Congestive heart failure in the elderly diabetic

C. Verny

Service de gériatrie, CHU de Bicêtre, Le Kremlin-Bicêtre, France

Abstract

The elderly diabetic is a potential congestive heart failure patient. Cardiac involvement is multifactorial, particularly ischemic conditions because of the accumulation at that age of vascular risk factors and therefore the frequency of coronary damages. The elderly diabetic very often has high blood pressure, with the risk of developing a hypertensive heart disease. Beyond these issues, the effects of chronic hyperglycaemia and insulin resistance on the heart specifically alter left ventricle compliance and therefore diastolic function, thus accelerating the effects proper to aging. No specific recommendation has been published on the management of the elderly diabetic with congestive heart failure. Even at an advanced age, with a clinical diagnosis of congestive heart failure that is sometimes difficult to make, the cardiological evaluation should be conducted rigorously within a global evaluation, and treatment should follow the same rules as in younger patients, with great caution given to the iatrogenic risks inherent to this population.

Résumé

Insuffisance cardiaque chez le diabétique âgé.

Le diabétique âgé est un insuffisant cardiaque en puissance. L’atteinte cardiaque est multifactorielle, en particulier ischémique du fait du cumul, à cet âge, des facteurs de risque vasculaire et donc de la fréquence de l’atteinte coronarienne. Le diabétique âgé est très souvent hypertendu, avec le risque de développer une cardiopathie hypertensive. En dehors de ces atteintes, les effets propres de l’hyperglycémie chronique et de l’insulinorésistance sur le cœur altèrent de façon spécifique la compliance du ventricule gauche et donc la fonction diastolique, accélérant ainsi les effets propres du vieillissement. Aucune recommandation spécifique n’a été publiée sur la prise en charge du diabétique âgé insuffisant cardiaque. Même dans le grand âge, devant un diagnostic clinique d’insuffisance cardiaque, parfois difficile à faire, l’évaluation cardiological doit être menée avec rigueur, au sein d’une évaluation globale, et le traitement doit suivre les mêmes règles que chez les plus jeunes, avec une grande prudence du fait des risques iatrogènes.

Key words: Diabetes mellitus; Elderly diabetic; Aging heart; Diabetic heart disease; Diastolic dysfunction; Congestive heart failure

Mots-clés : Diabète sucré ; Diabétique âgé ; Vieillissement cardiaque ; Cardiopathie diabétique ; Dysfonction diastolique ; Insuffisance cardiaque

Introduction

Studies on the Framingham population, from now roughly 30 years ago, have taught that diabetes was a risk factor for congestive heart failure (CHF), whatever the patient’s age [1]. Since then, the literature has enriched our knowledge on the specific effects of diabetes and chronic hyperglycaemia on the heart, in terms of structural, biochemical, and functional modifications.

In parallel, CHF has been recognized as a major health problem in the elderly subject, one of the leading causes of mortality and emergency hospitalization in the 65 years and above age group [2]. The increase in prevalence [3-5] and incidence [6] of CHF with age is explained by the effects of aging itself on the heart, progressively reducing the functional reserves, to which can be added one or several chronic cardiopathies: hypertensive, ischemic, or valvular.

Correspondence.

Adresse e-mail : christiane.verny@bct.aphp.fr

© 2007 Elsevier Masson SAS. Tous droits réservés.
The combined effects of diabetes and aging on the heart make CHF one of the major preoccupations of diabetes in the older subject. Diabetologists and cardiologists on the one hand [7] and cardiologists and geriatricians on the other hand [8-9] have combined their energies to establish recommendations on the treatment of these CHF patients, diabetic or old, some stressing the absence of demonstrated specificity in the management of CHF in the diabetic despite an increase in the frequency and severity of the disease [7], others concerned with “the absence of a major and specific particularity of the elderly subject concerning treatment of congestive heart failure” [8].

The latest US recommendations on management of the older diabetic emphasize the need to individualize the overall healthcare objectives and the glycaemic objectives [10], recalling how heterogeneous the elderly population is, both in terms of clinical presentation and duration of the disease’s progression, as well as in terms of complications of diabetes and of number of co-morbidities [11]. Despite the very high vascular risk in this population, the chapter on the specific management of CHF in this population remains to be written.

