Medical Management of Acute Heart Failure

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Acute Heart Failure

Definition

Rapid or gradual change in signs and symptoms in patients with chronic heart failure

or

New-onset heart failure that requires urgent therapy

Relevance

- **Heart failure is one of the most important health problems of our population**

- **It is the only cardiovascular disorder whose prevalence, incidence and mortality is steadily rising in spite of extraordinary advances in the diagnosis and management of other forms of cardiac disease**

Prevalence

- Heart failure afflicts over 5 million Americans and 15 million Europeans
- Cost in the U.S. is over 34 billion dollars per year, mainly related to hospitalizations
- Over 1 million hospitalizations with a primary diagnosis of HF occur each year in the U.S.
- That trend will likely continue due to an aging population, improved survival after MI and better prevention of sudden cardiac death
- AHF resulting in hospitalization is the most common and most expensive diagnosis-related group for Medicare patients

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Patient Characteristics

- Most patients have worsening chronic HF resulting in hospitalization, with the remaining 15% to 20% diagnosed with HF for the first time
- The mean age is 75 years and over one-half are women
- History of CAD is present in 60%, hypertension in 70%, diabetes in 40%, atrial fibrillation in 30%, and moderate to severe renal impairment in 20% to 30%
- Approximately 50% of patients have a relatively preserved systolic function, are older and more likely to be female
- Post discharge mortality and re-hospitalization rates reach 10 to 20% and 20 to 30%, respectively within 3-6 months

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Clinical Background

- **Onset of acute HF may occur in minutes, hours or few days with symptoms related to pulmonary congestion, low cardiac output or both**

- **Presenting symptoms related to pulmonary congestion prominently include: shortness of breath, particularly in the supine position, with minimal effort or at rest**

- **Symptoms associated to an acute decrease in cardiac output include: loss of alertness, confusion, lethargy, nausea, vomiting or lack of appetite**

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Clinical Signs

- **Most frequent clinical signs include:**
  - **Resting tachycardia**
  - **Tachypnea**
  - **Pulmonary rales**
  - **S3 gallop**
  - **Hemodynamic instability with rapid changes in vital signs.**

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Common Factors That Precipitate Hospitalization

- Noncompliance with medical regimen, sodium and/or fluid restriction
- Acute myocardial ischemia
- Uncontrolled high blood pressure
- Atrial fibrillation and other arrhythmias
- Recent addition of negative inotropic drugs (e.g., verapamil, nifedipine, diltiazem, beta blockers)
- Pulmonary embolism
- Nonsteroidal anti-inflammatory drugs
- Excessive alcohol or illicit drug use
- Endocrine abnormalities (e.g., diabetes mellitus, hyperthyroidism, hypothyroidism)

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Precipitants for Admission

- Hospitalization commonly results from congestion or fluid overload and not due to a low cardiac output
- Hospitalization for HF, in itself, is one of the most important predictors for rehospitalization
- Both in the U.S. and Europe, uncontrolled hypertension, ischemia, arrhythmias, exacerbation of chronic obstructive pulmonary disease with or without pneumonia, and noncompliance (dietary and/or medication) are major precipitants for admission
- In patients presenting with de novo HF, a significant number are diagnosed with an acute coronary syndrome

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Clinical Course

- Most patients have rapid symptomatic improvement with loop diuretics and have a relatively short hospital stay (mean 6 days; median 4 days in the U.S.)
- A comprehensive assessment is not often performed (e.g., cardiac catheterization, assessment for viable, but dysfunctional myocardium); this may result in underutilization of evidence-based therapies
- In patients admitted with worsening chronic HF, except for diuretic dose escalation, introduction of new or up-titration of evidence-based therapies (e.g., ACE inhibitors, beta-blockers) is <5% to 10%
- In fact, patients are often discharged on the same pre-admission medications
- In-hospital mortality (2% to 4%) may reach to 20% in patients with severe renal impairment and low systolic blood pressure, a group that represents <2% to 5% of the overall AHF population

