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Research Article

Nutritional Management of Chronic Heart Failure

H. Benalikhoudja, M. Abdelbaki

Cardiology and cardiovascular surgery department (EHS Dr Maouche Alger)

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Abstract:

Chronic heart failure is associated with a nutritional syndrome which is often complicated by a state of cachexia. This nutritional syndrome plays a pejorative role. When obesity is moderate, it has favorable consequences on morbidity and mortality. Nutritional syndrome results directly from two processes linked to the development of heart failure. The first results in a neuroendocrine disruption which leads to a reduction in anabolic factors and an increase in catabolizing factors, resulting in a hypercatabolic state and leading to cachexia. The second is the reduction of caloric consumption and absorption with a decrease in energy production and an increase in demand. Specific dietary intakes or supplements have been carried out with some success, but a consensus remains to be established.

Keywords: ICC, nutritional syndrome, cachexia, anabolism, catabolism, BMI.

Introduction

Chronic heart failure (CHF) is an inability of the heart to ensure a systemic flow adapted to the body's oxygen needs. The existence of a nutritional syndrome associated with CHF is a recent concept. Body mass index (BMI) is currently recognized as an important prognostic factor. Although it is known that cachectic patients have an unfavorable prognosis [1], several studies have demonstrated that a moderate BMI associated with CHF has a favorable effect on morbidity and mortality [2,3]. At an advanced stage, CHF is accompanied by cachexia resulting from a metabolic disorder with a poor prognosis.

Undernutrition worsened by age affects 16% of outpatients. It is correlated with NYHA stage, physical activity, duration and frequency of hospitalizations and mortality. Significant weight loss and a hypercatabolic state results from both neuroendocrine disturbances (imbalance between anabolic and catabolic factors), reduced caloric consumption and absorption (decreased energy production. Cachexia linked to CHF is defined as a weight loss of more than 6%, non-voluntary and not linked to depleting treatment during follow-up [4].

the wasting of peripheral muscles being correlated with the severity of the condition and contributing to exercise incapacity [5]. The catabolic process also affects the heart. A follow-up study of 37 patients shows a decrease in left ventricular mass in patients who developed cachexia, whereas it did not evolve in non-cachectic patients [6].

The aim of treatment in cases of CHF is to treat the symptoms, prevent episodes of decompensation, slow the progression of the disease, act on the nutritional syndrome to allow early diagnosis before the appearance of cachexia, and resistance to nutrition.

Mechanism of cachexia

Left heart failure leads to splanchnic hypoperfusion with ischemia of the digestive mucosa and an increase in intestinal permeability. This leads to translocation of bacteria and endotoxins (lipopolysaccharides) and elevation of pro-inflammatory cytokines. When the insufficiency progresses towards involvement of the right heart, it promotes splanchnic venous stasis. TNF-α is a powerful catabolic factor. It increases with the severity of the CHF. The production of cytokines leads to significant neuroendocrine stimulation and is associated with exercise intolerance [7]. Other cytokines, such as IL-2 and IL-6, increase during HF associated with cachexia [9, 10]. Stimulation of the renin-angiotensin-aldosterone system in CHF results in a notable increase in angiotensin II. It is a powerful anorexigenic factor, known to induce a loss of muscle mass via a modification of the level of IGF-1 (Insulin-like) Growth Factor 1). The SOLVD study showed that treatment with ACE inhibitors can reduce the appearance of cachexia by 19% [4].

Leptin (anorexigenic) would increase with the NYHA stage, but this point remains controversial. On the contrary, ghrelin levels increase in ICC. This process is considered as a compensatory mechanism for the catabolism/anabolism imbalance, intended to lower insulinemia and leptinemia.

The acceleration of the feeling of satiety has been attributed to an overstimulation of gastric peptide YY which slows gastric emptying [11]. Urinary leakage secondary to diuretic administration also contributes to worsening cardiac dysfunction [12].

Copper deficiency aggravates cardiomyopathy through mitochondrial fragmentation and myofibrillar disorganization. It severely impacts cardiac energy production and acts on contractility.

Calcium deficiency (coupled with vitamin D deficiency) affects salt sensitivity. In addition, it worsens osteopenia.

Magnesium deficiency worsens CHF, and some studies report improvement in left ventricular function with supplementation. Selenium is a major element in antioxidant defenses via glutathione peroxidase. Keshan dilated cardiomyopathy, a viral disease (coxsackie B3) triggered by selenium deficiency [13]. Myocardial lesions are of oxidative origin and associated with a glutathione peroxidase deficiency. In many other situations, similar cardiac damage associated with selenium deficiency is described, such kwashiorkor or selenium-free parenteral nutrition.

Thiamine (vitamin B1) deficiency, caused by insufficient intake and increased urinary loss from diuretics, affects energy metabolism. It is the essential cofactor of many enzymes, including mitochondrial pyruvate dehydrogenase, the key enzyme in glycolysis. Coenzyme Q10 (ubiquinone) is a key element in the production of ATP by ensuring the transfer of electrons along the mitochondrial respiratory chain. Heart failure leads to a loss of cardiac ubiquinone which can reach 50%, but also of other effectors of contraction such as carnitine and creatine [14].

