Review
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Management of Congestive Heart Failure and Pulmonary Edema

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Abstract

Congestive heart failure (CHF) is one of the most important condition received by emergency physcians. Current review describes the management of CHF and pulmonary edema based on the latest updates, with a priority in the 2016 guidelines of the European Society of Cardiology (ESC). In the latest guidelines, they were more algorithmic approach than previous ones. The importance of step treatment was emphasized based on these algorithms. It was seen that treatment and management were done with prognostic classifications especially Nohria-Stevenson classification.

Key words: Congestive Heart Failure, Pulmonary Edema

Introduction

Heart failure (HF) is defined as the condition of the heart muscle that does not pump blood to vital organs at a sufficient level[1]. The term of congestive HF is given to patients with breathlessness and edema whereby the heart fails to pump effectively[2]. The most recent data on the prevalence of HF in our country was obtained from the HAPPY study. According to this study, the prevalence of HF in our country is higher than in western countries, and the estimated prevalence is 6.9%[3].

The latest guideline of the European Society of Cardiology (ESC) for the management of HF was published in 2016. One of the most important changes in previous guidelines was related to HF classification. In this classification made according to left ventricular ejection fraction (LVEF). LVEF, below 40%; HF with low ejection fraction, patients with a LVEF in the range of 40–49%; heart failure with mid-range ejection fraction, LVEF ≥50%; HF with preserved EF[4, 5]. The Nohria-Stevenson classification, one of the prognostic classifications, was one of the highlights of the guideline. In this classification, the presence of congestion (dry-wet) condition of patients with HF and the presence of hypoperfusion (cold-warm) at rest are evaluated (Table 1). Killip classification is one of the prognostic classification used for acute coronary syndrome patients. The presence of pulmonary edema in this classification is considered to be Killip class III (Table 2). The New York Heart Association classification is the most well-known and widely used classification of CHF (Table 3)[6].

Epidemiology

The precipitants of acute HF are examined under two headings: cardiac and non-cardiac. Coronary ischemic diseases and hypertension are the most common causes of acute HF for in cardiac precipitants.

Cardiac precipitants;

- Heart valve diseases
- Cardiomyopathy
- Myocarditis
- Heart rhythm disorders

Combination of negative inotropic agents (verapamil, beta-blockers, diltizem, etc.)

- Non-compliance with treatment

Non-cardiac precipitants;

- Endocrinological diseases (diabetes, thyroid disorders)
- Pulmonary diseases (pulmonary embolism, asthma, COPD)
- Increased blood volume (anemia)
- Conditions leading to sodium retention such as renal failure, medication and addiction (steroids, excessive alcohol intake, illicit substance abuse)
- Other (cerebrovascular accident, surgery)

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Table 1. Classification of Nohria-Stevenson.

(-)		Presence of congestion during rest	
		(+)	
Presence of hypoperfusion at rest	(-)	warm and dry	warm and wet*
	(+)	cold and dry **	cold and wet

* Pulmonary congestion, orthopnea / paroxysmal nocturnal dyspnoea, peripheral (bilateral) edema, jugular venous dilatation, congestive hepatomegaly, acid, hepatojugular reflux
** Cold sweaty extremities, oliguria, mental confusion, dizines, narrowed pulse pressure

Table 2. Classification of Killip.

Class	Physical Examination Findings
I	No evidence of heart failure
II	S3 (+), ral, tachycardia in <50% of lung areas
III	S3 (+), > 50% of lung areas
IV	Cardiogenic shock

Pathophysiology

It is important to understand the working principle of the heart in order to understand the treatment modalities of HF. The cardiac output is the amount of blood that the heart pumps in one minute, which determines heart rate, contractility, preload and afterload. As the heart rate increases, cardiac output increases at the same rate. Similarly, as the contractile strength of the heart increases, the cardiac output increases with that rate[7, 8].

Unlike other parameters that affect HF, there is a confusion about the preload and afterload. Preload refers to ventricular wall tension (or wall stress) that occurs during the diastole period of the heart[8]. The Frank-Starling law is often used in the preliminary explanation. According to this law, the ventricle volume increases with the blood supply to the ventricle, which affects the tension of the ventricular muscle fibers, which allows the heart to pump blood more strongly. As the amount of air in the balloon increases, the tension of the balloon increases. When the balloon tension is maximized the balloon's mouth is opened, the balloon starts to discharge air more strongly. As seen in the Frank-Starling law and balloon sample, the most important factor affecting the preload is the increased wall tension as a result of the amount of blood flowing into the ventricle. Because of Frank-Starling law does not function correctly in HF, these patients should be given precautionary measures to reduce preload[9].