Differential Diagnosis

- HF resembles and should be distinguished from:
  - Conditions causing circulatory congestion due to sodium and water retention but with no disturbance of cardiac structure or function (e.g., renal failure)
  - Noncardiac causes of pulmonary edema (e.g., acute respiratory distress syndrome)
  - Obstructive airway disease
  - Noncardiac causes of sudden depression of cardiac output, such as hypovolemic shock or cardiac tamponade
  - Pulmonary embolism
  - Diffuse parenchymal lung disease
  - Pulmonary vascular occlusive disease
  - Diseases of chest wall and respiratory muscles

Diagnostic Approach

- A complete history and physical examination are the first steps in evaluating the structural abnormality or cause responsible for the development of HF
- Direct inquiry may reveal prior or current evidence of MI, valvular disease, or congenital heart disease
- Examination of the heart may suggest the presence of cardiac enlargement, murmurs, or a third heart sound
- Although the history and physical examination may provide important clues about the nature of the underlying cardiac abnormality, identification of the structural abnormality leading to HF generally requires invasive or noninvasive imaging of the cardiac chambers

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Non Invasive Imaging

- A comprehensive 2D-echocardiogram with Doppler flow studies is the single most useful diagnostic test in the evaluation of patients with heart failure

- Useful to determine whether abnormalities of myocardium, heart valves, or pericardium are present and which chambers are involved

- Three fundamental questions must be addressed:
  - If the LV ejection fraction (EF) preserved or reduced
  - Is the structure of the LV normal or abnormal
  - Are there other structural abnormalities such as valvular, pericardial, or right ventricular abnormalities that could account for the clinical presentation

- That information should be quantified with a numerical estimate of EF, measurement of ventricular dimensions and/or volumes, measurement of wall thickness, and evaluation of chamber geometry and regional wall motion
  - Most patients with symptoms and signs of HF have a LV ejection fraction less than 40%
  - The Doppler examination provides unique assessment of valvular function and permits detection of intracardiac shunts and estimation of the pulmonary artery pressure

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Two-dimensional and Doppler echo (continued)

- Doppler investigation of the diastolic mitral flow pattern is particularly useful in assessing the presence of LV diastolic dysfunction

  - Up to 50% of patients echocardiographically examined for CHF are found to have normal LV systolic function
  - A common example of LV diastolic dysfunction is seen in elderly hypertensives with LVH and a clinical picture suggestive of CHF, but whose LV systolic function is normal
  - Establishing a diagnosis of diastolic heart failure in such patients is crucial, as they may adequately respond to diuretics but could seriously deteriorate if exposed to vasodilator or positive inotropic therapy

Natriuretic peptides (BNP and NT pro BNP) assays

- BNP and N-terminal pro-BNP are hormones produced primarily by the ventricular myocardium and whose release is stimulated by volume expansion and myocardial wall stress
- Are relatively sensitive markers for the presence of HF with depressed ejection fraction
- Are also elevated in HF patients with a preserved EF, but to a lesser degree
- Their levels increase with age, renal impairment; are higher in women and can rise in right HF from any cause; levels can be falsely low in obese patients
- Levels may normalize in some patients following appropriate treatment
- Normal concentration in untreated patients is useful for excluding the diagnosis of HF
- Interpretation of BNP levels in patients that present with dyspnea helps in differentiating between cardiac and pulmonary causes:
    - <100 pg/mL very unlikely associated to HF
    - 100 to 500 pg/mL possibly related to HF
    - > 500 pg/mL supports a diagnosis of heart failure
        - » Most patients with acute HF and dyspnea have levels > 400 pg/mL
- Helpful in diagnosis and prognosis; as BNP has a short half-life, it is also useful in monitoring therapy

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Basic Laboratory Data

12-lead ECG: Screening for acute myocardial infarction, ventricular hypertrophy, arrhythmias or conduction defects; ventricular dyssynchrony (wide QRS)

