Heart failure in the elderly: A geriatric syndrome. Picture of the modern situation

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Abstract

Among the older patients' cohort, the aetiology of heart failure is peculiar and differs in many ways from the younger one, both in its epidemiology, diagnostic work-up and clinical presentation. Focusing on this population, we could assume that heart failure is a real geriatric syndrome, characterized by several features, which coexist with other comorbidities and require specific and targeted cares. It is therefore necessary to examine the global burden of heart failure and the patient’s history rather than the causal cardiomyopathy - frequently more than one in the elderly - facing with the condition, bearing in mind the quality of life even before its duration.

Introduction

Heart failure (HF) affects about the 16% of individuals 75 years of age and older, respect 4-5% of individuals 45 years and older [1], even with the limits due to different populations investigated and different criteria applied for the diagnosis of this disease.

Identifying a single condition determining alone HF in the elderly is pretty hard, while finding the main causes of HF in the general population is relatively easy (just citing the most frequent: ischaemic, hypertensive and heart valve disease-related) [2]. In geriatric cohorts, finding out several pathological events, concurrent to determine HF, is rather common [3]. Here, factors as polypharmacy, malnutrition and cognitive impairment play a fundamental role that is often underestimated. We can state that HF in the elderly is a multifactorial condition.

Current evidence

Heart failure is more than ever a geriatric topic: we are currently spectators of a progressive increase of the mean age at the first diagnosis of HF, which at the time of writing is of 80 years. HF can be considered a “geriatric epidemic”, partly because nowadays life expectation has been enormously elongated by early diagnosis and effective targeted therapies for several common diseases, which have also decreased the recurrence of acute events [4]. However, this has also led to the appearance of increasingly cases of chronic comorbidities.

Pathophysiology

Finding out an elderly patient affected by only a single cardiovascular disease that has led to HF is unexpected; there are more frequently various correlated conditions characterising a clinical status of HF. Furthermore, also typical and physiological ageing of cardiovascular system and vascular structures leads to HF (Table 1). From this point of view, HF is more like a physio-pathological condition than a pathological status derived from a defined disease.

The higher prevalence of HF in women and the higher number of preserved ejection fraction (pEF) are other points of interest which characterise this condition in older adults if compared with younger cohorts [5,6]. These findings are mainly due to the higher mean age and the lower incidence of ischaemic disease in females.

If we could draw a model of the “typical old HF patient”, it could be a woman of 85 years affected by hypertension and kidney impairment, not totally self-sufficient, with polypharmacy and low compliance to drug assumption and HFpEF.

In summary, we can conclude that the main features of HF as geriatric syndrome [7] are the following:

i. multifactorial aetiology;

ii. heterogeneity of presentation, with frequent overlaps between several co-existing morbidities;

iii. difficulty in the diagnostic work-up, mainly due to the impossibility to perform histological or instrumental tests and exams;

iv. chance to easily find out some aetiological indicators for certain risk factors;
v. role of other comorbidities in determining a worse outcome.
vi. Due to these reasons, there is the need to face with HF in the elderly with a multidimensional approach, evaluating from the beginning not only the usual linear model “targeted diagnosis - targeted therapy” but rather the global frame, giving value to the quality of life, the residual autonomy level of the patient and the familiar and social support, considering the polypharmacy risks and the comorbidities burden in order to guarantee the best work-up for such a challenging and tricky condition.

References


| Table 1. Main microscopic alteration of the cardiovascular system due to physiological ageing. |
| Cardiomyocytes | Decrease in function and number |
| Calcium metabolism | Alteration that leads to contractility impairment |
| Contractile proteins | Modification due to aging, with reduction in function and number |
| ATP use | Reduced efficacy |
| Fibrosis | Increased |
| Extracellular matrix metabolism | Alteration in pro-fibrotic direction |

ATP, adenosine triphosphate.