

Diagnostic and therapeutic problems in heart failure*

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ABSTRACT

Heart failure (HF), affecting an increasing number of patients, is associated with several diagnostic, therapeutic, socioeconomic problems. Despite considerable advances, prognosis in HF – both in systolic failure and in that with preserved systolic function – is poor, which in part results from not the fully understood pathophysiology of this disease, and therapeutic approaches used at present. Until recently, efforts of researchers focused on the development of effective therapeutic methods in more advanced stages of the disease, but their results were disappointing. Currently, more emphasis is placed on broadly understood methods of HF prevention. When starting HF treatment upon objective confirmation of the diagnosis, there are some primordial goals to be remembered, that if achieved, may translate into postponing the prospect of death and improving the patient's quality of life. Therapy of HF employs pharmaceutical agents that reduce mortality (β -blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, aldosterone antagonists) and attenuate symptoms (diuretics, digoxin, inotropes). Intensification of therapy depends on the functional NYHA class. In recent years, fate of patients with advanced HF has improved through the use of electrotherapy (cardioverters-defibrillators and[or] resynchronization devices). The key issue is to develop a uniform model of healthcare for HF patients, focused on an outpatient clinic system rather than a hospital.

According to a number of estimates, the incidence of heart failure (HF) will increase in the following decades, with advances in the treatment of acute cardiological conditions and ageing of populations being mostly to blame. At present, HF affects 2–10% of the population aged ≥ 65 years.¹ Despite advances, the prognosis in HF is fatal: approximately 50% of patients die within 5 years after the onset of symptoms, and in the group with terminal HF 50% of patients die within 1 year.² The pathophysiology of HF has not been fully understood. However it can be assumed that the primary myocardium injury triggers a number of mechanisms leading to neurohormonal stimulation, endothelial dysfunction and many secondary phenomena that affect the damaged myocardium, thus leading to remodeling of the left ventricle, impaired contractility, hypertrophy, fibrosis, apoptosis and electrophysiological disturbances.³ Until recently HF has been perceived as systolic HF, but over several recent decades there have been increasing numbers of HF patients with normal left ventricular systolic

function.⁴ It turned out that this HF is associated with a similar prognosis as systolic HF.^{5,6} Importantly, there are no therapeutic standards for this group of patients.

At the first contact with a patient, when a suspicion is made that complaints reported by him or her are consistent with the diagnosis of HF, it is worth to remember about practical hints, which permit avoiding a diagnostic error and planning of lifetime treatment for the patient.⁷ First of all, HF should be objectively confirmed by performing the ECG, the chest X-ray, echocardiography and a series of measurements of parameters indicating the function of multiple organs, as well as the presence of crucial factors for prognosis and the treatment introduced. After establishing the diagnosis of HF, the cause and stage of the disease should be determined. Only when the above questions have been answered, can the therapy begin, which in order to be effective has to meet the following objectives:

- 1) reduction of mortality

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- 2) improvement of the quality of life by attenuating HF symptoms and reducing hospitalization rate
- 3) prevention of HF progression and diseases leading to cardiac dysfunction.⁷

When beginning the therapeutic process with HF patients, one should remember about guidelines of American cardiac associations, which conditioned the achievement of different therapeutic goals on the disease stage.⁸ The first 2 stages have been termed as at-risk for HF: stage A – asymptomatic, without any cardiac structural or functional abnormalities and stage B – asymptomatic cardiac structural or functional abnormalities, in which the therapeutic goals have focused on the control of arterial pressure, lipid and carbohydrate metabolism, and the modification of concurrent risk factors. In these stages, in the process of individual treatment approach, one should consider the use of β -blockers and angiotensin-converting enzyme inhibitors (ACEI) or angiotensin receptor blockers (ARB) and implantable cardioverter-defibrillator (ICD). The following stages have been termed HF: stage C – cardiac structural and functional abnormalities with past or present HF symptoms, where the aims of the effective therapy are the same as in stages A and B, and restriction of dietary salt consumption, administration of diuretics in the case of overhydration and β -blockers and ACEI, and in selected patients the administration of aldosterone receptor antagonists, digoxin, resynchronization therapy and ICD implantation, whereas in stage D – resistant HF requiring specialized interventions – the principal therapeutic goals are consistent with accomplishment of the objectives recommended in the three previous stages, and in addition they include consideration of highly specialized therapeutic procedures.