1. Epidemiology

The incidence of CHF has doubled in diabetics compared to non diabetics, with age as one of the major risk factors [12]. This phenomenon has accelerated with time probably because of the improvements in living conditions and in the management of diabetes, CHF, and of the elderly subject in general. In the 65 years and older age group, of the incidental cases of CHF, 22% of men and 24% of women were diabetic between 1990 and 1994 (vs. 15% and 18%, respectively, 20 years earlier) [6]. In certain specific cardiological situations, diabetes significantly increases the risk of CHF. Diabetes increases the risk of CHF occurrence after a first episode of atrial fibrillation [13], or after myocardial infarction [14]. The occurrence of CHF during diabetes is probably related to the quality of glycaemic control. For some authors, every 1% increase in HbA1c corresponds to a 15% increase in the risk of developing CHF [15]. But the mechanisms are more complex, and modifications in the glucose metabolism preceding clinical diabetes, play a great role. Thus, one patient out of three will develop diabetes in the three years following the diagnosis of CHF [15].

The prevalence of diabetes among patients with CHF varies according to country as well as the diagnostic criteria of CHF and diabetes, but it is much higher than in the general population, ranging from 19% for a French study [5], to 28% of 70+-year-old patients hospitalized for CHF [16], to 36% in the Cardiovascular Health Study [17]. Of 31,600 US subjects 65 years and older hospitalized for CHF, 27% of whom were 85 years old or older, 40% were diabetics [18], a rate also found in a Canadian study [19]. Diabetics were also abundantly present in the interventional studies on CHF, 20% for the angiotensin-converting enzyme inhibitors (ACEIs) studies, 21% for beta-blockers [20] studies, and 28% for the angiotensin II receptor antagonists (ARA2) studies [21].

2. Effects of aging and diabetes on cardiac structure and physiology

The diabetic heart and the aged heart have a certain number of points in common. We report here the specific effects, excluding coronary vascular damages, which is discussed in another section.

2.1. The effects of aging

It is always difficult to separate out what is related to aging and what stems from the different cardiac pathologies that may have accumulated. During aging, a certain number of modifications appear, involving all of the cardiac structures and tissues. Some are only the consequences of the vascular system’s aging (an adaptive mechanism), others are primary. The primary element seems to be a decrease in the number of myocytes resulting in hypertrophy of the remaining myocytes and a relative increase in the fibroblasts producing collagen.

The pathophysiological processes leading to this loss have not yet been totally elucidated, but several mechanisms have been suggested, such as the accumulation of the terminal products of advanced glycation end products (AGE) or an overproduction of free radicals with a less effective repair system, participating in setting off myocyte apoptosis [2]. The functional consequences are a decrease in the elasticity and the compliance of the left ventricle (LV), with difficulty at passive filling. To maintain normal cardiac flow at rest, the heart digs into its adaptive reserve, increasing the active filling, i.e., the contribution of the atrial systole, with, on the echocardiogram, a modification of the E/A ratio (evaluating the respective roles in LV filling, the passive phase, and the contraction of the left atrium) [2]. This increase in effort results in structural changes in the left atrium, its dilatation, thus inducing a high prevalence, in older subjects, of supraventricular rhythm disturbances, such as atrial fibrillation. In addition, the decrease in elasticity and compliance and the increase in the amount of collagen in the vascular walls with age, bring about an increase in ejection resistance and thus contribute to myocardial hypertrophy [2, 22]. The clinical markers of this arterial aging are a progressive increase in pulse pressure, through an increase in systolic and a decrease in diastolic blood pressure. High pulse pressure is now recognized as a powerful vascular risk factor in the elderly subject [22-23]. Another sign of aging is the progressive decrease in the sensitivity of beta-adrenergic receptors. Thus, the heart rate at exercise, remains normal, not because of an increase in the heart rate, as in young subjects, but because of an increase in telediastolic volume of the left

ventricle [2]. Bringing together all these adaptive capacities to maintain a normal cardiocirculatory function as aging progresses explains the relative drop in functional reserves and the fragility of the system in an adverse situation that can lead to cardiac failure [2].

Independently of myocardial aging, a time effect is also observed on the valvular apparatus, such as dilatation of the rings and calcification of the valves, and on the conduction system, with a decrease in the number of pacemaker cells and fibrosis of the sinoatrial node [2].

