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Summary

Inflammatory bowel diseases (IBD) – Crohn's disease (CD) and ulcerative colitis (UC) are associated with various extraintestinal manifestations (EM). These manifestations may involve virtually any organ system. Neurological manifestations are not regarded as very common, but they are probably underdiagnosed. Patients with IBD have higher risk of developing multiple sclerosis (MS) regardless of therapy manner they use. There are quite a lot of publications describing relationship between developing multiple sclerosis and therapy with monoclonal antibody against tumor necrosis alpha (anti TNF- α). However, it remains to be shown whether anti-TNF-alpha itself causes MS or merely acts as a trigger, exacerbating preexisting subclinical CNS damage during chronic anti- TNF- α treatment. Majority of published cases support the second possibility. These results let us to define a hypothesis, that preselection of patients with IBD without any preclinical signs of demyelination before the initiation of anti TNF- a therapy may improve patient safety. The aim of our project was to develop neurological and magnetic resonance (MRI) screening of patients with IBD before anti TNF- α therapy initiation and subsequently exclusion of patients with high risk of developing MS. The second aim was to follow up on the neurological and magnetic resonance imaging (MRI) status of patients with IBD who were undergoing long-term therapy with anti- TNF- α and clarify the relationship between anti TNF- α therapy and developing MS in patients with IBD.

50 patients with Crohn's disease were included in the prospective phase of the study and observed for more than two years. Among these patients, 30 were treated with anti-TNF- α , whereas in the remaining 20, biological treatment was not indicated and they were followed as controls. All these patients underwent detailed examination at baseline and after approximately 1,5 year. 2 patients with signs of demyelination

were excluded from anti TNF- α therapy. One of them fulfilled McDonald criteria for MS. The second patient is observed with the diagnosis of radiologic isolated syndrome (RIS) without any clinical signs of MS to date. There were no significant changes in neurostatus or MRI findings either in the treated or control group after 1,5 year of observation period.

After evaluation of these results we performed statistic analysis of 97 patients with Crohn's disease, who were examined and observed in our department between 2008 and 2017. We haven't found any new case of demyelination in the group of patients on anti TNF- α therapy (78 patients).

The results of both analysis support our hypothesis, that long term anti TNF- α therapy in patients without any preclinical signs of

demyelination seems to be safe. Neurological screening of patients with IBD before the beginning of anti TNF- α can increase the safety of this treatment. Simultaneously we have confirmed higher incidence of demyelination among IBD patients regardless the therapy manner.

Key words – demyelination, multiple sclerosis, inflammatory bowel disease, Crohn disease, tumor necrosis factor alfa.