Heart failure (HF) is a syndrome of subjective and objective symptoms, underpinned by structural and/or functional disorders of the heart muscle. Unfortunately, its prevalence is increasing in our society (1-2% of the population). In the pathophysiology of HF, neurohormonal system disorders such as the renin-angiotensin-aldosterone system, vasopressin, and natriuretic peptides play a leading role. The natural course of the disease involves gradual worsening of the patient's condition with periods of acute cardiovascular decompensation. In about 80% of cases, patients present with fluid overload/congestion, requiring intensive treatment. However, the use of diuretics does not improve long-term prognosis. Moreover, during the course of the disease and intensive diuretic treatment, hyponatremia may occur, which is associated with a poorer prognosis. Therefore, despite advances in knowledge about HF, it remains associated with high mortality (about 20% within a year) and a high frequency of unplanned hospitalizations in the long-term (about 13 hospitalizations per 10 patients per year).

As a result of many years of research aimed at developing treatment methods to improve this unfavorable outcome, the effectiveness of several groups of drugs in improving long-term prognosis has been proven. According to the latest recommendations, these drugs should be used in the maximum doses tolerated by the patient. Unfortunately, the implementation of this therapy is far from effective. Registry data show that only about 40% of patients use the full set of recommended drugs. One of the main reasons for this is the tendency toward hypotension in patients, which prevents the initiation or increase of these drug doses. Additionally, for many years, in addition to pharmacological therapy, emphasis was placed on restricting sodium intake in patients. This was based on the fact that sodium is the dominant, highly osmotic extracellular microelement causing secondary water retention. This paradigm was accepted for many years, but recent studies have opposed this view. It has been shown that a diet based on strict sodium restriction does not improve prognosis in heart failure patients compared to a normal, balanced diet. Moreover, it has been demonstrated that supplementation of 3g of sodium chloride (equivalent to 1.2g of sodium) per day is safe, does not exacerbate congestion symptoms, but positively affects natriuresis, lowers renin levels, and raises blood pressure.

Based on the arguments presented above, the prospective, randomized, double-blind, placebo-controlled study described here has been designed. It involves the oral supplementation of 3g of sodium chloride in patients with chronic heart failure with reduced left ventricular ejection fraction and low serum sodium levels, who are not receiving optimal doses of heart failure medications. The aim is to determine whether this intervention will allow for increased doses of these medications, which could translate into improved prognosis. We will also assess the degree of sodium retention (reabsorption) and urine dilution following the administration of a continuously taken diuretic dose, as well as changes in aldosterone, renin, and NT-proBNP levels, which are indicators of neurohormonal activation and myocardial overload.

Each participant will be randomized to the study group or placebo, then will receive the supplementation preparation (sodium chloride vs. placebo) in specially prepared capsules that prevent identification of their contents. The study will include a series of in-person and telephone visits, during which the patient's clinical condition, severity of congestion symptoms, and serum and urine laboratory parameters will be assessed as indicators of changes in the body due to the intervention, and to evaluate the safety of supplementation. During visits, each study participant will be assessed for the possibility of increasing doses/introducing new heart failure medications. After three months of supplementation (sodium chloride vs. placebo), it will be discontinued, and patients will remain under observation for an additional three months, with a final visit scheduled at six months (three months after the end of supplementation) for a complete evaluation.

Positive results from this study could make a significant contribution to the treatment of heart failure patients with drugs proven to impact long-term prognosis, an improvement of which is crucial given the unfavorable epidemiological data presented above. Additionally, the study will contribute to a better understanding of the pathophysiology of this condition, particularly renal autoregulation of water-electrolyte balance. It seems that it is not diuretic treatment and dietary restriction, but neurohormonal balance and its effective maintenance that are key factors in keeping patients in a state of cardiovascular compensation.