Open Access Research Journal of Biology and Pharmacy

Journals home page: https://oarjbp.com/ ISSN: 2782-9979 (Online) OARJ OPEN ACCESS RESEARCH JOURNALS

(REVIEW ARTICLE)

Check for updates

Chronic heart failure: Clinical trends and modern approaches

Muyassar Gafurdjanovna Mukhamedova $^{\rm 1,\ *}$, Dildora Saidjanovna Narzullo
eva $^{\rm 2}$ and Guzal Alisherovna Nosirova $^{\rm 1}$

¹ Center for the development of professional qualification of medical workers, Tashkent, Uzbekistan. ² Bukhara State Medical Institute, Bukhara, Uzbekistan.

Open Access Research Journal of Biology and Pharmacy, 2022, 05(02), 001-006

Publication history: Received on 21 May 2022; revised on 26 June 2022; accepted on 28 June 2022

Article DOI: https://doi.org/10.53022/oarjbp.2022.5.2.0050

Abstract

Chronic heart failure (CHF) is one of the most pressing problems in modern cardiology, and the prevalence and diagnosis of this disease is increasing due to improvements in medical diagnosis and treatment, increased patient life expectancy, and advances in cardiology. This article discusses the specifics of the clinical course of CHF, modern trends in the course of the disease, early detection, analysis of causes and the criteria for the final classification in a broad sense, taking into account the latest data. At the same time, the latest achievements and modern views in the field are briefly described.

Keywords: Chronic heart failure; Etiology; Classification; Clinical picture

1. Introduction

Chronic heart failure is not a specific disease but a clinical syndrome with different etiology and pathogenesis. Chronic heart failure is one of the most deadly and common cardiovascular pathologies [1]. A number of complications, including hospitalization, lethal arrhythmias, and mortality, have been reported during the outbreak. In addition, CHF may be the last stage of many cardiovascular diseases, including myocardial infarction, valvular heart disease, and various cardiomyopathies [3]. As mentioned above, SLE is a pathological condition that occurs as a result of structural and functional diseases of the heart, with specific signs and symptoms. Several terms are used to assess the condition of patients with heart failure. Patients with left ventricular stroke volume and no signs and symptoms of heart failure may be referred to asymptomatic left ventricular systolic dysfunction.

2. Clinical findings

The most common clinical signs in SYUE include shortness of breath, swelling in the ankle or foot, and fatigue. Although the occurrence of these symptoms is not inevitable, their presence indicates that additional tests should be performed in patients to confirm the diagnosis. Although the causes and clinical course of left ventricular and right ventricular failure, are similar, symptoms such as fatigue and wheezing in heart failure accompanied by left ventricular failure, peripheral edema in right ventricular failure, and increased venous pressure are more common [4]. Due to compensatory mechanisms, no clear signs and symptoms may be observed in the early stages of heart failure. However, as the disease progresses, the following signs and symptoms: tachycardia (sensitivity 7%, specificity 99%), leg edema (sensitivity 10%, specificity 93%), dilatation of the jugular veins (sensitivity 39%, specificity 92%), abnormal pulmonary murmurs (wheezing) (sensitivity 60%, specificity 99%) is manifested to one degree or another [5]. Table 1 details the likelihood of encountering signs and symptoms of heart failure (Table 1). Patients who have signs and symptoms of heart failure and whose symptoms do not change within at least 1 month are referred to as stable CHF [6]. A sudden worsening of the condition in patients with stable CHF is called decompensated heart failure. Table 2 lists the

* Corresponding author: Muyassar Gafrdjanovna Mukhamedova Center for the development of professional qualification of medical workers, Tashkent, Uzbekistan.

Copyright © 2022 Author(s) retain the copyright of this article. This article is published under the terms of the Creative Commons Attribution Liscense 4.0.

factors leading to decompensation of heart failure (Table 2). While the New York Heart Association classification represents the severity of symptoms and functional status in heart failure, the Killip classification assesses the severity of patients after acute myocardial infarction [6].

