REVIEW OF RECENT ADVANCES & TECHNIQUES IN SURGICAL MANAGEMENT OF HEART FALIURE

Essay

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To my father my role model and the reason I chose this career, to my godfather the source of inspiration and the reason I'm still in this career and to all my professors and to my family who made this work possible

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WHAT IS HEART FAILURE

Definition of Heart Failure:

The clinical syndrome of HF is defined as the final common pathway that results from any structural or functional cardiac disorder that impairs the ventricle from either filling with (diastolic dysfunction) or ejecting blood (systolic dysfunction). The diverse causes of HF range from disorders of the pericardium, myocardium, endocardium, or great vessels. Despite the fact that the majority of patients with HF have symptoms secondary to impaired systolic function, it is important to recognize that symptoms may also arise due to abnormal filling. The overall prognosis of HF with preserved ejection fraction (HF-PEF) is less well defined, with certain observational series suggesting improved outcomes compared to HF with reduced ejection fraction (HF-REF), while other series have shown similar mortality for HF-PEF and HF-REF.[1]

Epidemiology:

Generally speaking, the prevalence of heart failure can be estimated at 1–2% in the western world and the incidence approaches 5–10 per 1000 persons per year. Estimates of the occurrence of heart failure in the developing world are largely absent. [2] [8]

Reliable estimates of the incidence of heart failure are available from the Rotterdam and Hillingdon heart failure studies. Both studies are population based and used an expert panel to establish the presence or absence of heart failure. In the **Hillingdon study** the incidence of heart failure increased from 0.2/1000 person years in those aged 45–55 years to 12.4/1000 person years in those aged >85 years. In **Rotterdam** the incidence increased from 2.5/1000 person years (age 55–64 years) to 44/1000 person years (>85 years or older). Heart failure occurs more frequently in men than in women (15 and 12 per 1000 person years, respectively). The higher figures in Rotterdam most probably reflect differences in methodology (including evaluation of all patients being prescribed diuretics or angiotensin converting enzyme (ACE) inhibitors in Rotterdam) rather than true differences in the incidence of heart failure. Interestingly, a more recent analysis from the Rotterdam study, although using a somewhat different methodology to identify incident heart failure, produced remarkably similar results. [2] [8]

Heart failure (HF) is the common end point to nearly every form of progressive cardiovascular disease. The prognosis for patients with HF is poor, and 20 % of those diagnosed with systolic HF will die within 1 year of diagnosis, with an annual mortality rate thereafter of 10 %. Moreover, HF heralds substantial morbidity and is associated with significant declines in physical and mental health, resulting in a markedly

decreased quality of life. Furthermore, HF continues to pose a tremendous economic burden on any healthcare system. In patients older than 65 years, it currently accounts for 20 % of all hospitalizations. Accordingly, there have been considerable efforts by insurance companies, federal agencies, and hospital administrators to reduce the rate of patients admitted to hospitals with this diagnosis.[2]

Many Middle Eastern countries have observed increases in the prevalence of the risk factors for the development of heart failure, including diabetes mellitus, obesity, and hypertension, with heart failure in the Middle Eastern population developing earlier than it is in their Western counterparts by at least 10 years. [3] [9]

Physiology of Heart Failure:

In its normal state, the heart's ventricles undergo filling at low pressures during diastole. The ventricles eject a percentage of this volume forward to the rest of the circulation during systole. HF occurs when either:

(1) The heart is unable to maintain its normal ejection fraction (EF), known as left ventricular systolic dysfunction.

(2) The heart maintains a normal EF but does so in the setting of elevated filling pressures, known as diastolic HF or HF with normal/preserved ejection fraction.

Left- and right-sided HF can occur independently. However, in advanced stages of HF, elevated pressures from the left side of the heart transmit pressure to the right side, precipitating right-sided HF.

Despite their interdependence in advanced HF, this chapter will focus on a discussion of left-sided HF to provide the clearest understanding of the physiology involved.[4] [5]

Left Ventricular Systolic Dysfunction/ Left-Sided HF:

Failure of the left ventricle to generate sufficient cardiac output results either from:

(a) Directly affect ventricular myocytes

(b) Secondarily from hemodynamic stress on the myocardium.