Chest film: For assessment of heart size, pulmonary congestion, pneumonia or pleural effusion

Laboratories: CBC, urinalysis, serum electrolytes (including calcium and magnesium), blood glucose, blood lipids, renal and hepatic function

Cardiac markers: If suspect myocardial ischemia or infarction

Arterial blood gases: In patients with severe respiratory distress or who experience respiratory failure or an abrupt change in mental status

Assessment of body weight and urine output

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Beware

- **Treatment should precede or occur simultaneously with the initial diagnostic evaluation in patients who present with:**
  - **Acute pulmonary edema**
  - **Cardiogenic shock**

- **A more conventional diagnostic approach could be followed in less urgent situations**

Hemodynamic In -Hospital Measurements by Balloon Flotation (Swan–Ganz) Catheter

- Pulmonary artery or pulmonary wedge pressure monitoring may be useful in critically ill patients
  - Not advised in other patients
  - Two primary hemodynamic determinants of acute HF:
    - Elevated LV filling pressures
    - Depressed cardiac output
    - Both frequently accompanied by an increase in systemic vascular resistance (SVR)
- Arterial pressure monitoring is useful in patients receiving vasodilators.

General Principles of Medical Management

- Adequate management of acute HF requires a prompt and accurate diagnosis with identification of:
  - Underlying cause
  - Involved pathophysiologic mechanisms
  - Precipitating factors
  - Exclusion of other potential causes of acute dyspnea or congestion based on the available basic clinical data plus standard laboratory tests and procedures

- Availability of BNP levels greatly improves the diagnostic accuracy

Aim of Medical Therapy

- The principal aim of therapy is to establish a prompt and effective therapeutic regime that leads to the relief of symptoms, the control of fluid overload, an increase in cardiac output and a decrease in vascular resistance
- That regime should lead to a short hospital stay and the expeditious initiation or re-adjustment of an oral therapeutic program that will slow disease progression and promote patient's long-term survival
- In general, patients with antecedent chronic CHF may be maintained in their prior drug therapy regarding ACE inhibitors or ACE receptor antagonists (ARBs), aldosterone antagonists or beta-blockers
- However, beta-blockers prescribed less than 2 weeks prior to decompensation may require discontinuation or dose reduction
- A 2-gram sodium diet and 1500 mL fluid restriction is generally indicated

Approach to Treatment

- Early phase of treatment typically takes place in the Emergency Room, where 80% of hospitalized patients present
- Evaluation and management often proceeds concomitantly
- After stabilization/treatment of life-threatening conditions, the key goals are improving the hemodynamics and the congestive symptoms
- It is recommended that the following common potential precipitating factors for acute HF be identified, as their recognition is critical to guide therapy:
  - Acute coronary syndromes/coronary ischemia
  - Severe hypertension
  - Atrial and ventricular arrhythmias
  - Infections
  - Pulmonary emboli
  - Renal failure
  - Medical or dietary noncompliance

Medical Therapy

Respiratory care

- Supplemental oxygen by nasal cannula or face mask is frequently warranted, with an aim to maintain the arterial oxygen saturation above 92%

- Endotracheal intubation and mechanical ventilation could be required when oxygenation fails, or in cases of hypercapnia or extremely altered mental status

- In those situations, the ventilator should be adjusted to maximize oxygenation and to minimize hypercapnia

General Plan for Pharmacologic Therapy

- Broadly speaking, patients with congestion but adequate perfusion are suitable candidates to receive intravenous diuretics, particularly if they have a satisfactory renal function

- Positive inotropic drugs should be employed in patients with signs of marked hypoperfusion or shock

- Vasodilator agents could be beneficial in both situations

- However, the most appropriate therapeutic strategy for management of the majority of patients hospitalized with acute CHF, those with congestion and reduced perfusion but not in shock, has not been fully defined

