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Heart failure (HF) is a prevalent and potentially life-threatening condition when not addressed. This article strives to delineate the various forms of heart failure, elucidating the stages, types of HF, and methods of diagnosis and treatment. Additionally, it underscores the significance of an interprofessional approach that includes patients, physicians, nurses, families, and caretakers.Objectives:Describe the different types and stages of heart failure.Identify the etiologies, epidemiology, clinical symptoms, and signs of heart failure.Highlight the significance of diverse therapeutic approaches in managing heart failure, including non-medical, medical, and interventional therapies.Outline the importance of the interprofessional team to improve outcomes and enhance care delivery in patients affected by heart failure.Access free multiple choice questions on this topic.Heart failure (HF) remains a prevailing cause of cardiovascular morbidity and mortality globally despite advancements in therapies and preventive measures. The Centers for Disease Control and Prevention (CDC)estimates that 6.7 million individuals aged 20or older in the United States are affected by HF. It is anticipated that the prevalence of HF will increase to 8.5 million Americans by 2030.Definition of Heart FailureHF is a multifaceted clinical syndrome arising from functional or structural impairment in the filling or ejection of blood by the ventricles, leading to a diverse range of symptoms.American College of Cardiology/ American Heart Association Stages of Heart FailureThe American College of Cardiology (ACC) and the American Heart Association (AHA) have outlined stages of heart failure to classify the progression and severity of the condition (Table 1. ACC/AHA Stages of Heart Failure).[1]Patients who have resolved symptoms and signs of HF with persistent left ventricular dysfunction are categorized as stage C and should receive appropriate treatment. If all HF symptoms, signs, and structural abnormalities completely resolve, the patient is deemed to be in remission from HF.Table 1. ACC/AHA Stages of Heart Failure. Classification of Heart Failure by Left Ventricular Ejection FractionPatients with HF are frequently classified by left ventricular ejection fraction (LVEF).[1]This classification system acknowledges the different prognoses and responses to guideline-directed medical therapy (GDMT) for patients with heart failure. The 2022 AHA/ACC/Heart Failure Society of America (HSFA) Guideline for the Management of Heart Failure identifies 4 classes of HF by LVEF (Table 2.Classification of Heart Failure by Left Ventricular Ejection Fraction).Table 2.Classification of Heart Failure by Left Ventricular Ejection Fraction. New York Heart Association Classification of Heart FailureThe NYHA classification of HF is a subjective evaluation by a clinician to delineate the functional capacity and symptoms of individuals diagnosed with ACC/AHA stage C or D heart failure. Serving as an independent predictor of mortality, the NYHA Classification is employed in clinical settings to assess the appropriateness of therapeutic interventions for patients in stage C or D of heart failure.[1] The 4 NYHA heart failure classes are:Class I (Mild HF): No restrictions in physical activity. Ordinary physical activity does not induce undue fatigue, palpitations, or dyspnea.Class II (Mild-to-Moderate HF): Slight limitations in physical activity. Comfortable at rest, but ordinary activities result in fatigue, palpitations, or dyspnea.Class III (Moderate-to-Severe HF): Significant restrictions in physical activity. Comfortable at rest, but less than ordinary activities lead to fatigue, palpitations, or dyspnea.Class IV (Severe HF): Unable to engage in physical activity without discomfort. Symptoms of heart failure are present at rest, and any physical activity exacerbates the discomfort.The etiology of HF will vary among LVEF classifications. The etiology of HF may be multifactorial, and individual cases may have multiple contributing factors.Etiologies of HFrEF:Common causes of HFrEF include myocardial infarction, ischemic heart disease, dilated cardiomyopathy, and viral infections affecting the heart muscle. Other contributing factors may include hypertension, valvular heart disease, and genetic