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Heart Failure in Family Medicine

Siniša Franjić[1,*]

[1]Faculty of Law, International University of Brcko District, Brcko, Bosnia and Herzegovina

Abstract

Heart failure is a serious condition in which the amount of blood squeezed out of the heart every minute is insufficient to meet the body's normal needs for oxygen and nutrients. Although some people wrongly believe that the term heart failure means that the heart has stopped, we must say that the term heart failure actually means that the heart has weakened and lost its ability to work. Heart failure has many causes including a many of diseases. Heart failure occurs most often in older people because they are much more likely to suffer from diseases that cause heart failure. Although heart failure gets worse over time, people with the condition can live for years.

Corresponding author: Siniša Franjić, Faculty of Law, International University of Brcko District, Brcko, Bosnia and Herzegovina, Tel: +387-49-49-04-60. Email: sinisa.franjic@gmail.com

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Introduction

Coronary artery disease and diabetes mellitus are the leading causes of heart failure in the United States [1]. It is estimated that 60–70% of patients having systolic heart failure have CAD (coronary artery disease) as the underlying etiology. CAD is a substantial predictor of developing symptomatic heart failure with LVSD (left ventricular systolic dysfunction) compared to asymptomatic LVSD. Heart failure is twice as common in diabetic patients and is one of the most significant factors for developing heart failure in women. This is likely due to the direct effect of diabetes in developing cardiomyopathy as well as the effects of CAD risk and progression. Poorly controlled hypertension and valvular heart disease also remain major precipitants of heart failure. Often overlooked risk factors in the development of heart failure are smoking, physical inactivity or obesity, and lower socioeconomic status. Tobacco is estimated to cause approximately 17% of heart failure cases in the United States, likely due to the enhanced CAD risk. Lower socioeconomic status may limit access to higher quality health care, resulting in decreased adherence to treatment of modifiable risk factors such as hypertension, diabetes mellitus, and CAD.

Heart failure is a clinical syndrome of symptoms and signs that may include fatigue, exercise intolerance, dyspnea, peripheral edema, and pulmonary congestion [2]. HF signs and symptoms result when the heart is unable to fill with or eject blood sufficient to perfuse body tissues and meet metabolic demands. "Heart failure" is preferred over the term "congestive heart failure" because up to one-third of ambulatory patients with HF do not manifest pulmonary or systemic congestion.

Heart failure occurs when the heart is unable to pump enough blood to meet the needs of the body [3]. Heart failure occurs gradually; as the heart fails, blood backs up in the veins and causes fluid to build up in the tissues. The symptoms of heart failure include edema (due to excess fluid in body tissues), shortness of breath, coughing and wheezing, tiredness, and fatigue. Most cases of heart failure result from damage to the heart from coronary artery disease or previous heart attack and hypertension.

Cardiac ischemia or coronary artery disease is the most common cause of HF compromising 60–70% of systolic HF [4]. Heart failure is increasing in older populations because of improved survivorship from treatments of CAD and other common causes of HF including hypertension, diabetes, and valvular disease. Many other cardiac conditions can eventually cause HF: arrhythmias (atrial fibrillation/flutter, heart block), cardiomyopathies (idiopathic, hypertrophic, restrictive, postpartum), and pericarditis.

High blood pressure or hypertension is, in general, a symptom-free condition where abnormally high blood pressure in the arteries increases the risk of developing problems such as stroke, aneurysm, heart failure, heart attack, and kidney damage [5]. For most people the word hypertension means excessive tension, nervousness or stress. In medicine, the term hypertension indicates the condition of high blood pressure regardless of the cause. High blood pressure is also called a "silent killer" because it usually does not cause symptoms for years until it causes damage to important organs. When measuring blood pressure, two values are recorded. The upper value represents the moment when the heart sticks (systole); while the lower value occurs during the relaxation of the heart between two stroke (diastole).

Diagnosis

A high index of suspicion is necessary to diagnose the syndrome of heart failure early in its clinical presentation, because of nonspecific signs and symptoms [1]. Patients are often elderly with comorbidity, symptoms may be mild, and routine clinical assessment lacks specificity. A prompt diagnosis allows for early treatment with therapies proven to delay the progression of heart failure and improve quality of life. Evaluation is directed at confirming the presence of heart failure, determining cause, identifying comorbid illness, establishing severity, and guiding response to therapy. Heart failure is a clinical diagnosis for which no single symptom, examination, or test can establish the presence or absence with certainty.