2.2. Effects of diabetes and insulin resistance on the heart

The effects of diabetes on cardiac structure and function are difficult to isolate from the usual coronary and hypertension context in these patients. Specific diabetic cardiomyopathy is now admitted, where high blood glucose and insulin resistance play a major role [24]. The so-called “metabolic” cardiopathy can even occur before the stage of clinical diabetes. During the Uppsala Longitudinal Study of Adult Men, in 70-year-old subjects followed up for nine years, who were free from valvular disease and CHF, insulin resistance, identified with the clamp technique, is a risk factor for the first hospitalization for CHF, independently of the diabetes and the usual risk factors [25]. Diabetes is one of the possible etiologies of the cardiomyopathies thus far described as primary: in an US population with a mean age of 66 years, diabetes is much more frequent in patients coming out of a hospitalization with a main diagnosis of idiopathic cardiomyopathy than in control subjects [26].

Most of the echocardiographic analyses show that, compared to controls, diabetics present with an increase in LV wall thickness, independent of the age, the existence of arterial hypertension, blood pressure, and body mass index (BMI) [24, 27-28]. This left ventricular hypertrophy (LVH) corresponds to concentric remodeling [29], without dilatation of the LV cavity [27-28]. In the Framingham population of subjects aged 52 to 59 years as a mean, this relation was only found in women [30]. No explanation has been advanced for this difference in the results other than the relatively young age of the subjects studied. This parietal hypertrophy is difficult to isolate from the effect of diabetes on the vascular walls, with an increase in the arterial stiffness index [27-28] and therefore an increase in post-load. An increase in the size of the left atrium was also found in diabetics, independently of BMI and sex [30-31].

Just as during aging, the histological anomalies come in two types: hypertrophy of the myocytes, and interstitial fibrosis [15]. Myocyte hypertrophy and LVH are correlated with insulin resistance markers and are explained by the trophic role of insulin via growth factors [24-25, 30, 32]. Interstitial fibrosis seems to depend more on high blood glucose, responsible for an increase in AGE production and the formation of free radicals [24, 33]. This fibrosis leads to decreased LV compliance, a decisive element of diastolic dysfunction. The innermost biochemical mechanisms of this cardiac disease have not been completely elucidated. They involve local phenomena of glucose toxicity but also lipid toxicity resulting in myocyte apoptosis [34-35].

Beyond these metabolic aspects, the situation is aggravated by the existence of an autonomic neuropathy, which is responsible for a decrease in vagal tonicity and therefore an increase in the heart rate with a reduction in filling time [15].

2.3. Consequences of aging and diabetes

The anomalies in LV diastolic function are at the forefront during diabetic heart disease. Therefore, 30%-60% of diabetics, even those with good glucose control, present echocardiographic signs of diastolic dysfunction [24, 35-36], independently of whether there is or not arterial hypertension. These differences in prevalence are in large part related to the difficulties of echographic identification of this dysfunction and to the choice of criteria. The progression of abnormalities of the diastolic phase related to age is thus greatly accelerated by diabetes [36], which also amplifies the changes induced by arterial hypertension [28].

3. Does diabetes modify the presentation of congestive heart failure in elderly subjects?

As mentioned above, the elderly diabetic is at very high risk of congestive heart failure, but also of coronary involvement and arterial hypertension. Therefore, even greater vigilance is called for.

3.1. Diagnosing congestive heart failure

In clinical practice, the question of diagnosing CHF in the elderly subject arises in two circumstances:

- during an intercurrent acute disease whose progression is complicated by the appearance of congestive signs, suggesting the decompensation of an underlying heart disease that has been silent to this point;
- progressive appearance of the classical symptoms of CHF, as the heart disease progresses, even without any acute problem.

To our knowledge, the clinical picture of CHF in the older diabetic has never been the subject of a specific study. The diagnostic procedure should be the same as for younger subjects: establish a positive diagnosis, describe the mechanism involved, and search for the cause and the possible transitory aggravating factors.

