ABC of heart failure

Clinical features and complications

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Clinical features

Patients with heart failure present with a variety of symptoms, most of which are non-specific. The common symptoms of congestive heart failure include fatigue, dyspnoea, swollen ankles, and exercise intolerance, or symptoms that relate to the underlying cause. The accuracy of diagnosis by presenting clinical features alone, however, is often inadequate, particularly in women and elderly or obese patients.

Symptoms

Dyspnoea

Exertional breathlessness is a frequent presenting symptom in heart failure, although it is a common symptom in the general population, particularly in patients with pulmonary disease. Dyspnoea is therefore moderately sensitive, but poorly specific, for the presence of heart failure. Orthopnoea is a more specific symptom, although it has a low sensitivity and therefore has little predictive value. Paroxysmal nocturnal dyspnoea results from increased left ventricular filling pressures (due to nocturnal fluid redistribution and enhanced renal reabsorption) and therefore has a greater sensitivity and predictive value. Nocturnal ischaemic chest pain may also be a manifestation of heart failure, so left ventricular systolic dysfunction should be excluded in patients with recurrent nocturnal angina.

Fatigue and lethargy

Fatigue and lethargy in chronic heart failure are, in part, related to abnormalities in skeletal muscle, with premature muscle lactate release, impaired muscle blood flow, deficient endothelial function, and abnormalities in skeletal muscle structure and function. Reduced cerebral blood flow, when accompanied by abnormal sleep patterns, may occasionally lead to somnolence and confusion in severe chronic heart failure.

Oedema

Swelling of ankles and feet is another common presenting feature, although there are numerous non-cardiac causes of this symptom. Right heart failure may manifest as oedema, right hypochondrial pain (liver distension), abdominal swelling (ascites), loss of appetite, and, rarely, malabsorption (bowel oedema). An increase in weight may be associated with fluid retention, although cardiac cachexia and weight loss are important markers of disease severity in some patients.

Physical signs

Physical examination has serious limitations as many patients, particularly those with less severe heart failure, have few abnormal signs. In addition, some physical signs are difficult to interpret and, if present, may occasionally be related to causes other than heart failure.

Oedema and a tachycardia, for example, are too insensitive to have any useful predictive value, and although pulmonary crepitations may have a high diagnostic specificity they have a low sensitivity and predictive value. Indeed, the commonest cause of lower limb oedema in elderly people is immobility, and pulmonary crepitations may reflect poor ventilation with infection, or pulmonary fibrosis, rather than heart failure. Jugular venous distension has a high specificity in diagnosing

Symptoms and signs in heart failure

Symptoms

Dyspnoea

Orthopnoea

Paroxysmal nocturnal dyspnoea

Reduced exercise tolerance, lethargy, fatigue

Nocturnal cough

Wheeze

Ankle swelling

Anorexia

Signs

Cachexia and muscle wasting

Tachycardia

Pulsus alternans

Elevated jugular venous pressure

Displaced apex beat

Right ventricular heave

Crepitations or wheeze

Third heart sound

Oedema

Hepatomegaly (tender)

Ascites

Common causes of lower limb oedema

- Gravitational disorder—for example, immobility
- Congestive heart failure
- Venous thrombosis or obstruction, varicose veins
- Hypoproteinaemia—for example, nephrotic syndrome, liver disease
- Lymphatic obstruction

Sensitivity, specificity, and predictive value of symptoms, signs, and chest x ray findings for presence of heart failure (ejection fraction <40%) in 1306 patients with coronary artery disease undergoing cardiac catheterisation

Clinical features	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	
History:				
Shortness of breath	66	52	23	
Orthopnoea	21	81	2	
Paroxysmal nocturnal dyspnoea	33	76	26	
History of oedema	23	80	22	
Examination:				
Tachycardia (>100				
beats/min)	7	99	6	
Crepitations	13	91	27	
Oedema (on				
examination)	10	93	3	
Gallop (S3)	31	95	61	
Neck vein distension	10	97	2	
Chest x ray examination:				
Cardiomegaly	62	67	32	

heart failure in patients who are known to have cardiac disease, although some patients, even with documented heart failure, do not have an elevated venous pressure. The presence of a displaced apex beat in a patient with a history of myocardial infarction has a high positive predictive value. A third heart sound has a relatively high specificity, although its universal value is limited by a high interobserver variability, with interobserver agreement of less than 50% in non-specialists.