predisposition.Etiologies of HFmrEF:The etiologies of HFmrEFare similar to HFrEF and HFpEF. Causes of HFmrEF may include a combination of myocardial infarction, ischemic heart disease, and underlying structural heart abnormalities that fall between those seen in HFrEF and HFpEF.Etiologies of HFpEF:Common causes of HFpEF include hypertension, atrial fibrillation, age-related cardiac changes, and underlying structural heart abnormalities such as hypertensive heart disease. Other contributing factors mayinclude diabetes, obesity, and chronic kidney disease.Other possible nonischemic etiologies ofHF include:Cardiotoxic medications, including some chemotherapeutic medicationsRheumatological or autoimmune diseasesEndocrinopathies, including thyroid disease, acromegaly, pheochromocytoma, and diabetesObesityFamilial, genetic, or heritable cardiomyopathies and cardiac diseasesDysrhythmias, including tachycardias, right-ventricular pacing, or frequent premature ventricular contractionsHypotensionInfiltrative cardiac diseases, including sarcoidosis, Fabry disease, hemochromatosis, and amyloidosisMyocarditisof any etiologyPeripartum cardiomyopathyStress cardiomyopathy such as Takotsubo and reverse Takotsubo cardiomyopathiesSubstance misuse, including alcohol, cocaine, and methamphetaminesCongenital heart diseasePulmonary hypertensioncausing right HFPulmonary embolism causing right HF.HF is a significant public health problem with a prevalence of over 5.8to 6.5 million in the U.S.[2][3]and around 26 million worldwide.[4]The expectation is that8 million people in the United States will have this condition by 2030, accounting for a 46% increase in prevalence.[5] At age 45 years, the lifetime risks for HF through age 75 or 95 years were 30% to 42% in white men, 20% to 29% in black men, 32% to 39% in white women, and 24% to 46% in black and higher BP and BMI at all ages led to higher lifetime risks.[6]The increase in HF prevalence does not necessarily have links with an increase in HF incidence. The aging of the population and modern therapies for cardiac patientsthat led to increased survival could explain the increase in prevalence even with a reduction in the incidence.[7]Information collected during the second 25-year period of the Framingham Heart Study reveals that the lifetime risk of Heart Failure with Preserved Ejection Fraction (HFpEF) is estimated at around 19.3%. This surpasses the approximate 11.4% lifetime risk associated with Heart Failure with Reduced Ejection Fraction (HFrEF). Notably, this trend appears more pronounced in women, with an apparent lifetime risk of HFpEF at 10.7%, compared to 5.8% for HFrEF. Additionally, these risks demonstrate variability based on ethnicity. Between 13%and 24% of patients with HF have HFmrEF.[8][9]Heart failure is a complex clinical syndrome that occurs when the heart is unable to pump or receive blood effectively, leading to inadequate perfusion of organs and tissues. The pathophysiology of heart failure involves various mechanisms, and it often develops as a result of underlying cardiovascular diseases or conditions that strain the heart.Following the initiation triggered by various risk factors such as coronary artery disease, tachyarrhythmias, valvular heart disease, myocarditis, hypertension, obesity, and diabetes, a cascade of mechanisms unfolds within the heart. Activation of the renin-angiotensin-aldosterone system, stimulation of the sympathetic adrenergic system, vasopressin secretion through beta receptor mediation leading to excessive water absorption, persistent elevation of inflammatory markers resulting in fibrosis, maladaptive intracellular calcium handling, ATP-related energy depletion, and increased production of reactive oxygen species collectively exert detrimental effects on the left ventricle. See Image.Pathophysiology of Heart Failure.Conversely, pathways involving natriuretic peptides and positive adaptations in intracellular calcium handling exhibit protective effects on the left ventricle. These mechanisms collectively impose strain on the heart, giving rise to the phenotypic expression of the heart failure spectrum. This expression manifests as changes in ventricular preload or afterload, variations in inotropy (increased or decreased contractility), lusitropy (the ability of the ventricle to relax), and chronotropy (heart rate regulation), ultimately