Table 1 Signs and	symptoms o	f heart failure
-------------------	------------	-----------------

		Sensitivity (%)	Specificity (%)	Causes
Symptoms	Excretion	66	52	Respiratory diseases, anemia, obesity, fear
	Paroxysmal nocturnal shortness of breath	33	76	Asthma, sleep apnea syndrome
	Orthopnea	21	81	Fear, obesity
	The presence of swelling	23	80	Venous insufficiency, hypoproteinemia, drugs, inactivity, pneumonia, aspiration pneumonitis, sepsis, liver disease, kidney disease
Signs	Increased pressure in the jugular veins	10	97	Pulmonary embolism, superior vena cava obstruction, pericardial fluid accumulation
	3 heart tone	31	95	Mitral regurgitation, fever, pregnancy
	Peripheral swelling	10	93	Venous insufficiency, hypoproteinemia, medications, inactivity
	Tachycardia	7	99	Arrhythmias, pain, fear, fever, hyperthermia, medications
	Crepitation	13	91	Pulmonary fibrosis, chronic obstructive pulmonary disease, pneumonia, lung abscess, bronchiectasis, bronchiolitis

Table 2 Factors leading to decompensation of heart failure

Acute weights	Slow weights	
Acute coronary syndrome	Infections	
Pulmonary artery embolism	Renal dysfunction	
Arrhythmias	Exacerbation of asthma or chronic obstructive pulmonary disease	
Hypertensive crisis	Arrhythmias	
Cardiac tamponade	Uncontrolled arterial hypertension	
Aortic dissection	Anemia	
Surgical complications	Hyperterioidism or hypothyroidism	
Peripartup cardiomyopathy	Failure to comply with treatment	

3. Reasons

A number of conditions can lead to chronic heart failure. Below details the pathological processes that can lead to CHF were stated. Determining the causes of SLE is very important in the proper conduct of treatment tactics. In addition, timely elimination of the causes leading to SUE is one of the main criteria of effective treatment.

Main causes of heart failure were shown below.

3.1. Myocardial disease

- Ischemic disease of the heart
- Hypertension
- Cardiomyopathies
 - Hypertrophic cardiomyopathy
 - o Dilated cardiomyopathy
 - o Restrictive cardiomyopathy
 - Peripartum cardiomyopathy
 - $\circ \quad \text{Infiltrative cardiomyopathy} \\$
 - Myocarditis
 - o Infectious
 - o Autoimmune
 - Eosinophilic
 - o Toxic

3.2. Valvular heart disease

- Mitral
- Aortal
- Triscubidal
- Pulmonary artery valve

3.3. Pericardial disease

- Exudative pericarditis
- Constructive pericarditis

3.4. Endocardia disease

- Endomiocardial disease
- Endocardial fibroelastosis
- Pheochromocytoma

3.5. Congenital heart disease

- Defects from left to right
- Left ventricular obstruction
- Individual ventricular anomaly
- Cyanotic heart disease

3.6. Rhythm disorders

- Tachyarthritis of the compartments
- Ventricular tachyarrhythmia
- Sinus node dysfunction

3.7. Cardiac circulatory disorders

• Atrioventricular block

3.8. Cases with high impact force

- Anemia
- Sepsis
- Thyrotoxicosis

- Peugeot disease
- Atriovenous fistula

3.9. Do not load with liquid

- Kidney failure
- Iatrogenic

3.10. Classification

Depending on the size of the left ventricle, the SYUE is divided into types with reduced ejection fraction (\leq 40%), moderately reduced ejection fraction (\geq 40% in the range of \leq 49%) and preserved ejection fraction (\geq 50%). Although HFmrEF is divided into a separate type, research on the epidemiology, pathophysiology, treatment and prognosis of patients in this category is still insufficient and remains unknown. HFmrEF itself is recommended to be divided into 3 sub-categories. These include the slightly improved group of HFmrEF (previously ejection fraction <40%), the unchanged group of HFmrEF (left ventricular ejection fraction in the range of 40-49%), and the group of HFmrEF worsened group (previously left ventricular ejection fraction > 50%). Of these, the improved and worsened groups account for 90% and the unchanged group for 10% [7, 8, 9].

