

Use of Beta-Blocker in Acute ST-Elevation Myocardial Infarction

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Abstract

This paper reported beta-blocker use in 21 STEMI patients over four years. The patients were between 50 - 65 years of age presenting with anterior, lateral, and inferior STEMI (ST-Elevation Myocardial Infarction). Seven of the patients were female, and 14 were male. They presented to an emergency room of a rural hospital that did not provide emergency percutaneous coronary angioplasty/stenting (PTCA/stenting). The hospital is about 70 minutes from a facility that provided PTCA/ stenting—all the patients presented with typical angina chest pain with ST elevation. They are hemodynamic stable. Most patients received Lopressor 35 mg IVP, with one receiving 115 mg in a 5 mg increment. They were chest pain-free and hemodynamically before leaving the ER for the transfer for PTCA/stent. The results demonstrated that beta-blockers are effective in relieving pain in STEMI patients. Further study is needed to determine its efficacy, safety, and how to use it.

Keywords

Beta-Blocker, Acute Myocardial Infarction

1. Introduction

Myocardial infarction (MI) occurs once every 40 seconds in the US, with an estimated annual incidence of 605,000 new cases and 200,000 recurrent cases [1] [2] and a prevalence of 3.0% for US adults aged 20 years and older during 2013-2016 [1]. ST-Elevation Myocardial Infarction (STEMI) remains a significant healthcare burden in the United States, with ≈750,000 cases annually [1] [2]. STEMI affects 500,000 Americans every year [3], for whom timely access to percutaneous coronary intervention (PCI) is critical to achieving better outcomes [4]. Due to the resource intensity of the procedure, however, only 37 per-

cent of all acute care adult hospitals in the United States offer any PCI lab capability [5]. In addition, less than 12 percent (574 of 4931) of all hospitals offer PCI for a high volume of STEMI patients (>40 primary PCI annually), with even fewer of these supporting round-the-clock availability [6]. This paper is based on retrospective and observational data. It is not planned research. Therefore, there is no control group or organized statistics. The author did not intend to publish the paper and keep a simple case count for professional interests. The author did not have access to the patient's medical records when he planned to write this manuscript due to changing jobs.

2. Results

The study used retrospective data from patients between 50 and 65 years of age presenting with anterior, lateral, and inferior STEMI from a rural community hospital over four years, which did not have the capability for percutaneous coronary angioplasty (PTCA)/stent. The patients had to be transferred to a facility 70 miles away, which provided the emergency PTCA/stent service. The data collection stopped at the end of 4 years after the hospital started emergency PTCA/stent for STEMI. Seven of the patients were female, and 14 were male. They presented to an emergency room of the rural hospital. The patients presented with typical angina chest pain with STEMI. They are hemodynamic stable, with heart rates of 80 to 100 beats per minute and systolic blood pressure between 170/95 to 150/80 mmHg. Most of the patients received Lopressor 35 mg IVP, with one receiving 115 mg in a 5 mg increment under the instruction of the cardiologist at the bedside. They were chest pain-free and hemodynamically before leaving the ER for the transfer for PTCA/stent. Intravenous beta-blockers were also used in non-STEMI patients; similar results were not presented or discussed in this paper.

The case receiving 115 mg of Lopressor was a 61-year-old male without significant past medical history and had normal functional status in his everyday life. He presented to the ER 30 minutes after 10/10 retrosternal chest pain, radiating to the left shoulder and arm and associated with shortness of breath. The patient is very anxious and unable to provide a detailed history. His blood pressure is 160/80 mmHg. His heart rate is 85 bpm. Oxygen saturation is 94%. The lungs are clear. His electrocardiogram (EKG) showed a 5 mm tombstone ST elevation II, III, and AVF consistent with acute inferior wall MI. Lopressor IVP at 5 mg with a total dose of 115 mg over 35 minutes under the instruction of a cardiologist, who was present at the bedside. His chest pain improved with each dose of the medication and eventually totally resolved. His EKG remained unchanged, with tombstone ST elevation. His blood pressure is 120/70 mmHg, and his heart rate is 62 bpm. He was transferred to a hospital 70 miles away, which provides emergency PTCA/stent. When he arrived at the catheterization laboratory of the referred facility, he remained chest pain-free and hemodynamically stable. He was talking and joking with the people in the catheterization labora-

tory. His EKG still demonstrated a 5 mm tombstone ST elevation in the inferior leads. Cardiac catheterization showed a 100% ostial lesion of the right coronary artery, which received a PTCA/stent. It resulted in the resolution of the ST elevation of his EKG. He made a complete recovery.