Symptoms

The most common manifestations of symptomatic heart failure are dyspnea and fatigue, but many conditions present with these symptoms [1]. Limited exercise tolerance and fluid retention may

eventually lead to pulmonary congestion and peripheral edema. Neither of these symptoms necessarily dominates the clinical picture at the same time. The absence of dyspnea on exertion is helpful to rule out the diagnosis of heart failure with a sensitivity of 84%. Other symptoms that are helpful in diagnosing heart failure include orthopnea, paroxysmal nocturnal dyspnea (PND), and peripheral edema. PND has the highest specificity: 84% of any symptom for heart failure. It is important to remember that no single clinical symptom has been shown to be both sensitive and specific. A substantial portion of the population has asymptomatic left ventricular systolic dysfunction, and the history alone is insufficient to make the diagnosis of heart failure. However, a detailed history and review of symptoms remain the best approach in identifying the cause of heart failure and assessing response to therapy.

Heart failure occurs if the heart is unable to perfuse body tissues adequately [6]. Cardiac output depends on three factors: preload, contractility, and afterload. Preload (also referred to as the left ventricular end diastolic pressure) is the pressure required to distend the ventricle at a given volume. The relationship of pressure and volume defines compliance. Contractility describes the functional state of the myocardial muscle. Normally, as preload increases, the cardiac output and the amount of blood pumped by the heart muscle increase. Afterload is the resistance against which the heart contracts and is clinically reflected by the systolic blood pressure.

Heart failure begins with symptoms that occur only during periods of stress, as during illness or exercise; but as the disease progresses, symptoms may occur with rest. Heart failure may be due to either systolic or diastolic dysfunction. Systolic dysfunction is characterized by decreased contractility of the left ventricle, resulting in a reduced ejection fraction. A decreased ejection fraction leads to a compensatory increase in preload to maintain cardiac output. Eventually, there is a limit to which increases in preload can compensate, and pulmonary congestion occurs, resulting in signs and symptoms such as orthopnea, paroxysmal nocturnal dyspnea (PND), rales, jugular venous distention (JVD), and edema.

Decreases in cardiac output trigger a host of compensatory mechanisms, including activation of the renin–angiotensin–aldosterone system, increased levels of catecholamines, and the secretion of atrial natriuretic hormone. These compensatory mechanisms result in systemic vasoconstriction, fluid retention, and increased afterload, which further inhi bits cardiac output, thus creating a vicious feedback cycle. Late changes effected by these compensatory mechanisms include myocardial and vascular remodeling and fibrosis.

Patients with diastolic cardiac failure are frequently elderly and female, with a history of hypertension, diabetes, and obesity [7]. Atrial fibrillation, if present, is usually paroxysmal, and a fourth heart sound (S_4 gallop) is often present in periods of normal sinus rhythm. In systolic heart failure, which can occur in all ages and more often in males, atrial fibrillation tends to be persistent, a third heart sound is present (S_3 gallop), and there is often a history of previous myocardial infarction. Classic signs of both types of failure include tachypnea, tachycardia (with pulsus alternans), JVD, rales, hepatojugular reflux, ascites, edema, hepatosplenomegaly, cephalization and congestion of pulmonary markings on chest radiography with or without cardiomegaly, and diminished oxygen saturation on pulse oximetry. Reasonable investigations in acute failure include the following: 12-lead ECG (electrocardiogram); chest radiograph; blood chemistries including BUN, creatinine, glucose, and electrolytes; BNP; CBC; TSH; liver function tests and lipids; urinalysis for protein and glucose; echocardiography; and pulse oximetry.

Hypertension

Left ventricular hypertrophy (LVH) is a predictor of CHF (congestive heart failure), as well as of coronary disease, stroke, and peripheral vascular disease [8]. The risk increases progressively with the increase in left ventricular muscle mass and with the severity of LVH by ECG criteria. The ECG and anatomical indicators of LVH (radiography and echocardiogram) contribute independently to the risk of CHF and other sequelae. Patients who have both are at greater risk than persons with either one. The ECG is less sensitive than the echocardiogram as a test for LVH, but economic factors make it more suitable as a routine test for patients with

hypertension. Biochemical markers of heart failure such as BNP (brain naturetic protein) and a metabolite of its precursor (NT-proBNP) have been found to have high sensitivity and specifi city for CHF. These tests have been mainly tested in the settings of emergency departments and cardiology outpatient clinics. Their usefulness in the setting of family practice remains uncertain, and, in any case they are not as yet widely available.

There are populations in economically undeveloped countries that do not show the rise of blood pressure with age characteristic of people in industrialized countries. When people emigrate from undeveloped to developed societies, they change to the pattern of the new environment. In North America and Europe, population studies have not shown significant differences in blood pressure between social classes as defined by occupation, income, and education. This is not the case, however, with mortality from hypertension -related disease. In both the United States and Britain, mortality is much higher among the poor and those with lower levels of education. In the United States, African Americans have a much higher mortality from hypertension-related disease than whites. The reasons for these differences are not clear. For family physicians, however, they do signify the need for special attention to patients in lower income groups, and especially to patients of African descent.