Afterload is the force that the ventricles should be able to pump blood to the body, which depends more on arterial blood pressure and vascular tone. An example of a bike ride uphill is given for the afterload. According to this example, a driver riding an uphill bike has more energy than a straight road. In this case, the slope of the hill should be reduced or the number of contractility and pedals should be increased. Hypertension is the most common cause of afterload increase and it is necessary to reduce blood pressure (reduce slope)[10, 11].

Clinical Features

Shortness of breath is the most common symptom of CHF. In the early period of CHF, shortness of breath during physical activity starts to develop during resting periods. In the more critical stages of CHF, there are more serious symptoms such as breathingonly in the sitting position (orthopnea) and waking up with air starvation (paroxysmal nocturnal dyspnoea, PND). Patients with chest pain, palpitation, cough, malaise, nausea, and mental status, as well as HF-specific symptoms such as orthopnea and PND, are also referred to the emergency department. Other non-specific symptoms include swelling of the abdomen (acid), scrotum and other parts of the body due to the decrease of venous circulation. While shortness of breath develops in the left HF, other symptoms (swelling, pain in the abdomen and extremities) are more likely to develop in the right HF[12].

Table 3. Classification of New York Heart Association Congestive Heart Failure

I (Mild)	No limitation of physical activity. Ordinary physical activity does not cause chest pain, breathlessness or palpitations
II (Mild)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in chest pain, breathlessness or palpitations
III (Moderate)	Marked limitation of physical activity without discomfort. Symptoms at rest can be present. If any physical activity is undertaken, discomfort is increased.
IV (Severe)	Unable to carry on physical activity without discomfort. Symptoms at rest can be present. If any physical activity is undertaken, discomfort is increased.

Physical Examination Findings

Cardiovascular findings[13];

- Tachycardia due to sympathetic nervous system activation: It should be kept in mind that users with medications that reduce heart rate, such as beta blockers, may develop CHF without tachycardia.
- S3 hearing with auscultation
- Heart rhythm disorders: Atrial fibrillation, ventricular ectopic pulses, reduced stroke volume-induced pulse disorders (pulsus alternans; a strong weak pulse, pulsus paradoxus; systolic pressure less than 10 mmHg during inspiration)
- Abnormal values in blood pressure (patients with CHF in the emergency room mostly present with systolic pressure> 140 mmg)
- Low pulse pressure level due to stroke volume reduction (normally 30-50 mmHg)

Respiratory findings[14];

- Tachypnea
- Rales with auscultation and wheezing
- Cheyne-Stokes respiration in more severe cases (respiration characterized by periods of hyperpnea, hypopnea, apnea**)**

Other findings[15];

- Jugular vein fullness: Since there is no valve between the vena cava and the right atrium, right atrial pressure changes lead to fluctuations in the vena cava. The patient's body is increased by 45 degrees and the measurement is made.
- Hepato-jugular reflux: This is the state of the jugular vein fullness after pressure to the liver, > 4 cm in the venous fullness.
- Hepatomegaly, right upper quadrant sensitivity
- Gode-releasing edema: from the bones to the feet on the back, in the legs
- Cold, pale and damp skin due to cyanosis

Diagnostic Approach

It is vital that diagnostic studies are performed correctly and quickly in order to detect the precipitating causes of acute HF patients and to initiate the treatment as soon as possible. In addition, the patient's history, additional cardiovascular diseases, if any, and the medications used should be noted. Diagnostic tools in CHF patients are examined under four diagnostic tools: electrocardiography (ECG), lung X-ray, laboratory tests and echocardiography.

The most specific findings of lung X-ray in CHF are pulmonary venous congestion, pleural effusion, interstitial or alveolar edema and cardiomegaly. In addition, lung X-ray is used to exclude alternative diagnoses such as pneumonia. However, it should be kept in mind that lung X-ray may be completely normal in 20% of patients. Echocardiography should be planned primarily for hemodynamically unstable patients, such as cardiogenic shock, and for patients who are thought to be structural and functional cardiac disorders.

BNP or NT-proBNP should be studied for each patient suspected of acute CHF. Although BNP <100, NT-proBNP <300 pg / mL excludes acute HF, the rise of natriuretic peptides does not always confirm acute HF. It should be noted that there are many cardiac (atrial and ventricular tachyarrhythmias, pulmonary embolism, myocarditis) and non-cardiac causes (chronic obstructive pulmonary disease, advanced age, renal failure and liver diseases, severe burns, hormonal disorders) that cause the elevation of natriuretic peptides. It should also be underlined that natriuretic peptides may be low in end-stage decompensated heart failure. Cardiac troponins have been studied by many clinicians in CHF. Elevated cardiac troponins due to myocyte injury in acute coronary syndromes are known to be associated with higher prognosis and mortality in CHF[16]. Procalcitonin has been used to make antibiotic decisions in additional cases of infection (pneumonia)[17]. Other laboratory tests, such as BUN, electrolytes, are evaluated before deciding on the discharge of the CHR patient.

