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Heart failure and elderly population – a systematic review

Niewydolność serca u osób w wieku podeszłym – przegląd literatury

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Streszczenie

Niewydolność serca (HF) jest jedną z najczęstszych chorób układu sercowo-naczyniowego, której przyczyną rozwoju są głównie choroba niedokrwienna serca, zaburzenia metaboliczne lub endokrynologiczne, czy też nadciśnienie tętnicze lub wady zastawkowe. Szacuje się, że niewydolność serca dotyczy około 64 milionów ludzi na świecie, a liczba ta wzrasta z roku na rok. Na przełomie ostatnich dekad zaobserwowano dwukrotny wzrost liczby chorych z niewydolnością serca, a zachorowalność obecnie jest na poziomie 1-2% w krajach rozwiniętych. Objawy niewydolności serca różnią się w zależności od tego, czy dysfunkcji uległa lewa, prawa, czy obie komory, ale w każdym z przypadków można zaobserwować typowe objawy podmiotowe takie jak uczucie zmęczenia, duszność oraz objawy przedmiotowe tj. wzrost ciśnienia w żyłach szyjnych, trzeszczenie nad polami płucnymi lub obrzęki obwodowe. W ramach diagnostyki wykonuje się badania laboratoryjne oraz badania obrazowe, a leczenie opiera się na odpowiedniej farmakoterapii w celu ograniczenia postępu choroby i zredukowania śmiertelności. *Geriatria 2021; 15: 232-242. doi: 10.53139/G.20211527*

Słowa kluczowe: niewydolność serca; osoby starsze; kardiologia

Abstract

Heart failure (HF) is one of the most common diseases of the cardiovascular system, the causes of which are mainly coronary heart disease, metabolic or endocrine disorders, hypertension, or valvular disease. As for today, it is estimated that heart failure affects approximately 64 million people worldwide, and this number is increasing every year. At the turn of the last decades, a two-fold increase in the number of patients with heart failure has been observed, and the incidence is currently at the level of 1-2% in developed countries. Symptoms of heart failure vary depending on whether the left, right, or both ventricles are dysfunctional, but in each case, typical symptoms such as fatigue, shortness of breath, and signs such as increased pressure in the jugular veins, crackling crackles, pulmonary or peripheral edema can be seen. As part of diagnostics, laboratory tests and imaging tests are performed, and treatment is based on appropriate pharmacotherapy to limit the progression of the disease and reduce mortality. *Geriatria 2021; 15: 232-242. doi: 10.53139/G.20211527*

Keywords: heart failure; elderly population; cardiology.

Introduction

Heart failure (HF) is one of the most common diseases of the cardiovascular system, the causes of which are mainly coronary heart disease, metabolic or endocrine disorders, hypertension, or valvular disease. Symptoms of HF vary depending on whether the left, right, or both ventricles are dysfunctional, but typical symptoms such as fatigue, shortness of breath, and signs such as increased pressure in the jugular veins, pulmonary crackles, and peripheral edema can be seen in each case. HF leads to a decrease in cardiac output, which results in a reduction in the supply of an adequate blood volume to meet the metabolic needs of tissues, or maintains an adequate blood supply, but with increased ventricular filling pressure to maintain cardiac output [1]. HF is classified into acute heart failure (AHF) and chronic heart failure (CHF). AHF is a sudden onset or worsening of symptoms and/or

signs of HF requiring urgent intervention. AHF may be diagnosed for the first time or more commonly occurs as CHF decompensation [2]. CHF, in turn, is caused by the presence of coexisting factors, e.g., cardiovascular diseases which contribute to the deterioration of the heart's function and consequently lead to the development of CHF. The prevalence of HF increases exponentially with age and paradoxically with the emergence of newer intervention techniques in the treatment of ischemic myocardial disease, which, while improving the patient's health and quality of life at a given time, also increase the likelihood of developing HF among these patients [3].

General classification of patients with HF

Currently, HF is classified according to the value of a left ventricular ejection fraction (LVEF). This division includes patients with HF with preserved LVEF (LVEF ≥50% – HFpEF), HF with reduced LVEF (LVEF ≤40% – HFrEF), and patients in the so-called "gray zone", i.e., with LVEF between 41 and 49% – HFmrEF. Differentiation of patients with HF by LVEF is important because of the heterogeneous etiology of each HF subtype [4].

