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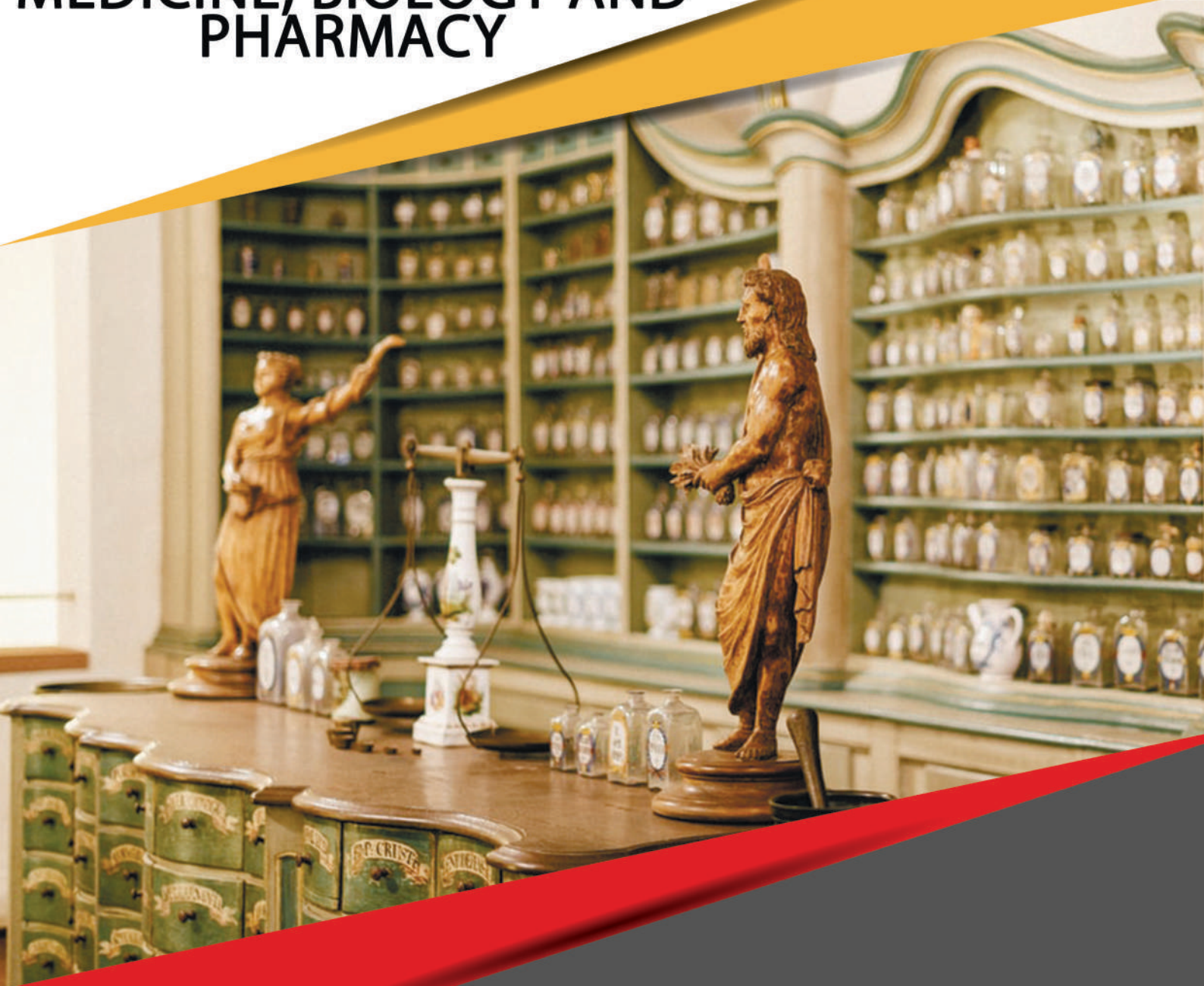

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SECTION 8. THERAPY

8.1 Neutrophil-to-lymphocyte ratio as a prognostic marker of adverse events of heart failure

Introduction. Heart failure (HF) is one of the leading causes of morbidity and mortality worldwide. It is established that simultaneously with the increase of the elderly, the prevalence of HF among the population increases, as well as the number of hospitalizations for HF, which is an important problem for health care [319]. HF is a pathological condition that is manifested by the inability of the circulatory system to provide the metabolic needs of the body. The classic symptoms are decreased exercise tolerance (due to shortness of breath and fatigue) [320] and fluid retention.

Chronic heart failure (CHF) is a progressive clinical syndrome that adversely affects the patient's [321] quality of life and mainly affects the elderly; the incidence doubles in men and triples in women with each decade after age 65 [322]. The European study EPidemiologia da Insuficiencia Cardiaca e Aprendizagem reported that the prevalence of heart failure in the group 25-49 years is 1.36%, in the 50-59-year-old group - 2.93%, in the 60-69-year-old group - 7.63% , in the group of 70-79 years - 12.67% and 16.14% in patients older than 80 years [323]. The prevalence of this phenotype of CHF is subject to large fluctuations and depends on the demographic situation in general and the intensity of correction of risk factors for cardiovascular disease (CVD) [324].

The 2016 European Society of Cardiology recommendations, a new classification of HF was introduced, according to which in addition to HF with reduced HFrEF (less than 40 %) and preserved left ventricular ejection fraction HFpEF (50 % or more) separated HF with intermediate left ventricular ejection fraction HFmrEF (40-49 %). At present, the epidemiology, pathophysiology, treatment and prognosis for the "gray zone" of HF with an intermediate fraction of left ventricular ejection remain insufficiently studied [325].