On the other hand, according to the European guidelines HF therapy is dependent on the stage of the disease; the more advanced HF, the more intensive treatment.⁷ 2 classes of drugs are used in therapy i.e. those reducing mortality (ACEI/ARB, β -blockers, aldosterone receptor antagonists) and those alleviating HF symptoms (diuretics, digoxin and inotropes).

The achievements of the recent 20 years have markedly improved the prognosis of patients with HF. Due to the use of ACEI, β -blockers, ARB, aldosterone receptor antagonists and statins as well as electrotherapy (resynchronization therapy and/or ICD), a 30–40% mortality reduction in patients with mild and moderate HF and a 50–60% mortality reduction in those with severe HF have been achieved. Unfortunately, these data derive from randomized controlled trials and are highly discrepant from the outcomes in patients in the so-called real world.⁹

Unsatisfactory outcomes of treatment in HF patients prompt the further quest for new classes of pharmaceutical agents and optimization of electrotherapy and invasive therapeutic procedures. This search focuses on, among other things,

achieving new therapeutic goals in different HF stages, the accomplishment of which would translate into better prognosis. Among these goals there are: anemia, renal insufficiency, hyperuricemia, excessive inflammatory stimulation, cardiac cachexia and depression, which are independent predictors of poor prognosis.^{10–12} By influencing the above goals, there is hope to postpone the HF progression, in consequence leading to death.

In the recent years, a great achievement in HF treatment was a wide use of invasive HF treatment methods to clinical practice, i.e. ICD implantation and (or) resynchronization therapy. The current guidelines recommend ICD implantation in selected symptomatic patients with left ventricular ejection fraction (EF) <30–35%, not earlier than 40 days after myocardial infarction, who receive optimal pharmacotherapy.⁷ Whereas the use of resynchronization therapy should be considered in patients with HF in III–IV NYHA class with EF <35% and QRS duration of ≥ 120 ms.⁷ In part of cases of severe HF in NYHA class III to IV, with EF <35% and QRS width of ≥ 120 ms, the resynchronization therapy with an ICD option can be considered.⁷

In some patients with HF revascularisation procedures can be considered, which in order to be effective must be performed in patients with viable myocardium.^{13,14} Lack of viability renders the distant effectiveness of revascularization methods comparable with effectiveness of conservative treatment.

While treating patients with advanced HF should be considered the need of qualification for heart transplantation. The very fact of positive qualification is not equivalent to the performed transplantation. This status has many causes, and it is unlikely that in the following years the number of transplantations will dramatically increase not only worldwide, but also in Poland. Therefore the data on survival of patients after heart transplantation several years ago in comparison with conservatively treated individuals currently awaiting the transplant, should be considered optimistic. It has been demonstrated that survival in these 2 groups of patients is comparable. These results provided the basis for verification of current qualification standards for heart transplant developed several years ago, which for clear reasons did not take into account the latest achievements in the field of pharmacotherapy and invasive treatment of patients with advanced HF.

In order to solve at least some problems associated with HF, the development of a model of care of Polish patients seems to be crucial. It is not a hospital that admits severely decompensated patients that should be in the center of this system, but rather an outpatient care system accomplishing the program of complex support and education of patients and their environment. This system should be based on a constant contact of the patient and their family with a doctor or a nurse trained in solving problems of patients

with HF, and possible hospitalisation whenever such a necessity arises.

In conclusion, it should be objectively stated that taking care of patients with HF is an exceptionally difficult challenge for healthcare professionals. Poor prognosis, a number of problems regarding the quality of life of the patients, as well as the lack of an efficient system of outpatient care are the reasons why we taste the bitterness of defeat when HF is combatted. One should also remember that once HF begins, it usually progresses despite elimination of the trigger, and its course is frequently insidious; at any time – despite the optimal therapy – cardiac death can occur, interrupting the natural history of HF development.

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