folia medica

Folia Medica 65(6):915-921
DOI: 10.3897/folmed.65.e102777

Original Article

The Role of Postoperative Trimetazidine Therapy in On-Pump Coronary Artery Bypass Surgery

Kamen Stanev[1], Antonia Yaneva[2], Asen Ivanov[1], Todor Gonovski[1]

[1] *Clinic of Cardiosurgery, St George University Hospital, Plovdiv, Bulgaria*

[2] *Department of Medical Informatics, Biostatistics and E-learning, Faculty of Public Health, Medical University of Plovdiv, Plovdiv, Bulgaria*

Corresponding author: Kamen Stanev, Clinic of Cardiosurgery, St George University Hospital, 66 Peshtersko Shose Blvd., 4000 Plovdiv, Bulgaria; Email: kkamen_st@abv.bg; Tel.: +359 32 602800

Received: 28 Feb 2023 ♦ **Accepted:** 18 Apr 2023 ♦ **Published:** 31 Dec 2023

Citation: Stanev K, Yaneva A, Ivanov A, Gonovski T. The role of postoperative trimetazidine therapy in on-pump coronary artery bypass surgery. Folia Med (Plovdiv) 2023;65(6):915-921. doi: 10.3897/folmed.65.e102777.

Abstract

Introduction: Coronary artery bypass surgery remains the gold standard in the treatment of patients with ischemic heart disease. However, the increased oxidative stress caused by the release of free radicals during the ischemia-reperfusion time is a well-known pathophysiological process during and after coronary revascularization procedures. It may lead to reversible and irreversible myocardial injury.

The focus of this prospective single-blinded randomized controlled trial is to investigate and analyze the effectiveness of the drug trimetazidine on reducing postoperative myocardial ischemia-reperfusion injury.

Aim: We evaluated the effects of trimetazidine on reactive oxygen species that may arise from myocardial ischemia-reperfusion period or systemic inflammation after coronary artery bypass grafting (CABG) surgery.

Materials and methods: The study included 90 patients divided into two groups who underwent elective coronary artery bypass surgery between March 2018 and October 2018. The patients in one of the groups received 35 mg trimetazidine twice daily as soon as they were extubated. The remainder of the pharmaceutical therapy was the same for all participants. Preoperative and postoperative levels of several blood-based biochemical markers including malondialdehyde (MDA), creatinine kinase-MB fraction (CK-MB) and high-sensitivity troponin T (hs-TnT) were measured. The data was classified and analyzed by the timing of sample collection.

Results: The results indicate that postoperative trimetazidine medication reduces MDA production, resulting in reduced oxidative stress and improved cardiac cell protection via antioxidant status augmentation. The follow-up was 6 months after the surgery. The Minnesota Living with Heart Failure Questionnaire was used to assess the quality of life of patients, and the results were excellent.

Conclusions: Postoperative trimetazidine therapy leads to improvement of the myocardial cell metabolism and thus reduction in post CABG ischemia-reperfusion injury.

Keywords

coronary artery bypass grafting, oxidative stress, postoperative results, trimetazidine

INTRODUCTION

Coronary artery disease (CAD) is one of the leading causes of death worldwide, including in Bulgaria, accounting for 66% of all ischemic heart disease-related deaths, including acute myocardial infarction. It is difficult to pinpoint the precise point at which human civilization became aware of the coronary artery disease. It is common knowledge that Leonardo da Vinci was the first to study the coronary arteries. The first person to claim that the reduced blood flow through the coronary arteries is the cause of CAD was Friedrich Hoffmann (1660–1742).[1] The idea that a blockage in a coronary artery's blood flow causes a myocardial infarction was first put forth by Adam Hammer in 1876.[2]

Direct identification of stenotic and occlusive atherosclerotic lesions in coronary arteries during an individual's lifespan became possible after Sones and Shirley at the Cleveland Clinic developed coronary angiography in the early 1960s.[3] This was definitely a watershed moment in cardiovascular care history.