The heart and blood vessels (arteries and veins) are essentially a pump with outgoing and returning vessels, which unlike rigid pipes, are aff ected by the pump pressure, the flow of blood, and the environmental and other factors that influence the tone of the smooth muscles that line these channels [9]. It is indeed this tone in the arteries (called peripheral resistance) that is particularly important in raising the blood pressure. There is an orchestral-like arrangement among all of these factors that functionally harmonize to produce our blood pressure. Factors that change any part of this balanced arrangement have the potential of raising the blood pressure. For example, substances that raise the arterial tone may elevate the blood pressure. These include some anti-inflammatory drugs, some medications for asthma, oral contraceptives, caffeine, nicotine, and cocaine, as well as other illicit drugs.

The blood pressure is recorded by two figures, one (systolic) over the other (diastolic). Blood pressure is recorded by a pressure cuff being placed around the upper arm, inflating the cuff , and then listening with a stethoscope at the site of the brachial artery at the elbow joint just below the cuff. As the cuff is deflated, tapping sounds are first heard, at which level the mercury measurement can be made to assess the systolic reading. When the sounds disappear, the reading made then is the diastolic pressure. The normal systolic blood pressure is 120 or less (expressed as millimeters of mercury) over diastolic pressure of 80 or less.

While descriptions of mild, moderate, and severe hypertension have been used for decades, new advances have made it clear that hypertension is best staged by assessment of the levels of systolic and diastolic pressures. This change has come about because of the realization that the word "mild" has conveyed an idea of unimportance. In fact, about 70 % of those with diastolic hypertension, and more than half of the deaths and disability that are attributable to hypertension, have occurred with levels of diastolic blood pressure between 90 and 104. The level of systolic blood pressure is also important, especially since it is a major contributor to complications that occur because of hypertension, which include mortality, coronary heart disease, strokes, heart failure, and kidney failure.

Types

Left-Sided Heart Failure

Left-sided failure may be due to hypertension (high blood pressure), anaemia, hyperthyroidism (overactivity of the thyroid gland), a heart valve defect (such as aortic stenosis, aortic incompetence, or mitral incompetence), or a congenital heart defect (see heart disease, congenital). In all of these conditions, the left side of the heart must work harder than normal to pump the same amount of blood [10]. Sometimes, the heart can compensate for the extra workload by an increase in the size of the left side and in the thickness of its muscular walls, or by an increase in the heart rate. This compensation is only temporary, however, and heart failure eventually follows.

Other causes of left-sided heart failure include

coronary artery disease, myocardial infarction (heart attack), cardiac arrhythmias (irregularities of heart rhythm), and cardiomyopathy (disease of the heart muscle). In cardiomyopathy, the pumping power of the heart is reduced to a point where it can no longer deal with its normal workload.

Whatever the underlying cause, in left-sided heart failure the left side of the heart fails to empty completely with each contraction, or has difficulty in accepting blood that has been returned from the lungs. The retained blood creates a "back pressure" that causes the lungs to become congested with blood. This condition leads to pulmonary oedema (excess fluid in the lungs), of which the main symptom is shortness of breath, eventually even when at rest. The patient may awaken at night with attacks of breathlessness, wheezing, and sweating.

Right-Sided Heart Failure

Right-sided failure is most often caused by pulmonary hypertension (raised blood pressure in the arteries supplying the lungs). This is itself caused by left -sided heart failure, or a lung disease such as chronic obstructive pulmonary disease (see pulmonary disease, chronic obstructive). Rightsided failure can also be due to a heart valve defect, such as tricuspid incompetence, or to a congenital heart defect.

In all types of right-sided heart failure, there is back pressure in the circulation from the heart into the venous system, causing swollen neck veins, enlargement of the liver, and oedema (excess fluid in body tissues), especially swelling of the legs and ankles. In addition, the intestines may become congested, causing discomfort and indigestion.

Examination

The clinical examination is helpful to assess the degree of reduced cardiac output, volume overload, and ventricular enlargement [1]. It can also provide clues to noncardiac causes of dyspnea. A third heart sound, S_3 (ventricular filling gallop), on examination is specific for increased left ventricular end-diastolic pressure and decreased left ventricular ejection fraction. It has the best specificity of any exam finding for heart failure: 99%. Thus, a third audible sound along with a displaced cardiac apex are both very predictive and effectively rule in a diagnosis of left ventricular systolic dysfunction. Volume overload from heart failure can present with many signs on examination. The presence of jugular venous distention and a hepatojugular reflex can be present and moderately effective in diagnosing heart failure. Other signs such as pulmonary rales (crackles), a murmur, or peripheral edema have a smaller but helpful effective in diagnosing heart failure.

