Background and Aims: Non-alcoholic fatty liver disease (NAFLD) is the most common liver disease in the world; its prevalence has increased recently, accompanied by global obesity pandemic. It is a complex entity that arises from numerous genetic, environmental, behavioral and social factors. Non-alcoholic steatohepatitis (NASH) is part of the disease progression and is a preamble to more severe complications such as cirrhosis and hepatocellular carcinoma. Currently, the only tool to diagnose NASH is liver biopsy. The aim is to identify the different microRNAs (miRNAs) involved in NAFLD progression.

Method: 117 patients were recruited; liver biopsy and a blood sample were obtained; 30 were submitted to microarray assays and classified according to controlled attenuation parameter (CAP) and NAFLD activity score (NAS). Patients with CAP ≤ 232 dB/m, 0-point NAS and histopathological report without alterations, were the control group; with CAP ≥ 290 dB/m, NAS with 1 to 3 points and histopathological report with steatosis in more than 5% of hepatocytes, were the NASH group. Samples, liver function tests, as well as fasting cholesterol, triglycerides, and glucose levels, were determined. RNA was extracted from liver tissue to analyze the miRNAs differential expression using the GeneChip miRNA 4.0 microarray; expression levels were compared with the Affymetrix TAC software using a fold change parameter ≥ 2 and FDR ≤ 0.05; FDR ≤ 0.001.

Results: Regarding the anthropometric characteristics, BMI had a significantly higher levels of CXCR6 expression compared to patients with NASH. Conversely there was a trend towards higher expression of CXCR6 on Th1 cells (CD4+CXCR3+) sampled from patients with NASH compared to those without (p = 0.059).

Conclusion: To our knowledge, this is the first study of immune cell phenotype in multiple anatomical compartments in this patient group. We find evidence of a pro-inflammatory, liver-homing phenotype in peripheral blood and adipose tissue and a dominant type 1 immune phenotype in all 3 compartments. Further investigation of the role of the adaptive immune system in the pathogenesis of NASH is warranted.