(a) Direct Injury to Ventricular Myocardium:

Direct injury to ventricular myocytes with subsequent loss of contractile function is observed most often in the case of myocardial infarction. After an extensive myocardial infarction, the infarcted tissue is no longer able to generate contractile activity, and therefore overall cardiac output is decreased. Furthermore, the myocardium adjacent to the infarcted area attempts to compensate for the loss of contractile tissue by undergoing remodeling. In this process, a programmed remodeling of the non-infarcted tissue is generated by both an

increased hemodynamic strain and the activation of local cytokines and systemic neurohormones. Although remodeling allows the myocardium to compensate in some measure initially, over time these changes transmit further stress to the adjacent tissue, ultimately, propagating worsening HF[5]

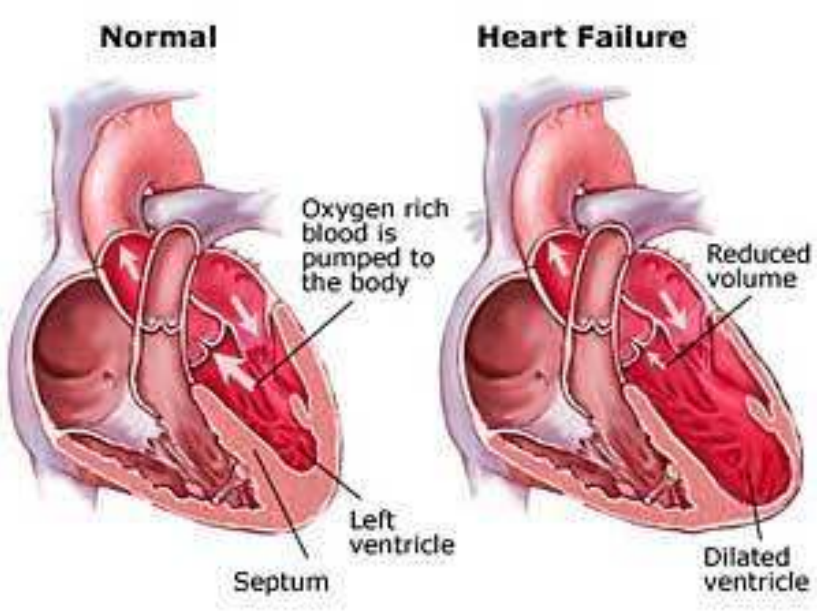

Fig. (1): Changes in heart failure

(b) Hemodynamic Stress on the Ventricular Myocardium:

Left ventricular systolic dysfunction also develops secondarily to the hemodynamic stress of a chronic pressure load (termed "afterload") or volume load (termed "preload") on the ventricular wall. Increased afterload is observed in patients with aortic stenosis and in patients with uncontrolled hypertension. Similarly, increased preload is seen in patients

with chronic mitral and/or aortic insufficiency, intracardiac shunts, and arteriovenous fistulas. In response to these disease processes, which impose sustained hemodynamic stress on the ventricular wall, the heart muscle undergoes a pathological hypertrophy. (5)

