HEART FAILURE- A MAJOR PUBLIC HEALTH PROBLEM

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Keywords: *heart failure, natriuretic peptide, public health problem*

Abstract: *Heart failure is one of the most common cardiac disease that affects people of all ages worldwide. Mechanisms that cause the disease began to be increasingly well understood. The most important are: Frank-Starling mechanism, myocardial hypertrophy and neurohormonal mechanisms. The most common cause of heart failure is the acute coronary syndrome. In fact, they say that almost any heart disease left untreated can lead to heart failure. The NYHA classification is used to determine the clinical evaluation of the disease. Echocardiography is the most frequently used imaging method to assess heart failure and B-type natriuretic peptide determination is becoming a routine laboratory examination used in this disease.Treatment of heart failure is trying to improve the patients' life, to slow disease progression and reduce mortality.*

Cuvinte cheie: *insuficienţa cardiacă, peptide natriuretice, problemă de sănătate publică*

Rezumat: *Insuficienţa cardiacă reprezintă una dintre cele mai frecvente boli cardiace care afectează populaţia de toate vârstele, pe plan mondial. Mecanismele care produc această boală încep să fie tot mai bine înţelese. Dintre acestea, cele mai importante sunt reprezentate de: mecanismul Frank-Starling, hipertrofa miocardică şi mecanismele neurohormonale.Cea mai frecventă cauză a insuficienţei cardiace o constituie sindromul coronarian acut. De fapt, se spune că aproape orice boală cardiacă lăsată netrată poate conduce la apariţia insuficienţei cardiace. Pentru evaluarea clinică a insuficienţei cardiace se foloseşte clasificarea NYHA. Ecocardiografia reprezintă cea mai frecvent folosită metodă de evaluare imagistică a insuficienţei cardiace, iar dozarea peptidului natriuretic de tip B începe să devină o examinare de laborator utilizată de rutină în această boală. Tratamentul insuficienţei cardiace încearcă să îmbunătăţească calitatea vieţii pacienţilor, să încetinească progresia bolii şi să reducă mortalitatea.*

INTRODUCTION

Heart failure is a syndrome manifesting as the inability of the heart to fill with or eject blood due to any structural or functional cardiac conditions.

In terms of incidence, prevalence, morbidity, and mortality, the epidemiologic magnitude of heart failure (HF) is staggering. According to the American Heart Association, heart failure is a condition that affects nearly 5.7 million Americans of all ages and is responsible for more hospitalizations than all forms of cancer combined. It is the number 1 cause for hospitalization among Medicare patients. With improvement in survival of acute myocardial infarctions and a population that continues to age, heart failure will continue to increase in prominence as a major health problem in the United States and worldwide.(1,2,3)

Pathophysiology

Regardless of the precipitating event, the common pathophysiologic state that perpetuates the progression of heart failure is extremely complex. Compensatory mechanisms exist on every level of organization from sub-cellular all the way through organ-to-organ interactions. Only when this network of adaptations becomes overwhelmed does heart failure ensue. In this section, we focus on those adaptations that represent significant therapeutic targets in the treatment of heart failure.(9)

Most important among these adaptations are the (1) Frank-Starling mechanism, in which an increased preload helps to sustain cardiac performance; (2) alterations in myocyte regeneration and death; (3) myocardial hypertrophy with or without cardiac chamber dilatation, in which the mass of contractile tissue is augmented; and (4) activation of neurohumoral systems, especially the release of norepinephrine by adrenergic cardiac nerves, which augments myocardial contractility and includes activation of the renin-angiotensin-aldosterone system (RAAS), sympathetic nervous system (SNS), and other neurohumoral adjustments that act to maintain arterial pressure and perfusion of vital organs. In acute heart failure, the finite adaptive mechanisms that may be adequate to maintain the overall contractile performance of the heart at relatively normal levels become maladaptive when trying to sustain adequate cardiac performance.(9)

The primary myocardial response to chronic increased wall stress is myocyte hypertrophy, death/apoptosis, and regeneration.[2] This process eventually leads to remodeling, usually the eccentric type. Eccentric remodeling further worsens the loading conditions on the remaining myocytes and perpetuates the deleterious cycle. The idea of lowering wall stress to slow the process of remodeling has long been exploited in treating heart failure patients.

The reduction of cardiac output following myocardial injury sets into motion a cascade of hemodynamic and neurohormonal derangements that provoke activation of neuroendocrine systems, most notably the above-mentioned adrenergic systems and RAAS. The release of epinephrine and norepinephrine, along with the vasoactive substances

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Article received on 02.08.2010 and accepted for publication on 21.02.2011
ACTA MEDICA TRANSILVANICA June 2011; 2(2)187-189

endothelin-1 (ET-1) and vasopressin, causes vasoconstriction, which increases afterload, and, via an increase in cyclic adenosine monophosphate (cAMP), causes an increase in cytosolic calcium entry. The increased calcium entry into the myocytes augments myocardial contractility and impairs myocardial relaxation (lusitropy).