The diagnostic criteria for CHF in elderly subjects are the same as for younger subjects [8]. However, the sensitivity and specificity of each sign or symptom are lower in older subjects, mainly because of the frequency of associated diseases [9, 22]. The pulmonary signs (shortness of breath, abnormalities on auscultation) can be attributed to a chronic respiratory disease or to the sequelae of tuberculosis for example, or the existence of anemia. These patients’ reduced
mobility can prevent shortness of breath with exercise and therefore considerably delay the diagnosis of CHF. Edemas of the lower limbs can result from deficiencies (hypalbuminemia) or iatrogenic causes (calcium channel blockers). CHF in advanced age can have an atypical presentation, such as isolated major and progressively increasing weakness, or confusion/delirium syndrome, in particular in patients with dementia [9].

The electrocardiogram (ECG) should obviously be done systematically. Although there are no electrical signs typical of CHF, the ECG is normal in only 5% of CHF cases. Thus, in such cases of normal ECG, diagnosis of CHF should be reconsidered. ECG permits to find signs indicative of ventricular hypertrophy, coronary heart disease, or arrhythmia problems [8]. A chest x-ray can provide images characteristic of pulmonary edema or cardiomegaly (if the examination is done on a patient in the supine position, the cardiothoracic index cannot be interpreted), but can also help in the differential diagnosis when there are pleural calcifications, tuberculosis sequelae, bronchial ectasia, or images of pulmonary fibrosis that must be explored by a CT examination. The dosage of type B natriuretic peptide can also greatly assist when the clinical diagnosis is difficult [8]. Its interpretation is being evaluated in the very old subject: the rate increases with age and kidney failure, and is higher in women. However, the threshold above which the type B natriuretic peptide has the best positive predictive value is yet debated, but is probably between 300 and 400 ng/l [37].

The key test is echocardiography, which should be systematic when there is any suspicion of CHF, even at very advanced ages [8-9]. First of all, the mechanism responsible for the heart failure can be established. No clinical sign can differentiate systolic CHF, with a decrease in the left ventricle ejection fraction (LVEF), whose main etiology is coronary disease, and diastolic dysfunction, characterized by normal LVEF, but with a defect in LV compliance, resulting in difficulties filling the LV [19]. These two types are clearly distinct entities, from both a pathophysiological and a morphological/functional point of view [32]. The echocardiography then points to the etiologic diagnosis: ischemic or hypertensive heart disease in most patients, or valvular heart diseases in others.

3.2. Diastolic congestive heart failure

CHF of the elderly subject is characterized by a high frequency of diastolic CHF because of the effects of aging itself, on the one hand (but which cannot alone explain the heart failure), and the frequency of arterial hypertension on the other. According to the European criteria, diastolic CHF is defined by the coexistence of CHF signs, a LVEF over 45%, and left ventricle telediastolic pressure greater than 16 mmHg [32]. Of elderly CHF subjects, 30%-50% have preserved LVEF [19, 22, 38], with differences between studies depending on the echocardiographic criteria retained. In the Cardiovascular Health Study, for example, 63% of the CHF patients aged 65 years or more had a LVEF ≥55%, and 15% between 45% and 54% [17]. On account of the LV compliance defect's repercussion on the left atrium, the prevalence of atrial fibrillation is higher during diastolic CHF, with the risks of embolic complications requiring specific treatment [19, 21].

Does diabetes modify this distribution? No formal response can be given. Indeed, for some authors, diastolic CHF is even more frequent in diabetic elderly subjects [22]; others claim that patients with a LVEF <40% are more often diabetic, probably through coronary damages [19]. Female sex, advanced age, the presence of arterial hypertension and chronic obstructive lung disease are, however, significantly associated with diastolic CHF [19, 38].

Beyond accounting for the mechanism of CHF, echocardiography can specify the cause or causes of cardiac involvement, often hypertensive or ischemic in diabetics, so as to better adapt treatment.

3.3. Effects of diabetes and age on the prognosis of congestive heart failure

The main prognostic factors used in the literature are mortality, frequency of hospitalizations, and other vascular events, in particular cerebral events, and to a lesser degree because they are more difficult to evaluate, the impacts on quality of life.