Depending on the location of the deficiency, YU is divided into left ventricular, right ventricular, or biventricular heart failure. CHF is divided into acute and chronic heart failure depending on the pace of development, time. Alternatively, high pulse is divided into strong CHF and low pulse is divided into strong CHF, depending on the contraction of the heart. High-impact CHF is a rare condition in which the cardiac index is higher than 2.5-4.4 at rest and the vascular system resistance is low [10]. Examples of this are often anemia, hyperthyroidism, vitamin B1 deficiency. This is often due to low blood volume and pressure, activation of the sympathy-adrenal system and renin-angiotensin-aldosterone systems, increased secretion of antidiuretic hormone, which in turn leads to left ventricular enlargement, left ventricular remodeling, and UE. Heart failure with low pulse force is relatively more common and is characterized by a lack of strength of heart contractions when metabolic requirements are increased. Left ventricular dysfunction as a result of extensive myocardial infarction, right ventricular dysfunction as a result of acute pulmonary artery embolization, and biventricular dysfunction are among the leading causes of low-impact heart failure. Recently, the concept of HFpEF with low resistance to physical activity has been proposed by a number of scientists [911,12,13]. This occurs as a result of impaired oxygen uptake into the skeletal muscles during exercise or defects in oxygen delivery.

According to The New York Heart Association, heart failure is divided into the following functional classes:

- Class I: CHF has no difficulty in physical movement; Normal physical activity does not cause symptoms to occur.
- Class II: CHF causes some restrictions on physical activity; patients feel comfortable in a relaxed state, but normal physical activity causes symptoms of heart failure.
- Class III: CHF causes significant restrictions on physical activity; while patients feel enslaved in a relaxed state, some normal movements lead to the appearance of symptoms of chronic heart failure.
- Class IV: Patients are unable to perform any physical activity, and symptoms of heart failure bother them even at rest.

The American College of Cardiologists and the American Heart Association (ACC / AHA) recommend that heart failure be divided into the following stages [14, 15, 16, 17].

- Stage A: The risk of developing SLE is very high, but there are no signs of heart failure or structural heart disease.
- Stage B: Structural heart disease is present but there are no CHF symptoms.
- Stage C: There are signs of structural heart disease as well as CHF.
- Stage D: Treatment-resistant heart failure requiring specialized care.

4. Conclusion

CHF is a polyetiological pathological syndrome, which has several classifications depending on the number of causative factors, the diversity of the clinical picture, the degree of dysfunction of the parts involved in the pathological process, and the subjective and objective appearance of the patient. Identifying the causes and correctly classifying patients with SLE is important in correcting treatment tactics, improving prognosis, and improving patients 'quality of life.

Compliance with ethical standards

Acknowledgments

Authors thank to Center for the development of professional qualification of medical workers.

Disclosure of conflict of interest

The authors have no potential conflict of interest to declare.

