**Background and Aims:** Limited data are available on hepatic steatosis (HS) among human immunodeficiency virus (HIV) patients uninfected with hepatitis C virus (HCV). Little is known about the relationship between cardiovascular (CVD) risk and HS in HIV mono- and/or HCV co-infected patients.

**Aims** of the study were to assess prevalence of HS and its risk factors in HIV-patients and to evaluate whether HS correlated with advanced liver fibrosis (ALF) and/or CVD risk.

**Methods:** Fifty seven HIV mono- and 61 HIV/HCV co-infected patients were consecutively enrolled. The “bright liver echopattern” (BL) was used for HS diagnosis. ALF was defined as “liver stiffness” ≥ 9.5 kPa by transient elastography (TE). Main parameters of liver function, glycaemia, total cholesterol (T-Chol), triglycerides (TG), HDL-C, HIV and HCV viral load, duration of highly active anti-retroviral therapy (HAART) and CD4+ cell count were recorded. CVD risk was appreciated using the 10-year Framingham risk score (FRS) and the diagnosis of metabolic syndrome (MS) performed according to ATP III criteria.

**Results:** In the whole HIV-population HS prevalence was 52.5% (54.4% in HIV mono- and 50.8% in co-infected patients (P = not significant). HS was associated with lipodistrophy, TG values (P < 0.0001), MS (P < 0.0004) and T-Chol levels (P < 0.001) in both HIV-groups (table). In HIV mono-infected patients HS was linked with HAART exposure > 1 year (P < 0.01). By multivariate analysis only TG levels (P < 0.02) and FRS (P < 0.05) were independently associated with HS in both HIV groups. No correlation was observed between HS and ALF, measured by TE.

**Conclusion:** HS was common in HIV-patients occurring in about half of the population. HS was linked with FRS but was not correlated with ALF. The lack of correlation between HS and ALF may be due to the limited number of patients. Several studies have reported the association of HS with multiple CVD risk factors, including MS and IR. HS and CVD shared several risk factors in our HIV patients and for this reason we suppose that HS should be considered as an early marker of CVD even in this population.

### Table 1. Diagnostic performance of CAP

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total HIV</th>
<th>BL− (n = 56)</th>
<th>BL+ (n = 62)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lipodistrophy, n (%)</td>
<td>23 (41%)</td>
<td>47 (76%)</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>MS, n (%)</td>
<td>5 (9%)</td>
<td>23 (37%)</td>
<td>&lt;0.0005</td>
<td></td>
</tr>
<tr>
<td>T-Chol, mg/dl.</td>
<td>170 ±39</td>
<td>201 ±53</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>TG, mg/dl. (range)</td>
<td>111 (50–380)</td>
<td>180.5 (49–615)</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
</tbody>
</table>

**Conclusion:** HS was common in HIV-patients occurring in about half of the population. HS was linked with FRS but was not correlated with ALF. The lack of correlation between HS and ALF may be due to the limited number of patients. Several studies have reported the association of HS with multiple CVD risk factors, including MS and IR. HS and CVD shared several risk factors in our HIV patients and for this reason we suppose that HS should be considered as an early marker of CVD even in this population.

### 1304

**THE PERFORMANCE OF CONTROLLED ATTENUATION PARAMETER (CAP) FOR THE NON-INVASIVE EVALUATION OF STEATOSIS USING FIBROSCAN®: PRELIMINARY RESULTS**

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**Background and Objective:** Steatosis is a common histological finding in patient with chronic liver disease (CLD). Up to now liver biopsy (LB) was the gold standard to diagnose steatosis. Controlled Attenuation Parameter (CAP) is a novel non-invasive parameter measuring the attenuation of ultrasound generated by Fibroscan®. We aim to assess diagnostic value of CAP in predicting steatosis in CLD. To our knowledge, this is the first independent validation study on the CAP vs liver biopsy.

**Aim** was to assess whether CAP could be used as a non-invasive diagnostic method for the detection of steatosis in patients with chronic liver disease (CLD).