resulting in permanent morphological alterations in the left ventricle.In heart failure with reduced ejection fraction, the left ventricle enlarges and weakens, and the pressure-volume relationship reveals a reduction in stroke volume, an elevation in left ventricular end-diastolic pressure, and an increase in left ventricular end-diastolic volume. Heart failure with preserved ejection fraction is associated with hypertrophy and abnormal lusitropy, and the pressure-volume relationship indicates an elevation in end-diastolic pressure along with a reduction in stroke volume and left ventricular end-diastolic volume.Common histopathological findings in heart failure with preserved ejection fraction include fibrosis, hypertrophy, inflammation, and amyloidosis. Identifying amyloid traditionally involves Congo red staining, although its effectiveness varies based on staining quality and laboratory expertise. Crystal violet is an alternative stain that offers more consistency and better visibility under conventional light microscopy.In glycogen storage disorders, enlarged myocytes with cytoplasmic vacuoles containing glycogen are observed, highlighted by periodic acidshiff staining. Hypertrophic cardiomyopathy is characterized by myocyte disarray, requiring at least 20% of the overall section area for diagnosis. This condition presents with haphazardly arranged myocytes or myofiber bundles and increased interstitial fibrosis.Heart failure with reduced ejection fraction is most commonly caused by coronary artery disease. Following myocardial infarction, histologic changes, including interstitial edema, can be appreciated within 4-12 hours. Early ischemia, characterized by a neutrophilic inflammatory cell infiltrate, occurs in the first 72 hours. The subsequent stage (three to five days) involves the removal of myocytes, replaced by lymphocytes, pigment-laden histiocytes, and myofibroblasts. Two to four weeks post-infarction, collagen deposition increases, and inflammation decreases. By one to two months, dense collagen scar forms, and residual inflammatory cells are minimal.In nonischemic dilated cardiomyopathy, microscopic findings include nonspecific myocyte hypertrophy and myocardial fibrosis. Sarcoidosis exhibits patchy mononuclear inflammatory infiltrates with noncaseating granulomas, often amidst prominent myocardial fibrosis. Lyme carditis is characterized by a perivascular pattern of inflammation, resembling a "road map" and a cellular composition of lymphohistiocytic with frequent plasma cells. Giant cell myocarditis pathology involves florid myocarditis with lymphocytes, histiocytes, and multinucleated giant cells, with numerous eosinophils distinguishing it from sarcoidosis. Myocarditis histopathology involves inflammation associated with myocyte injury and various cellular compositions, including lymphocyte-predominant, lymphohistiocytic, or mixed cellular patterns with eosinophils and/or neutrophils.[10]A comprehensive history and physical examination are essential for all patients suspected of having heart failure (HF), as the diagnosis relies heavily on clinical symptoms and signs. This evaluation should encompass an analysis of risk factors and potential causes of HF. Regardless of ejection fraction (EF), HF symptoms exhibit similarities.Symptoms of Heart Failure:Shortness of breath, orthopnea, and paroxysmal nocturnal dyspneaPersistent cough or wheezingEdema (generalized or lower extremity)Fatigue and TirednessLack of appetite and nauseaConfusion and impaired thinkingPalpitationsSigns of Heart Failure:Pulses alternansElevated jugular venous pressureDisplaced LV apex beatCardiac cachexiaSinus tachycardiaRight ventricular heaveBilateral rales/ crepitations or a cardiac wheeze on lung examPresence of S3Pitting edema in dependent areasTender hepatomegalyAscitesRegular assessment of HF symptoms and signs during clinic visits is crucial for monitoring therapy response and stability. Vital signs and volume status should be evaluated at each visit to ensure a comprehensive understanding of the patient's condition.Following the initial assessment through a detailed history and physical examination, it is advisable to incorporate further diagnostic investigations. These may include an electrocardiogram, complete blood count, and a comprehensive metabolic panel encompassing liver function