Monitoring of Therapy

- Effect of HF treatment should be monitored with careful measurement of
  - Fluid intake and output
  - Vital signs
  - Body weight, determined at the same time each day
- Clinical signs (supine and standing) and symptoms of systemic perfusion and congestion
- Daily serum electrolytes, urea nitrogen, and creatinine concentrations should be measured during the use of IV diuretics or active titration of HF medications

Medical Therapy

Diuretics

- Prompt intravenous administration of diuretics, such as furosemide, bumetanide or torsemide, is indicated in all patients with acute HF, with pulmonary congestion, but whose systemic perfusion is adequate
- Therapy should begin in the ER or outpatient clinic without delay, as early intervention is associated with better outcomes
- If patients are already receiving loop diuretic therapy, the recommended starting dose is at double the daily oral dose with reassessment every two to four hours
- Urine output and signs and symptoms of congestion should be serially assessed, and diuretic dose should be titrated accordingly to relieve symptoms and to reduce extracellular fluid volume excess
- Monitoring of daily weight, supine and standing vital signs, fluid input, and output is a necessary part of daily management
- Assessment of daily electrolytes and renal function should be done while intravenous diuretics or active HF medication titration is being undertaken

Medical Therapy

Diuretics

- When a patient with congestion fails to respond to initial doses of intravenous diuretics, several options may be considered:
  - Increase the dose of the loop diuretic to ensure that adequate drug levels reach the kidney
  - Add a second type of diuretic, typically a thiazide (metolazone or intravenous chlorothiazide) or spironolactone, to improve diuretic responsiveness
  - Consider continuous infusion of the loop diuretic
    - By continuous delivery of the diuretic to the nephron, rebound resorption occurring during the time blood levels of diuretic are low is avoided and ototoxicity risk may actually be reduced
  - If all diuretic strategies are unsuccessful, ultrafiltration or another renal replacement strategy may be reasonable
    - Ultrafiltration moves water and small to medium-weight solutes across a semipermeable membrane to reduce volume overload

General Principles of Medical Management

Diuretics

- Their use requires monitoring of the acid-base status and serum electrolytes, as acidosis and alkalosis may further depress myocardial function, and a low serum potassium or magnesium may give rise to serious complications

- Moreover, excessive reduction of the pulmonary capillary wedge pressure may cause postural hypotension, a decrease in stroke volume, an increase in systemic vascular resistance and the activation of the renin-angiotensin aldosterone system with subsequent sympathetic activation and increased water and sodium retention

- Use of diuretics as sole therapeutic agents has been reported to diminish the glomerular filtration rate with further compromise of renal function

- Although the use of diuretics may result in the effective relief of symptoms, their impact on mortality has not been well studied

Diuretics

- Loop diuretics
  - The intravenous form is usually preferred, especially when significant pulmonary edema is present
  - Specific agents
    - Furosemide
      - IV dosage: initial, 20 mg (maximum, 400 mg/d)
      - PO dosage: initial, 20–40 mg 1–2 times daily (maximum, 400 mg/d)
    - Bumetanide
      - IV dosage: initial, 0.5 mg (maximum, 2 mg/d)
      - PO dosage: initial, 0.5–1.0 mg 1–2 times daily (maximum, 10 mg/d)
    - Torsemide
      - IV dosage: initial, 5 mg (maximum, 20 mg/d)
      - PO dosage: initial, 10 mg 1–2 times daily (maximum, 200 mg/d)
  - Side effects
    - Metabolic alkalosis
    - Hypokalemia
    - Hyperuricemia
    - Hyperglycemia
    - Weakness
    - Nausea
    - Dizziness
- Ultrafiltration
  - Is reasonable for patients with refractory congestion who fail to respond to diuretics, especially those with underlying renal insufficiency to remove excess fluid
  - No evidence yet that ultrafiltration offers any benefit over pharmacologic diuresis in patients who respond to drug therapy and its safety data are still lacking

