Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.292573.3412 Volume 31, Issue 10 October. 2025
Manuscript ID:ZUMJ-2405-3412
DOI:/10.21608zumj.2024.292573.3412

ORIGINAL ARTICLE

Study on the Effects of Sodium-glucose Cotransporter 2 Inhibitors on Myocardial Ischemia-Reperfusion in type ll Diabetic Male Rats

Nermeen A Zaitoun[1], Salah M. Ibrahim[1], Reham H. Ibrahim[1], Ashraf S. Abdalsalam[1*], Ahmed Algazeery[2]

[1] Physiology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt
[2] Zoology Department, Faculty of Science, Zagazig University, Zagazig, Egypt

***Corresponding author:**
Ashraf Saleh Abdalsalam Saleh
Email:
ashrafbendaba2017@gmail.com

Submit Date 25-05-2025
Revise Date 02-06-2025
Accept Date 04-06-2025

Abstract

Background: Regardless of the presence of diabetes, the sodium glucose co-transporter-2 inhibitor empagliflozin (EMPA) lowers hospitalization and death rates for heart failure after myocardial infarction. Although results point to an innate ability for cardioprotection, the exact mechanism needs more explanation in ischemia and reperfusion injuries.

The aim of the study was to clarify role of empagliflozin (SGLT2i) in ischemia and reperfusion injury by reducing infarct size and ameliorating oxidative stress.

Methods: This study was carried out at Physiology Department, Faculty of Medicine, Zagazig University on 48 albino rats that were divided into 6 groups, Group IA: rats were received d chronic oral vehicle daily for 7 days before ischemia reperfusion. Group IB: Rats receiving oral administration of EMPA 1.5 h before ischemia reperfusion (EMPA-Acute). Group IC: Rats receiving chronic oral EMPA treatment daily for 7 days before ischemia reperfusion (EMPA-Chronic). Group ⅡA: control diabetic rats were received chronic oral vehicle daily for 7 days before ischemia reperfusion. Group ⅡB: Diabetic rats receiving acute oral administration 1.5h before ischemia reperfusion (EMPA-Acute). Group ⅡC: Diabetic rats receiving chronic oral EMPA treatment daily for 7 days before ischemia reperfusion (EMPA-Chronic).

Results: There was a significant decrease in infarct size when compared chronic non diabetic with chronic diabetic albino rat groups also there was significant decrease in SOD, MDA in chronic EMPA receiving groups diabetic and non-diabetic.

Conclusions: Empagliflozin has a strong role in reducing infarct size and improving oxidative stress in ischemia and reperfusion injury in induced type ll diabetes and non-diabetic rats.

Keywords: Sodium-glucose Cotransporter 2 Inhibitors; Myocardial Ischemia-Reperfusion; Diabetes.

INTRODUCTION

Diabetes mellitus has emerged as one of the most important and prevalent problems in recent years. Right now, it ranks eighth in the world's leading causes of mortality, with 5.2 million deaths, or 82.4 deaths per 100,000 people. In Egypt, DM is a conundrum that is becoming worse very quickly. As per UN estimates, Egypt's population is projected to reach 102,334,404 by the middle of 2020. The International Diabetes Federation (IDF) estimates that

15.2% of Egyptian adults have diabetes, a number that may possibly be underestimated **[**1]. Diabetes is a significant risk element for the emergence of heart disease (CVD). Both macrovascular problems like coronary artery disease and peripheral vascular disease, as well as microvascular ailments like retinopathy, neuropathy, and nephropathy, and carotid artery disease, become more prevalent as diabetes persists longer. Beneficial methods for coronary artery revascularization Interventions such as pharmacological thrombolysis, angioplasty, PCI, and CABG (coronary artery bypass grafting) are among the methods that can successfully resuscitate the ischemia myocardium and restore myocardial blood reperfusion. But a sudden recovery to oxygenated blood flow could cause significant ischemia/reperfusion (I/R) damage down the road as well as potentially fatal arrhythmias [2].

The effectiveness of current reperfusion therapy is significantly limited by lethal reperfusion injury, and ventricular fibrillation has been observed to occur more frequently during reperfusion than during coronary artery closure [3].

Interventions such as pharmacological thrombolysis, angioplasty, PCI, and CABG (coronary artery bypass grafting) SGLT2 in the renal proximal tubule were done. It accomplishes this by increasing the excretion of glucose in urine, which lowers blood glucose levels without requiring insulin. The heart-protective properties of this pharmacological family, which also includes ertugliflozin, dapagliflozin, canagliflozin, and empagliflozin (EMPA), have been validated by several clinical investigations [4].

