

## THE PHARMA INNOVATION - JOURNAL

# Dyslipidemia and Heart Failure: The Analysis of 2-years Observation

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Heart failure (HF) is important medical and social problem. The rate of new cases of heart failure is 10 per 1000 individuals aged over 65 years, 52.3% of all heart failure patients die within five years of their initial diagnosis. Lipid metabolism is modified risk factor of cardiovascular diseases. The aim of our study was to evaluate the dynamic of lipids metabolism in patients with HF. We observed of 35 patients with HF II-III FC (NYHA). Our results showed that nobody observed persons had target LDL-C level. Thus, the lipid-lowering strategy was imperfect in all observed patients with HF. This direction of therapy should be revised (active life-style modification, raising of statines doses or additional prescription other lipid-lowering medicaments).

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**Keyword:** heart failure, treatment, dyslipidemia

### 1. Introduction

Cardiovascular disease (CVD) due to atherosclerosis of the arterial vessel wall and to thrombosis is the foremost cause of premature mortality and of disability-adjusted life years (DALYs) in world, and is also increasingly common in developing countries [1]. In the European Union, the economic cost of CVD represents annually ~€192 billion in direct and indirect healthcare costs [1]. The main clinical entities are coronary artery disease (CAD), ischaemic stroke, and peripheral arterial disease (PAD).

All CVD resulted in chronic heart failure (HF). As a result of the aging population and medical advances resulting in falling mortality for ischemic heart diseases, the incidence of heart failure is likely to continue to increase in the years to come. This will pose a challenge to both funders and service providers [2, 3]. The rate of new cases of heart failure is 10 per 1000 individuals aged over 65 years [4]. In addition to

considerable morbidity, heart failure is also associated with an unfavorable prognosis and thus high disease-related mortality. Overall, 52.3% of all heart failure patients die within five years of their initial diagnosis [4].

Lipid metabolism can be disturbed in different ways, leading to changes in plasma lipoprotein function and/or levels. This by itself and through interaction with other cardiovascular (CV) risk factors may affect the development of atherosclerosis. Elevation of total cholesterol (TC) and low-density lipoprotein-cholesterol (LDL-C) has received most attention, particularly because it can be modified by lifestyle changes and drug therapies. The evidence showing that reducing TC and LDL-C can prevent CVD is strong and compelling, based on results from multiple randomized controlled trials. TC and LDL-C levels continue therefore to constitute the primary targets of therapy [5].

The aim of our study was to evaluate the dynamic of lipids metabolism in patients with HF.

## 2. Material and Methods

We observed of 35 patients with HF II-III FC (NYHA). The diagnosis was verified by laboratory and instrumental methods according to European Society of Cardiology recommendations (2013, 2014). All patients were randomized into two groups: 14 patients received of basic therapy medications (diuretics, RAAS blocker, beta-blocker, statine and acetylsalicylic acid); rest of 21 patients (second group) additionally received of Ivabradine (Coraxan, Les Laboratoires Servier, France) in doses 5 or 7, 5 mg twice a day (due to heart rate). The levels of total cholesterol, triglycerides (TG), cholesterol of low density lipoproteins (LDL-C), cholesterol of high density lipoproteins (HDL-C) were examined. All patients were examined on randomization, after 1 and 2 years of observation. The study was performed in accordance with the Helsinki Declaration and Good Clinical Practice

Guideline. The study was approved by the local ethics committee and written informed consent was obtained from all patients. Statistical analyses were performed using the Statistica 6.1 (Stat Soft, Tulsa, OK, USA). Statistical significance was assumed at  $p < 0.05$ .

## 3. Results and Discussion

In cohort of observed patients 30 (85.7%) were males. The middle age was 64,  $6 \pm 6$ , 83 years. The average Body Mass Index (BMI) is  $29.79 \pm 0.91$  kg/m<sup>2</sup>. Only 2 patients had normal body weight. 19 persons were overweight (54.29%). 11 patients had obesitas of I degree, 3 – II-nd.

Due all period of observation we didn't found changes in main values of lipids' metabolism (see table 1), except HDL-C level, which significant increased at the end of 1 year of treatment: from  $1.16 \pm 0.07$  mmol/l to  $1.28 \pm 0.06$  mmol/l ( $p = 0.01$ ). Similar results were received during detail analysis of two subgroups of treatment (see table 2 and 3).

**Table 1:** The Dynamics of Lipidogram in Patients with HF (All Observed).

Value, mmol/l	Randomisation	After 1 year	After 2 years
Total Cholesterol	$4.71 \pm 0.22$	$4.78 \pm 0.22$ $p = 0.91$	$4.84 \pm 0.22$ $p = 0.64$
Triglycerides	$1.78 \pm 0.24$	$1.64 \pm 0.18$ $p = 0.18$	$1.61 \pm 0.17$ $p = 0.39$
LDL-Cholesterol	$2.74 \pm 0.17$	$2.83 \pm 0.18$ $p = 0.18$	$3.06 \pm 0.19$ $p = 0.11$
HDL-Cholesterol	$1.16 \pm 0.07$	$1.28 \pm 0.06$ $p = 0.01$	$1.25 \pm 0.06$ $p = 0.18$

**Table 2:** The Dynamics of Lipidogram in Patients with HF (Group with Ivabradine).

Value, mmol/l	Randomisation	After 1 year	After 2 years
Total Cholesterol	4.37±0.29	4.65±0.28 p=0.38	4.78±0.29 p=0.21
Triglycerides	1.35±0.13	1.43±0.15 p=0.44	1.49±0.21 p=0.36
LDL-Cholesterol	2.59±0.23	2.85±0.24 p=0.17	3.04±0.28 p=0.06
HDL-Cholesterol	1.16±0.09	1.27±0.08 p=0.07	1.27±0.08 p=0.36

**Table 3:** The Dynamics of Lipidogram in Patients with HF (Group with Basic Treatment).

Value, mmol/l	Randomisation	After 1 year	After 2 years
Total Cholesterol	5.20±0.28	4.97±0.38 p=0.55	4.94±0.33 p=0.62
Triglycerides	2.41±0.54	1.95±0.39 p=0.06	1.80±0.54 p=0.22
LDL-Cholesterol	3.06±0.23	2.80±0.29 p=0.54	3.03±0.20 p=0.83
HDL-Cholesterol	1.15±0.09	1.28±0.08 p=0.08	1.23±0.08 p=0.21

Treatment targets of dyslipidaemia are primarily based on results from clinical trials. In nearly all

lipid-lowering trials the LDL-C level has been used as an indicator of response to therapy.

Therefore, LDL-C remains the primary target of therapy in most strategies of dyslipidaemia management. The most recent Cholesterol Treatment Trialists' Collaboration (CTT) meta-analysis of several trials involving >170 000 patients confirmed the dose-dependent reduction in CVD with LDL-C lowering. Every 1.0 mmol/L (~40 mg/dL) reduction in LDL-C is associated with a corresponding 22% reduction in CVD mortality and morbidity [6].

According modern recommendation of CVD prevention of European Cardiology Society all observed patients are very high risk persons. For patients with very high CV risk, the treatment target for LDL-C is <1.8 mmol/L (less than ~70 mg/dL) or a  $\geq 50\%$  reduction from baseline LDL-C.

Our results showed that nobody observed persons had target LDL-C level.

#### 4. Conclusions

Thus, the lipid-lowering strategy was imperfect in all observed patients with HF. This direction of therapy should be revised (active life-style modification, raising of statines doses or additional prescription other lipid-lowering medicaments).

#### 5. References

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