Epidemiology

As for today, it is estimated that HF affects approximately 64 million people worldwide, and this number is increasing year by year [5]. At the turn of the last decades, a two-fold increase in the number of patients with HF has been observed, and the incidence is currently at the level of 1-2% in developed countries [6]. In addition, an increase in the incidence of HF has been demonstrated among people ≥ 70 years of age, which is now >10% [7]. It is associated with an aging population and prolonged exposure to risk factors for the development of heart failure. The risk of developing the disease in relation to gender is 28.5% among women and 33% among men over 55 years of age [8]. HF also carries a high rate of mortality and hospital readmissions – about 40% of patients die within 5 years of the first hospitalization due to HF [9]. In Poland as many as 53% of patients with this diagnosis are re-hospitalized, of which 25% of this group undergoes hospitalization within 30 days of discharge from hospital [9]. HF mortality is projected to increase to approximately 25% in the coming years [10]. Currently, HFpEF is more often diagnosed than HFrEF, and patients with HFpEF are more often women whose main cause is atrial fibrillation or arterial hypertension more often than myocardial infarction [11].

Common risk factors

Risk factors for the development of HF can be divided into three categories, which are myocardial disease, preload and afterload disorders, and heart rhythm disturbances.

Myocardial disease

This category is dominated by ischemic heart disease, which is the most common cause of HF development [12]. The subsequent cause is toxic heart damage, which is distinguished, e.g., through the abuse of psychoactive substances, drugs, or heavy metals. Other causes include inflammation-related damage and the immune response, infiltrative heart disease, metabolic and genetic disorders. Examples of diseases from this group include endocarditis, myocarditis, neoplastic infiltration, amyloidosis, thyroid disease, diabetes, metabolic syndrome, or cardiomyopathies [13].

Preload and afterload disorders

This category includes the second leading cause of HF, which is hypertension (HT), but also heart valve disease, ventricular septal defects, rupture of the free wall or interventricular septum, aortic dissection, and chest trauma. It also includes diseases of the pericardium and endocardium, e.g., pericarditis, or hypereosinophilic syndrome, as well as high cardiac output syndrome, which is present, for example, in severe anemia or during pregnancy [14]. Diseases leading to changes in preload and afterload lead to impaired venous return and LVEF, thus limiting the possibility of adequate metabolic supply of tissues

Heart rhythm abnormalities

Cardiac arrhythmias that can cause HF to include both tachyarrhythmias, such as AF, and bradyarrhythmias, such as sinus node disease [6]. As aforementioned, AF is the most common arrhythmia in the elderly population, and its incidence increases with age being 4,2% in 60-69 age group and 13,4% in 80-89 age group [15]. AF has the greatest share in the development of HF, which may be both a complication and a cause of HF [16]. When AF is the cause, the clinical course is milder in contrast to other causes of HF development [17]. In turn, when AF is a complication of CHF, increased mortality and risk of stroke are observed [18].

Atrial fibrillation (AF) can be both a cause of and a complication of HF. AF leads to the development of HF due to asynchrony of systolic and diastolic functions of the heart, which leads to an increase in left atrial pressure, a decrease in arterial pressure, and a decrease in cardiac output [19].

Acute coronary syndrome

AHF and CHF are common complications of the acute coronary syndrome (ACS). According to the ESC-HF-LT study, patients hospitalized for AHF were older than those with CHF, they were more often women (37.4% vs.28.8% in the case of CHF), and these patients experienced an episode of ischemic heart disease more often (53,8% vs 43.1% in the case of CHF) [20]. However, the development of AHF has been more frequently observed in men who had a previous episode of ischemic heart disease and underwent percutaneous coronary intervention [13]. Due to the prolongation of life expectancy, ACS becomes more frequent in the elderly population and for now, more than 50% of admissions for ACS are for patients that are ≥ 65 years of age [21]. Consequently, this leads to a situation in which older people are more likely to experience AHF after ACS. ACS results in ischemia and, consequently, myocardial necrosis, which depends on which coronary artery is narrowed and the degree of narrowing of the coronary arteries by atherosclerotic plaque. This situation leads to a deterioration of the contractility of the heart, which in turn causes the development of systolic HF. In the GRACE risk score model, the most sensitive predictor of in-hospital mortality compared to patients without HF was the Killip scale in which rales and/or S3 had 2 times, pulmonary edema had 3 times and cardiogenic shock had 4 times the risk of death [12]. The risk of developing HF was the same for STEMI and NSTEMI patients [22].

