GUT MICROBIOTA AND ITS ASSOCIATION WITH SMALL INTESTINAL BACTERIAL OVERGROWTH IN PATIENTS WITH HYPERLIPIDEMIA

K. Kvit¹, N. Kharchenko²

¹Danylo Halytsky Lviv National Medical University, Therapy №1 and Medical Diagnostics, Lviv, Ukraine,²Shupyk National Healthcare University of Ukraine, Gatroenterology, Dietology and Endoscopy, Kyiv, Ukraine

Abstract category: 4.2.: Nutrients and gut function

Introduction: The variety of microbes colonizing the human gut is almost 10 times more than the total cells in a human. Current evidence suggests that the intestinal microbiota is involved in cardiovascular disease occurrence with the host-microbe interaction regulating metabolic pathways. Recent studies suggest that the characteristics of the gut microbiota are altered in hyperlipidemia patients, and also, that small intestinal bacterial overgrowth (SIBO) contributes to the pathogenesis of this condition. However, such associations remain poorly investigated and characterized. The LPS from intestinal flora bacteria can induce a chronic subclinical inflammatory process with further atherosclerosis development. Additionally, microbiota injuries through activation of TLR4 pathway could impact on lipid metabolism in the liver.

Aims & Methods: The aim of this study was to examine the composition of gut microbiota and its correlation relationship with SIBO in patients with hyperlipidemia. TLR4 serum level was examined as one of the proinflammatory factors that could play a substancial role in gut-liver-cholesterol axis.

105 patients with hyperlipidemia (average age 42.52±2.6) with an average BMI of 26.08±0.81 were examined in Danylo Halytsky Lviv National Medical University (Lviv, Ukraine). 52 control subjects (average age 37.38±2.6), an average BMI of 24,01±0.75. All control subjects had normal lipid range and no history of coronary disease. Determination of microbial composition at the level of major microbial phyla was carried out by identification of Bacteroidetes, Firmicutes, and Actinobacteria DNA with quantitative realtime PCR (qRT-PCR), using gene-targeted primers. The examination of lactulose breath test was proved to patients of both groups. Quantative detection of TLR4 in the serum was realized by using the Cusabio Elisa kit.

Results: The composition of microbiota was significantly different in separate groups of species between patients with and withour hyperlipidemia. Bacteroidetes in main group amounted to $40.70\pm25.43\%$, Firmicutes – $39.43\pm23.77\%$, Actinobacteria – $8.95\pm9.41\%$, while in the control group - Bacteroidetes $52.98\pm13.62\%$, Firmicutes $33.85\pm12.47\%$, Actinobacteria – $5.36\pm1.76\%$.

Firmicutes/Bacteroidetes index in the main group averaged 2.98 ± 1.61 , in control – 0.75 ± 0.54 . Negative correlation between Bacteroidetes and Firmicutes (r=-0.84), Bacteroidetes and Actinobacteria (r=-0.52), and Bacteroidetes and Firmicutes/Bacteroidetes index (r=-0.74) was marked in patients of the main group. Additionally, there was a positive correlation between tryglicerides, cholesterol and Actinobacteria (r=0,48). SIBO prevalence was 58,5% in main group, while in control - 31%. TLR4 serum level was 2.67±1,05 ng/mL in hyperlipidemia group, 1.23±0.99 ng/mL in controls (normal range 1,25-2,5 ng/mL).

Conclusion: There is an essential role of microbiota in lipid metabolism due to the fact of SIBO prevalence in patients with hyperlipidemia in comparison with the control group (58,5% vs. 31%). Moreover, the TLR4 serum level that is usually activated by LPS as the result of SIBO was higher more than the normal range in patients with hyperlipidemia (2.67±1,05 ng/mL). Additionally, the composition of the microbiota was different between both groups - Actinobacteria and F/B index were significantly higher in patients with hyperlipidemia. Actinobacteria was in correlational relationship with triglycerides and cholesterol level.

Nothing to disclose: Yes

Keyword 1: microbiota

Keyword 2: cholesterol

Keyword 3: TLR4

1. I have read and I accept the Privacy Policy and General Terms and Conditions: Yes

2. I confirm that I previewed this abstract and that all information is correct. I accept that the content of this abstract cannot be modified or corrected after the submission deadline and I am aware that it will be published exactly as submitted.: Yes

3. I confirm that the submission of the abstract constitutes my consent to publication (e.g. conference website, programmes, other promotions, etc.).: Yes

4. I herewith confirm that the contact details saved in this system are those of the corresponding author, who will be notified about the status of the abstract. The corresponding author is responsible for informing the other authors about the status of the abstract.: Yes

Has this abstract been presented at a national meeting?: $\ensuremath{\mathsf{No}}$

Has this abstract been presented at DDW/AGA/AASLD?: No

Has this abstract been presented at another international meeting?: No

Has this abstract been previously published?: No

This abstract should be considered as Translational/Basic Science abstract: $\ensuremath{\mathsf{No}}$

This abstract should be taken into consideration for the Today's science; Tomorrow's medicine symposia on the topic: 'Immunity and GI disorders': No

This abstract should be considered as Paediatric abstract: No

Travel grant application: Yes