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REVIEW

Acute heart failure syndrome

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Introduction

Heart failure can be defined as a clinical syndrome in which a structural or functional cardiac abnormality impairs the capacity of the ventricle to fill or eject enough blood for the requirements of the body. Acute heart failure syndrome represents a complex, heterogeneous set of clinical conditions, all with the common denominators of congestion of the lungs and dyspnoea.[1] There are multiple phenotypes of acute heart failure syndrome (AHFS), but there is no real consensus on definition. Furthermore, there is a lack of evidence-based management of these patients in the emergency room.

Clinical manifestations

In general, the clinical manifestations of heart failure depend on the rate at which the syndrome of heart failure develops to form possible different phenotypes.[2]

Clinical phenotypes

When heart failure develops gradually, there is time for the compensatory mechanisms to develop (such as neurohormonal stimulation with sympathetic and renin-angiotensin activation), and the classical syndrome of chronic persistent heart failure develops. The vast majority of cases of AHFS seen in clinical practice involve underlying chronic heart failure, followed by the development of an episode of acute decompensation, with worsening symptoms and signs, leading to presentation in the emergency room or hospitalisation with AHFS.

A normal healthy person can suddenly develop acute heart failure. This can occur with an acute myocardial infarction or with a rupture of a cardiac valve, such as in infective endocarditis, or a tachyarrhythmia with a very fast rate. All of these can precipitate acute heart failure (new-onset acute heart failure).

Transient heart failure can develop in conditions of myocardial injury, causing a type of "stunning"of the myocardium. Examples of this can be seen in acute brain injury, some forms of takotsubo syndrome or myocardial injury in acute myocarditis.

Clinical picture

The presenting profiles of AHFS are:[1,3]

- *An elevated blood pressure:* This can be a reflection of pulmonary congestion (pulmonary oedema with crepitations in the lung), as well as the lack of systemic congestion (increased jugular venous pressure, peripheral pitting oedema and a large, tender liver.) Many patients have normal ejection fraction.
- *A normal or moderately elevated blood pressure:* This develops slowly with systemic congestion. There may be very few signs of pulmonary congestion.
- *Low blood pressure (< 90 mmHg):* Many patients have renal impairment.
- *Profound depression of cardiac output:* This refers to cardiogenic shock.
- *Flash pulmonary oedema:* This is a severely elevated blood pressure with an abrupt onset.
- *Combination:* Acute coronary syndrome with acute heart failure.

History of the patient

The history may reveal well-recognised risk factors for heart failure, such as hypertension and other risk factors for atherosclerosis. A family history of heart failure (cardiomyopathy risk), the consumption of cardiac toxins, such as alcohol and recreational drugs (e.g. cocaine), and chemotherapy, are additional risk factors for heart failure (stage A heart failure). A previous cardiac event (myocardial infarction and myocarditis), or a heart murmur, implies structural abnormality which increases the risk of heart failure (stage B heart failure).

Differential diagnosis

The important differential diagnosis is adult respiratory distress syndrome (ARDS), a condition of acute lung injury with pulmonary oedema and bilateral lung infiltrates, very similar to cardiac pulmonary oedema.

Investigations

The **electrocardiogram** is helpful in diagnosing an acute myocardial ischaemic event, left ventricular hypertrophy or an arrhythmia.

A chest X-ray may show bilateral lung infiltrates (pulmonary oedema) and also cardiomegaly. Signs of congestion may be lacking in a chest radiograph in up to 15% of cases.[3]

The natriuretic peptides, **brain natriuretic peptide** and N-terminal of the prohormone **brain natriuretic peptide,** may be helpful in cases when dyspnoea cannot be ascribed with clinical certainty to heart failure, and is helpful in excluding acute heart failure, as demonstrated in the Breathing Not Properly trial.[3] They also help to diagnose heart failure in uncertain cases.

Elevated troponin levels diagnose acute coronary syndrome, and are also used as a prognostic factor in heart failure.

Blood gas analysis may show hypoxaemia, and with oxygen therapy, a ventilation or perfusion defect which responds to oxygen can assist in the diagnosis of heart failure to differentiate it from a shunt in ARDS with no or little response to oxygen therapy.

Management

The current emergency room goals of therapy are to relieve congestion (dyspnoea), restore haemodynamics, achieve euvolaemia and avoid harm (myocardial and renal injury caused by the treatment).

Oxygen therapy

Oxygen therapy must be instituted.

Intravenous diuretics

An intravenous diuretic is the mainstay of therapy as it reduces the preload of the heart and increased dieresis, which relieves congestion in the lungs. The majority of experience is with the use of furosemide (Lasix®), a loop diuretic with rapid onset of action. Diuretics can either be given as a bolus method or as a continuous intravenous infusion, with equal efficacy. Other loop diuretics can be used, such as torasemide. The loop diuretics can have detrimental effects, such as worsening renal function and increased mortality.[4] Overdiuresis should be avoided. Other more experimental treatments remove fluid when diuretics fail, and these may be important in future, such as vasopressin antagonists, adenosine antagonists and ultrafiltration.

An aggressive reduction of blood pressure is necessary in cases presenting with an elevated blood pressure and pulmonary oedema.

Intravenous inotropes

Positive inotropic drugs are typically used in patients with decompensated systolic heart failure, and showing the signs and symptoms of end-organ dysfunction due to hypoperfusion.[5] All positive inotropes are associated with an increase in mortality. Dobutamine is the drug most often used, and raises blood pressure solely by increasing cardiac output. Norepinephrine can be used in the small group of patients with vasodilatation and hypotension. Milrinone is not better than dobutamine, but may be preferred in patients who were on beta blockers before admission. New inotropes being developed include omecamtiv mecarbil and levosimendan.

Vasodilators

Nitrates are effective in reducing vascular resistance through vasodilatation with a significant haemodynamic effect being achieved within 30 minutes. Intravenous and oral nitrates are effective, as is nitrate ointment, in relieving dyspnoea. They are commonly used together with inotropes, and are necessary to reduce blood pressure in cases presenting with elevated blood pressure. However, there are few studies on the use of nitrates. Intravenous nitroprusside and nesiritide are other vasodilators that are used in special circumstances.

Serelaxin, a vasoactive peptide hormone, was tested in the Relaxin in Acute Heart Failure (RELAX-AHF) trial. It improved dyspnoea and reduced total mortality (driven by a reduction in mortality from other cardiovascular causes and sudden death without an apparent impact on mortality from heart failure).[6]

Noninvasive positive pressure ventilation with continuous positive airway pressure has been successfully used to treat acute pulmonary oedema.[7]

Assistance of the myocardium

Intra-aortic balloon pump and pulsatile left ventricular assist devices are available in special circumstances.

It is important to establish the cause of the heart failure, as well as the acute precipitant for acute decompensated heart failure and to treat it.

Patients presenting with acute heart failure should receive anticoagulation as they are at high risk of thrombotic complications, such as pulmonary embolism.

On discharge from hospital, it is important that patients are started on evidence-based, life-saving therapy of chronic heart failure, such as renin-angiotensin system inhibitors (angiotensin-converting enzyme inhibitors or angiotensin-specific receptor blockers) and beta blockers.

According to the guidelines, morphine is not considered for use any more, although small doses can be considered, especially in the very anxious.[8]

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