Arterial hypertension

Arterial hypertension (HT) can contribute to the development of both systolic HF and diastolic HF. In the case of diastolic HF, HT leads to left ventricular hypertrophy due to increased afterload. Hypertrophy causes an increased demand for oxygen and leads to an increase in left ventricular filling pressure. This situation leads to impair subendocardial perfusion, myocardial hypoxia, and deterioration of the diastolic function of the heart [23]. Such a course leads to the development of more frequently diagnosed diastolic heart failure [23]. It is more often diagnosed among women in whom HF and atrial fibrillation predominate amongst comorbidities as opposed to myocardial infarction which occurs less frequently. Moreover, patients with this type of HF are more often older than those with systolic HF [24,25]. A hypertensive crisis in HT may lead to the development of AHF. This situation develops because of a sudden increase in blood pressure ≥180/120 mmHg in people with left ventricular diastolic dysfunction, which worsens the already impaired ejection fraction and contributes to blood congestion in the pulmonary circulation [26]. The development of systolic HF is most often caused by an imbalance between the demand of the heart muscles for oxygen and its supply, which leads to cardiomyocyte hypoxia, necrosis, and worsening of contractility of the heart [23]. Hypertension or hypertensive crisis alone is not the only risk factor for developing HF. Also important are the nocturnal drop in blood pressure, the absence of which is associated with a twice higher risk of developing HF. Isolated systolic arterial hypertension also increases the risk of developing HF [27].

Hyperthyroidism

Hyperthyroidism and related to it HF are more common in the elderly due to existing diseases of the cardiovascular system [28]. The state of hyperthyroidism leads to decreased contractility of the heart muscle, decreased diastolic compliance, and pulmonary congestion [29].

Obesity and diabetes

Obesity and diabetes are common diseases among the elderly [30]. Increased concentration of glucose and free fatty acids circulating in the bloodstream leads to abnormal lipid deposition and the enlargement of existing fat tissue, both visceral and subcutaneous. Overload of cardiomyocytes with lipids leads to enlargement of the heart and its damage due to lipotoxicity [31]. This toxicity develops through the activation by lipids of signaling pathways involved in the development of inflammation, including protein kinase C and nuclear factor κ, which interfere with the insulin signaling pathway [32]. This leads to development of insulin resistance, which reduces the consumption of glucose by cardiomyocytes and promotes the use of free fatty acids, which finally leads to increased formation of free oxygen radicals and diastolic dysfunction of the heart muscle [33].

Pulmonary embolism

Pulmonary embolism leads to tricuspid regurgitation, increased pressure, worsening contractility, dilatation, and, consequently, right ventricular dysfunction. This situation is further aggravated by the displacement of the interventricular septum towards the left ventricle, which worsens its filling in diastole. As a result, blood flow to the left ventricle deteriorates and the cardiac output becomes decreased which leads to the development of HF [34].

Acute mechanical causes

In the case of rupture of the heart muscle, the heart is volumetrically overloaded and is not adapted to such conditions, e.g., in the case of papillary muscle rupture and acute mitral valve regurgitation. Otherwise, for example, rupture of the free wall of the heart may result in loss of blood from the heart to the pericardium, cardiac tamponade, and impaired diastolic function of the heart.

Valvular defects

Valvular defects that may contribute to the development of HF or worsen its symptoms include aortic stenosis (AS), aortic regurgitation (AR), and mitral regurgitation (MR). In the case of AS, the afterload of the left ventricle is increased which leads to left ventricular hypertrophy and its remodeling. AR contributes to the progressive widening of the left ventricle, leading to its dysfunction. MR leads to left atrial hypertrophy and its remodeling and, consequently, to remodeling of the left ventricular muscle [7].

Right ventricle disfunction

The cause of the development of HF may also be the dysfunction of the right ventricle due to its volume or pressure overload. While the primary cause of right ventricular dysfunction is pulmonary hypertension caused by left ventricular dysfunction, it should be noted that right ventricular dysfunction may be caused by such as right ventricular infarction, arrhythmogenic right ventricular cardiomyopathy, and valvular dysfunction, which may eventually lead to for HF development [35].

