**AFTER**

The current treatment for rheumatoid arthritis (RA) includes many medications with various chemical structures and pharmacological properties. Suppression of the inflammation progress is their common mechanism of action.

Methotrexate (MT) plays a special role among the advanced medicines used to treat RA and other rheumatoid and non-rheumatoid inflammatory diseases.

Regarding other effects of MT on the cardiovascular system, some studies show that patients with RA that have been treated with MT show an approximately 60% decrease in cardiovascular mortality rate compared with patients that received other basic anti-inflammatory medications. Therefore, the favorable impact of MT on the cardiovascular system (CVS) may be linked to the main pharmacological effects of this medicine. Specifically, this includes the increase in adenosine formation. It has been confirmed that when adenosine interacts with macrophage adenosine receptors, it activates enzymes that participate in the metabolism and transport of cholesterol from the vascular wall to the liver.

Furthermore, previous research suggests that for the patients with RA that already have atherosclerotic vascular damage, the use of MT is associated with an increased risk of cardiovascular mortality.

Additionally, there are no references in the literature on the MT impact on the functional state of CVS. Specifically, no cardiodynamic data exists on the progress of cardiovascular damage in patients with RA.

Therefore, the purpose of this investigation was to study the MT impact on the endothelial dysfunction indices in patients with RA.

Experimental materials and methods. We examined 102 patients with RA that underwent inpatient treatment at the Rheumatology Department of the National Rheumatology Center, which is based at the I Clinic of TMA. The RA diagnosis was confirmed using the classification criteria proposed by the American Association of Rheumatology (AAR). The control group consisted of 20 healthy middle-aged individuals.