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Introductory Chapter: Heart Failure - A Multifaceted Syndrome

Giuseppe Rescigno

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<http://dx.doi.org/10.5772/intechopen.85478>

1. Introduction

Heart failure (HF) is a worldwide issue. This syndrome is estimated to be present in more than 37 million of patients in the world [1]. In Western countries, this is mainly related to ischemic heart disease, hypertension and diabetes mellitus. In developing and Third-World countries, the causes are more varied. There is still a high incidence of rheumatic disease and end-stage valvulopathies. However, ischemic heart disease is increasing due to the spread of risk factors (diabetes, hypercholesterolemia, smoke). Progressive ageing of the population is another factor that has greatly increased the incidence of HF over the years. The number of hospital admissions with HF as a diagnosis in the USA tripled from 1.27 million in 1979 to 3.86 million in 2004 [2].

Nevertheless, despite major improvements of medical, interventional and surgical treatment, the mortality of the patients with advanced HF is still high; a recent analysis of Medicare patients showed a 50% mortality at 3 years [3].

Apart from the clinical burden in terms of morbidity and mortality, the financial impact of HF is impressive. A forecasting of the impact of heart failure in the USA showed an increase of total expenditure from 21 billion to 53 billion between 2012 and 2030 [4].

2. Classifications

The American College of Cardiology and AHA practice guidelines for chronic HF published in 2001 proposed a classification that includes four stages of HF [5]. Stages A and B are early phases without an overt syndrome and are alerting states. Stage A patients are at risk for HF related to conditions such as hypertension, atherosclerotic heart disease and diabetes mellitus. Stage B

patients have developed structural heart disease from a variety of potential insults to the heart muscle, i.e. myocardial infarctions or valvular heart diseases, but still asymptomatic. Stages C and D are the symptomatic phases of HF. Therapeutic interventions, including dietary salt restriction, medications and implantable devices (pacemakers and defibrillators), are indicated for patients with symptomatic heart failure (stage C). In the end stages of HF (stage D), patients present marked symptoms at rest or with minimal activity despite optimal medical therapy.

Another important classification of HF is based on the left ventricular ejection fraction (EF) value. We generally consider the HF as a low (reduced, <40%) EF syndrome (HFrEF). There is indeed an almost equal number of patients in HF with a preserved EF (>50%) (HFpEF). In between we find patients with moderately reduced EF (40–49%, HFmrEF). The HFpEF shows normally a diastolic LV dysfunction [6].

3. Diagnosis

The diagnosis of HF may be sometimes challenging. There are the classical signs and symptoms that represent the definition of the syndrome (shortness of breath, fatigue, elevated JVP, crackles, ankle swelling, gallop rhythm). However, there are also less specific ones; for instance, depression could be a symptom of HF, as well as confusion, in particular in the elderly. In the older people, in particular, there may be other conditions that make the diagnosis more difficult (i.e. chronic pulmonary diseases). In stable patients, the measurements of natriuretic peptides may help. NT-proBNP level > 125 pg/mL or BNP level > 35 pg/mL should prompt the request for an echocardiography if one of the classical signs or symptoms is present [7]. Transthoracic echocardiography represents the cheapest assessment that may give a certain diagnosis. Care should be taken when an HFpEF is present. In these patients a diastolic dysfunction should be demonstrated to make diagnosis.

Other imaging modalities could be used in doubtful cases as magnetic resonance imaging (MRI) or gated single-photon emission CT (SPECT). MRI is extremely accurate in measuring volumes, masses and EF of both ventricles. The gadolinium-enhanced modality allows a precise assessment of fibrosis, scars and inflammation (myocarditis). Amyloidosis, sarcoidosis, Chagas disease, non-compaction cardiomyopathy and haemochromatosis are similarly well defined by MRI [8].

4. Prevention

The value of prevention in HF has is now well established. The most effective way is a thorough control of hypertension if present [9]. A mild alcohol intake and a regular physical activity are also extremely beneficial. A prompt pharmacological intervention in asymptomatic patients is also recommended; the use of ACE inhibitors has clearly shown to reduce mortality and hospital admissions in patients without a clinically evident HF but with a low EF [10].