The days when Dr Lillehei and his surgical team performed the first open-heart surgery utilizing controlled cross-circulation and Dr Dennis et al. used the first pump oxygenators are long gone thanks to Dr. Gibbon's invention of the heart-lung machine, which was recognized as one of the biggest inventions of the mid-20th century. This marked the beginning of the modern era of heart surgery and the treatment of coronary artery disease.[4-6]

The first reported arterial graft used in the treatment of CAD was the left internal mammary artery (LIMA) anastomosed to the left anterior descending (LAD) coronary artery done in 1964 by Kolesov in Leningrad while Favaloro in 1967 became the first surgeon to perform coronary artery bypass grafting (CABG) surgery with the saphenous vein (SVG) as a conduit.[7,8] Energy metabolism disorders are the common pathological basis of many chronic diseases including the cardiovascular ones.[9] Recent studies suggested that the development of atherosclerosis and CAD is accompanied by energy reduction in the mitochondria, which in fact are the main energy producers and cellular metabolism regulators.[10,11]

Mitochondrial dysfunction can directly promote cell death, inflammation, and oxidative stress and ultimately modify normal cell metabolism.[12] This pathophysiological change can be seen in the cardiac cell homeostasis as well, occurring during the reperfusion injury processes in variety of conditions such as angina, myocardial infarction, as well as in CABG surgery, percutaneous coronary interventions (PCI), etc.

The overproduction of free radicals, such as superoxide anion and hydrogen peroxide, causes myocardial reperfusion injury. These free radicals are removed by a series of scavenger enzymes and antioxidants in the cardiac cell.[13,14]

The cellular damage can be either reversible or irreversible depending on a variety of conditions such as duration of the ischemia, the existence of collateral circulation, the rapidity of restoration of physiological pH at the time of reperfusion, etc.[15,16]

Regardless of the fact that complete revascularization is achieved, a significant percentage of patients who have undergone CABG surgery or PCI will experience a recurrence of anginal symptoms.[17-19]

Therefore, optimal post-procedural medical therapy will remain a substantial part of the antianginal treatment. Beta-blockers and nitrates are first-choice anti-ischemic agents, while nicorandil, ivabradine, ranolazine, and trimetazidine are recommended as second-line treatment drugs.[20-22]

Trimetazidine is a well-known anti-ischemic metabolic modulator. The manifestation of its cardio-protective action is by modulating the cardiac metabolism through inhibition of long-chain 3-ketoacyl-CoA thiolase activity resulting in free fatty acid oxidation reduction and shifting substrate utilization from fatty acid to glucose without affecting the hemodynamics.[23] Therefore, it is an excellent complementary drug to the CAD treatment improving the endothelial function and vasodilatory effect on coronary arteries.[24] It also has short- and long-term beneficial effects in diabetes patients by decreasing the fasting glucose plasma levels without affecting the lipid file.[25]

AIM

The aim of our study was to investigate the effects of trimetazidine on reactive oxygen species that may arise from myocardial IR or systemic inflammation after CABG surgery. For this purpose, a number of laboratory markers, including CPK-MB, TrT, and MDA, as a trace of oxidative stress, and functional indicators were measured in a six-month period after surgery.

MATERIALS AND METHODS

The study was approved by the local medical Ethics Committee and all the patients gave their informed consent.

The study design was a prospective, single-blinded, randomized controlled trial. A total of 93 patients underwent elective isolated CABG surgery at our center between March 2018 and October 2018. Of these, 2 patients developed acute renal failure requiring continuous central veno-venous hemodiafiltration and one patient required prolonged mechanical ventilation (>48 h) and were excluded from the trial.

The final study cohort comprised 90 patients **(Table 1)**, preoperatively divided into 2 groups: group 1, the study group: 34 male and 11 female patients (n=45), who received 35 mg of trimetazidine twice daily immediately after extubation, and group 2, the control group: 33 male and 12 female patients (n=45), who were administered placebo. The remaining medical therapy was identical to all of the participants: acetylsalicylic acid (ASA) 100 mg daily, rosu-

vastatin 10 mg daily, metoprolol 50 mg twice daily, perindopril 5 mg daily, and gliclazide 60 mg daily.