In addition, the activation of the RAAS leads to salt and water retention, resulting in increased preload and further increases in myocardial energy expenditure. Increases in renin, mediated by decreased stretch of the glomerular afferent arteriole, reduced delivery of chloride to the macula densa and increased beta1-adrenergic activity are responses to decreased cardiac output. This results in an increase in angiotensin II (Ang II) levels and, in turn, aldosterone levels. This results in stimulation of the release of aldosterone. Ang II, along with ET-1, is crucial in maintaining effective intravascular homeostasis mediated by vasoconstriction and aldosterone-induced salt and water retention.(9)

As heart failure advances, there is a relative decline in the counterregulatory effects of endogenous vasodilators, including nitric oxide (NO), prostaglandins (PGs), bradykinin (BK), atrial natriuretic peptide (ANP), and B-type natriuretic peptide (BNP). This occurs simultaneously with the increase in vasoconstrictor substances from the RAAS and adrenergic systems. This fosters further increases in vasoconstriction and thus preload and afterload, leading to cellular proliferation, adverse myocardial remodeling, and antinatriuresis with total body fluid excess and worsening congestive heart failure symptoms.

ANP and BNP are endogenously generated peptides activated in response to atrial and ventricular volume/pressure expansion. ANP and BNP are released from the atria and ventricles, respectively, and both promote vasodilation and natriuresis. Their hemodynamic effects are mediated by decreases in ventricular filling pressures, owing to reductions in cardiac preload and afterload. BNP, in particular, produces selective afferent arteriolar vasodilation and inhibits sodium reabsorption in the proximal convoluted tubule. BNP inhibits renin and aldosterone release and, therefore, adrenergic activation as well. Both ANP and BNP are elevated in chronic heart failure. BNP, in particular, has potentially important diagnostic, therapeutic, and prognostic implications.(4,5,6,7)

Causes of Heart Failure

For heart failure to occur, there must be an unresolved impairment of the heart that compromises its ability to work as a pump. The source of this can be a cutoff of blood supply, an increase in workload due to high blood pressure caused by non-functioning valves or a genetic predisposition. Heart failure can be worsened by a poor diet and lifestyle.

Coronary Artery Disease (CAD)

This is the most common cause of heart failure in the U.S. today. CAD causing obstruction to the coronary arteries prevents blood flow and, therefore, oxygen delivery to the heart. CAD is a manifestation of atherosclerosis, which can affect any artery of the body. Risk factors for CAD also include smoking, high cholesterol, hypertension, and diabetes.

Hypertension

This is more commonly known as high blood pressure. It is a condition that is treatable and simple to diagnose with a blood pressure cuff. Although most individuals will not have symptoms, hypertension is detected by a simple measurement with a blood pressure cuff and stethoscope. It is also a risk factor for CAD, stroke, peripheral vascular disease, or kidney impairment.

Valvular Heart Disease

A condition that occurs when the valves between the chambers of the heart are faulty, either due to birth defect or injury.

Cardiomyopathy

A disease of the heart muscle. This can be one of many varieties. It can arise because of genetic causes, a viral infection, or consumption of toxins (lead, alcohol, etc.). In peripartum cardiomyopathy, women who have recently given birth can develop heart muscle impairment. In many cases, the condition is called "idiopathic", which means it has occurred of uncertain origin or cause.

In addition to those causes above, the following factors also can play a role in the evolution of heart failure:

1. family history of heart failure
2. diabetes
3. marked obesity
4. heavy consumption of alcohol, or drug abuse
5. failure to take medications
6. large salt intake in diet
7. sustained rapid heart rhythms

Clinical

The NYHA classification of heart failure is widely used in practice and in clinical studies to quantify clinical assessment of heart failure. Breathlessness, a cardinal symptom of LV failure, may manifest with progressively increasing severity as (1) exertional dyspnea, (2) orthopnea, (3) paroxysmal nocturnal dyspnea, (4) dyspnea at rest, and (5) acute pulmonary edema. Other cardiac symptoms of heart failure include chest pain/pressure and palpitations. Patients often manifest noncardiac symptoms of heart failure like anorexia, nausea, weight loss, bloating, fatigue, weakness, oliguria, nocturia, and cerebral symptoms of different severity ranging from anxiety to memory impairment and confusion.

Laboratory Studies

The release of BNP (B-type natriuretic peptide) appears to be in direct proportion to ventricular volume and pressure overload. BNP is an independent predictor of high LV end-diastolic pressure and is more useful than ANP or norepinephrine levels for assessing mortality risk in patients with heart failure. (2,3,4)

Imaging Studies

Echocardiography determines LV/RV size and function, LV wall motion abnormalities, valvular function and abnormalities, diastolic function, presence or absence of pericardial abnormalities or intracardiac masses; evaluates intracardiac filling pressures.

Treatment

Although heart failure is a serious condition that progressively gets worse over time, certain cases can be reversed with treatment. Even when the heart muscle is impaired, there are a number of treatments that can relieve symptoms and stop or slow the gradual worsening of the condition.(8)

The goals of heart therapy are to:

- Relieve symptoms and improve quality of life
- Slow disease progression
- Reduce the need for emergency room visits and hospitalization
- Help people live longer

Treatment options depends on the type, cause, symptoms and severity of the heart failure. Usually, more than one therapy is used. These options include:

- Treating the underlying causes
- Lifestyle changes
- Medications
- Surgery

Heart failure is a major public health problem in

industrialized countries, the prevalence of which appears to be rising over the last decade. Recent studies, however, suggest that survival in heart failure may be improving, concurrent with the advent of improved medical therapy.

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