The process SGLT2 inhibitors' mechanism of cardiovascular system protection from the harmful effects of hypoglycemia is currently the subject of numerous investigations. The primary research areas are energy metabolism, oxidative stress, inflammatory response, cardiac fibrosis, and electrolyte balance [5].

Empagiflozin treatment for cardiac ischemia-reperfusion may have certain infarct-sparing effects, according to a number of studies [6]. We therefore endeavor to elucidate the potential relationship between empagliflozin and cardioprotective effects that decrease ischemia/reperfusion injury.

METHODS

This expiremintal study 48 male albino rats weighing 180-200 g. maintained for acclimation at a steady 23 °C, with a 12-hour light-dark cycle and unrestricted access to food and water. Prior to any research, the rats were housed at an animal facility for a period of two weeks. The institutional Animal Care and Use Committee and the physiology department approved the experimental protocol. Zagazig University (ZU –IACUC) Approval number is ZU-IACUC /3/F/439/2022.

The Physiology Department at Zagazig University's Faculty of Medicine conducted this study. Total 36 albino rats were divided into 6 groups, each group contains 6 rats; Group IA: rats were receive d chronic oral vehicle daily for 7 days before ischemia reperfusion. Group IB: Rats receiving oral administration of EMPA 1.5 h before ischemia reperfusion (EMPA-Acute). Group IC: Rats receiving chronic oral EMPA treatment daily for 7 days before ischemia reperfusion (EMPA-Chronic). Group IIA: control diabetic rats were received chronic oral vehicle daily for 7 days before ischemia reperfusion. Group IIB: Diabetic rats given acute oral medication 1.5 hours prior to reperfusion following ischemia (EMPA-Acute). Group C: Oral EMPA therapy administered to diabetic rats on a daily basis for seven days prior to ischemia reperfusion (EMPA-Chronic).

Induction of high fat diet (HFD) streptozotosin (STZ) of type ll diabetes

A two-week high-fat diet (HFD) consisting of 57.5% fat, 26.9% carbohydrates, and 15.6% protein was used to induce diabetes type II. Following the HFD, an A 25 mg/kg STZ diluted in citrate buffer solution was injected intraperitonealLy. Rats that maintained blood glucose levels higher than during fasting 250 mg/dl for 48 hours following the treatment of STZ were classified as diabetic rats [7].

Induction of ischemia and reperfusion:

Rats were put to sleep in an induction chamber using a mixture of 8 percent sevoflurane (Sevorane, AbbVIE A/S, Copenhagen, Denmark) and ambient air (flow rate: 0.8 L/min). The rats were put on board in the supine position, intubated, and attached to a mechanical ventilator after anesthesia was induced.The four expanded limbs were fastened to the board's sides.The myocardium is accessible through a pericardiotomy via lateral chest compression and a left-sided thoracotomy through the fourth and fifth ribs. The left anterior descending coronary artery (LAD) will be located and ligated using a 4-0 silk suture, approximately 2 mm distally from the location where the left atrial appendage and pulmonary conus meet [8]. To confirm ischemia by looking for alterations in the ECG together with paling and hypokinesia. After the myocardium had been in ischemia for 30 minutes, the ligature was removed, and the patient was kept alive for three hours in order to get cardiac enzyme samples.

Estimation of infarct size (Figure 1):

Following excision, the hearts were cut into five 2 mm thick slices using a rat heart slicer matrix as a guide after being frozen at -80 °C. The slices were then treated with 1% 2,3,5-tetrazolium after that chloride (TTC) for three minutes in order to identify the infarction regions.

Oxidative stress assessment:

By measuring the amounts of malondialdehyde (MDA) and superoxide dismutase (SOD), oxidative stress was evaluatedinside cardiac tissue [9].

Biochemical markers:

Measurements were made of serum glucose, insulin, HOMA IR, creatinine kinase, and troponin levels during fasting.

Statistical analysis

All information was statistically examined using IBM SPSS Statistics for Windows, Version 23.0 (IBM Corp., Armonk, NY, 2015). In quantitative data, the mean ± SD was used for expression. More than two groups of normally distributed variables were compared using the Anova test (f). P-values less than 0.05 were regarded as statistically significant.

RESULTS

In non-diabetic group: There was no significant difference between control, acute, chronic non diabetic Albino rates groups regarding serum fasting insulin, Homa IR, or Fasting Blood Glucose (Table 1).

In diabetic group: Homa-IR, fasting blood glucose, and fasting insulin were significantly increased in control diabetic compared to acute, chronic diabetic albino rat group, ($p<0.05$). Also, Homa-IR, fasting blood glucose, and fasting insulin were significantly increased in acute diabetic compared to chronic diabetic albino rat group (Table 1).

