There were no significant intergroup differences in the prevalence of hypertension, coronary artery disease, obesity, hyperlipidemia, prior stroke, and diabetes mellitus (p>0.05). However, patients with LAAT had a higher prevalence of prior MI (10% vs 2.8%, p=0.01), renal impairment (mean eGFR 71.2 [54; 84] vs 90.0 [64; 103], p=0.015), and heart failure with reduced EF (40% vs 7.2%, p<0.001).

According to the results of TTE, patients didn't have significant differences in left atrial diameter (46.8 [43; 50] mm vs 44.2 [41; 46] mm, p=0.06) and left atrial volume (74.6 [61; 85] mm3 vs 63.8 [52; 72] mm3, p=0.056). However, the left atrial volume index (LAVI) was higher in patients with LAAT (37.6 [31; 42] mm3 vs 30 [24; 36] mm3, p=0.007). Moreover patients with LAAT had lower left ventricular ejection fraction (LVEF) (49.2 [44; 60]% vs 57.4 [55; 62]%, p=0.019) and higher right atrial area (26.7 [23; 30] mm2 vs 23.8 [21; 27] mm2, p=0.02).

It is interesting to say that patients didn't have differences in values of end-diastolic volume of the left ventricle (54.8 [51; 58] mm vs 52 [48; 56] mm, p=0.22), but patients in the LAAT group had significantly higher end-systolic volume of the left ventricle (41.6 [33; 45] mm vs 36.1 [32; 40] mm, p=0.03) and left ventricular mass index (147.4 [119; 164] g/m2 vs 121.3 [99; 136] g/m2, p=0.003).

Conclusion. Patients with LAAT had higher values of left atrial and right atrial diameters and indices, as well as lower LVEF. Further use of those parameters could help predict LAAT development in patients with non-valvular AF.

ЛИТЕРАТУРА

1. Identifying Patients at High Risk of Left Atrial Appendage Thrombus Before Cardioversion: The CLOTS-AF Score / L. Segan [et al.] // J Am Heart Assoc. – 2023. – Vol.12, №12. – P. e029259. doi: 10.1161/JAHA.122.029259.

2. CHA_2DS_2-VASc score stratifies mortality risk in patients with and without atrial fibrillation / C.S. Harb [et al.] // Open Heart. – 2021. – Vol. 8, №2. – P. e001794. doi: 10.1136/openhrt-2021-001794.

CEREBRAL TOXOPLASMOSIS IN PRIMARY DIAGNOSED HIV-INFECTION (CLINICAL CASE)

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Introduction. *Toxoplasma gondii* belongs to the phylum Apicomplexa, which is a diverse group of protists. They are mostly intracellular parasites and may cause potentially serious disease in animals and humans. Almost 25% of the patients with

HIV infection and along the disease's natural course latent *T. gondii* infection will gradually progress into cerebral toxoplasmosis. Epidemiological studies by Ze-Dong Wang *et al*., 2017 reveal that if a late diagnosis is made, the results show considerable morbidity and mortality.

Aim of the study: presenting a clinical case of Cerebral toxoplasmosis in a patient with primary diagnosed HIV-infection.

Materials and methods. Our study was performed by collecting relevant patient data from the department, retrospective analysis of patients data and information from online journals.

Results and discussion. A 37-year-old male patient presented to hospital with complains of productive cough with sputum, runny nose, temperature of 38^0C, series of generalized convulsive seizures and generalized weakness. The patient had previously visited the district therapist and was prescribed cefuroxime which was ineffective. X-ray examination of the thorax, no pathology was identified. Patient's condition gradually worsened over the following few days, and then was admitted to the nearest hospital.

On physical examination, he was found to have pale skin, had a GCS of 14 with slurred speech and answered questions incorrectly and with delay. He was disorientated in time and place. His vitals: temperature: 36.9°C, blood pressure: 125/75mmHg, heart rate: 89 bpm, respiratory rate: 18 within the normal limits. On palpation of the lymph nodes, posterior cervical, anterior cervical, submandibular were up to 0.5 cm in diameter and not adherent to surrounding tissues, palpation was painless. On neurological examination, rigidity of occipital muscle, Kernig's and Brudzinski's signs were negative, but he was found to have right-sided spastic hemiparesis.

ELISA for HIV IgG antibodies was performed and became positive. HIV-infection was confirmed by RNA HIV detection – viral load 2.9×10^5 cop/ml. Severe immunosuppression with CD4+T-lymphocytes 12cells/µl was revealed. Computer tomography of brain was performed and there were changes left parietal lobe, the changes were non-specific to inflammation or ischemia. Therefore, MRI was performed and showed typical changes for toxoplasmosis. ELISA confirmed the presence of toxoplasmosis IgG antibodies.

Clinical diagnosis: HIV-infection, clinical stage 4 toxoplasmic meningoencephalitis, Candidiasis of the oral mucosa. Severe weight loss (weight loss of more than 10% of body weight).

Co-trimoxazole 480 mg (8 tablets/day) was then immediately prescribed as initial therapy and was continued 6 weeks. He was then started on Acryptega (Dolutegravir/ Tenofovir/ Lamivudine-50/300/300) (1 tablet/day) after two weeks of Co-trimoxazole therapy till present (lifelong) and for secondary prophylaxis Co-trimoxazole 480 mg (4 tablets/day) was given on 22.01.24 until CD4 >200cells/µl and will stay on this level at least 3 months.

Conclusion. Clinical case of late HIV infection diagnosis in 37-year-old patient manifested as acquired cerebral toxoplasmosis with rapid progression of the disease attributed the severe immunosupresion. Treatment of this patient according to the

above protocol showed significant improvement in patient's mental status and eventually became stable. He was advised to receive lifelong ART and continuous monitoring of CD4+ counts and viral load (HIV status).

CHANGES OF INTESTINAL MICROBIOME AND THERAPEUTIC STRATEGIES FOLLOWING A SEVERE BURN

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Introduction. Serious side effects from severe burns include multiple organ failure, infection, and a high mortality rate. The greatest microbial resource in the individual's body, the intestinal microbiome, is heavily involved in this pathological mechanism. Following a serious burn, it is usual to have gut dysbiosis and breakdown of the intestinal epithelial barrier, which can result in microbes' translocation into the circulation and adjacent organs.

Aim of the study. Thus this research detailing alterations in the intestinal mucosal barrier's function and the gut flora following severe burns. And also discussed the possibilities and difficulties associated with microbial treatment.

Materials and methods. A comprehensive analysis of the publications discussing dysbiosis of the intestinal microbiota ,alterations in the intestinal mucosal barrier and therapeutic approaches after a severe burn, was carried out.

Results and discussion. Dysbiosis of intestinal microbiota following burns. Following a severe burn obligatory anaerobes and Bifidobacterium, which decreased over time, and opportunistic pathogens including Escherichia coli and enterococci, which greatly increased in number. Following the course of therapy, the gut microbiota started to remodify. As a result, the majority of opportunistic infections declined, eventually returning to normal. While helpful bacteria steadily proliferated [1].

During the initial phases of burn gut dysbiosis increased and some probiotic microorganisms, such butyrate-producing bacteria, may have decreased, while potentially harmful bacteria increased. Research revealed that in the early post-burn stage, Proteobacteria was abundant, mostly accompanied by an increase in Escherichia and Shigella species and a drop in Firmicutes/Bacteroidetes levels [3].

Changes in intestinal mucosal barrier. The development and incidence of mechanical barrier damage brought on by severe burns are either directly or