**Methods:** We recruited 71 patients enrolled from the clinic of Hepatology and Gastroenterology at University Hospital, Palermo, Italy. The diagnosis of steatosis was based on liver biopsy. The mean age of the patients was 50.07 years, 73.2% females, who underwent LB and Fibroscan® (both CAP and liver stiffness measurement) were enrolled. The mean number of portal spaces and the mean length of liver biopsy specimens were 13 and 15 mm respectively. Steatosis was assessed by a single expert pathologist, blinded to Fibroscan® and clinical data as: S0: steatosis in less than 5% of hepatocytes, S1: 6–32%, S2: 33–100%. The diagnostic performance of CAP was assessed using sensitivity (Se), specificity (Sp), positive (PPV) and negative predictive value (NPV), positive (+LR) and negative (−LR) likelihood ratios and area under ROC curves (AUROC).

**Results:** There were 35 patients with S0, 26 with S1 and 10 with S2. The mean CAP value was 231.94 ±52.37 (201.50±35.85 in S0 patients, 247.96±44.33 in S1, respectively 293.80±48.60 in S2, p < 0.0001). CAP values were significantly different between steatosis grades (S0–S1: p < 0.0001, S1–S2: p = 0.011, respectively S0–S2: p < 0.0001).

Diagnostic performance of CAP to detect the different grades of steatosis is summarized in Table 1.

**Conclusion:** CAP is a promising non-invasive tool to detect steatosis in CLD patients.
inflammatory markers (high sensitivity CRP, interleukins-6 and -8, TNF-α and adiponectin) were available for all patients. Liver stiffness measurement (LSM) by transient elastography was performed in 39 and liver biopsy in 29 patients. Histological lesions were evaluated blindly by a single liver histopathologist (Brunt’s classification).

Results: Among anthropometric and body composition indices, MedDietScore was inversely correlated with waist circumference (r = -0.269, p = 0.047) and tended to correlate with visceral fat (r = -0.242, p = 0.075). With regard to biochemical and inflammatory markers, MedDietScore was positively correlated with log-HDL (r = 0.287, p = 0.034) and log-adiponectin (r = 0.304, p = 0.024) and negatively with insulin levels (r = -0.369, p = 0.006) and insulin resistance index-HOMA (r = -0.381, p = 0.004). MedDietScore tended to correlate also with interleukin-8 concentrations (r = -0.241, p = 0.089). The above correlations with biochemical and inflammatory markers remained significant after adjustment for waist circumference. MedDietScore inversely correlated with LSM (r = -0.353, p = 0.028), histological stage (rho = -0.554, p = 0.007) and histological severity of steatosis (r = -0.518, p = 0.013). Patients with simple fatty liver reported significantly higher MedDietScore compared to those with steatohepatitis-NASH (34.3±4.6 vs. 28.9±3.2, p = 0.003).

Conclusions: Greater adherence to the MD is associated with lower insulin resistance and higher HDL and adiponectin levels and is also associated with less hepatic steatosis and most importantly less liver fibrosis, estimated by both transient elastography and liver histology. Moreover, lower adoption of the MD is observed in patients with NASH compared to those with simple fatty liver.

1306  
METABOLIC SYNDROME AND SURROGATE MARKERS OF INSULIN RESISTANCE IN NONALCOHOLIC FETTY LIVER DISEASE (NAFLD), CHRONIC HEPATITIS C (CHC) AND B (CHB) IN BULGARIA

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It is well known that insulin resistance (IR) and metabolic syndrome (MetS) correlated with the progression of liver disease in patients with NAFLD and chronic hepatitis C (CHC), but the comparison between both diseases, as well as with chronic hepatitis B (CHB) is not established yet. In this study we compared the prevalence and signs of MetS and surrogate markers of IR in patients with NAFLD, CHC and CHB, and healthy volunteers. The parameters of MetS, fasting and during OGTT glucose and insulin were investigated in patients with NAFLD (n=250), genotype 1 CHC (n=366), CHB (n=334), and 211 healthy volunteers. The risk for coronary heart disease according Framingham Risk Score (FRS) was also assessed. MetS was more frequent in NAFLD (52%) and CHC (50.5%) compared to CHB (33%) and healthy volunteers (31%, p <0.01). The mean fasting glucose level was higher in NAFLD cases, but frequency of DM was similar in NAFLD group (32%), and CHC (30%), and lower – in CHB (20%). Impaired fasting glucose, impaired glucose tolerance and diabetes mellitus as well as increased levels of the fasting insulin and those on OGTT, and HOMA-IR were presented mostly in patients with steatosis, and those patients with CHC (p <0.001). The mean FRS was significantly higher in NAFLD (p<0.001) and CHC with metabolic steatosis (p<0.01) compared to CHC without fatty liver, CHB and healthy volunteers.