In comparison between non diabetic and diabetic Albino rats: the current study's findings revealed a markedly higher of SFI, associated with significant decrease inHOMA-IR ($p<0.001$), and Fasting Blood Glucose ($p<0.001$), when compared control, acute, chronic non diabetic groups with those in diabetic groups (Table 1).

In non-diabetic group: There was a significant increase of Troponine ng/ml, CK-MB IU/L, infarct size when compared control, with acute, chronic non diabetic

albino ratgroups. Also, there was significant increase of Troponine ng/ml, CK-MB IU/L, when compared acute with chronic non diabetic albino rat groups. While, there was no significant difference between acute with chronic no diabetic albino rat groups, regarding infarct size (Table 2).

In diabetic group: there was a significant increase of Troponine ng/ml, CK-MB IU/L, infarct size when compared control, with acute, chronic diabetic albino rat groups. Also, there was a significant increase of Troponine ng/ml, CK-MB IU/L, when compared with acute with chronic diabetic albino rat groups. While, there was no significant difference between acute with chronic diabetic albino rat groups, regarding infarct size (Table 2).

In comparison betweennon-diabetic and diabetic Albino rats: the current study's findings indicated a considerable rise in of Troponine ng/ml, CK-MB IU/Lwhen compared control, acute, chronic non diabetic groups with those in diabetic groups. When comparing the infarct sizes of the diabetic albino rat group, acute no diabetic, and control groups, there was no discernible difference. Even though there was a notable decline in infarct size when compared chronic non diabetic with chronic diabetic albino rat groups (Table 2).

In non-diabetic group: there was a significant increase of MDAwhen compared control, with acute, chronic non diabetic albino rat groups. Also, there was a significant increase of MAD, when compared acute with chronic non diabetic albino rat groups. Regarding SOD; there was significantly decreased of SOD when compared control, with acute, chronic non diabetic albino rat groups. Also, there was significantly decreased of SOD, when compared acute with chronic non diabetic albino rat groups (Table 3).

In diabetic group: there was a significant increase of MDAwhen compared control, with acute, chronic diabetic albino rat groups. Also, there was a significant increase of MAD, when compared acute with chronic diabetic albino rat groups. Regarding SOD; there was significantly decreased of SOD when comparedcontrol with acute and chronic diabetic albino rat groups, ($p<0.001$). Also, there was a significant decrease of SOD, when compared acute with chronic diabetic albino rat groups (Table 3).

In comparison between non-diabetic and diabetic Results of the current investigation indicated a noteworthy decline in albino rats of MDA of control, acute, chronic non diabetic groups when compared with control, acute, chronic diabetic group and a significant increase of SOD of control, acute, chronic non diabetic groups when compared with control, acute, chronic diabetic group (Table 3).

Table (1): Effect of empagliflozin on serum fasting insulin levels, HOMA IR and fasting blood glucose in all groups

Variables	Groups	Control group n.8	Acute Group n.8	Chronic Group n.8	f	p	
Serum fasting insulin (uIU/ml)	Non diabetic	24.9±0.70	25.81±1.28	25.31±0.99	1.23	0.31	
	Type ll diabetes	37.1±1.12	33.47±0.73	29.12±1.88	28.4	.0001*	P1=0.006 P2=0.0001 P3=0.002
T ^p		23.4 0.0001	16.06 0.0001	8.7 0.0001			
Fasting Blood	Non diabetic	98.5±5.55	99.63±3.58	98±5.26	.233	.791	

Variables	Groups	Control group n.8	Acute Group n.8	Chronic Group n.8	f	p	
Glucose (mg/dl)	type ll diabetes	376.7±48.55	323±22.4	266 ±34.75	18.1	.0001*	P1=0.025 P2=0.0001 P3=0.016
T ^p		16.1 0.0001*	27.8 0.0001*	13.5 0.0001*			
HOMA IR	Non diabetic	6.06±0.30	6.33±0.36	6.1±0.43	1.22	0.32	
	Type ll diabetes	19.15±0.99	15.53±1.69	12.98±1.33	40.9	.0001*	P1=0.0001 P2=0.0001 P3=0.004
T ^p		35.8 0.0001*	15.03 0.0001*	13.9 0.0001			

Non diabetic group : Control, Acute SGLT, Chronic SGLT
Diabetic group: Type ll diabetes control,Acute SGLT9 in type ll diabetes, Chronic SGLT9 in type ll diabetes
F:Anova test, p>0.05 no significant, , *p<0.05 significant,
P1: 0.15, compare control group with acute group
P2: 0.88 , compare control group with chronic group
P3: 0.014, compare acute group with chronic group
t:student t test compare between two group