Treatment and Management

First of all, the patient should be monitored, vital signs (blood pressure, number of breaths, pulse oximetry) should be evaluated and ECG should be taken at regular intervals. Although urinary catheter is not necessary, urine output should be followed. The ESC 2016 guideline recommends investigating acute HF into two phases, the urgent phase of the emergency contact and the immediate phase (60-120 minutes) after initial contact. In the urgent phase, the patient should be evaluated in terms of cardiogenic shock and respiratory failure. If there is cardiogenic shock in this evaluation; the patient should be provided with circulation support (pharmacological and mechanical). In the presence of respiratory failure, oxygen, CPAP / BPAP and / or endotracheal intubation should be performed. İmmediate phase where the precipitant causes are investigated and then switched. It should be kept in mind that a multidisciplinary approach is required for patients with CHF. The ESC 2016 guideline proposes an algorithm based on the Nohria-Stevenson classification (hot and dry, hot and wet, cold and dry, cold and wet) for the early management of patients with CHF[4].

Although routine oxygen therapy is not recommended for non-hypoxemic patients in acute HF, continuous oxygen monitoring should be performed and the patient should be monitored. Performed pH and lactate form arterial blood gas. Non-invasive mechanical ventilation (CPAP, BiPAP)

should be considered in patients with SpO_2 <90% and pale number> 25 / min(ESC 2016 recommendation level 2A, level of evidence C). Endotracheal intubation should considered with Hypoxemia (PaO_2 <60 mmHg), hypercapnia ($PaCO_2$> 50 mmHg) and acidosis (pH <7.35)[18].

Patients with acute symptoms of CHD should be given 20-40 mg of the first visit to the emergency department and IV furosemide should be given as an oral dose for chronic CHF patients. As in the DOSE study, patients with high-dose diuretics (2.5 times the oral dose) recovered faster[19]. These high dose ratios, which result in greater weight changes, cause temporary disturbances in renal function, but long-term effects are limited. Furosemide may given as bolus or infusion according to the symptoms and clinical condition. Vasodilators show effect, reducing venous and arterial tone, which also reduces the preload and afterload. In patients with systolic blood pressure > 90 mmHg, vasodilator (nitroglycerin 10-20 µg / min, nitroprusside 0.3 µg/kg/min) should be started. Conversely, patients with hypotensive and hypoperfusion symptoms should be given inotropic agents (2-20 µg/kg/min dobutamine, 3-5 µg/kg/dk dopamin and in the presence of severe hypotension the dose of dopamine >5 µg/kg/dk) and vasopressors (norepinephrine dose 0.2-1.0 µg/kg/dk). However, these agents may cause myocardial ischemia and arrhythmias due to adrenergic effects, ECG monitoring is required.

Levosimendan (2016 ESC recommendation level 2B)[20] is one of the most important therapeutic agents in the recent HF guidelines. By increasing the calcium sensitivity of contractile proteins in the myocardium, the inotropic effect leads to a decrease in peripheral vascular resistance (preload and afterload) by opening ATP-dependent potassium channels in vascular smooth muscles[21]. In contrast to other inotropic agents, levosimendan has an inotropic effect without increasing myocardial oxygen consumption. This is the reason why they are preferred primarily in patients with CHD due to coronary artery disease. Levosimendan improves cardiac output by better correction of hemodynamics than dobutamine and leads to a decrease in PCWP (pulmonary capillary end pressure)[22]. Although there are conflicting results on the effects on mortality, studies on reducing mortality are more prevalent[23]. The recommended loading dose is 6-12 µg/kg/min in 10 minutes and the infusion dose is 0.05-0.2 µg/kg/min.

Among other treatment agents, digoxin is recommended in the presence of AF and rapid ventricular rate (>110 bpm) and given in boluses 0.25-0.5 mg i.v. Although it was thought that morphine reduced anxiety in CHF patients and relieved breathlessness, some studies showed that mortality increased[24]. Ultrafiltration is recommended by ESC as one of the alternative treatment options in cases where there is no response to medical agents (diuretics, etc.) (recommendation level 2B, level of evidence B). In patients with acute renal failure, this level of recommendation is 2A, and the level of evidence is C[25]. Other conditions accompanying these patients requiring ultrafiltration; severe hyperkalemia (K +> 6.5 mmol / L), severe acidemia (pH <7.2), BUN> 150 mg / dL and serum creatinine> 3.4 mg / dL.

Criteria for ICU admission for acute HF;

- The need for intubation
- Hypoperfusion symptoms
- SpO_2 <90% despite oxygen support
- The use of accessory muscles, number of breaths> 25 / min

In conclusion, it should be evaluated whether there is cardiogenic shock and / or respiratory failure in the patients with acute symptoms of CHF in the emergency department. Accordingly, circulatory and respiratory support should be given. On the other hand, by performing diagnostic studies, the presipitant causes should be determined and treated quickly.

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