The distribution of patients with CHF by LVEF is based on the fact that the disease is based on various etiological factors. Framingham Heart Study proposes to classify heart rate according to the root cause of the disease: coronary heart disease (CHD), systemic hypertension, valvular heart disease or other diseases. Patients with CHF are more likely to suffer from heart valve disease, hypertension and atrial fibrillation [326]. Compared to patients with HFrEF, patients with HFpEF are usually elderly women with obesity and comorbidities such as diabetes, chronic lung disease, cancer, hypothyroidism, anemia, and chronic kidney disease [327]. Most patients with HF have multimorbidity, and the number of patients with three or more chronic comorbidities has increased from 68% in 2002 to 87% in 2020 [328].

Aim of the study. To study the parameters of diagnostic significance of the neutrophil-to-lymphocyte ratio (NLR) is in the prediction of lethal outcome and systemic inflammatory response syndrome in patients with heart failure.

Currently, the study of the role of systemic inflammation and immune activation in the development and progression of HF is the focus of researchers. Systemic inflammation is not inflammation in the full sense of the word; it reflects pro-inflammatory changes, which are manifested by the accumulation in the blood of chemokines and cytokines - mediators of inflammation, dissolved forms of their receptors, adhesion molecules, activation of cellular elements - leukocytes, lymphocytes, monocytes, platelets [329]. Cytokine-mediated systemic subclinical inflammation has been shown to be associated with activation of the sympatho-adrenal and renin-angiotensin systems, endothelial dysfunction, pro- and antioxidant imbalances, and other pathogenetic links of CHF. Given this, it becomes clearer the role of the immune inflammatory process in the induction and progression of myocardial remodeling and, accordingly, in increasing the severity of CHF [330].

When determining markers of systemic inflammation in patients with HF of ischemic origin, increased levels of proinflammatory cytokines (such as tumor necrosis factor-α (TNF-α), interleukin-1) (IL-1), interleukin-6 (IL-6) and their soluble receptors), with the progression of CH. Hyperexpression of proinflammatory cytokines impairs the course of HF through mechanisms of inhibition of protein biosynthesis,

intracellular glucose transport, activation metalloproteinases, inhibition myocardial and peripheral muscle contractility, formation of nitric oxide by endothelium, and muscle stimulation and apoptosis stimulation [331].

The study of the above markers of inflammation is limited due to their high cost and necessitates the search for available markers that could be used both to assess the adverse course of HF and to determine the risk of complications or adverse prognosis.

In clinical practice, a more accessible method is to determine the markers of inflammation by the content of leukocytes and the study of the leukocyte formula according to the general blood test.

White blood cell counts are a classic marker of systemic inflammation in CVD, but data on their association with HF are conflicting.

Many studies show the involvement of neutrophils in the progression of HF, as they respond to inflammation by promoting overexpression of pro-inflammatory cytokines, namely α-TNF, IL-6, which have a destructive effect on the myocardium, leading to decreased pumping function of the heart and complications [332]. As regulators of both innate and adaptive immune responses, neutrophils can influence chronic immune response and affect the function of dendritic cells as well as lymphocytes. It is believed that lymphopenia is more common in stressful conditions such as HF, due to activation of the hypothalamic-pituitary-adrenal system. Activation of this link leads to the secretion of cortisol, and increased levels of cortisol lead to a decrease in the relative concentration of lymphocytes. Lymphopenia, found in patients with acute or chronic heart failure, regardless of etiology correlates with the severity of the disease and is a negative prognostic marker associated with increased mortality [333].

A number of researchers have proposed the definition of NLR as an additional marker of systemic inflammation, which appeared as a prognostic indicator of poor prognosis in CVD [334]. This indicator can be determined by dividing the number of neutrophils by the number of lymphocytes. NLR provides information on two pathophysiological pathways: neutrophils (associated with a rapid immune response

and elevated levels of free radicals responsible for tissue damage) and lymphocytes (associated with a chronic adaptive immune response) [333].

Increased NLR levels indicate endothelial damage and dysfunction as a result of increased neutrophil secretory activity, which can lead to an unfavorable prognosis in CVD [335]. According to the literature, in modern cardiology, studies have shown that NLR is an independent predictor of outcome in patients with stable coronary heart disease and a predictor of short - and long-term mortality in patients with acute coronary syndromes ((ST-segment elevation myocardial infarction) (STEMI)) and heart transplantation [333]. In addition, NLR can also be used to risk stratification in patients with both CHF and acute HF decompensation. According to American colleagues, a higher rate of NLR is associated with increased mortality within 30 days in acute decompensation of HF [336]. Therefore, the studied indicator is higher, the worse the prognosis.

In a study on HF patients performed by Yan et al. it was shown that high NLR is associated with a higher rate of major cardiovascular events in elderly patients with HF [333]. A multicenter study conducted by Delcea et al. [333] confirmed that SNL correlates with disease severity and is a prognostic marker associated with an increased risk of mortality.

Thus, understanding the pathophysiology of HF and the role of inflammatory markers can optimize the clinical management of patients with HF and reduce adverse clinical consequences. More and more works confirm the idea of SNL monitoring as an auxiliary marker of severity and, most importantly, poor prognosis in patients with HF.