In patients with pre-existing chronic heart failure, other clinical features may be evident that point towards precipitating causes of acute heart failure or deteriorating heart failure. Common factors that may be obvious on clinical assessment and are associated with relapses in congestive heart failure include infections, arrhythmias, continued or recurrent myocardial ischaemia, and anaemia.

Clinical diagnosis and clinical scoring systems

Several epidemiological studies, including the Framingham heart study, have used clinical scoring systems to define heart failure, although the use of these systems is not recommended for routine clinical practice.

In a patient with appropriate symptoms and a number of physical signs, including a displaced apex beat, elevated venous pressure, oedema, and a third heart sound, the clinical diagnosis of heart failure may be made with some confidence. However, the clinical suspicion of heart failure should also be confirmed with objective investigations and the demonstration of cardiac dysfunction at rest. It is important to note that, in some patients, exercise-induced myocardial ischaemia may lead to a rise in ventricular filling pressures and a fall in cardiac output, leading to symptoms of heart failure during exertion.

Classification

Symptoms and exercise capacity are used to classify the severity of heart failure and monitor the response to treatment. The classification of the New York Heart Association (NYHA) is used widely, although outcome in heart failure is best determined not only by symptoms (NYHA class) but also by echocardiographic criteria. As the disease is progressive, the importance of early treatment, in an attempt to prevent progression to more severe disease, cannot be overemphasised.

Complications

Arrhythmias

Atrial fibrillation

Atrial fibrillation is present in about a third (range 10-50%) of patients with chronic heart failure and may represent either a cause or a consequence of heart failure. The onset of atrial fibrillation with a rapid ventricular response may precipitate overt heart failure, particularly in patients with pre-existing ventricular dysfunction. Predisposing causes should be considered, including mitral valve disease, thyrotoxicosis, and sinus node disease. Importantly, sinus node disease may be associated with bradycardias, which might be exacerbated by antiarrhythmic treatment.

Atrial fibrillation that occurs with severe left ventricular dysfunction following myocardial infarction is associated with a poor prognosis. In addition, patients with heart failure and atrial fibrillation are at particularly high risk of stroke and other thromboembolic complications.

Ventricular arrhythmias

Malignant ventricular arrhythmias are common in end stage heart failure. For example, sustained monomorphic ventricular



Gross oedema of ankles, including bullae with serous exudate

Precipitating causes of heart failure

- · Arrhythmias, especially atrial fibrillation
- Infections (especially pneumonia)
- Acute myocardial infarction
- Angina pectoris or recurrent myocardial ischaemia
- Anaemia
- Alcohol excess
- Iatrogenic cause—for example, postoperative fluid replacement or administration of steroids or non-steroidal anti-inflammatory drugs
- Poor drug compliance, especially in antihypertensive treatment
- Thyroid disorders—for example, thyrotoxicosis
- Pulmonary embolism
- Pregnancy

European Society of Cardiology's guidelines for diagnosis of heart failure

Essential features

Symptoms of heart failure (for example, breathlessness, fatigue, ankle swelling)

and

Objective evidence of cardiac dysfunction (at rest)

Non-essential features

Response to treatment directed towards heart failure (in cases where the diagnosis is in doubt)

NYHA classification of heart failure

Class I: asymptomatic

No limitation in physical activity despite presence of heart disease. This can be suspected only if there is a history of heart disease which is confirmed by investigations—for example, echocardiography

Class II: mild

Slight limitation in physical activity. More strenuous activity causes shortness of breath—for example, walking on steep inclines and several flights of steps. Patients in this group can continue to have an almost normal lifestyle and employment

Class III: moderate

More marked limitation of activity which interferes with work. Walking on the flat produces symptoms

Class IV: severe

Unable to carry out any physical activity without symptoms. Patients are breathless at rest and mostly housebound

tachycardia occurs in up to 10% of patients with advanced heart failure who are referred for cardiac transplantation. In patients with ischaemic heart disease these arrhythmias often have re-entrant mechanisms in scarred myocardial tissue. An episode of sustained ventricular tachycardia indicates a high risk for recurrent ventricular arrhythmias and sudden cardiac death.

