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ЖУРНАЛ ГЕПАТО-ГАСТРОЭНТЕРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

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IVABRADINE WITHOUT CLINICAL HEART FAILURE IN STABLE CARDIOVASCULAR DISEASE

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ANNOTATION

Heart beat is a recognized indicator cardiovascular system risk. According to research conducted in the past, the anti-arrhythmic medicine ivabradine has the potential to enhance the outcomes for stable cardiovascular disease, failure of the left ventricle, and at beats per minute of at least 70.

Key words: Ivabradine, coronary artery disease, and the risk of cardiovascular disease.

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ПРИМЕНЕНИЕ ПРЕПАРАТА ИВАБРАДИНА ПРИ СТАБИЛЬНОМ СЕРДЕЧНО-СОСУДИСТОМ ЗАБОЛЕВАНИИ БЕЗ КЛИНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ

АННОТАЦИЯ

Сердцебиение является общепризнанным показателем риска сердечно-сосудистой системы. Согласно исследованиям, проведенным в прошлом, антиаритмический препарат ивабрадин потенциально может улучшать исходы стабильного сердечно-сосудистого заболевания недостаточности левого желудочка и при частоте ударов не менее 70 в минуту.

Ключевые слова: ивабрадин, ишемическая болезнь сердца, риск сердечно-сосудистых заболеваний.

Introduction: When the body's metabolic functions create an undue burden on the heart because it cannot pump enough blood, A disease that is referred to as heart failure (HF) can develop. According to the most recent set of guidelines offered by the American College of Cardiology Foundation and the American Heart Association (ACCF/AHA), A defect, either structural or functional, in the ability of the ventricles to fill or to discharge blood. can cause the complicated medical condition known as heart failure (HF) [1-3, 15-20]. A deterioration in the structure or function of ventricular filling or blood ejection can result in heart failure.1 High prevalence and poor clinical outcomes

are related with HF, a serious public health issue. Around 5 million Americans are impacted in the U.S., and more than 500,000 new cases are identified yearly. 2,3 HF is the most common reason for hospitalization in people over 65. 4 Each year, more than 1 million patients with HF as their primary diagnosis are hospitalized, costing Medicare more than \$17 billion overall [4,8-11]. 4 Despite the fact that medical treatment has led to a substantial improvement in outcomes, admission rates following HF hospitalization are still high, with more than 50% of patients returning within six months of discharge. Any disorder that causes a change in left ventricular (LV) structure or function can put a

patient at risk for developing heart failure (HF). The primary cause of HF is coronary artery disease (CAD). Ejection fraction (EF) of 50% or more is normal or preserved in about 50% of HF patients [12-14]. Patients who have been diagnosed with heart failure can be separated into two distinct groups: those who have heart failure but have preserved ejection fraction and those who do not. (HFpEF) and those who have heart failure with decreased ejection fraction (HFrEF; also known as systolic HF).: (HFpEF; formerly diastolic HF). HFpEF is defined by aberrant ventricular relaxation and inadequate ventricular filling, whereas HFrEF is characterized by reduced myocardial contractility and inadequate ventricle emptying. HFrEF is also characterized by inadequate ventricular emptying. Dyspnea, exhaustion, and indicators of volume overload, such as peripheral edema and pulmonary rales, are among the primary clinical symptoms of HF.

The most widely used technique for determining the severity of functional restrictions in HF patients is the New York Heart Association (NYHA) functional classification scheme [5-6]. Although it can be challenging to determine a person's prognosis, a poor prognosis is associated with the onset of clinical heart failure (HF). Patients who have symptoms when at rest (NYHA class IV) have an annual death rate that ranges from 30% to 70%, whereas patients who experience symptoms while engaged in moderate activity (NYHA class II) only have a mortality rate that ranges from 5% to 10%.

A higher heart rate is a known sign of higher cardiovascular risk in individuals, including healthy persons as well as individuals who currently suffer from cardiovascular disease. This is the case regardless of whether the individual has cardiovascular disease or not. By suppressing the heart rate, ivabradine can lower heart rate while having no impact on blood pressure or the function of the left ventricle during systole. If there is pacemaker current in the sinoatrial node. It has been established that it helps people with stable angina pectoris symptoms by lowering ischemia. This helps those who have angina pectoris. Patients who suffer from systolic heart failure may find that ivabradine helps them get better results. On the other hand, a post hoc analysis found that ivabradine improved outcomes for patients with a heart rate of 70 beats per minute or higher, particularly in those who suffered from angina. Patients who had been given a diagnosis of angina were especially likely to experience this issue. These findings were consistent with what was revealed during the trial in which ivabradine was tested on patients suffering from coronary artery disease and left ventricular systolic dysfunction. We conducted a large-scale randomized controlled study of ivabradine in individuals with stable coronary artery disease who did not have any clinical indications of heart failure in order to give more data in support of these findings. The title of the clinical trial was An Investigation on the Morbidity and Mortality Benefits of the I-F Inhibitor Ivabradine in Patients Suffering from Coronary Artery Disease (SIGNIFY).

Relevance. Ivabradine was added to the usual background medicine in individuals who had coronary artery disease that was sta-

ble but did not have any clinical signs of heart failure, to lower heart rate. This was done in order to achieve the desired outcome of lowering the heart rate. Ivabradine treatment did not result in an improved prognosis for the patients, despite the fact that it was given to them.

Purpose of Study. In the participants of our trial who had stable coronary artery disease and no clinical signs of heart failure, we investigated the efficacy of adding ivabradine to the standard medical therapy that is recommended by guidelines. These participants were included because they met the criteria for the trial. We did not find any evidence that ivabradine was beneficial in lowering the risk of cardiovascular events.

Methods. In addition to the standard clinical trial, we also carried out an inquiry using ivabradine that was controlled by randomization, blinding, and placebo. A total of 19,102 patients who met the following criteria for background therapy were included in the study: they had stable coronary artery disease, a heart rate of at least 70 beats per minute is considered to be abnormal and no clinical signs of heart failure (includes 12,049 individuals with angina that prevented them from participating in strenuous physical activity (class II on the scale used by the Canadian Cardiovascular Society, which ranges from I to IV, with higher classes suggesting greater restrictions on physical activity). Participants in the study were actual patients. Patients were given either a placebo or ivabradine, both of which could be taken up to twice a day in quantities of up to ten milligrams each. The dosage was selected with the goal of achieving a heart rate in the range of beats per minute of 55 to 60 when using the medicine. The major composite served as the endpoint. Measure that counted either myocardial infarction that did not cause mortality or cardiovascular causes. This measure was chosen as the primary end point since it was easier to analyze.

Results. Individuals that are suffering from coronary artery disease that has been stable (19,102) who had a heart rate of at least 70 beats per minute, we added ivabradine to conventional background therapy in a blinded, randomized, placebo-controlled experiment (included 12,049 individuals with angina that limited their ability to participate in physical activity [class II on the Canadian Cardiovascular Society scale, which spans from I to IV, with higher classes indicating greater limitations on physical activity]. Your heart rate ought to be somewhere in the range of 55 and 60 beats per minute. Patients were given either ivabradine in doses of up to 10 mg two times day or a placebo. Patients took 20 milligrams four times day. The study's primary endpoint was a composite measure that included both fatal and nonfatal myocardial infarctions.

Conclusion. Ivabradine was added to the standard background medicine in order to slow a stable case of coronary artery disease patients' heart rate and did not have any clinical signs of heart failure. This was done in order to achieve the desired outcome of decreasing heart rate. The patients did not see an improvement in their prognosis as a result of the treatment of ivabradine.

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