ORIGINAL RESEARCH PAPER

Cardiology

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DIAGNOSIS AND MANAGEMENT OF ACUTE HEART FAILURE

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ABSTRACT

Acute Heart Failure is a common clinical syndrome which is responsible for a significant number of hospital admissions. It requires early diagnosis and prompt and effective treatment so that morbidity and mortality associated with it can be reduced. Clinical features include tachycardia, tachypnea, dyspnea, hypoxia, chest congestion etc. Mainstay of treatment are diuretics and vasodilators.

Acute heart failure is among the common causes for admission in hospitals worldwide (1,2). AHF can be defined as the new onset or recurrence of symptoms and signs of heart failure requiring urgent or emergent therapy leading to hospitalization.

Epidemiology:
The overall number of admissions for AHF continue to increase as a consequence of the aging of the population, improved survival after acute myocardial infarction and effective prevention of sudden cardiac death. AHF is more likely in older age group (over 70 years).

Upto 50% of patients may have normal LV ejection fraction (HFpEF). Hypertension, coronary artery disease, and dyslipidemia are most common associated comorbidities (3,4). Other conditions such as diabetes mellitus, COPD, chronic renal insufficiency, peripheral vascular disease, obesity, atrial fibrillation are also frequent.

Pathophysiology:
Impairment of left ventricular function (systolic, diastolic, or both) is found in almost all such patients. Various extracardiac factors may be important in precipitating AHF. Stimulation of sympathetic nervous system and RAAS (renin angiotensin aldosterone system) leads to vasoconstriction, salt and water retention, and increase in diastolic filling pressures and clinical symptoms. Presence of hypertension, tachycardia, and myocardial ischemia further exaggerates the heart failure (5).

Evaluation:
On admission, patients' condition is evaluated in order to:
1. Establish a definitive diagnosis of AHF.
2. Emergent treatment of life-threatening shock or respiratory failure.
3. Identify and treat any clinical triggers.
4. Risk stratification and rapid implementation of appropriate therapy.(6)

The patient may be classified into any of the three syndromes:
1. Decompensated heart failure - Group of patients with worsening signs and symptoms in background of chronic heart failure. This represents largest group of patients.
2. Acute hypertensive heart failure - hypertension is a key trigger in these patients due to anxiety, dyspnea leading to sudden worsening. Frank pulmonary edema is more common in these patients.
3. Cardiogenic shock. This is relatively uncommon. Patients present with features of organ hypoperfusion.

The common symptoms of AHF include dyspnea, leg discomfort, abdominal discomfort, fatigue, sleep disturbance, or depression.

Physical Examination:
AHF is clinical diagnosis. Volume overload may manifest as chest rales, pleural effusion, blood pressure is usually high and presence of hypotension is a predictor of poor outcome. Elevated jugular venous pressure, presence of LVS3, edema, ascites, hepatomegaly, all are common. In severe cases, extremities may be cool with skin discoloration.

Bio Markers:
The natriuretic peptides BNP (brain natriuretic peptide) and NT - pro BNP (N - terminal pro brain natriuretic peptide) are well established as a class I indication for diagnosis.

Management:
Hospitalization is required for most patients with AHF. They need emergent therapy which should start in emergency/outpatient setting.

1. Oxygen administration in presence of hypoxemia with use of non-invasive ventilator is required for patients with cardiogenic pulmonary edema. Early administration may prevent deterioration and need for intubation and mechanical ventilation later.
2. Intravenous loop diuretics (furosemide, torsemide, bumetanide) are the main stay of treatment (7) especially in those with features of systemic venous congestion or edema.
3. IV vasodilators such as nitroglycerin or nitroprusside are required in most patients with high BP. Nesiritide is recombinant human B-type natriuretic peptide and is identical to endogenous BNP. It causes vasodilation in venous and arterial vasculature resulting in significant drop in ventricular filling pressures and increase in cardiac output.
4. Specific precipitating factors such as sudden onset atrial fibrillation, acute coronary syndrome, right ventricular heart failure must be looked for and treated accordingly.
5. Comorbid conditions which are present should be managed based on their assessment.
6. Progressively worsening renal dysfunction (cardiorenal syndrome) may lead to refractory heart failure due to poor response to loop diuretics. This necessitates increase in dose or use of combination of diuretics.
7. Close hemodynamic monitoring of volume status is essential so that therapy is not inadequate.
8. Hypotension (SBP below 90 mmHg) is a bad prognostic sign, necessitating the use of intravenous inotropes. Among the most frequently used are Dopamine, Dobutamine, Epinephrine and inodilators such as milrinone. Other vasopressors such as phenylephrine and norepinephrine are potent alpha1 agonists and may induce marked vasoconstriction which may lead to end-organ hypoperfusion and tissue necrosis.

Recent advances
Although many trials of newer agents have been carried out none have shown potential benefit. Vasodilators such as relaxin, natriuretic peptides, neurohumoral antagonists and inotropic agents are being developed.

Conclusion
Acute heart failure is one of the most difficult emergencies to manage and prevent over the long term. Management includes treating the manifestations of the syndrome rather than the

pathophysiology. Better understanding of the central derangements and better targeting of the treatment will improve the prognosis. Newer therapies are being developed which may further improve the management of these patients.

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