Sustained polymorphic ventricular tachycardia and torsades de pointes are more likely to occur in the presence of precipitating or aggravating factors, including electrolyte disturbance (for example, hypokalaemia or hyperkalaemia, hypomagnesaemia), prolonged QT interval, digoxin toxicity, drugs causing electrical instability (for example, antiarrhythmic drugs, antidepressants), and continued or recurrent myocardial ischaemia. β Blockers are useful for treating arrhythmias, and these agents (for example, bisoprolol, metoprolol, carvedilol) are likely to be increasingly used as a treatment option in patients with heart failure.

Stroke and thromboembolism

Congestive heart failure predisposes to stroke and thromboembolism, with an overall estimated annual incidence of approximately 2%. Factors contributing to the increased thromboembolic risk in patients with heart failure include low cardiac output (with relative stasis of blood in dilated cardiac chambers), regional wall motion abnormalities (including formation of a left ventricular aneurysm), and associated atrial fibrillation. Although the prevalence of atrial fibrillation in some of the earlier observational studies was between 12% and 36%—which may have accounted for some of the thromboembolic events—patients with chronic heart failure who remain in sinus rhythm are also at an increased risk of stroke and venous thromboembolism. Patients with heart failure and chronic venous insufficiency may also be immobile, and this contributes to their increased risk of thrombosis, including deep venous thrombosis and pulmonary embolism.

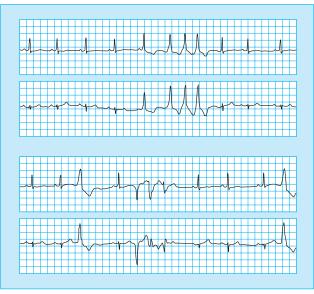
Recent observational data from the studies of left ventricular dysfunction (SOLVD) and vasodilator heart failure trials (V-HeFT) indicate that mild to moderate heart failure is associated with an annual risk of stroke of about 1.5% (compared with a risk of less than 0.5% in those without heart failure), rising to 4% in patients with severe heart failure. In addition, the survival and ventricular enlargement (SAVE) study recently reported an inverse relation between risk of stroke and left ventricular ejection fraction, with an 18% increase in risk for every 5% reduction in left ventricular ejection fraction; this clearly relates thromboembolism to severe cardiac impairment and the severity of heart failure. As thromboembolic risk seems to be related to left atrial and left ventricular dilatation, echocardiography may have some role in the risk stratification of thromboembolism in patients with chronic heart failure.

Prognosis

Most long term (more than 10 years of follow up) longitudinal studies of heart failure, including the Framingham heart study (1971), were performed before the widespread use of angiotensin converting enzyme inhibitors. In the Framingham study the overall survival at eight years for all NYHA classes was 30%, compared with a one year mortality in classes III and IV of 34% and a one year mortality in class IV of over 60%. The prognosis in patients whose left ventricular dysfunction is asymptomatic is better than that in those whose left ventricular dysfunction is symptomatic. The prognosis in patients with congestive heart failure is dependent on severity, age, and sex, with a poorer prognosis in male patients. In addition, numerous prognostic indices are associated with an adverse prognosis,

Predisposing factors for ventricular arrhythmias

- · Recurrent or continued coronary ischaemia
- Recurrent myocardial infarction
- Hypokalaemia and hyperkalaemia
- Hypomagnesaemia
- Psychotropic drugs—for example, tricyclic antidepressants
- Digoxin (leading to toxicity)
- Antiarrhythmic drugs that may be cardiodepressant (negative inotropism) and proarrhythmic



24 Hour Holter tracing showing frequent ventricular extrasystoles

Complications of heart failure

Arrhythmias—Atrial fibrillation; ventricular arrhythmias (ventricular tachycardia, ventricular fibrillation); bradyarrhythmias Thromboembolism—Stroke; peripheral embolism; deep venous thrombosis; pulmonary embolism

Gastrointestinal—Hepatic congestion and hepatic dysfunction; malabsorption

Musculoskeletal—Muscle wasting

Respiratory—Pulmonary congestion; respiratory muscle weakness; pulmonary hypertension (rare)

Morbidity and mortality for all grades of symptomatic chronic heart failure are high, with a 20-30% one year mortality in mild to moderate heart failure and a greater than 50% one year mortality in severe heart failure. These prognostic data refer to patients with systolic heart failure, as the natural course of diastolic dysfunction is less well defined

including NYHA class, left ventricular ejection fraction, and neurohormonal status.