In conclusion the metabolic disturbances of patients genotype 1 CHC with steatosis are similar to those in NAFLD, and more frequent and intensive than in CHB.

1307  
HIGH LEVELS OF URSODEOXYCHOLIC ACID ACT AS FXR ANTAGONIST AND DEPELTE LIVER CHOLESTEROL DUE TO INCREASED BILE ACID SYNTHESIS IN MORBIDLY OBESE PATIENTS

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Aim: Ursodeoxycholic acid (UDCA) was shown to improve insulin resistance and steatosis in mice. The efficacy and possible modes of action of UDCA treatment in human non-alcoholic fatty liver disease (NAFLD) have been debated. We aimed to explore potential mechanism of UDCA action in patients with morbid obesity awaiting Roux-en-Y gastric bypass surgery.

Methods: Forty morbidly obese patients were randomized to UDCA (20 mg/kg/day three weeks before surgery) or no treatment (controls). Serum liver function tests, lipids, bile acids and markers of insulin resistance/diabetes (OGTT, HOMA) were obtained before and after treatment. During surgery, biopsies were taken from the liver for histology and gene as well as protein expression studies.

Results: Three patients dropped out; UDCA 1 (diarrhea), controls 2 (pregnancy, bleeding). Completers of both groups were well matched by gender (female, 68.4 vs. 77.7%), age (42.8±12.3 vs. 38.5±10.1 years), BMI (41.9±4.6 vs. 40.6±3.9 kg/m²), HOMA (5.1±2.5 vs. 6.6±3.9) and OGTT (IGT or T2DM, 37% vs. 50%). NAS scores were: no, 11 vs. 13; borderline, 4 vs. 4; definite 3 vs. 4. UDCA despite significantly (p<0.05) expanding the BA pool 10.6±7.6 fold (≤ 55.3 mmol/L; UDCA >90%) increased bile acid synthesis as measured by serum C4 (7α-hydroxy-cholest-4-ene-3-one), CYP7A gene expression, and serum levels of primary bile acids CDCa and CA. Circulating FGFl9 decreased by 18% (p=0.05). Significant increases in gene expression levels of key regulators of lipid turnover (SREBP2, SCD, HMGCR) were reflected by significantly decreased serum LDL-cholesterol and increased triglycerides. UDCA significantly decreased ALT, AST and γGT but did not affect HOMA, glucose tolerance, adiponectin and lectin.

Conclusion: Changes in serum lipid and gene expression profiles in UDCA treated, morbidly obese patients indicate hepatic cholesterol depletion as a result of increased bile acid formation due to FXR-antagonistic effects of very high UDCA levels in noncholestatic livers.

1308  
THE FIB-4 SCORE RELIABLY EXCLUDES ADVANCED FIBROSIS IN A DIABETIC COHORT WITH NAFLD

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Background: 20–30% of the population are estimated to have NAFLD. Amongst these, type 2 diabetics are recognised to be at highest risk of advanced steatohepatitis. The EASL position statement on NAFLD supported screening diabetics for NAFLD. There is a need for a non-invasive tool to screen diabetics for advanced fibrosis. The FIB-4 score has been shown to be effective in a general NAFLD cohort; however, the relative merits of this and similar scores have not been determined in a diabetic population.

Aim: To assess performance of simple non-invasive tests for fibrosis in diabetic patients with biopsy-proven NAFLD.

Methods: Patients who were reviewed in our hospital fatty liver clinic between 1999–2009 were included. Liver biopsies were assessed using the Kleiner score. The FIB-4 and NAFLD fibrosis scores were calculated from blood tests taken within 6 months of liver biopsy.