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Congestive heart failure

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Abstract

Background: Heart failure is a complex clinical syndrome in which the heart cannot pump enough blood to meet the body's requirements. Heart failure is a complex clinical syndrome that results from a functional or structural heart disorder impairing ventricular filling or ejection of blood to the systemic circulation this produces a reduction in cardiac output, which then becomes inadequate to meet the peripheral demands of the body. Heart failure can severely decrease the functional capacity of a patient and increase mortality risk. It is imperative to diagnose and effectively treat the disease to prevent recurrent hospitalizations, improve quality of life, and enhance patient outcomes. Coronary artery disease and diabetes mellitus have become the predominant predisposing factors for heart failure. Other structural causes of congestive heart failure (CHF) include hypertension, valvular heart disease, uncontrolled arrhythmia, myocarditis, and congenital heart disease.

Keywords: Clinical syndrome, ejection of blood, congestive heart failure (CHF)

Introduction

Heart failure is a complex clinical syndrome that may include fatigue and shortness of breath on exertion (and in advanced cases, at rest), orthopnea, paroxysmal nocturnal dyspnea, nocturia, mental status changes, anorexia, and abdominal pain. Patients have different symptoms based on clinical severity. The severity of heart failure is defined symptomatically, and the most commonly used system is the New York Heart Association (NYHA) functional classification [13]. Patients are grouped according to the degree of effort needed to elicit heart failure symptoms. Class I patients exhibit symptoms only at exertion levels similar to those achieved readily by healthy individuals, whereas class II patients have symptoms on ordinary exertion. Class III patients have symptoms on minimal exertion, and class IV patients have symptoms at rest.

This structural remodeling of the heart produced by cardiac dysfunction results in increased preload and afterload. In turn, the increased size causes increased wall stress, thus worsening cardiac performance [30].

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: Systemic Examination

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: Systemic Examination

Lipid profile		Blood sugars		Coagulation profile		Others	
T. Cholesterol	136mg/dL	HBA1C	5.7%	PT	24.8sec	Magnesium	1.95mg/dL
TG	70mg/dL			INR	2.4	Calcium	9.3mg/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

Use of cardio protective aspirin does not attenuate the risk of Congestive Heart Failure with celecoxib, rofecoxib, diclofenac, ibuprofen or naproxen. Findings of interaction between NSAIDs and CV comorbidities were not robust to the definition of the NSAID exposure, and for naproxen and hypertension, were even counterintuitive.

Conclusion

Congestive heart failure has plagued humankind for as long as recorded history. Heart failure indeed is a complex disease and so far has been a major cause of morbidity and mortality in developing and developed countries. A standardized medical therapy has been successful in the early stages of HF. Advanced stages of HF require frequent hospitalization due to the presence of severe HF and or associated co-morbid conditions, which require strict implementation of an appropriately individualized multidisciplinary approach and quality measures to reduce re-admissions. While pharmacological management has a limited role in advanced cases of HF, novel therapeutic agents, such as regenerative and gene therapy, are in the developmental stages and need further refinement before their approval for the treatment of HF.

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