Age is recognized by all to be a risk factor for mortality in CHF [22]. In the CHARM (Candesartan in Heart Failure: assessment of reduction in mortality and morbidity) study, in an evaluation of prognosis based on all-cause mortality, or the occurrence of death due to cardiovascular disease, or hospitalization for CHF, three major risk factors were found: age over 60 years, diabetes, and LVEF <45% [39]. In the VALIANT (Valsartan in Acute Myocardial Infarction Trial) study investigating postinfarction CHF, during the 3-year follow-up, age was associated with an increase in the risk of overall mortality, cardiovascular mortality and cardiovascular events. After 75 years of age, atrial fibrillation was significantly more frequent [40]. This age impact can have several explanations, such as the frequency and seriousness of the co-morbidities, the increase in the frequency of social problems, whose influence on the hospitalization rate specifically for CHF has been described [41], but also the insufficiency of effective medical prescriptions. In the VALIANT study for example, the subjects who were 85 years of age and older, at inclusion and at three years, received significantly fewer beta-blockers, platelet antiaggregants, and statins [40]. For others, the excess mortality in elderly patients with CHF is related to the unjustified under-use of ACEIs [42-43]. This overcautiousness in prescribing medications is probably multifactorial: the fear of an immediate iatrogenic effect canceling out the medium-term benefits of these drugs, the lake of interventional studies in the oldest subjects, and the absence of trials studying diastolic CHF specifically [18]. The frequency of atrial fibrillation...
A certain number of poor prognosis factors have been demonstrated in follow-up studies of elderly CHF patients. In the Cardiovascular Health Study, in subjects 65 years of age or older [17], but also in a survey on patients with a mean age of 89 years who were institutionalized [44], the risk of death was greater when LVEF was altered. Another prognostic factor that has been consistently found is kidney failure, a sign of renal hypoperfusion and secondary activation of the sympathetic and renin-angiotensin-aldosterone systems [44-45]. Huynh et al. attempted to establish a prognostic score based on a 14 year follow-up study lasting 14 years, in patients 70 years of age and older who were hospitalized for CHF. The mean survival was 2.5 years, but with highly heterogeneous values. In multivariate analysis, the factors of poor prognosis that were identified were the following: age 75 years or older, natremia less than 135 mEq/l, co-morbidities (dementia, lower-limb arteritis, coronary heart disease), systolic arterial pressure less than 120 mmHg, and a blood urea nitrogen level greater than 10.7 mmol/l. At 6 months, mortality ranged from 7.9% for patients with no or one of these risk factors to 59.5% for patients who had four or more [16]. This prognostic score can be useful in choosing drugs or the more appropriate treatment for the patient.

Does diabetes account for the prognosis for CHF? The results are controversial. It seems that for relatively young CHF subjects, diabetes may be responsible for an excess morbidity and mortality [15, 39], more specifically during ischemic cardiac diseases, as shown in the BEST (Beta-blocker Evaluation of Survival Trial) study in patients with a mean age of 61 years [46]. In the older patients, however, diabetes did not seem to be a factor of poor prognosis [16, 44], whatever the type of cardiopathy, systolic or diastolic [19].

4. Treatment of congestive heart failure in the elderly diabetic

Today there are no specific recommendations for the elderly diabetic.

4.1. The global objectives of patient management

The global objectives were reviewed recently in a consensus conference organized by the French Cardiology Society (SFC) and the French Society of Geriatrics and Gerontology (SFGG). The recommendations are the same as in young subjects: reducing the congestive symptoms, mortality, and the number and duration of hospitalizations, slowing down the progression of the disease, and improving the patient’s quality of life [9]. Diabetes does not modify these objectives. In the elderly diabetic, however, three problems arise:

- Does CHF treatment depend on age?
- Does CHF treatment efficacy vary with diabetes?
- What is known on diastolic CHF treatment?