Vasodilators

- The addition of vasodilators such as nitroprusside, nitroglycerin, or nesiritide to the HF regimen of the hospitalized patient might be appropriate in patients with adequate blood pressure and ongoing congestion not sufficiently responsive to diuretics and standard oral therapy

- Regardless of the agent used, the clinician should make certain that intravascular volume is, in fact, expanded and that the patient's blood pressure can tolerate the addition of the vasodilating drug

- Intravenous nitroglycerin, primarily through its venodilation effects, lowers preload and may help to more rapidly reduce pulmonary congestion

- Patients with HF and hypertension, coronary ischemia, or significant mitral regurgitation are often cited as ideal candidates for the use of intravenous nitroglycerin

- However, tachyphylaxis to nitroglycerin may develop rather quickly and up to 20% of those with HF may develop resistance to even high doses

Vasodilators

- Sodium nitroprusside is a balanced preload-reducing venodilator and afterload-reducing arteriodilator that also dilates the pulmonary vasculature
  - Data demonstrating efficacy are limited, and invasive hemodynamic blood pressure monitoring is typically required
- Nitroprusside has the potential for producing marked hypotension and is usually used in the intensive care setting; longer infusions of the drug have been associated with thiocyanate toxicity, particularly in the setting of renal insufficiency
- Nitroprusside is potentially of value in severely congested patients with hypertension or severe mitral valve regurgitation complicating LV dysfunction
- Nesiritide (human BNP) reduces LV filling pressure but has variable effects on cardiac output, urinary output, and sodium excretion
  - The severity of dyspnea is reduced more rapidly compared to diuretics alone
- Because nesiritide has a longer effective half-life than nitroglycerin or nitroprusside, side effects such as hypotension may persist longer
  - Conservative dosing of the drug (i.e., no bolus) and use of only the recommended doses may reduce complications.

Vasodilators

- Require monitoring of arterial pressure
- Nitroglycerin IV
  - Dosing: Start 20 µg/min and increase in 20 µg increments until patient symptoms are improved or pulmonary capillary wedge pressure (PCWP) is decreased to 16 mmHg without reducing systolic blood pressure below 80 mmHg
    - Maximum doses: 40–400 µg/min
    - Most common side effect: headache, which often resolves during continued therapy
    - Long-term (>48 h) use is associated with hemodynamic tolerance
- Nitroprusside IV
  - Dosing: Initiate at 10 µg/min and increase by 10–20 µg every 10–20 min as tolerated until PCWP is decreased to 16 mmHg without reducing systolic blood pressure below 80 mmHg
    - Maximum doses: 30–350 µg/min
    - Major limitation: side effects from cyanide (predominantly as GI and central nervous system manifestations)
    - Suspected cyanide toxicity is treated by decreasing or discontinuing the nitroprusside infusion
    - Long-term (>48 h) use is associated with hemodynamic tolerance

Vasodilators (continued)

- Nesiritide IV
  - Recombinant form of BNP
  - Dosing: bolus (2 µg/kg) followed by a fixed-dose infusion (0.01–0.03 µg/kg per min)
  - Max: 0.01–0.03 µg/kg per minute
  - Side effects:
    - Headache is less common than with nitroglycerin
  - Nesiritide was approved for the treatment of AHF in the U.S. in 2001; retrospective data raised the hypothesis that it may worsen renal function and increase post-discharge mortality
  - Safety and efficacy of nesiritide is being tested in a large international trial (ASCEND-HF [Double-Blind, Placebo-Controlled, Multicenter Acute Study of Clinical Effectiveness of Nesiritide in Subjects With Decompensated Heart Failure] trial)

Vasodilators

Side effects

- Hypotension is the most common side effect of all 3 vasodilating agents
  - Less so with nesiritide
  - Their use is frequently associated with bradycardia, particularly nitroglycerin
  - Pulmonary artery vasodilation
    - May lead to worsening hypoxia in patients with underlying ventilation -perfusion abnormalities