The exclusion criteria included emergent or redo-operation, severe left ventricular dysfunction with left ventricle ejection fraction (LVEF) below 40%, permanent atrial fibrillation (AF), renal or liver dysfunction, pulmonary disease, and insulin-dependent diabetes.

Series of pre- and postoperative assessments were obtained for all patients such as: transthoracic echocardiogram: 12 hours before the operation, 1 hour after the operation, on discharge, and six months after the CABG procedure **(Table 2)**.

The CK-MB **(Table 3)** and hs-TrT **(Table 4)** blood levels were examined 12 hours prior to, 12 hours after the operation, and 6 months after the procedure.

The blood samples for the MDA **(Table 5)** evaluation were taken 12 hours prior to surgery and 12 hours, 1 and 6 months after the surgery.

To minimize the impact of the surgical team members on the results, all surgical procedures were performed by the same surgical team.

Operative technique

All patients were operated using standard cardiopulmonary bypass (CPB) technique, and the myocardium was protected using intermittent cold-blood cardioplegic solution, administered in retrograde and antegrade fashion. After a full median sternotomy, the LIMA was harvested by a no-touch technique as a pedicled graft and treated with papaverine solution. The SVG was harvested from both lower legs simultaneously with the LIMA. After harvesting the conduits, the patient was heparinized (300 U/kg), and CPB initiated in a standard fashion. The aorta was clamped and the cardioplegic solution administered. Distal anastomosis was performed: SVG to the right coronary artery (RCA) and the circumflex artery (RCX), and the LIMA to the LAD. The aorta was declamped and after that partially clamped with Satinsky clamp to perform the proximal SVG anastomoses to the RCA and RCX. After the end of the procedure and CPB termination, the heparin was fully reversed using protamine.

RESULTS

The demographic and preoperative characteristics of the patients are summarized in **Table 1**. The study group's mean age at the time of surgery was 62.97±9.45 years, with 34 male patients (75.6%); the control group's mean age was 64.18±6.54 years, with 33 male patients (73.3%). All patients had three-vessel CAD. Eleven patients (24%) in the study group had previously been deployed a total of 16 stents, while 14 patients (31%) in the control group had previously been implanted a total of 19 stents. Overall logistic euroSCORE II (European System of Cardiac Operative Risk Evaluation) was low for both groups: 1.6±0.4 and 1.5±0.7, respectively.

Every patient received coronary artery bypass grafting which was performed using CPB. There was no significant difference between the 2 groups regarding sex, age, BMI, duration of the CPB, and aortic cross-clamp time or the total number of grafts. There were no hospital deaths.

Table 1. Demographic and preoperative characteristics

Characteristic	Group 1, study group (n=45)	Group 2, control group (n=45)	*P*
Sex (male) (%)	34 (75.6%)	33 (73.3%)	>0.05
Age (years)	62.97±9.45 (55–72)	64.18±6.54 (52–75)	>0.05
BMI	29.3 (23.1–32.4)	29.6 (24.2–31.9)	>0.05
Diabetes type II (%)	45 (100%)	45 (100%)	>0.05
Arterial hypertension (%)	68.8% (n=31)	64% (n=29)	>0.05
Hyperlipidemia (%)	66.6% (n=30)	68.8% (n=31)	>0.05
Elective (n)	100% (n=45)	100% (n=45)	>0.05
Patients with stent deployment (n)	24% (n=11)	31% (n=14)	>0.05
Total number of stents deployed (n)	16	19	>0.05
EuroSCORE II	1.6±0.4	1.5±0.7	>0.05

Table 2. Intraoperative data

Characteristic	Group 1, study group (n=45)	Group 2, control group (n=45)	*P*
CPB time (min)	43.7±3.3 (38–51)	43.5±3.8 (38–55)	>0.05
Ao Cross-clamp time (min)	27.4±2.5 (22–31)	28.2±2.7 (21–34)	>0.05
Number of bypass grafts performed (n)	135	135	>0.05
Venous bypass grafts (n)	90	90	>0.05
Off-pump procedures (n)	nil	nil	-

There were no significant differences between the two groups in the CK-MB, hsTrT and MDA baseline values or the preoperative hemodynamic data **(Tables 3, 4, 5, 6)**.