tests, electrolytes, and renal function tests. Additionally, considering urinalysis, lipid panel, hemoglobin A1c, thyroid-stimulating hormone, and iron studies can provide a more comprehensive understanding of the patient's health.For a more in-depth cardiac evaluation, essential labs involve measuring NT pro-brain natriuretic peptide and brain natriuretic peptide. An NT-pro-brain natriuretic peptide exceeding 125 pg/ml and a brain natriuretic peptide equal to or greater than 35 pg/ml are reliable indicators for assessing heart failure. A transthoracic echocardiogram is crucial for assessing left ventricular ejection fraction, while a chest x-ray proves beneficial when suspecting pulmonary vascular congestion. If a transthoracic echocardiogram doesn't suffice for evaluating left ventricular function, alternative modalities like cardiac MRI, cardiac CT angiogram, or radionuclide imaging should be considered (see Image.Assessment of Patients With Suspected Heart Failure Flowsheet).[11]Further specialized tests may be warranted when classifying heart failure based on ejection fraction. These include a stress echocardiogram, exercise treadmill stress test, cardiac CT angiogram or nuclear imaging and a coronary angiogram for suspected ischemia, a polysomnogram for suspected sleep apnea, an autoimmune panel for autoimmune diseases, genetic counseling and testing for familial cardiomyopathy (hypertrophic cardiomyopathy, idiopathic dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia, isolated noncompaction cardiomyopathy), 24-hour blood pressure monitoring for hypertensive cardiomyopathy, and heart rhythm monitoring for arrhythmias.Other diagnostic tools involve a transesophageal echocardiogram for valvular heart disease, cardiac MRI for structural heart disease, urine toxicology for drug abuse-related cardiomyopathy, and endocardial biopsy for giant cell myocarditis. Performing right heart catheterization and cardiopulmonary exercise testing frequently provides vital information for managing heart failure. Serum and urine immunofixation with a technetium 99 pyrophosphate scan for cardiac amyloidosis. Additionally, urine or serum HCG can be employed to evaluate heart failure further. (Figure 2)Pharmacologic therapy for Heart Failure (HFrEF, HFmrEF, and HFpEF)Beta-blockers (Class I) reverse sympathetic activation in HFrEF, improving survival, reducing heart failure hospitalizations, and increasing left ventricular ejection fraction (LVEF). Randomized controlled trials support Carvedilol, metoprolol succinate, and bisoprolol.Angiotensin receptor-neprilysin inhibitor (ARNI) sacubitril/valsartan demonstrated reduced cardiovascular and all-cause mortality in HFrEF compared to enalapril in the Prospective Comparison of ARNI with ACE-I to Determine Impact on Global Mortality and Morbidity in Heart Failure (PARADIGM-HF) trial. In a predefined subgroup analysis of participants in the Prospective Comparison of ARNI with ARB Global Outcomes in HFpEF (PARAGON-HF), it was found that sacubitril-valsartan, in comparison to valsartan alone, not only decreased the overall risk of heart failure-related hospitalizations but exhibited a more pronounced reduction in women with HFpEF compared to men. The average left ventricular ejection fraction (LVEF) in the study participants was 57%. It is indicated for symptomatic HFrEF, HFmrEF, and HFpEF.ACE inhibitors (ACEi) or angiotensin receptor blockers (ARBs) (Class IA) are the primary therapies for chronic heart failure. ACE inhibitors inhibit angiotensin-converting enzyme (ACE), preventing the formation of angiotensin II, leading to natriuresis, diuresis, and a reduction in arterial blood pressure, subsequently reducing afterload. Numerous clinical trials demonstrated survival benefits and reduced hospitalization in chronic symptomatic heart failure with reduced ejection fraction (HFrEF). While ARBs may be considered in patients intolerant to ACE inhibitors. ARB is beneficial in HFmrEF and HFpEF. However, there is no substantial evidence to support ACEI benefit in HFpEF. The routine combination of ACEi and ARB is not recommended.Sodium-glucose cotransporter 2 inhibitors (SGLT2i) (Class 1A recommendation for HFrEF, Class 2A recommendation for HFpEF and HFmrEF) have shown positive results in reducing HF hospitalization and mortality. Contraindications include symptomatic hypotension, SBP