Assessment of endothelial dysfunction in patients with ankylosing spondyloarthritis due to previous coronavirus infection

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Introduction. The priority of vascular damage in the pathogenesis of ankylosing spondyloarthritis (AS) is one of the current issues. Damage to the vascular endothelium in the early stages of ankylosing spondyloarthritis, changes in the hemostasis system with the subsequent development of atherosclerotic processes in the vessels correlate with the severity of the disease. We studied the severity of endothelial dysfunction by determining endothelin- 1 in serum and determining the vasoregulatory function of the endothelium by Doppler ultrasound diagnostics of the brachial artery.

Materials and methods. The study involved 80 patients diagnosed with ankylosing spondyloarthritis who underwent COVID-19. The average duration of AS disease was 8.3 ± 0.6 years. Patients were divided into the main group - 40 patients with AS and confirmed IgG analysis for COVID-19 established by ELISA (mean age 55.2 ± 1.3 years, 36 men and 14 women), control group - 40 patients with AS who did not tolerate COVID-19 (mean age 53.5 ± 1.6 years, 30 men and 10 women). All patients underwent general clinical, laboratory and instrumental diagnostic methods, assessment of disease activity according to the VAS scale and BASDAI, BASMI, ASDAS indices to establish the diagnosis of AS. To determine endothelin-1 as the main marker of endothelial dysfunction, the patients' venous blood serum was taken and examined by ELISA. ELISA KIT for ENDOTHELIN-1 (USA) was used as a reagent. To determine the vasoregulatory function of the endothelium, all patients underwent Doppler US diagnosis of the brachial artery with a reactive hyperemia test. The study was conducted two times with the application of the tonometer cuff in the projection of the brachial artery at the level of 5 cm above the ulnar fossa, air injection up to 200 mm Hg and the determination of flow-dependent vasodilation before and after the sample.

Results. Analysis of the obtained results of both groups showed the highest disease activity in the main group according to the VAS scale, BASDAI, BASFI, ASDAS 7.2 ± 0.4, 8.2 ± 0.6, 7.4 ± 0.5, 4.2 ± 0.9 indices, respectively. While control patients had low BASDAI, BASFI, ASDAS, and VAS scales 4.3 ± 0.2, 3.1 ± 0.8, 2.1 ± 0.7, 5.1, ± 0.4 ($p < 0.05$), respectively. Endothelin-1 was high in both groups - 231± 1.8 pg/mL and 187,5± 1, 6 pg/mL ($p < 0.05$), but an increase of 1.5 times was observed in the main group than in the control group. Flow-dependent dilation rates were lower in the main group than in the control group (11.1 ±5.6 and 16.1±5%; $p<0.03$). A direct correlation relationship was established between flow-dependent dilation and endothelin-1 scores in the main and control groups ($r = 0.55$, $p = 0.003$; $r = 0.22$, $p = 0.04$, respectively).

Conclusion. Endothelial dysfunction is a leading pathogenetic mechanism for the development of vascular inflammation in patients with ankylosing spondyloarthritis. Coronavirus infection increases the severity of endothelial dysfunction, leading to irreversible disorders of the hemostasis system, atherosclerotic vascular changes up to the development of circulatory insufficiency, which further affects the quality of life and the development of formidable complications.