3. Discussion

The data presented demonstrated and, at least, suggested that beta-blockers could provide symptom relief and that they are safe. At the early stage of the author's practice as a cardiologist, he had given patients nitroglycerine, morphine, and Lopressor 5 mg intravenous push X3 as recommended. It seemed the beta-blocker was more effective than nitroglycerin and morphine, which might lower the blood pressure and limit the use of beta-blockers. These findings lead to a literature search. It had been reported that there was a significant catecholamine surge in acute myocardial infarction, which can cause damage to the myocardium [7]-[9]. Beta-blockers may decrease this damage to the myocardium [7]. It has been demonstrated that beta-blockers have morbidity and mortality benefits in patients with acute coronary syndromes [10]. There were no systematic studies on the efficacy, safety, and dose of beta-blockers in treating acute coronary syndrome. Due to the facts above, the author of the paper started to give the patients beta-blockers as described based on chest pain and hemodynamic tolerance of each patient in this study.

During the last 30 years, PTCA/stent has become more available for treating acute myocardial infarction, especially STEMI. Beta-blocker is not routinely used in the critical management of acute coronary syndrome/acute myocardial infarction on the presentation of STEMI before the PTCA/stenting. The current paper raises interest in beta-blockers' role in managing acute coronary syndrome.

Treatment goals for acute coronary syndrome/STEMI include eliminating the symptoms and lowering the damage to the myocardium. No doubt, opening the stenotic or occluded artery is an essential treatment that causes ischemia and triggers a cascade of events. The overstimulation of beta-receptors also plays a significant role in the symptoms, such as chest pain, increase in blood pressure and heart rate, and damage to the myocardium. It has been demonstrated in Takotsubo syndrome, which may present with chest pain, ST elevation (to a lesser extent) [11] [12], increased cardiac enzymes, regional wall motion abnormalities, and a global decrease in left ventricular systolic dysfunction without significant stenosis in the coronary artery. It is associated with a catecholamine surge, suggesting the catecholamine surge may contribute to chest pain, myocardium damage, and ST elevation in STEMI. It may be the reason why the beta-blocker used in acute coronary syndrome relieves chest pain and provides morbidity and mortality benefits since it may decrease the damage to the myocardium [13].

Since the PTCA/stent has become widely available, the potential benefits of

the beta-blocker have been overshadowed. Especially emergency/urgent PTCA is not available in many hospitals. The acute IV use of beta-blockers may benefit those patients. Even in the hospitals providing emergency PTCA/stent, concurrent use of beta-blockers in the ER may help the patient since overstimulation of beta-receptors may still cause damage to the myocardium after opening the occluded artery. In this report, patients received higher doses (115 mg total) of Lopressor, likely due to an acute catecholamine surge during the events. The high dose of the beta-blocker used in the study was only applied to patients with acute coronary syndrome.

There are concerns about the use of beta-blockers in the presentation of acute coronary syndromes, especially STEMI. In this study, the patients with definitive diagnoses of STEMI were hemodynamically stable at presentation. There were no cases of STEMI being hemodynamically unstable during those four years, which need to be excluded from the study. It suggests that the patients presented with true STEMI are younger and healthier, which may require further study. Furthermore, the patients presented with CHF, hypotension, bradycardia, and atrioventricular node block should not receive beta-blockers. There is another concern that the right coronary artery stenosis/occlusion may cause right ventricular infarction, leading to hypotension, bradycardia, and AV block. The beta-blocker may make the situation worse. In the current study, a case receiving 115 mg Lopressor had occluded right coronary artery. He did not have any complications related to right ventricular infarction. In other cases, the right coronary artery stenosis/occlusion demonstrated by cardiac catheterization (not included in this report) did not have complications. The right ventricular infarction during right coronary stenosis/occlusion is an uncommon possibility. If it occurs, a Beta-blocker will be contraindicated. If beta-blockers improve myocardial ischemia, the chance of right ventricular infarction may decrease. In addition, the author has not encountered a case of right ventricular infarction. We should not avoid the significant benefit of beta-blockers due to a remote possibility. In addition, I have treated NSTEMI and unstable angina in about 100 cases since I started managing STEMI in 2007, as described in this paper. It has been very effective, with a nearly 100% success rate without a single case of complication. Again, this is retrospective and observational data without detailed information about the patients and no statistics due to the reasons above.

4. Conclusion

The use of beta-blockers in STEMI at presentation to the ER may provide quick symptom relief and potentially decrease damage to the myocardium, resulting in long-term mortality and morbidity benefits. The dose of beta-blockers will be dependent on the response of each patient. This conclusion may apply to other sub-groups of acute coronary syndrome, such as unstable angina and NSTEMI. Further investigation is needed to verify the study's results and demonstrate its safety and efficacy, including how to use it, due to its retrospective and observational nature.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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