Intravenous Inotropic Drugs

- Use of intravenous inotropes such as dopamine, dobutamine, and milrinone may be considered in patients with either predominantly low output syndrome (e.g., symptomatic hypotension) or combined congestion and low output
- These agents may help relieve symptoms due to poor perfusion and preserve end-organ function in those with severe systolic dysfunction and dilated cardiomyopathy
- Their greatest value is in patients with relative hypotension and intolerance or no response to vasodilators and diuretics
- Clinicians should be cautioned that the use of these drugs portends a very poor prognosis and that a thorough hemodynamic assessment must be undertaken to ensure that the low output syndrome is responsible for the presenting clinical signs and symptoms
- Likewise, clinicians should not use a specific blood pressure value that might or might not mean hypotension, to dictate the use of inotropic agents
- Rather, a depressed blood pressure associated with signs of poor cardiac output or hypoperfusion (e.g., cold clammy skin, cool extremities, decreased urine output, altered mentation) should prompt a consideration for more aggressive intravenous therapy

Intravenous Inotropic Drugs

- Dobutamine requires the beta-receptor for its inotropic effects, while milrinone does not; this may be a significant consideration for patients already maintained on beta-blocking drugs

- Furthermore, milrinone has vasodilating properties for both the systemic circulation and the pulmonary circulation

- Although these agents are widely utilized in the treatment of acute HF, their definite indication is in patients who present signs of hypoperfusion or cardiogenic shock

- Recent studies have discouraged their routine utilization in patients with either acute or chronic heart failure due high mortality, increased risk of hospitalization, aggravation or induction of arrhythmias and ischemic events

- Their primary direct hemodynamic effects are to increase cardiac index, decrease systemic vascular resistance and indirectly decrease ventricular filling pressures

Inotropic Agents

- Dobutamine
  - Most commonly used combination of inotropic and vasodilator agent for the treatment of acute HF
  - Dosing: Continuous infusion, at an initial infusion rate of 1–2 µg/kg per min
  - Higher doses (>5 µg/kg per min) are frequently necessary for severe hypoperfusion
  - Little added benefit of increasing the dose above 10 µg/kg per min
  - Patients maintained on chronic infusions for >72 h generally develop tachyphylaxis and require increasing doses

- Milrinone
  - Dosing: bolus dose of 0.5 µg/kg per min, followed by a continuous infusion rate of 0.1–0.75 µg/kg per min
  - More effective vasodilator than dobutamine
  - Produces a greater reduction in LV filling pressures
  - Greater risk of hypotension

- Dopamine
  - Dosing: initial 1–2 µg/kg per minute; maximum 2–4 µg/kg per minute

Vasoconstrictors

- These agents are used to support the systemic blood pressure in patients with acute heart failure:
  - Dopamine for hypotension
    - Generally the first choice for therapy in situations where modest inotropy and pressor support are required
    - Dosing: initial 5 µg/kg per minute; maximum 5–15 µg/kg per minute
  - Epinephrine
    - Dosing: initial 0.5 µg/kg per minute; maximum 50 µg/kg per minute
  - Phenylephrine
    - Dosing: initial 0.3 µg/kg per minute; maximum 3 µg/kg per minute
  - Vasopressin
    - Dosing: initial 0.05 units/minute; maximum 0.1–0.4 units per minute

Vasoconstrictors

Caution

- Their prolonged use may lead to renal and hepatic failure and gangrene of the limbs

- Should be reserved for true emergency situations

Acute HF and Suspected Acute MI

- When patients present with acute HF and a known or suspected acute myocardial infarction due to occlusive coronary disease, especially when there are signs and symptoms of inadequate systemic perfusion, urgent cardiac catheterization and revascularization is reasonable, if likely to prolong meaningful survival

Invasive Hemodynamic Monitoring

- Invasive hemodynamic monitoring should be performed to guide therapy in patients with respiratory distress or with clinical evidence of impaired perfusion in whom the adequacy or excess of intracardiac filling pressures cannot be determined from clinical assessment