Patophysiology

During aging, many changes occur in the human heart, both at the cellular and metabolic levels, which can eventually lead to the development of HF. Among the elderly population dominates HF with a preserved ejection fraction [36]. The changes leading to its development can be divided into 3 groups. These are cardiomyocyte dysfunction, changes in calcium metabolism, and imbalance in the metabolism of the extracellular matrix. Each of the processes listed above contributes to the deterioration of heart muscle function and ultimately causes HF.

Cardiomyocyte disfunction

Ischemia or aging leads to a decrease in telomere length, death of cardiomyocytes, and the reduction of myocardial cell renewal resulting in a decrease in the number of myocytes, hypertrophy of the remaining muscle cells, and an increase in the thickness of the left ventricular wall, and deterioration of myocardial contractility [37].

Alteration in calcium management

In an aging heart, metabolism and regulation of cellular calcium resources deteriorate, leading to the deterioration of contractility and relaxation of the heart muscle. Other causes of the pathogenesis of HF in the elderly are contractile proteins, which begin to resemble those from the hypertrophic heart and reduce the efficiency of ATP utilization. As a result of the above-mentioned factors, the diastolic dysfunction of the myocardium develops, and the cardiovascular reserve is reduced [1].

Imbalance in extracellular matrix metabolism

Reduced number of cardiomyocytes and deterioration of their functions lead to increased collagen production and fibrosis. Myocardial fibrosis is caused by the high activity of the renin-angiotensin-aldosterone system, inflammatory pathways, and oxidative stress, which are constantly active in the elderly and people with HF. This situation leads to an increase in the stiffness of the aorta and the wall of the left ventricle, followed by an increase in preload, dilation of the left ventricle, and its systolic dysfunction [38].

AHF – classification, signs, and symptoms, diagnostic and treatment

Classification of AHF

AHF can occur as a first episode of HF, but is more common as an acute decompensation of CHF [39]. The AHF classification is based equally on the observation

of the clinical picture, the search for the most common causes of AHF, and physical examination. First, AHF is divided according to the level of systolic blood pressure (SBP) into AHF with preserved SBP (SBP 90-140 mmHg), with increased SBP (SBP >140 mmHg) and AHF with reduced SBP (SBP <90 mmHg). In most cases, there are patients with normal or elevated SBP [40]. Further subdivisions are the division of AHF due to the presence of congestion and hypoperfusion (Table I) and the Kilip Kimball classification (Table II), which are based on the physical symptoms characteristic of HF. The Kilip Kimball classification applies to patients in whom HF was induced by AMI.

Table I. Forrester clasiffication

Hypoperfusion	Edema	Profile
No	No	Warm and dry
Yes	No	Cold and dry
No	Yes	Warm and wet
Yes	Yes	Cold and wet

Table II. Killip and Kimball scale

Class I	No heart failure, rales and/or S3
Class II	Heart failure: Rales < 50% lungs, S3 and venous hypertension
Class III	Severe heart failure: pulmonary edema, rales > 50% lungs
Class IV	Cardiogenic shock

Signs and symptoms of AHF

Symptoms characteristic for AHF are dyspnoea, orthopnea, tachypnoea, symmetrical crackling sounds over the pulmonary fields, as symptoms of left ventricular failure. Right ventricular failure manifests itself through symmetrical swellings on the lower limbs, hepatomegaly, ascites, and jugular vein dilatation. Ventricular dysfunction is also accompanied by symptoms of cardiac hypoperfusion such as muscle weakness, decreased exercise tolerance, restlessness, confusion, pale, cool skin, peripheral cyanosis, threadlike pulse, oliguria, respiratory disturbance, respiratory failure. Predominant symptoms in the elderly population are fatigue, dyspnea, and confusion [39].

Diagnostic of AHF

Among the most common causes of AHF are acute coronary syndrome, uncontrolled arterial hypertension, arrhythmia, acute mechanical cause, and pulmonary embolism, which are included in the acronym CHAMP [2]. The diagnosis of AHF should be based on three stages, in which one should try to confirm or exclude the most common causes of AHF. The first step is to gather a history to obtain information about potential factors for the development of AHF and past cardiovascular diseases. Thereafter, signs and symptoms should be assessed for congestion and hypoperfusion. Ultimately, congestion and hypoperfusion should be confirmed or ruled out by auxiliary tests. As part of laboratory diagnostics, the concentration of natriuretic peptide, cardiac troponins, urea, creatinine, sodium, potassium, TSH, glucose, liver enzymes, and blood morphology are measured.