4.2. Particularities in the treatment of congestive heart disease in elderly subjects

The theoretical rules of CHF treatment present no specificity in relation to age. Management should be global, extending beyond simple prescription of drugs, with training for the patient and his family (weight monitoring, adapting diuretic doses, maintaining good eating habits and adapted physical exercise), with preventive actions such as flu and pneumonia vaccinations [8]. The indications of the different classes of drugs, depending on the type of CHF and how severe it is, are the same as in younger subjects [8]. However, in the therapeutic discussion, notions of frailty, the influence of co-morbidities and multiple medications intervene at each stage, with an evaluation of the risk/benefit ratio that should take all of these factors into account. The prescription for an elderly patient with CHF includes more and more medications, 7.5 on average in the early years of the century, with the indications for each one generally indisputable in the attempt to reduce cardiovascular morbidity and mortality, to improve symptoms, and to treat the associated diseases, the heaviest of which being diabetes [18]. Yet the problems of compliance and drug interactions are directly correlated with the number of drugs. There has been no interventional study specifically conducted in very old subjects. However, every time the results of large studies have been analyzed as a function of age, no difference in efficacy compared to younger subjects have been noted but with an increase of adverse drug even an increase of adverse drug events [3]. For example, in CIBIS II (Cardiac Insufficiency Bisoprolol Study), bisoprolol reduced overall mortality by 32% in subjects 71 years of age and older (vs. 31% before 71 years) and was as effective in these two age groups in reducing the rate of hospitalization for CHF [45]. In the Val-Heft (Valsartan Heart Failure Trial) study on systolic CHF, compared to placebo, valsartan reduced the risk of first hospitalization for CHF by 28.7% in subjects 65 years and older, with no impact on mortality, but an identical improvement in quality of life before and after 65 years of age [3].

Even statins, frequently indicated in older diabetics, could be beneficial during CHF. In one cohort of subjects aged 65 years and more, discharged from the hospital with a diagnosis of CHF, 1- and 3-year survival rates were significantly higher in patients discharged on statins [47]. Several very recent publications underscore this potentially beneficial effect, whether or not the CHF was ischemic [48-49], explained by the action of statins on endothelial functions, inflammation, and the autonomic nervous system [50-51]. However, today there has been no prospective randomized trial. The frequency of iatrogenic accidents at this age requires that precautions in prescribing and in monitoring be strictly respected, such as blood pressure monitoring and...
creatinine and kaliemia dosage in the week following the introduction or increase in dosage of an ACEI or an ARA2, or the monitoring of hydroelectrolytic balance with diuretics [8]. The ACEIs should be proposed at all stages of CHF. They are effective on 1-year survival rates, even in older subjects with borderline high blood pressure or those with kidney failure [52]. A salt-free diet is not indicated over the long term in older subjects because of the high risk of inducing anorexia and secondarily denutrition. However, it may be useful on a transitory basis during decompensation episodes, when the congestive signs are at the forefront.

4.3. Treatment of congestive heart failure in diabetics

Nor have there been studies specifically conducted in diabetics. Nevertheless, the high prevalence of diabetes in all the trials investigating CHF justifies an analysis of the results based on the existence or absence of this co-morbidity. In a recent meta-analysis of the large randomized trials on the impact of ACEIs and beta-blockers in systolic CHF, the effect of ACEIs on total mortality was the same, whether the patients were diabetic or not, with a 15% risk reduction [20]. The reduction in the relative risk of death when taking beta-blockers was not significant in non diabetics (35% vs. 23% in diabetics), but showed a much greater reduction in diabetics [20]. In the BEST study (bucindolol vs. placebo in patients with a mean age of 61 years and older with a LVEF <35%), the beta-blocker was also effective in diabetics in terms of mortality and number of hospitalizations for CHF or for all causes [46], as in the CIBIS II study [45]. The therapeutic rules are therefore the same in diabetics, with the choice of drugs possibly influenced by the existence of coronary heart disease and arterial hypertension, which are very frequently associated.

4.4. Treatment of diastolic congestive heart failure

Currently, there are no specific recommendations for managing diastolic CHF, but trials are in progress [53]. The classical drugs can be used, depending on the clinical stage of the disease. Inhibition of the renin-angiotensin-aldosterone system using ACEIs or ARA2 can theoretically improve LV compliance and endothelial function, with a beneficial action on fibrosis and LV hypertrophy over the long term [24], which nevertheless persists in more than 40% of diabetics treated with ACEIs, perhaps because of dosages that are too low [14]. In the CHARM study (in which one-quarter of the patients were 75 years old or older), candesartan has been shown to be more effective than placebo in reducing the number of hospitalizations for CHF [54], including for patients with preserved LVEF, but had no effect on mortality in this case [55]. Through their effect on the heart rate, the beta-blockers as well as calcium channel blockers can improve diastolic function by increasing LV filling time [8]. In the CHARM study, AF was associated with a higher rate of morbidity events in the group with LVEF >40% [21]. In cases of AF, maintaining or restoring sinusal rhythm may therefore be logical, but the efficacy has not yet been demonstrated. The antialdosterone diuretics theoretically exert a positive effect against myocardial remodeling and fibrosis, but their clinical efficacy has only been proven in systolic dysfunctions [24]. The loop diuretics are indicated during episodes of decompensation, when congestive signs are present [8].