References

- [1] Coronel R, de Groot JR, Van Lieshout JJ. Defining heart failure. Cardiovascular research. 2011; 50(3): 419–422.
- [2] Alyavi A, Uzokov J. Treatment of stable angina pectoris: focus on the role of calcium antagonists and ACE inhibitors. Ont Health Technol Assess Ser. 2017; 15(9): 1-12.
- [3] Mukhamedova M, Alyavi BA, Uzokov JK, Babaev MA, Kamilova SE. P120 Relationship between left ventricular global function index and cardiac systolic functions in patients with chronic ischemic disease of the heart and diabetes mellitus. European Heart Journal-Cardiovascular Imaging, 20(Supplement_3), jez. 2019; 147-008.
- [4] GBD. Disease and Injury Incidence and Prevalence Collaborators, "Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016," Lancet. 2016; 390: 1211–1259.
- [5] Uzokov J, Alyavi A, Alyavi B. Influence of combination therapy of rosuvastatin and telmisartan on vascular and metabolic profile in hypercholesterolemic patients with metabolic syndrome. Atherosclerosis. 2007; 263: e241.
- [6] Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE, Jr, Colvin MM, Drazner MH, Filippatos GS, Fonarow GC, Givertz MM, Hollenberg SM, Lindenfeld J, Masoudi FA, McBride PE, Peterson PN, Stevenson LW, Westlake C. 2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America. Circulation. 2007; 136(6): e137–e161.
- [7] Uzokov J. Influence of abnormal lipid components in statin-naive young adults: Is there any gap?. European journal of preventive cardiology. 2020; 27(8): 868-869.
- [8] Iskhakov S, Uzokov J, Kamilova S. Comparative analysis of the inflammatory biomarkers in patients with stable coronary artery disease and metabolic syndrome. Atherosclerosis. 2019; 287: e171.
- [9] Babaev M, Alyavi AL, Alyavi BA, Uzokov JK. P2533 Influence of l-arginine aspartate on vascular markers in hypertensive patients with metabolic syndrome. European Heart Journal. 2018; 39(suppl_1): ehy565-P2533.
- [10] Usarov M, Mamatkulov X, Uzokov J, Hojiev S, Yakhshilikov D, Dadaev S. Ps 11-56 Efficacy Of Combination Therapy Using Nebivalol And Trimetazidine In Hypertensive Patients With Metabolic Syndrome And Stable Angina. Journal of Hypertension. 2016; 34: e349.
- [11] Lutfullayevich AA, Anisxonovich AB, Kamilovich UJ, Ismatovich AS, Ulugbekovich KN. GW28-e0699 Cardiovascular risk stratification and gender differences in hypertensive patients with metabolic syndrome. Journal of the American College of Cardiology. 2017; 70(16S): C138-C139.
- [12] Alyavi AL, Alyavi BA, Tulyaganova DK, Aliahunova MY, Sabirzhanova ZT, Nuritdinova SK, Sayfiev NY. Features of Inflammatory Markers in Patients With Coronary Heart Disease. International Journal of Healthcare and Medical Sciences. 2018; 4(10): 188-192.
- [13] Lutfullayevich AA, Anisxonovich AB, Kamilovich UJ, Ismatovich AS, Ulugbekovich KN. GW28-e0698 Telmisartan with amlodipine versus lisinopril with amlodipine on home blood pressure variability in patients with metabolic syndrome. Journal of the American College of Cardiology. 2017; 70(16S): C138-C138.
- [14] Mukhamedova M, Uzokov JK, Orziev DZ, Mukhitdinova OY, Payziev DD, Narzullaeva DS, Rakhimova DA. Features of serum bilirubin in non ST elevation acute coronary syndrome. European Heart Journal: Acute Cardiovascular Care. 2022; 11(Supplement_1): zuac041-110.
- [15] Mukhitdinova O, Alyavi BA, Ubaydullaeva ZZ, Uzokov JK, Mukhamedova MG, Rakhimova DA, Orziev DZ. Changes of blood D-dimer level after COVID-19 in patients with coronary heart disease. European Heart Journal: Acute Cardiovascular Care. 2022; 11(Supplement_1): 041-136.

- [16] Uzokov J, Alyavi A, Alyavi B, Azizov S. Influence of Combined Therapy on Inflammatory State and Proinflammatory Cytokines in Patients with Coronary Artery Disease and Metabolic Syndrome. Eur Cardiol. 2020; 15(15):27.
- [17] Mukhamedova MG, Narzullaeva DS, Uzokov JA. Efficacy of rosuvastatin on lipid parameters and vascular and inflammatory markers in patients with metabolic syndrome and coronary artery disease. Journal of critical reviews. 2020; (7): 19.