Laboratory tests

Primarily any patient with suspected AHF or CHF should undergo appropriate laboratory tests. The basic tests, in this case, include the concentration of natriuretic peptide, which is often increased in these patients (BNP, NT-proBNP, or MR-proANP). In the case of a patient with acute dyspnoea and suspected AHF, the concentration of BNP enables the differentiation of AHF from non-cardiac causes of acute dyspnea.

Auxiliary tests

An ECG is performed to detect a recent myocardial infarction or to confirm the presence of new arrhythmias, an X-ray to assess lung congestion, and an echocardiogram to assess cardiac output, pulmonary artery pressure, and right and left ventricular filling pressures.

Treatment of AHF

Treatment of AHF is a three-step process. The first step is to monitor hemoglobin oxygen saturation and measure blood pH and PaCO2 in venous or arterial blood in people with cardiogenic shock. Oxygen therapy is recommended for people with SpO2 <90% or PaO2 <60 mmHg [7]. Non-invasive methods of ventilation with positive airway pressure are recommended in patients with respiratory disorders such as respiratory rate >25/min and SpO2 <90% [7]. It should be started as soon as possible to limit any further deterioration in respiratory function. If non-invasive respiratory failure cannot be controlled and the patient's condition worsens, intubate the patient, and use invasive ventilation techniques. The main pharmacotherapeutics used in the treatment of AHF are diuretics, vasodilators, vaso-

constrictors, and positive inotropic drugs. Patient status according to Forrester's classification has a significant impact on the choice of pharmacotherapy (Table I).

When neither congestion nor hypoperfusion predominates, the patient should be treated according to the symptoms observed, such as hyperkalemia, symptomatic hypotension, or hypoperfusion. When a profile with predominant congestions is observed, treatment depends on the presence or absence of associated hypertension.

In the absence of hypertension, the preferred medications are diuretics, vasodilators, and renal replacement therapies when previous treatment of hypertension is ineffective. In contrast, when hypertension predominates, vasodilators and diuretics are recommended. A hemodynamic profile with predominant hypoperfusion suggests fluid therapy and inotropic positive drugs.

In the last hemodynamic profile with simultaneous hypoperfusion and congestion, treatment depends on the presence of hypotension. Its presence suggests the use of inotropic positive drugs, diuretics, vasoconstrictive drugs, and, as a last resort, mechanical circulatory support, while its absence allows the use of vasodilators, diuretics, and inotropic positive drugs. Of the diuretics, loop diuretics are recommended. Thiazide diuretics should be added to the treatment if no adequate effect is achieved. Among vasodilator drugs, the use of nitroglycerin is suggested. The inotropic positive drugs used in the treatment of AHF are mainly dobutamine and dopamine. Norepinephrine is used as a vasoconstrictive treatment.

Anticoagulant prevention is recommended in patients who have not previously received anticoagulant therapy and have no contraindications to such treatment. It is used to reduce the risk of deep vein thrombosis and pulmonary embolism. The use of low molecular weight heparin is recommended as part of this type of prevention.

CHF – classification, signs, and symptoms, diagnostic and treatment

Classification of CHF

The NYHA classification (Table III) is used to assess the severity of symptoms and the degree of exercise intolerance, while the ACCF and AHA classification (Table IV) describes the stages of HF based on structural changes and symptoms. This allows pharmacotherapy to be adapted to the current condition and relieve symptoms of HF.

Table III. NYHA classification

Class	Symptoms
I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation or dyspnoe
II	Slight limitation of physical activity. Comfortable at rest but ordinary physical activity results in fatigue, palpitation or dyspnoe
III	Marked limitation of physical activity. Comfortable at rest but less than ordinary activity results in fatigue, palpitation or dyspnoe
IV	Unable to carry out any physical activity without discomfort. Sympotms at rest. If any physical activity is undertaken, discomfort is increased.