- Invasive hemodynamic monitoring can also be useful for carefully selected patients with acute HF who have persistent symptoms despite empiric adjustment of standard therapies, and:

  - Whose fluid status, perfusion, or SVR or PVR are uncertain
  - Whose systolic pressure remains low, or is associated with symptoms, despite initial therapy
  - Whose renal function is worsening with therapy
  - Who require parenteral vasoactive agents
  - Who may need consideration for advanced device therapy or transplantation

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Mechanical and Surgical Interventions

- If pharmacologic interventions fail to stabilize patient with refractory HF, mechanical and surgical interventions may provide effective circulatory support
  - Intraaortic balloon counterpulsation
  - LV assist device
  - Cardiac transplantation

General Principles of Medical Management
Mechanical Assistance

Intraaortic Balloon Counterpulsation

- Intraaortic balloon pump (IABP) insertion is to be considered in some patients when signs and symptoms of low cardiac output persist in the presence of high filling pressures and threaten organ dysfunction, such as renal or liver failure

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Mechanical Assistance

Short or Long-term Mechanical Support

- A line of last resort in therapy could be the necessity to install short or long-term mechanical support with a ventricular assist device

- This measure might eventually be necessary when all of the above measures fail and before irreversible organ dysfunction occurs, as a bridge to recovery or cardiac transplantation

Transition to Oral Therapy

- In all patients hospitalized with HF, both with preserved and low EF, transition should be made from IV to PO diuretic therapy with careful attention to PO diuretic dosing and monitoring of electrolytes

- With all medication changes, the patient should be monitored for supine and upright hypotension, worsening renal function and HF signs/symptoms

Initiation of Beta Blockers

- Initiation of beta blockers is recommended after optimization of volume status and successful discontinuation of IV diuretics, vasodilators and inotropic agents

- Beta blockers should be initiated at a low dose and only in stable patients

- Particular caution should be used when initiating beta blockers in patients who have required inotropes during their hospital course

General Principles of Medical Management

Disposition

- After stabilization, patients are to be kept in an oral therapeutic regime in agreement with their particular clinical characteristics and any accompanying co-morbid conditions, so as to assure the full resolution of the symptoms prior to hospital discharge

- Medications should be reconciled in every patient and adjusted as appropriate on admission and upon discharge from the hospital

- It is recommended that treatment with oral therapies known to improve outcomes (ACE inhibitors or ARBs and beta-blockers) be continued in most patients with reduced ejection fraction who experience an exacerbation of HF requiring hospitalization, in the absence of hemodynamic instability or contraindications

- Initiation of oral therapies with ACE inhibitors or ARBs and beta blockers is recommended in stable patients prior to hospital discharge, that were not receiving such therapies prior to their admission

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Disposition

- Comprehensive written discharge instructions for all patients with a hospitalization for HF and their caregivers is strongly recommended, with special emphasis on the following six aspects of care:
  - Diet
  - Discharge medications (with a special focus on adherence, persistence, and up titration to recommended doses of ACE inhibitor/ARB and beta blockers)
  - Activity level
  - Follow-up appointments
  - Daily weight monitoring
  - What to do if HF symptoms worsen

Conclusions

- Hospitalization for AHF represents a significant and growing health care burden
- Patients are characterized by their heterogeneity in terms of mode of presentation, pathophysiology, and prognosis
- Most patients symptomatically improve during hospitalization; however, their early post-discharge rehospitalization and mortality rates continue to be extremely high
- Worsening signs and symptoms and neurohormonal and renal abnormalities occurring soon after discharge may contribute to these high post-discharge event rates
- Currently available assessment modalities combined with recent advances in cardiovascular therapies provide present-day opportunities to improve post-discharge outcomes
- Further investigation into pathophysiologic targets and novel approaches to clinical trial design are needed
- Improving post-discharge outcomes is the most important goal in the management of AHF patients