5. Treatment of diabetes in congestive heart failure patients

In the latest US recommendations on the management of diabetes in the elderly subject, this point was not covered [10].

5.1. Blood glucose objectives

Through AGEs, chronic high blood glucose has a specific role in the pathophysiology of myocardial fibrosis and therefore in the compliance defect, that led to diastolic dysfunction. Theoretically, at least at an early stage, strict blood sugar control should be able to slow down LV remodeling. However, currently there is no clinical proof of this benefit [56]. In elderly subjects, setting individualized glycemic goals is a fundamental element in managing diabetes, mainly based on gerontological criteria [57]. The influence of CHF on the choice of goals is complex and probably depends on the clinical stage.

5.2. Glucose-lowering drugs

The specific effects of oral antidiabetics on cardiac function, in particular diastolic, are unknown [56]. However, several factors require great caution in the use of oral antidiabetic drugs in elderly subjects with CHF. Indeed, this is a frail population that risks frequent decompensations during any intercurrent acute disease and kidney failure, permanent or transitory, during congestive episodes. Metformin treatment can thus be complicated by lactic acidosis in cases of acute congestive heart disease. The use of glitazones in a context of potential CHF was the subject of recent recommendations [58]. The insulin-sensitizing effect of glitazones could have a major positive impact on prevention or treatment of myocarditis hypertrophy related to hyperinsulinism. However, water and salt retention is nearly always observed with this treatment, manifesting by edema and/or weight gain. This makes the risk of CHF 1.6-1.8 times higher. It is therefore recommended that glitazones not be prescribed in cases of known LV dysfunction, whatever the stage, or to stop them in case of symptoms suggestive of CHF [58].
6. What is the indication for echocardiography in the asymptomatic elderly diabetic?

There are no specific recommendations. The high frequency of cardiac abnormalities in this population is an argument for extending the echocardiography indications. To make it more cost-effective, it may be necessary to target the subjects at the greatest risk, for example, subjects with micro-or macroproteinuria. In subjects of a mean age of 60 years, it has been shown that albuminuria was significantly associated with LV function, either systolic or diastolic, even after adjustment for age, sex, BMI, systolic blood pressure, duration of diabetes, the existence of coronary heart disease, and LV mass [59]. Some studies are currently evaluating the rate of diabetes, the existence of coronary heart disease, and prevention should be emphasized: bringing blood pressure to normal levels and optimizing blood glucose control.

7. Conclusion

Age, diabetes, and arterial hypertension are risk factors for CHF. The myocardial changes observed during normal aging and during diabetes are very similar, with diabetes considered an accelerator of aging. Beyond systolic CHF, most often related to coronary disease, the most frequently encountered problem in elderly diabetics is diastolic dysfunction of the left ventricle, with preserved LVEF, aggravated by the coexistence of arterial hypertension. Few formal data are available today on the treatment of this type of CHF, and prevention should be emphasized: bringing blood pressure to normal levels and optimizing blood glucose control.

References

[7] Charbonnel B, Bouhanick B, Le Feuvre C et le groupe de travail. Systematically dosing BNP and only requesting ultrasound in cases of high levels has been proposed [14], but the validity of this measure has not yet been demonstrated. The problem then becomes interpreting the rates in very old subjects. Once the echocardiography has been performed, what should be done with the result? If there is an abnormality in the segmental kinetics, diagnosis is oriented toward coronary disease, with specific management and intensive treatment, if possible, of the associated vascular risks. If there is systolic dysfunction, even asymptomatic, treatment with ACEIs and beta-blockers should be discussed. If there is asymptomatic isolated diastolic dysfunction, no therapeutic intervention benefits from formal clinical proof [36]. More aggressive management of arterial hypertension seems logical all the same to limit the development of LV hypertrophy, an independent risk factor of morbidity and mortality [14].