Table IV. ACCF and AHA classification

A. Patients at high risk for developing HF in the future but no functional or structural heart disorder	B. A structural heart disorder but no symptoms at this stage
C. Previous or current symptoms of heart failure in the context of an underlying structural heart problem, but manager with medical treatment	D. Advanced disease requiring hospital-based suport, a heart transplant or palliative care

Signs and symptoms of CHF

The symptoms of CHF depend on which ventricle is failing. In the presence of left ventricular failure, exercise-induced dyspnea or dyspnea at rest, paroxysmal nocturnal dyspnea, dry cough, wheezing, tachypnea, and abnormal respiratory sounds may be observed. In the case of right ventricular failure, the characteristic symptoms are symmetrical peripheral edema, nocturia, abdominal pain, constipation, ascites, hepatomegaly, excessive jugular vein filling, positive hepatocervical symptom, and Kussmaul's symptom. Failure of both ventricles leads to signs and symptoms such as exercise intolerance, fatigue, rapid weight changes, oliguria, palpitations, dizziness, depression, apex beat shifted to the side, abnormal heart rate, tachycardia, presence of third or fourth heart sounds, pale skin and Cheyn Stokes breath [41].

Diagnostic of CHF

Among the most common causes of CHF, there are acute coronary syndrome, arterial hypertension, diabetes, obesity, and older age [42].

Diagnostics should be based on laboratory and auxiliary tests. As part of laboratory tests, the concentration of B-type natriuretic peptide (BNP ≥ 35 pg/ml, NT-proBNP ≥ 125 pg/ml, enables the diagnosis of HF) should be assessed. Its concentration increases in patients before symptoms appear. Laboratory tests also show normocytic anemia, increased activity of AST/ALT and LDH, and hyperbilirubinemia. In turn, as part of auxiliary examinations, ECG, echocardiography, chest X-ray, CT, or MRI are performed to determine the exact cause of HF.

Treatment of CHF

Treatment of CHF is based on reducing CHF mortality, preventing subsequent CHF hospitalizations, and reducing symptoms associated with CHF, and improving patients quality of life.

Patients with HFrEF

This group includes patients with symptoms suggestive of HF on general examination and LVEF ≤40% on echocardiography. Angiotensin-converting enzyme inhibitors (ACEIs), beta-blockers (BBs), and mineralocorticosteroid receptor antagonists (MRAs) are currently recommended for initial therapy [43]. Dapagliflozin or empagliflozin should be added to therapy to reduce the risk of cardiovascular death and improve the patient's overall condition [44]. ACEIs or angiotensin receptor antagonists (ARBs) along with BBs are drugs that improve prognosis and reduce hospitalization in patients with HFrEF. Other drugs that can be used to treat HFrEF are ivabradine, digoxin, and CRT. Ivabradine decreases mortality and reduces the incidence of re-hospitalization for HF in patients with HFrEF, LVEF ≤35%, NYHA II-IV, sinus rhythm, and resting HR ≥75 beats/min [45]. Digoxin can be used in patients with sinus rhythm and symptomatic HFrEF to reduce the risk of hospitalization for HF [6].

Patients with HFmEF

Patients with symptoms of HF and LVEF 41-49% are eligible. More often, this group of patients includes men, young people, with a higher risk of ACS as the main cause of HF development [46]. Therapy is based on the use of ACEIs, ARBs, BBs, MRAs, ARNIs, and mechanical treatments for HF mentioned above.

Patients with HFpEF

This group includes patients with symptoms of HF, LVEF ≥50%, and imaging evidence of structural abnormality or left ventricular dysfunction. These patients are more likely to be women, elderly, with atrial fibrillation, chronic kidney disease, and other comorbidities [47]. As of today, there is no definite treatment regimen for this form of HF due to the still ongoing research on the use of drugs in the treatment of HFpEF such as perindopril, candesartan, irbesartan, spironolactone, digoxin, and sacubitril/valsartan.

Changes revolutionizing treatment of CHF

According to the latest reports, drugs from the group of angiotensin receptor antagonists and neprilysin inhibitors (ARNIs) and SGLT-2 inhibitors may significantly improve the condition of HF patients [7]. These drugs, due to their multidirectional effects, reduce the progression of symptoms in HF, reduce the severity of symptoms, and reduce the risk of death from HF.