Deficiency of taurine (amino acid involved in calcium homeostasis and regulation of blood pressure), folate and vitamin C have a detrimental effect on cardiac activity

The care:

The ICC and its nutritional component justify its early consideration. Unfortunately, few interventional studies highlight the adaptation of nutritional treatment to the evolution of CHF. If the general cardiovascular recommendations of learned societies (including the limitation of salt to 2 g/d) are suitable for the patient in the early phases of CHF, they are insufficient for patients engaged in a process of malnutrition.

As long as weight loss remains moderate, oral nutrition supplemented with supplements is considered, but beyond a significant loss of body mass, artificial nutrition may be necessary. Even if this approach remains controversial, some studies show that it is possible to feed even unstable patients with enteral nutrient intake [15], parenteral nutrition being reserved for patients developing digestive complications).

Before any ICC, it is necessary to measure the ingestates, measure the body compartments with the calculation of the BMI (report:

waist/hip), evaluate the albumin level [16], measure body composition by bioimpedance metry (fat mass, lean mass), take an ECG, a chest x-ray, a liver test (cytolysis, cholestasis), assessment hemostasis, FNS, blood ionogram, renal panel, BNP (B-type natriuretic peptide) and NT-pro BNP. (Precursor of BNP), measure nutritional plasma proteins: (albumin, prealbumy, transferrin), measure inflammatory plasma proteins: (reactive protein (CRP), orosomucoid, ceruloplasmin (alpha2 globulins).

Management of CHF begins with changes in lifestyle habits, replacement of Nacl with Kcl (1 to 2 g/day). A fluid intake of 1.2 liters/day (all liquids combined).) and in case of end-stage renal failure, strict fluid restriction must be observed: 750 ml/d)

It will be necessary to act on aggravating factors such as alcohol, tobacco), balance blood pressure and diabetes; act on obesity and overweight. It will also be necessary to assess weight loss (weigh yourself twice a week and note your weight). The diet must be high in protein and high in calories, and nutritional intake must be divided (4 meals and 2 snacks) [[17].

Renutrition requires:

The beginning phase requires 40-50kcal/kg/d with 1.5-2g/kg/d of protein. the dose of carbohydrates is 2.5 to 3 times the protein intake (55-60%) of the energy intake and insulin as needed.

The maintenance phase requires 30-40kcal/kg/d and 1-1.5g/kg/d of protein, the carbohydrate dose is 2.5 to 3 times the protein intake: 55-60% of energy intake up to to recover lean mass. Vitamin C supplementation is 2 – 3g/d, vit E: 600-1200IU/d, Minerals (magnesium (0.6 to 1 g/d), selenium (0.8 mg/d) d for 3 months, then 0.2 to 0.4 mg/d), Carnitine: 3 g/d, Q10: ubiquinone: 150 mg/d, Taurine: 3 g/d, Arginine: -6-8g/l: minimum dose to improve dysfunction endothelial; -18- 20g/l: maximum dose without side effects) [17] Omegas are at a dose of 3-1 g/day (EPA and DHA) according to the recommendations of the American Heart Association [18]. In case of coronary artery disease, it is advisable to give 1 g/day EPA and DHA.) If the triglyceride level is very high we can go up to: 4 g/day (EPA and DHA). Supplementation for 9 months with Ca, Mg, Zn, Cu, Se, Vit A, B1, riboflavin, B6, folate, B12, vit C, E, D, coenzyme Q10 increases the left ventricular ejection fraction by 5, 3% and a significant increase in quality of life [18]

Hawthorn (Crataegus oxyacantha) supplementation allows resistance to exercise, reduces shortness of breath and fatigue; it acts on the strength of the heart muscle (vasodilators, antiarrhythmic). Ghrelin increased cardiac mass and LV ejection fraction as well as exercise tolerance [19]. Anamoreliéne, a molecule which mimics the action of ghrelin, promotes protein synthesis rather than their degradation. The dose is 100 mg/day orally [20] The drop in GH, IGF1 and IGFBP3 in cases of CHF of ischemic origin [21], we recommend an injection of growth hormone at a rate of 10 mg/kg/day, it acts on the FE and the modification of the lipid profile (atheroma). It plays a protective role against atherosclerosis [21] Cardiac cachexia treated with high doses of GH for 1 week to 3 months showed an increase in muscle mass and physical capacities [22,23]

The intake of testosterone has an effect on the relaxation of smooth muscle. It is the same mechanism of action as nifedipine, (via) calcium channels. Testosterone has anti-ischemic, anti-inflammatory, anabolic, vasomotor effects, improves insulin sensitivity [24].

The contribution of anti-TNF agents has led to an improvement in quality of life, an increase in ejection fraction and walking distance [25, 26,27]. Sports activity such as Tai chi: improves quality of life, increases resistance to exercise and quality of sleep [28]

Conclusion

Undernutrition is therefore a frequent and negative factor in the development of CHF, in terms of morbidity and mortality. It

must be taken into consideration as early as possible as a component of the pathophysiology of CHF, in monitoring by the cardiologist and in treatment. The specific therapy of this treatment is increasingly studied, but is not yet the subject of a consensus and the recommendations remain to be clarified, especially since they must take into account both the evolution of cardiac function and that of nutritional status. Furthermore, we still have much to discover about more specific aspects of particular and complex malnutrition. If we want to prevent or at least delay the functional cardiac consequences. In recent years, doctors have become aware of the problem, as evidenced by the increase in research work. The effectiveness of early management of the nutritional component on cardiac function is beginning to be documented, but a consensus remains to be established on the appropriate measures depending on the evolution.

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