Euro Heart Congress 2020: Integrative metabolic therapeutic approach for symptomatic patients with left ventricular dilatation and reduced ejection fraction

Mila Jakovljevic
Polyclinic for Cardiovascular Diseases and Prevention, Croatia

Abstract

Introduction: Myocardial energetic has a central role in patho-physiology of heart failure. The heart is an example of specialized type 1 (red) striated muscle in that is continuously active and reliant principally on aerobic metabolism for its energy supply. However, failing of heart is by insufficient ATP supply is currently lacking. Over the last couple of years, an increasingly complex picture of mechanisms evolved that suggest that potentially metabolic intermediate and redox states could play the dominant role for signaling that eventually results in left ventricular remodeling and contractile dysfunction. In the patho-physiology of heart failure, mitochondria emerge in the cross fire defective excitation, contraction coupling and energetic demand which may provoke oxidation stress as an important upstream mediator of cardiac re-modeling and cell death. Thus further therapies may be guided towards restoring defecte ion homeostatic and mitochondrial redox shifts rather than aiming solely at improving the generation of ATP. Our hypothesis was that cardiac metabolic therapeutic approach must be integrative improving both substrate utilization and complete substrate oxidation i.e., supporting normal mecha-nism of energy production without increased generation of reactive oxygen species. Since the energy metabolism is linked to cardiac function, we accessed the effect of the inte-grative metabolic approach on the functional ability and quality of patients with heart failure and reduced ejection fraction.

Aim: The aim was to study the effects of integrative metabolic and therapeutic approach of symptomatic patients with left ventricular dilatation and reduced ejection fraction.

Methods &Results: Heart failure with reduced ejection fraction (HFrEF) happens when the left side of your hear does not pump blood out to the body as well as normal. The types of heart failure are based on measurement called ejection fraction. The ejection fraction measures how much the blood inside the ventricle is pumped out with each contraction. The left ventricle squeezes and pumps some (but not all) of the blood in the ventricle out to your body. A normal ejection is more than 55%; this means that 55% of total blood in the left ventricle is pumped out with each heart beat. Heart Failure With Ejection Fraction happens when the muscle of left ventricle is not pumping as well as normal. The ejection is 40% or less. The amount of blood is being pumped out of the heart is less than the body needs. A reduced ejection fraction can happen because the left ventricle is enlarged and cannot pump normally. Three main heart types describe heart failure according to the left ventricular ejection fraction (EF) and the differentiation between these types is important due to different demographics, co-morbidities and response to therapies:

- Heart failure with reduced ejection fraction (HFrEF): EF greater than or equal to 40% and reduced ejection fraction less than or equal to 40%.
- Heart failure with preserved EF (HFrEF): EF greater than or equal to 50%.
- Heart failure with mid-range EF (HFmrEF): EF is 41 to 49 % per European guidelines and 40 to 49% the US guidelines.

All patients with HFrEF have comitant diastolic dysfunction; in contrast, diastolic dysfunction may occur in the absence of systolic function. More than two-thirds of heart fail-ures are attributable to ischemic heart disease, COPD, hyper-tensive heart disease & rheumatic heart disease. Other pos-sible heart failure include congenital heart disease, myocardi-tis, infiltrative disease, peperutum, cardiomyopathy, human immune virus, amyloidosis, substance abuse, long standing alcohol use, obesity, diabetes mellitus, hyperthyroidism, pul-monary hypertension, constrictive pericarditis (can cause HFrEF), pulmonary embolism (can cause HF) and chemotherapies (like doxorubicin).

We investigated 33 patients with left ventricular dilatation>60 mm/and reduced ejection fraction <40% in 76% supportive complementary therapy/CST. Prior to each CST period, ther-a py was optimized for one month. CST is consisted of a 10 day session. In addition to optimization the patients were treated with carnitine, L-arginine, Magnesium, vitamin-b, co-enzyme Q-10, vitamin –c, vitamin-e, and selenium while lying for 30 minutes inside a pulsed electromagnetic field with intensity up to 30 micro teslas and inhaling negatively ionized oxygen. Before and after each CST period, patients were asked to evaluate the equality of life using the MINNESOTA LIVING with heart failure questionnaire and the visual ana-logue scale VAS, EF, LVIdD, NYHA classes were determined. Statistical analysis was based on t-test, spearman’s rank correlation coefficient WILCOXONS signed ranks test. The longest period observation was 122 months.

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After administrating metabolic supportive therapy, a statistically significant improvement was noticed in particular items of MLHFQ in emotional and physical dimensions. The values of VAS and EF are increased where as the values of NYHA and LYID decreased significantly.

**Conclusion:** HFpEF is not a true cardiac disease, but rather a systemic disease with morphological and functional heterogeneity. Clinical manifestations HFpEF appear to be the consequences of other underlying pathologies not are insufficiently treated. HFpEF emerges as a model with pro-inflammatory cardiovascular and non-cardiovascular co-existing co morbidities, frequently present in the elderly, constituting a potential geriatric syndrome. Integrative metabolic therapeutic approach significantly improved the functional capacity and quality of life in the patients of HFpEF

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