In summary, the data suggests that postoperative trimetazidine treatment does not lead to improvement in the LVEF, but leads to decrease in MDA production, and therefore to oxidative stress reduction and better myocardial cell protection by antioxidant status augmentation **(Table 7)**.

Regarding the overall hospital stay, there was a trend (with marginal statistical significance) for a shorter hospital stay in the study group.

Table 3. LVEF

Characteristic	Group 1, study group (n=45)	Group 2, control group (n=45)	*P*
12 hours preoperatively (%)	44.1±0.2 (41–51)	45.2±2.7 (41–51)	>0.05
1 hour post-operatively (%)	43.8±5.7 (38–48)	44.6±3.2 (39–51)	>0.05
At discharge (%)	47.5±2.8 (43–53)	47.8±2.9 (43–55)	>0.05
6 months after surgery (%)	54.4±3.2 (48–62)	53.9±3 (49–57)	>0.05

Table 4. MB-CK levels

Characteristic	Group 1, study group (n=45)	Group 2, control group (n=45)	*P*
12 hours preoperatively (U/l)	2.9±0.2 (2.4–3.6)	2.9±0.2 (2.4–3.5)	>0.05
12 hours post-operatively (U/l)	37.9±5.7 (29–49)	37±5.9 (27–49)	>0.05
6 months after surgery (U/l)	3.1±0.4 (2.1–3.8)	3.2±0.3 (2.7–3.8)	>0.05

Table 5. hs-TrT levels

Characteristic	Group 1, study group (n=45)	Group 2, control group (n=45)	*P*
12 hours preoperatively (U/l)	19.9±1.1 (18.3–22.1)	20.5±2.1 (18.3–31.1)	>0.05
12 hours post-operatively (U/l)	187.2±8.9 (165–206)	186.8±12.5 (166–221)	>0.05
6 months after surgery (U/l)	19.4±1.1 (17.9–22)	19.6±1.2 (18.1–22.3)	>0.05

Table 6. MDA levels

Characteristic	Group 1, study group (n=45)	Group 2, control group (n=45)	*P*
12 hours preoperatively, (μmol/ml)	236±11 (223–243)	232±12 (219–244)	>0.05
12 hours post-operatively (μmol/ml)	287±18 (269–303)	284±20 (264–300)	>0.05
1 month after surgery (μmol/ml)	227±9 (211–233)	270±14 (260–282)	<0.05
6 months after surgery (μmol/ml)	211±7 (205–216)	231±13 (221–239)	<0.05

Table 7. Postoperative data

Characteristic	Group 1, study group (n=45)	Group 2, control group (n=45)	*P*
Cardiac ward stay (hrs)	23.4±2.9 (17–29)	23.3±2.6 (18–28)	>0.05
In-hospital stay (days)	6.8±0.8 (5–8)	7.1±0.7 (5–8)	<0.05
Total blood loss (ml)	255.7±60.6 (170–410)	276±66.2(180–440)	>0.05
Postoperative angiogram (n)	45	45	>0.05
Graft occlusion (n)	nil	nil	

The follow-up went for 6 months after the surgery. There were no ischemic incidents in all patients. The quality-of-life assessment by Minnesota Living with Heart Failure Questionnaire was conducted and revealed excellent results.

DISCUSSION

We performed this study in open-heart surgery patients, because the best investigative model of myocardial ischemia-reperfusion (IR) injury is the cardioplegic-arrested heart during heart surgery.