Angiotensin receptor antagonists and neprilysin inhibitors (ARNI)

A new group of drugs that combines drugs acting on the RAAS system and the system of neutral endopeptidases (valsartan + sacubitril). By inhibiting neprilysin, ARNI reduces the degradation of natriuretic peptides and bradykinins, which increases the concentration of A and B natriuretic peptides, which reduce the release of renin and aldosterone and bind to the neprilysin receptor and intensify diuresis, natriuresis, improve relaxation of the heart muscle and inhibit heart remodeling. The AT1 inhibitor reduces vasoconstriction, sodium, and water retention and inhibits cardiac remodeling [48]. Additional benefits of the use of ARNI drugs include improved quality of life, reduced severity of HF symptoms, reduced incidence of diabetes requiring insulin therapy, reduced incidence of GFR reduction, and reduced demand for loop diuretics [49,50]. These drugs are intended for patients with symptomatic HFrEF, LVEF ≤35%, increased concentration of B-type natriuretic peptide (BNP ≥150 pg/ml, NT-proBNP ≥ 600pg/ml), NYHA II-III and GFR> 30ml/min/1,73m^2.

SGLT-2 inhibitors

Recent studies have shown that both in patients with type 2 diabetes and in non-diabetics diagnosed with HF, the use of this group of drugs has shown positive effects in terms of reducing cardiovascular complications, reducing HF mortality, and reducing the frequency of readmissions because of HF [51,52]. The action of this group of drugs is based on the inhibition of the sodium-glucose co-transporter type 2 (SGLT-2), which increases the release of glucose in the urine. This effect is obtained without the risk of hypoglycemia and independently of insulin. In addition, these drugs also contribute to the reduction of sodium reabsorption from urine and increase its delivery to the dense macula, which by regulating the diameter of the afferent arterioles enables protection against hyperfiltration and loss of albumin in the urine. This improves hemodynamics, reduced plasma volume and blood pressure, leading to a reduction in left ventricular preload and afterload [53].

Other methods of treating HF

They are used when pharmacological treatment is not fully sufficient and HF symptoms worsen or do not improve. The main non-pharmacologic treatments for HFrEF include implantable cardioverter-defibrillator (ICD), cardiac resynchronization therapy (CRT), or left ventricular assist devices. All these devices prevent sudden cardiac death, reduce the severity of symptoms, and improve quality of life.

Complications of HF

The deterioration of the heart function due to multi-level changes in patients with HF entails numerous complications, which together may lead to a shortened survival rate of patients or worsen their quality of life, which is bad because of HF itself. The most common complications of HF include supraventricular arrhythmias, ventricular arrhythmias, thromboembolic complications, and brittleness syndrome. The elderly population, due to the higher prevalence of diseases responsible for the development of HF, are at greater risk of developing the following complications.

Arrhytmias

Cardiac arrhythmias are common in patients with HF, and their type depends on the cause of HF development. In AF, the most common arrhythmia in patients with HF, it may be caused by left ventricular dysfunction, valvular disease, or sinus node disease [54]. Ventricular arrhythmias, on the other hand, are common in the end stage of HF and are most often triggered by myocardial infarction [19].

Thromboembolic complications

These complications are predisposed by reduced cardiac output, local reduction in contractility of the heart walls, and atrial fibrillation. Each of these causes disturbs the outflow of blood from the atria and ventricles to the peripheral vessels, which predisposes to the formation of blood clots, which eventually, reaching the pulmonary or carotid arteries, cause pulmonary embolism or ischemic stroke [12].

Brittleness syndrome

It is a symptom complex that occurs in chronically ill patients, including those with HF, which includes an unintentional loss of body weight (≥5 kg per year), fatigue, muscle weakness, slower walking, and low physical activity [55]. HF contributes to the development of malnutrition, lack of appetite, or deterioration of nutrient absorption from the gastrointestinal tract, which in turn leads to macro- and micronutrient deficiencies and translates into changes in skeletal muscles at the cellular, metabolic, and functional levels [56]. Brittleness syndrome contributes to an increase in the frequency of hospitalizations and significantly worsens the prognosis of the patient with HF [57].

Summary

Diseases such as hypertension, obesity, and atrial fibrillation are very common among the elderly. These and other diseases that the elderly may suffer from contributing to the development of HF. Moreover, the presence of these diseases may exacerbate HF-related complications and contribute to an increased risk of death. Due to the increasing probability of HF development with age and the demographically aging population in Poland, this is a serious problem. Even though most people with HF cannot be cured completely, appropriate prophylaxis, meaning adequate treatment of concomitant diseases, and early detection of HF can reduce the progression of HF, improve the quality of life, extend life, and reduce mortality due to HF.

Conflict of interest
None

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