International Journal of Applied Research 2021; 7(9): 180-182

ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor: 8.4
IJAR 2021; 7(9): 180-182
www.allresearchjournal.com
Received: 23-09-2021
Accepted: 26-08-2021

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Congestive heart failure

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Abstract
Congestive heart failure is a syndrome that can be caused by a variety of abnormalities, including pressure and volume overload, loss of muscle, primary muscle disease or excessive peripheral demands such as high output failure. In the usual form of heart failure, the heart muscle has reduced contractility. This produces a reduction in cardiac output, which then becomes inadequate to meet the peripheral demands of the body. The 4 primary determinants of left ventricular (LV) performance are generally altered as follows: (1) There is an intrinsic decrease in muscle contractility. (2) Preload or left atrial filling pressure is increased, resulting in pulmonary congestion and dyspnea. (3) Although systemic blood pressure is often reduced, there is an increase in systemic vascular resistance (afterload), which can further reduce cardiac output. (4) Heart rate is generally increased as part of a compensatory mechanism associated with an increase in sympathetic tone and circulating catecholamines. In patients with coronary disease, there is often an imbalance between myocardial oxygen supply and demand. An increase in heart size may be particularly deleterious by increasing wall tension because of the Laplace relation and increasing myocardial oxygen consumption.
Intrinsic compensatory mechanisms include an increase in catecholamines, which increase contractility and heart rate in an attempt to maintain cardiac output; cardiac muscle hypertrophy, which helps maintain cardiac function; a rise in LV filling pressure, which can optimize performance according to the Frank-Starling mechanism; and an increase in peripheral arterial-venous oxygen extraction so as to maximize the oxygen delivered for a given cardiac output. Although these compensatory mechanisms are initially helpful, many of them may actually be excessive, such as an increase in catecholamines and systemic vascular resistance.

Keywords: Heart failure, NYHA classification, Preload, afterload

Introduction
Heart failure (HF) is a clinical syndrome caused by structural and functional defects in myocardium resulting in impairment of ventricular filling or the ejection of blood. The most common cause for HF is reduced left ventricular myocardial function; however, dysfunction of the pericardium, myocardium, endocardium, heart valves or great vessels alone or in combination is also associated with HF. Some of the major pathogenic mechanisms leading to HF are increased hemodynamic overload, ischemia-related dysfunction, ventricular remodeling, excessive neuro-humoral stimulation, abnormal myocyte calcium cycling, excessive or inadequate proliferation of the extracellular matrix, accelerated apoptosis and genetic mutations.

Classification of HFs
The New York Heart Association (NYHA) functional classification defines four functional classes as:

- Class I: HF does not cause limitations to physical activity; ordinary physical activity does not cause symptoms.
- Class II: HF causes slight limitations to physical activity; the patients are comfortable at rest, but ordinary physical activity results in HF symptoms.
- Class III: HF causes marked limitations of physical activity; the patients are comfortable at rest, but less than ordinary activity causes symptoms of HF.
- Class IV: HF patients are unable to carry on any physical activity without HF symptoms or have symptoms when at rest.

The American College of Cardiology/American Heart Association (ACC/AHA) staging system is defined by the following four stages:

- Stage A: High risk of heart failure, but no structural heart disease or symptoms of heart failure;
- Stage B: Structural heart disease, but no symptoms of heart failure;
- Stage C: Structural heart disease and symptoms of heart failure;
- Stage D: Refractory heart failure requiring specialized interventions.

Case Report

Diagnosis: Congestive Heart Failure, Global Hypokinesia, Severe Mr, Mild TR, Trivial Ar, Severe PAH, NSR, Normal LV Size & Moderate LV Dysfunction, LVEF ~ 34%

History & Examination

This patient non diabetic, non-hypertensive, with no family h/o CAD, Presented with c/o retrosternal chest pain, radiating to back associated with dyspnea on exertion & palpitation since 15 days, consulted in trauma & emergency, diagnosed as Congestive Heart Failure and Referred here for further management. No H/o syncope. O/E BP= 126/86 mmHg, Pulse = 106 Bp/m, regular, JVP- Normal, CVS- S1 normal, P2 loud, pansystolic murmur present at apex.
R/S- B/L basal crepts present. Patient was admitted for management.

ECG - NSR, Left Axis Deviation,

ECHO: Normal LV Size & Moderate LV Dysfunction, LVEF ~ 34%, Global Hypokinesia, Moderate To Severe Mr, Mild TR (RVSP 76.43 Mmhg),
Trivial Ar, Severe PAH, Ra & RV- Normal Size

Hospital Course: Patient was managed conservatively with Lasix, Ramipril, Bisoprolol, Ivabradine, Spironolactone and other supportive measures.

Status at Discharge: Stable

Future Plan: Medical Management

Investigations

USG Abdomen: S/O Moderate Left Sided Pleural Effusion, Simple Hepatic Cyst in Segment V of Liver

Table 1: Biochemistry Report

CBC		RFT		LFT		Thyroid profile		Viral marker	
TRBC	**4.68millon/ul**	**Urea**	**40mg/dL**	**AST**	**36U/L**	**T4**	**7.7ug/dl**	**COVID-19(17.03.21)**	**NEG**
HB	13.8gm%	Creatinine	1.2mg/dL	ALT	128U/L	T3	0.66ng/ml		
TLC	5910/ul	UA	10.5mg/dL						
Platelet	131000/ul	Na	136mEq/L						
Urine R/M	WNL	K	4.4mEq/L						

Table 2: Lipid Profile

Lipid profile		Blood sugars		Coagulation profile		Others	
T. Cholesterol	**136mg/dL**	**HBA1C**	**5.7 %**	**PT**	**24.8sec**	**Magnesium**	**1.95 mg/dL**
TG	70mg/dL			INR	2.4	Calcium	9.3 mg/dL
HDL	17mg/dL						
VLDL	14mg/dL						
LDL	105mg/dL						

Systemic Examination

CNS- Conscious
RS- B/L basal crepts present
P/A- Within normal limits
CVS- S1 normal, P2 loud, pansystolic murmur present at apex.

Discussion

Despite the advancement in medicine, management of heart failure (HF), which usually presents as a disease syndrome, has been a challenge to healthcare providers. This is reflected by the relatively higher rate of readmissions along with increased mortality and morbidity associated with HF.

Conclusion

Congestive heart failure has plagued humankind for as long as recorded history. Only in recent years have we been able to recognize it as a condition brought about by disease, trauma, and even congenital abnormalities, anything that causes the heart itself to function in an abnormal and stressful manner for extended lengths of time. Now we can anticipate its development by recognizing the conditions that cause it to occur. We even have the screening tools to measure it, such as the New York Functional Assessment and the American Heart Association/American College of Cardiology Heart Failure Staging Tool, so we can better focus on early treatment and aggressive control with medications, invasive procedures, and especially with lifestyle changes. These areas will be discussed in detail in sections two and three of this special three-part series, Congestive Heart Failure.

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