Reperfusion of ischemic areas, especially reperfusion with oxygen, may contribute further to the tissue damage known as reperfusion injury. IR injury causes microvascular damage, reversible contractile dysfunction known as myocardial stunning and in some cases irreversible myocyte damage and finally, reperfusion can cause necrosis of the ischemic myocardium. Many studies have confirmed that IR during heart surgery increases oxygen free radicals and highly active molecules, such as the O_2, OH, and H_2O_2. These radicals have important roles in the microvascular dysfunction of organ disorders and these functional abnormalities can lead to organ death, a consequence of IR injury after cardiac arrest. Increased oxidative stress can also cause oxidation of proteins and lipids, which may lead to molecular and cellular dysfunction. Oxygen free radicals react with polyunsaturated lipids in membranes, creating lipid peroxidation products that can inhibit protein synthesis and alter enzyme activities. Oxidation of polyunsaturated fatty acids of membrane phospholipids can cause membrane disintegration, mitochondrial dysfunction, and Ca^{2+} overload. Free radicals react with membrane-bound lipids and lead to lipid peroxidation.

CPB is associated with greater than normal lipid peroxidation and with an imbalance in the status of antioxidants.

Malondialdehyde, which is produced during the lipid peroxidation of polyunsaturated fatty acids, is a marker for the oxidative destruction of cellular membranes. It induces a negative cardiac mechanical effect. An increase in the level of antioxidants may make the heart more resistant to IR injury.

Administration of antioxidants reduces the cardiac cell injury and dysfunction during open heart surgery and during acute MI.[15]

MDA was measured pre- and post-operatively as oxidative stress marker.

Trimetazidine is an anti-ischemic agent; it inhibits the long-chain mitochondrial 3-ketoacyl coenzyme A thiolase enzyme in the myocyte, which in turn causes partial inhibition of fatty acid oxidation and an increase in glucose oxidation. It increases the antioxidant capacity and protects against oxygen free radical-induced toxicity, counteracts the Ca^{2+} overload, and reduces the area of necrosis. Trimetazidine is conventionally used mainly for patients with coronary or cerebrovascular disease. It has no effects on coronary flow, contractility, or heart rate, suggesting that it acts by directly improving myocardial energy metabolism. Trimetazidine can bind to mitochondria and it significantly increases the rate of glucose oxidation and reduces the rate of fatty acid oxidation. It is metabolized in the liver and excreted via urine, so patients with renal failure or liver dysfunction were not included in this study.[14-16]

Postoperative treatment with trimetazidine is associated with an increase in the major antioxidant enzyme systems, but did not change postoperative hemodynamics, and therefore it has an important role in protecting against IR injury by increasing endogenous antioxidants. Some authors have found significantly lower cardiac troponin-T levels in a trimetazidine pretreatment group during CABG. The increase in endogenous antioxidants and stability of the MDA levels in the systemic venous samples indicate regulation of oxidative stress in the whole body, and may not be directly connected to the heart.[23-25]

CONCLUSIONS

In our study, there was no difference in improvement in LVEF 6 months after CABG surgery between groups, but there was a decrease in the MDA levels and shortening of the hospital stay in the trimetazidine group compared to the control group. Because of the protective effect of trimetazidine against IR injury, it must be considered in the medical treatment protocol after a CABG surgery.

Conflicts of Interest

The authors have no conflicts of interest to declare.

Patient consent to publish

Obtained.

REFERENCES

1. Story C, Cherney K, Nall R. The History of Heart Disease. Healthline. 2018. Available from: https://www.healthline.com/health/heart-disease/history
2. Lie JT. Centenary of the first correct antemortem diagnosis of coronary thrombosis by Adam Hammer (1818-1878): English translation of the original report. Am J Cardiol 1978; 42(5):849–52.
3. Sones Jr FM, Shirey EK. Cine coronary arteriography. Mod Concepts Cardiovasc Dis 1962; 31:735–8.
4. Lillehei CW, Cohen M, Warden HE, et al. The direct-vision intracardiac correction of congenital anomalies by controlled cross circulation: results in 32 patients with ventricular septal defects, tetralogy of Fallot, and atrioventricular communis defects. Surgery 1955; 38:11–29.
5. Dennis C, Spreng DS, Nelson GE, et al. Development of a pump-oxygenator to replace the heart and lungs: an apparatus applicable to human patients and application to one case. Ann Surg 1951; 134:709–21.