Survival can be prolonged in chronic heart failure that results from systolic dysfunction if angiotensin converting enzyme inhibitors are given. Longitudinal data from the Framingham study and the Mayo Clinic suggest, however, that there is still only a limited improvement in the one year survival rate of patients with newly diagnosed symptomatic chronic heart failure, which remains at 60-70%. In these studies only a minority of patients with congestive heart failure were appropriately treated, with less than 25% of them receiving angiotensin converting enzyme inhibitors, and even among treated patients the dose used was much lower than doses used in the clinical trials.

Some predictors of poor outcome in chronic heart failure

- High NYHA functional class
- Reduced left ventricular ejection fraction
- Low peak oxygen consumption with maximal exercise (% predicted value)
- Third heart sound
- Increased pulmonary artery capillary wedge pressure
- Reduced cardiac index
- Diabetes mellitus
- Reduced sodium concentration
- Raised plasma catecholamine and natriuretic peptide concentrations

Cardiac mortality in placebo controlled heart failure trials

Trial	Patients' characteristics	Ischaemic heart disease (%)		Cardiovascular mortality		
			Treatment	Treatment (%)	Placebo (%)	Follow up (years)
CONSENSUS	NYHA IV (cardiomegaly)	73	Enalapril	38	54	1
SOLVD-P	Asymptomatic (EF $< 35\%$)	83	Enalapril	13	14	4
SOLVD-T	Symptomatic (EF < 35%)	71	Enalapril	31	36	4
SAVE	Postmyocardial infarction (EF < 40%)	100	Captopril	17	21	4
V-HeFT I	NYHA II-III (EF $<45\%$)	44	H-ISDN	37	41	5
V-HeFT II	NYHA II-III (EF $< 45\%$)	52	Enalapril	28	34*	5
PRAISE	NYHA III-IV (EF $< 30\%$)	63	Amlodipine	28	33	1.2

EF ejection fraction. SOLVD-P, SOLVD-T = studies of left ventricular dysfunction prevention arm (P) and treatment arm (T). H-ISDN = hydralazine and isosorbide dinitrate.

Treatment with angiotensin converting enzyme inhibitors prevents or delays the onset of symptomatic heart failure in patients with asymptomatic, or minimally symptomatic, left ventricular systolic dysfunction. The increase in mortality with the development of symptoms suggests that the optimal time for intervention with these agents is well before the onset of substantial left ventricular dysfunction, even in the absence of overt clinical symptoms of heart failure. This benefit has been confirmed in several large, well conducted, postmyocardial infarction studies.

Sudden death

The mode of death in heart failure has been extensively investigated, and progressive heart failure and sudden death seem to occur with equal frequency. Some outstanding questions still remain, however. Although arrhythmias are common in patients with heart failure and are indicators of disease severity, they are not powerful independent predictors of prognosis. Sudden death may be related to ventricular arrhythmias, although asystole is a common terminal event in severe heart failure. It has not been firmly established whether these arrhythmias are primary arrhythmias or whether some are secondary to acute coronary ischaemia or indicate in situ coronary thrombosis. The cause of death is often uncertain, especially as the patient may die of a cardiac arrest outside hospital or while asleep.

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The table on the sensitivity, specificity, and predictive value of symptoms, signs, and chest *x* ray findings is adapted with permission from Harlan et al (*Ann Intern Med* 1977;86:133-8).

The ABC of heart failure is edited by C R Gibbs, M K Davies, and G Y H Lip. CRG is research fellow and GYHL is consultant cardiologist and reader in medicine in the university department of medicine and the department of cardiology, City Hospital, Birmingham; MKD is consultant cardiologist in the department of cardiology, Selly Oak Hospital, Birmingham. The series will be published as a book in the spring.

^{*}Treatment with H-ISDN.