Other exam findings can assist in determining causes of heart failures or assess other differential diagnosis. Cardiac murmurs may be an indication of primary valvular disease. Asymmetric rales or rhonchi on the pulmonary examination may suggest pneumonia or chronic obstructive pulmonary disease (COPD). Dullness to percussion or auscultation of the lungs could indicate pleural effusion. Examination of the thyroid can exclude thyromegaly or goiter, which could cause abnormal thyroid function precipitating heart failure. Hepatomegaly can indicate passive hepatic congestion.

Management

The management of a HF (heart failure) patient when the follow-up in the office includes the goals of enhancing quality of life, preventing hospitalizations due to worsening status, and preventing disease progression and avoidable death [11].

To enhance quality of life, you should routinely assess the patient's functional capacity (NYHA class), monitor HF signs and symptoms, and review self-care strategies with the patient or family. Changes in NYHA class (New York Heart Association) indicate disease progression and need for more intensive therapy or possible conversations about end-of-life and prognosis. The patient's weight and volume status with its corresponding signs (peripheral edema, crackles on lung exam) may prompt discussion of diet and medication adherence, medication adjustments (diuretics). Changes in dyspnea may direct the conversation to exercise prescription or further evaluation of ischemic heart disease, or rate control if atrial fibrillation is present.

Management of HF requires a partnership between the physician and patient. At each encounter, inquire about specific self-care tasks such as monitoring daily weights, symptom monitoring (understanding symptoms), dietary and medication adherence, following exercise prescription, and avoiding medications that

worsen symptoms. Many hospitalizations can be avoided if patients follow these simple self-care tasks and understand how to intervene when signs of early volume overload appear.

To prevent disease progression and its attendant consequences such as hospitalizations, decline in functional status, and premature death requires close attention to control the upstream contributing causes of HF and medication regimens with demonstrated benefit. Achieving optimal control of the conditions that have caused or contribute to HF applies to both systolic and diastolic HF. Thus, you should help the patient keep his or her blood pressure under 140/90, achieve lipid target goals, keep the hemoglobin A1c between 7.0 and 7.5%, manage ischemia to prevent ongoing cardiac damage, and keep the heart rate in patients with atrial fibrillation between 60 and 80 beats per minute (bpm). Case management is another care option that can help HF patients follow treatment guidelines, attack barriers to behavior changes, and reduce hospitalizations.

Role of the Nurse

Even within the same profession, differences exist among its practitioners [12]. Different senses need to be trained to meet the domains of nursing practice in different areas. Consider the nurse working in critical and acute care. He needs to retrain his senses to identify and manage life-sustaining physiological functioning in unstable patients. Th is means developing knowledge and observational skills to detect early signs of instability, such as a sudden change in cardiovascular functioning as evidenced by a change in the quality of a patient's pulse. Pulse is caused by pressure exerted on the arterial wall causing expansion of the vessel for a brief moment as the wave of pressure passes. All arteries demonstrate a pulse, but only a few are accessible to observation. Th e nurse may observe signs of cardiovascular distress by noticing the color of the patient's skin (sight) or detecting a faint sigh or murmur (sound); however, observation of the patient's pulse depends on touch. By gently asserting pressure on the arterial site, the nurse notes rate, strength, and quality. Consider a rapid pulse rate. It may be an indication of tachycardia, or a compensatory mechanism as a result of a systemic infection, or a sign that the patient is going into shock. Bradycardia, a slow pulse, may indicate a heart transmission block when the impulse from the sino-atrial node does not reach the ventricle and the ventricular contractions slow. The strength of the pulse also indicates different aspects of heart function: A weak pulse may indicate left heart failure due to the myocardium's inability to reach full stoke volume, or a loss of volume, such as that which happens with a bleed. In these cases, retraining the senses requires the nurse to learn how to locate a pulse; how to exert just enough pressure to feel the pulse so that its rate can be counted; and how to develop the proprioreceptors in his own fingertips to distinguish between a thready pulse, a pounding pulse, and so forth. The nurse also has to interpret the information in light of the information he has about cardiovascular physiology and to relate that information to knowledge of this particular patient (e.g., the patient's normal pulse, medical history, and current medical condition), in order to determine whether changes in pulse quality and rate are significant.

Conclusion

The pathophysiology of heart failure is complex and there is no single cause, so we say that it is more of a clinical syndrome than a specific disease. Structural and functional causes of heart failure include ischemic heart disease as the leading cause, then high blood pressure, heart valve disease, congenital heart disease, cardiomyopathy. Risk factors include viral infections, anemia, heart failure, diabetes, thyroid disease, kidney disease, some medications. Other risk factors include smoking, obesity, excessive alcohol consumption, and a high fat diet and salt.

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