6. Gibbon JH. Application of a mechanical heart and lung apparatus to cardiac surgery. Minn Med 1954; 37:171–85.
7. Olearchyk AS. Vasilii I. Kolesov: a pioneer of coronary revascularization by internal mammary-coronary artery grafting. J Thorac Cardiovasc Surg 1988; 96(1):13–8.
8. Favaloro RG. Saphenous vein autograft replacement of severe segmental coronary artery occlusion: operative technique. Ann Thorac Surg 1968; 5(4):334–9.
9. Naviaux RK. Metabolic features and regulation of the healing cycle - a new model for chronic disease pathogenesis and treatment. Mitochondrion 2019; 46:278–97.
10. Ballinger SW, Patterson C, Knight-Lozano CA, et al. Mitochondrial integrity and function in atherogenesis. Circulation 2002; 106:544–9.
11. Wallace DC. Mitochondrial genetics: a paradigm for aging and degenerative diseases? Science 1992; 256:628–32.
12. Yu E, Calvert PA, Mercer JR, et al. Mitochondrial DNA damage can promote atherosclerosis independently of reactive oxygen species through effects on smooth muscle cells and monocytes and correlates with higher-risk plaques in humans. Circulation 2013; 128:702–12.
13. Zweier JL, Talukder MH. The role of oxidants and free radicals in reperfusion injury. Cardiovasc Res 2006; 70(2):181–90.
14. Dhalla NS, Elmoselhi AB, Hata T, et al. Status of myocardial antioxidants in ischemia-reperfusion injury. Cardiovasc Res 2000; 47(3):446–56.
15. Hausenloy DJ, Yellon DM. Myocardial ischemia-reperfusion injury: a neglected therapeutic target. J Clin Invest 2013; 123(1):92–100.
16. Piper HM, Garcia-Dorado D, Ovize M. A fresh look at reperfusion injury. Cardiovasc Res 1998; 38(2):291–300.
17. Cameron AA, Davis KB, Rogers WJ. Recurrence of angina after coronary artery bypass surgery: predictors and prognosis (CASS Registry). Coronary Artery Surgery Study. J Am Coll Cardiol 1995; 26(4):895–9.
18. Pocock SJ, Henderson RA, Seed P, et al. Quality of life, employment status, and anginal symptoms after coronary angioplasty or bypass surgery. 3-year follow-up in the Randomized Intervention Treatment of Angina (RITA) Trial. Circulation 1996; 94:135–42.
19. Boden WE, O'Rourke RA, Teo KK, et al. COURAGE trial coprincipal investigators and study coordinators. Design and rationale of the Clinical Outcomes Utilizing Revascularization and Aggressive DruG Evaluation (COURAGE) trial Veterans Affairs Cooperative Studies Program no. 424. Am Heart J 2006; 151(6):1173–9.
20. Task Force Members, Montalescot G, Sechtem U, Achenbach S, et al. 2013 ESC guidelines on the management of stable coronary artery disease: the Task Force on the management of stable coronary artery disease of the European Society of Cardiology. Eur Heart J 2013; 34(38):2949–3003.
21. Da Silva RMFL. Additional and emerging drugs for standard therapy for patients with stable angina. E-Journal of Cardiology Practice 2017; 15(11).
22. Kallistratos MS, Poulimenos LE, Manolis AJ. Stable angina pectoris: which drugs or combinations to use in which patients. E-Journal of Cardiology Practice 2017; 15(8).
23. Kantor PF, Lucien A, Kozak R, et al. The antianginal drug trimetazidine shifts cardiac energy metabolism from fatty acid oxidation to glucose oxidation by inhibiting mitochondrial long-chain 3-ketoacyl coenzyme A thiolase. Circ Res 2000; 86:580–88.
24. Di Napoli P, Chierchia S, Taccardi AA, et al. Trimetazidine improves postischemic recovery by preserving endothelial nitric oxide synthase expression in isolated working rat hearts. Nitric Oxide 2007; 16:228–36.
25. Fragasso G, Piatti PM, Monti L, et al. Short- and long-term beneficial effects of trimetazidine in patients with diabetes and ischemic cardiomyopathy. Am Heart J 2003; 146(5):854.

