT were recruited. The cutoff CAP value for the diagnosis of HS was 238 dB/m.

**Results** Of the study population, 146 (43.7%) patients had HS. During the follow-up period (median 38.6 months), 303 (90.7%) and 25 (7.5%) patients experienced complete virological response (CVR) (HBV DNA p=0.380). However, lower CAP value was independently associated with the higher probability of CVR achievement (hazard ratio [HR]=0.996; p=0.004) and HBeAg loss among HBeAg positive patients (HR=0.989; p=0.031). The cumulative incidence of HBeAg loss among HBeAg positive patients was significantly higher in patients without HS than that of patients with HS (p=0.022, log-rank test).

**Conclusions** The HS was not correlated with HCC development in patients who initiated AVT using entecavir and tenofovir. However, HS was negatively correlated with the risk of CVR achievement and HBeAg loss among HBeAg positive patients.

**IDDF2018-ABS-0030**

**CLINICAL AND HISTOLOGICAL FEATURES OF PATIENTS WITH ALCOHOLIC LIVER DISEASE**

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10.1136/gutjnl-2018-IDDFabstracts.192

**Background** Alcohol-attributable burden on global health is increasing, and the relationship between population alcohol consumption and liver-related deaths is strong. Longstanding scientific and clinical work has led to a relatively thorough, if