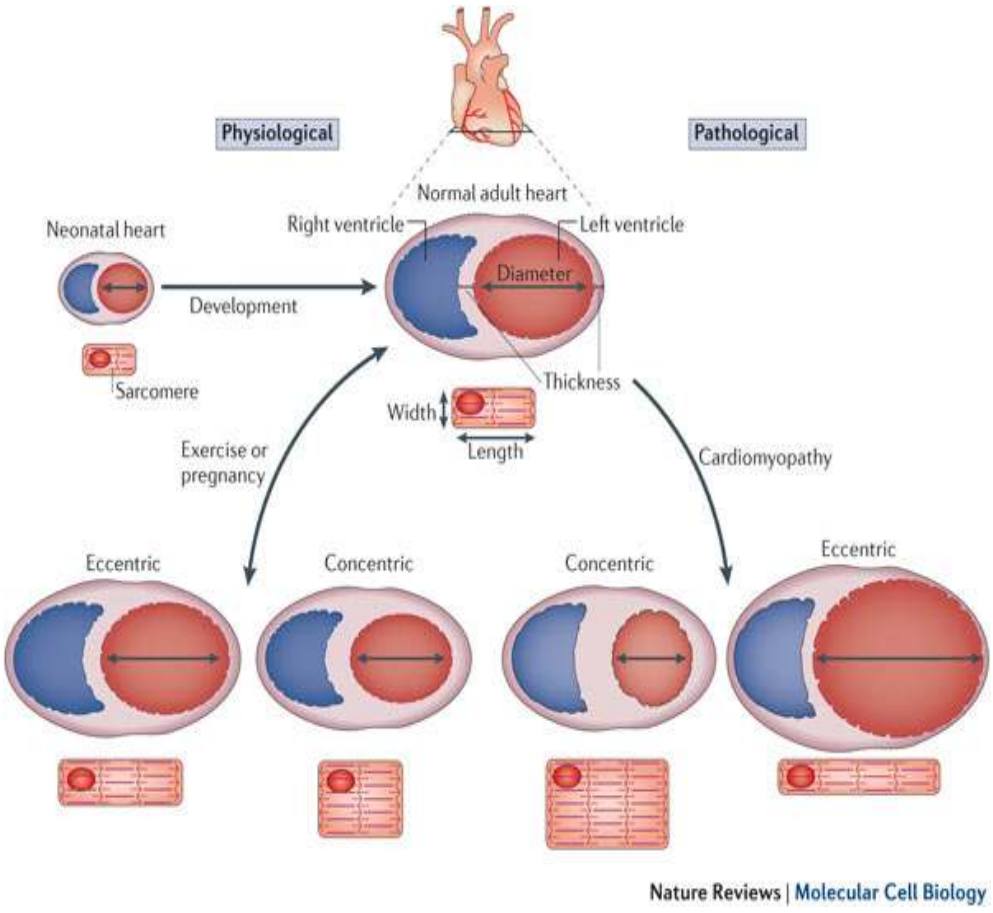

Fig. (2): Pathophysiology of heart failure

This is the early phase of remodeling. In cases of increased afterload, the ventricle undergoes concentric hypertrophy, which is characterized by an increased ventricular

wall thickness in comparison to wall cavity size. In cases of increased preload, the ventricle undergoes eccentric hypertrophy, characterized by an increase in chamber volume with normal or reduced wall thickness. (5)

Whereas cardiac hypertrophy can be a normal physiologic response to exercise, allowing for an increase in mass and improvement in contractility, pathologic hypertrophy involves no improvement in contractility. Rather, it allows the ventricle to maintain contractile force temporarily until it can no longer overcome the increased hemodynamic stress. As mentioned, the increased wall stress also promotes the production of inflammatory cytokines. These cytokines have been shown to have deleterious effects on contractile proteins by altering their expression and by triggering pathways involved in myocyte apoptosis. Cytokine and neurohormonal production have been shown to occur in later phases of remodeling. Eventually the muscle fibers accumulate collagen and fibrose. This eventually leads to left ventricular dilatation, further loss of contractile function, and thus reduced systolic function.(5)

Heart Failure with Normal/Preserved Ejection Fraction:

Diastolic dysfunction and diastolic HF have different meanings. In both cases, the ventricle becomes less compliant, leading to impaired/abnormal ventricular filling, as measured by echocardiography or other imaging modalities.

Diastolic dysfunction refers only to impaired/ abnormal filling by imaging; diastolic HF instead refers to diastolic dysfunction with clinical symptoms and signs of HF. To more clearly make a distinction between these two entities, the term "diastolic HF" is now substituted by a relatively new construct referred to as Heart Failure with Normal/Preserved Ejection Fraction (HFNEF). Approximately 50 % of the overall HF population has a normal left ventricular ejection fraction (LVEF). In comparison to patients with HF and low LVEF, these individuals are more likely to be women and more likely to be older. They also have a higher likelihood of obesity, hypertension, renal failure, atrial fibrillation, and anemia. [4] [5]

The clinical syndrome of HF in these individuals can be as profound as those patients with HF symptoms and low LVEF. Similarly, the prognosis of patients with clinical HF and normal LVEF is only minimally better in comparison to those with patients with a low LVEF. These two entities also share common etiologies.[1,6]

As mentioned, aortic stenosis and poorly controlled hypertension often lead secondarily to left-sided heart failure. Prior to the development of left-sided HF, the ventricle remodels via a mechanism of concentric hypertrophy, known as left ventricular hypertrophy (LVH), as it works to preserve cardiac output. With LVH, there is often impaired ventricular relaxation and thus higher ventricular filling pressures. LVH is

therefore a common cause of HFNEF since higher ventricular filling pressures can cause "backup" of fluid into the lungs despite normal LV contractility. The other major causes of HFNEF are also attributable to impaired ventricular relaxation and include transient myocardial ischemia, infiltrative processes that deposit into the myocardial architecture creating a restrictive cardiomyopathy, and hypertrophic cardiomyopathy. Infiltrative processes involve the intercalation of toxins, diseases, or infections into the myocardium. The following are examples of common infiltrative sources: chemotherapy, amyloidosis and other connective tissue diseases, alcohol from long-term abuse, and human immunodeficiency virus (HIV) and other viruses. Genetic and myopathic disorders such as Duchenne Muscular Dystrophy can also produce a restrictive cardiomyopathy.(6)

As mentioned, patients with left-sided HF and HFNEF not only share similar etiologies but often have similar clinical presentations. However, the mechanisms by which the left ventricle acts to maintain stroke volume in these two groups of patients are different. In HF with low LVEF, the eccentric or dilated left ventricle acts to maintain stroke volume via the Frank-Starling mechanism. By this mechanism, the left ventricle's increased compliance accommodates for greater ventricular filling and thus a greater end-diastolic volume (EDV). This permits a greater stroke volume with each subsequent contraction and thus a way to preserve forward