Роль послеоперационной терапии триметазидином в операциях аорто-коронарного шунтирования с искусственным кровообращением

Камен Станев[1], Антония Янева[2], Асен Иванов[1], Тодор Гоновски[1]

[1] *Клиника кардиохирургии, УМБАЛ „Св. Георги“, Пловдив, Болгария*

[2] *Кафедра медицинской информатики, биостатистики и электронного обучения, Факультет общественного здравоохранения, Медицинский университет – Пловдив, Пловдив, Болгария*

Адрес для корреспонденции: Камен Станев, Клиника кардиохирургии, УМБАЛ „Св. Георги“, бул. „Пещерско шосе“ № 66, 4000 Пловдив, Болгария; Email: kkamen_st@abv.bg; тел.: +359 32 602800

Дата получения: 28 февраля 2023 ♦ **Дата приемки:** 18 апреля 2023 ♦ **Дата публикации:** 31 декабря 2023

Образец цитирования: Stanev K, Yaneva A, Ivanov A, Gonovski T. The role of postoperative trimetazidine therapy in on-pump coronary artery bypass surgery. Folia Med (Plovdiv) 2023;65(6):915-921. doi: 10.3897/folmed.65.e102777.

Резюме

Введение: Аортокоронарное шунтирование остаётся золотым стандартом лечения пациентов с ишемической болезнью сердца. Однако повышенный окислительный стресс, вызванный высвобождением свободных радикалов во время ишемии-реперфузии, является хорошо известным патофизиологическим процессом во время и после процедур коронарной реваскуляризации. Это может привести к обратимому и необратимому повреждению миокарда.

Целью этого проспективного одиночного слепого рандомизированного контролируемого исследования является изучение и анализ эффективности препарата триметазидин в снижении послеоперационного ишемического-реперфузионного повреждения миокарда.

Цель: Мы оценили влияние триметазидина на активные формы кислорода, которые могут возникнуть в результате периода ишемии-реперфузии миокарда или системного воспаления после операции аортокоронарного шунтирования (CABG).

Материалы и методы: В исследование включены 90 пациентов, разделённых на две группы, оперированных планово и перенёсших изолированное аортокоронарное шунтирование в период с марта 2018 г. по октябрь 2018 г. Пациенты одной из групп получали триметазидин по 35 mg 2 раза в сутки сразу же после родов. Они были экстубированы. Остальная часть фармацевтической терапии была одинаковой для всех участников. Были измерены предоперационные и послеоперационные уровни нескольких биохимических маркеров крови, включая малоновой диальдегид (MDA), фракцию креатининкиназы-MB (CK-MB) и высокочувствительный тропонин T (hs-TnT).

Данные были классифицированы и проанализированы по времени сбора проб.

Результаты: Результаты показывают, что послеоперационное лечение триметазидином снижает выработку MDA, что приводит к снижению окислительного стресса и улучшению защиты сердечных клеток за счёт повышения антиоксидантного статуса. Наблюдение осуществлялось через 6 месяцев после операции. Оценка качества жизни с помощью Миннесотского опросника качества жизни больных сердечной недостаточностью показала отличные результаты.

Заключение: Послеоперационная терапия триметазидином приводит к улучшению метаболизма клеток миокарда и, следовательно, к снижению ишемически-реперфузионного повреждения после CABG.

Ключевые слова

аортокоронарное шунтирование, окислительный стресс, послеоперационные результаты, триметазидин