(MVD). Liver ultrasound was used to diagnose and grade the NAFLD (grade 1–3). NAFLD fibrosis score (NFS) was calculated, and patients were stratified to low, indeterminate, and high probability for advanced liver fibrosis.

**Results** Total of 85 patients with median age of 40 years (IQR 35–43) and predominated by males (84.7%). Thirty-three (38.9%) had ST elevation myocardial infarction (STEMI), 32 (37.6%) had Non-STEMI and 20 (23.5%) had unstable angina. MVD was demonstrated in 36.5%, SVD in 24.7%, mild CAD in 31.8%, and no apparent CAD in 7.1% of the patients. Median Syntax score was 16 (IQR 9.0–22.3). NAFLD was diagnosed in all, 85 (100%) patients, with 13 (15.2%), 36 (42.4%) and 36 (42.2%) patients had grade 1, 2 and 3 liver steatosis respectively. NFS detected low advanced fibrosis probability in 60 (70.6%) patients, indeterminate probability in 24 (28.2%) patients and high probability in only 1 (1.2%) patient. No significant correlation between grades of NAFLD with ACS subtypes (p=0.72), severity of CAD (p=0.882) and Syntax score (p=0.982). No significant association between NFS and ACS subtypes (p=0.232), severity of CAD (p=0.445) and Syntax score (p=0.624, r=0.07).

**Conclusions** NAFLD is highly prevalent in young patients presented with ACS, and it should be routinely screened in our clinical practice. However, in a small cohort, we observed no significant correlation between severity of NAFLD and severity of CAD among young ACS patients.

**Background** Since 1976, the laboratory diagnosis of spontaneous bacterial peritonitis (SBP) has been established by ascetic fluid (AF) polymorphonuclear-leucocyte (PMN) count ≥250/mm³ with or without the AF culture result in cirrhotic patients. We aimed to reevaluate whether the current cutoff count of PMN would be still optimal to diagnose SBP in cirrhotics having a hepatocellular carcinoma (HCC) or not.

**Methods** This preliminary study included 136 consecutive patients having cirrhosis with (n=60) or without HCC (n=76) and the diagnosis of CTPV was made after extensive workup for other diseases, presenting with features of portal hypertension next to cirrhosis. Two disease entities rare entity and usually is a sequela of EHPV. Cavernous transformation of the portal vein is a rare entity and usually is a sequela of EHPV.

**Results** Complete blood count revealed biconcave and adequate platelets with microcytic, hypochromic anaemia. PBS revealed Microcytic cells with hypochromicity, anisocytosis and poikilocytosis. Liver function tests, Hepatitis B and C screening were negative. Fibroscan of the liver revealed intermediate fibrosis. Colour flow Doppler ultrasound of the abdomen is compatible with portal hypertension of the portal vein and splenomegaly. MRI of the whole abdomen revealed portal hypertension with marked splenomegaly, cavernous transformation of the portal vein and collateral vessel formation. There’s also focal biliary ectasia and cholecytolithiasis. Repeat gastroscopy revealed 3 esophageal varices.

**Conclusions** In conclusion, NCPH is the most common cause of portal hypertension next to cirrhosis. Two disease entities in NCPH, namely NCPF/IPH and EHPVO are distinct diseases, presenting with features of PHT – variceal bleed, splenomegaly and near normal liver functions. Likely pathogenesis is early age, portal inflammation/infection in a prothrombotic individual although we still have to work up this patient for thrombotic risk. The diagnosis needs exclusion of cirrhosis in NCPF/IPH and presence of portal cavernoma in EHPVO. Slow hepatic dysfunction due to parenchymal extinction and portal biliopathy is a late event in EHPVO. Effective management of PHT and its complications results in excellent 5 and 10 years survival.