5. Treatment

The pharmacological treatment of HF is clearly codified in several national and international guidelines [11]. The cornerstones remain the diuretics, beta blockers, ACE inhibitors and mineralocorticoid receptor antagonists; recently new medications have been used with promising results in subcategories of HF patients like the ivabradine and sacubitril valsartan. However, a multidisciplinary discussion about every single patient and a close monitoring of the clinical effectiveness are important [12]. The management of acute HF decompensation is more complex and less codified.

HF patients may benefit of a few non-pharmacological interventions able mainly to improve survival. The mortality in HF is to be cancelled not infrequently sudden and related to arrhythmic events. The most recent guidelines about HF clearly states that 'An implantable cardioverter-defibrillator (ICD) is recommended to reduce the risk of sudden death and all-cause mortality in patients who have recovered from a ventricular arrhythmia causing haemodynamic instability, and who are expected to survive for >1 year with good functional status'. This is a recommendation IA, therefore with a very strong evidence. Moreover, the ICD is also strongly recommended in patients with an EF <35% after 3 months of optimal medical treatment and when life expectancy is more than 1 year [7]. On the contrary, when the patient is in NYHA Class IV and is not a candidate for cardiac resynchronisation therapy (CRT), ventricular assist device (VAD) or heart transplantation, the ICD is formally contraindicated.

The cardiac resynchronisation therapy (CRT) is a non-pharmacological/non-surgical treatment that represents a consistent adjunct for HF management. It consists in a synchronous pacing of both ventricles. There are clear indications, mainly related to ECG parameters and the clinical status of the patient [7]. Nowadays, the CRT devices have frequently defibrillating or pacing features. This treatment has shown to improve the quality of life and, partly, the survival of HF patients [13, 14]. However, there are non-responders to CRT that sometimes are difficult to identify before the implantation. As a rule, ischemic patients respond less favourably; on the contrary, female subjects are better responders.

The last resource for end-stage HF patients or when a refractory acute HF develops are mechanical circulatory support devices. In case of acute decompensation, extracorporeal, non-durable life support systems can be used to unload the ventricles. When the condition is chronic, a left ventricular assist device (LVAD) can be indicated. There are five conditions [15] that may prompt the consideration of a MCS:

- A. Bridge to decision or bridge to bridge: The use of short-term MCS (ECMO) in patients with cardiogenic shock until haemodynamics and end-organ perfusion are stabilised, contraindications for long-term MCS are excluded (brain damage after resuscitation) and additional therapeutic options including long-term VAD therapy or heart transplant can be evaluated.
- B. Bridge to candidacy: The use of MCS (usually LVAD) to improve end-organ function in order to make an ineligible patient eligible for heart transplantation.

- C. Bridge to transplantation: The use of MCS (LVAD or BiVAD) to keep the patient alive who is otherwise at high risk of death before transplantation until a donor organ becomes available.
- D. Bridge to recovery: The use of MCS (typically LVAD) to keep the patient alive until the heart recovers enough to remove MCS.
- E. Destination therapy: The long-term use of MCS (LVAD) as an alternative to transplantation in patients with end-stage HF ineligible for transplantation or long-term waiting for heart transplantation.

The technology behind LVAD is continuously evolving, and smaller and less invasive models are expected in the next few years.

Heart transplantation still represents a viable treatment modality for end-stage heart failure. Major issues are the shortage of donor organs and many possible complications like organ rejection, neoplasms, coronary artery disease of the graft, infections and renal failure. The indications and contraindications are well codified [16]. There is no randomised study; however, survival and quality of life are greatly improved. Management of immunosuppressant medications is of outmost importance.

6. Conclusion

Heart failure is a worldwide health issue; however, new diagnostic modalities, drugs, devices and multidisciplinary approaches will certainly be able to better manage these complex patients.

Author details

Giuseppe Rescigno

Address all correspondence to: grescigno@mac.com

Department of Cardiothoracic Surgery, New Cross Hospital, Royal Wolverhampton Trust, Wolverhampton, United Kingdom

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