Table (2): Effect of empagliflozin on troponin, CKMB and infarct size in all groups

Variables	Groups	Control group n.8	Acute Group n.8	Chronic group n.8	F	P	Post hoc test P1	P2	P3
Troponine ng/ml	Non diabetic	7.47±0.24	6.49±0.17	5.95±0.35	34.9	0.0001	.0001	.0001	0.001
	Type ll diabetes	9.41±0.62	8.59±0.25	7.66±0.49	27.99	0.0001	.007	.0001	0.002
T ^p		12.5 0.0001	29.5 0.0001	12.7 0.0001					
CK-MB (IU/L)	Non diabetic	565.8±24.45	464.57±15.18	408.19±35.06	36.7	0.0001	.0001	.0001	0.001
	type ll diabetes	599.8±28.31	511.49±29.05	471.25±23.9	46.7	0.0001	.0001	.0001	0.022
T ^p		2.6 0.022	4.05 0.001	4.2 0.001					
infarct size(%)	Non diabetic	31.75±3.54	28±2.27	25.5±2.45	10.4	0.001	0. 043	0.001	0.27
	Type ll diabetes	32.13±3.04	28.75±2.43	27.5±0.93	8.6	0.002	0.025	0.002	0.87
T ^p		0.23 0.82	0.64 0.53	2.2 0.049					

Non diabetic group : Control, Acute SGLT, Chronic SGLT
Diabetic group: Type ll diabetes control,Acute SGLT9 in type ll diabetes, Chronic SGLT9 in type ll diabetes
F:Anova test, p>0.05 no significant, , *p<0.05 significant,
P1: compare control group with acute group
P2: compare control group with chronic group
P3: compare acute group with chronic group
t:student t test compare between two group

Table (3): Effect of empagliflozin on MDA and SOD in all groups

Variables	Groups	Control group n.8	Acute Group n.8	Chronic group n.8	F	P	Post hoc test P1	P2	P3
MDA (nmol/gm)	Non diabetic	14.79±3.41	5.79±1.84	4.05±1.33	34.6	0.0001	0.001	.0001	0.002
	Type ll diabetes	18.32±3.01	10.19±2	6.15±0.89	66.7	0.0001	.0001	.0001	0.003
T ^p		2.2 0.045*	4.6 0.0001*	3.7 0.002*					
SOD (IU/ L)	Non diabetic	2.19±0.40	4.26±0.88	6.16±0.74	64.4	0.0001	0.001	.0001	.0001
	Type ll diabetes	1.47±0.35	3.09±0.48	4.6±1.02	42.5	0.0001	.0001	0001	0.001
T ^p		3.8 0.002*	3.3 0.005*	3.5 0.002*					

SOD:superoxide dismutase .
MDA:Malonaldehyde .
Non diabetic group : Control, Acute SGLT, Chronic SGLT
Diabetic group: Type ll diabetes control,Acute SGLT9 in type ll diabetes, Chronic SGLT9 in type ll diabetes
F:Anova test, p>0.05 no significant, , *p<0.05 significant,
P1: compare control group with acute group
P2: compare control group with chronic group
P3: compare acute group with chronic group
t:student t test compare between two group

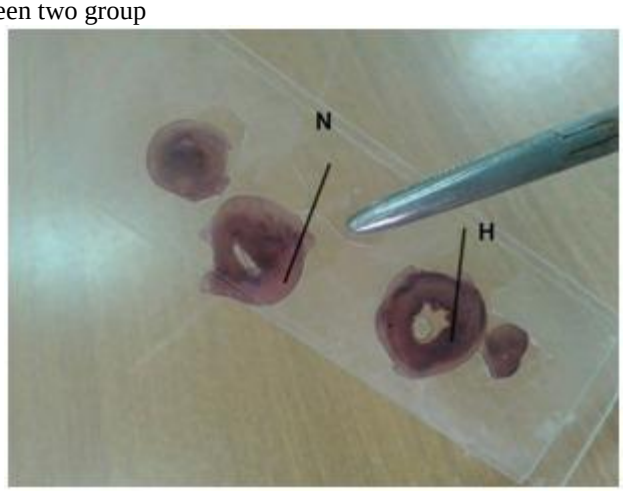

Figure (1): Left ventricle slices stained by nitro blue tetrazolium stain in control non diabetic rat. N: Unstained necrotic tissue, H: Stained viable tissue

DISCUSSION

In clinical procedures like thrombolysis, percutaneous transluminal coronary angioplasty, and coronary bypass surgery—which are the standard treatment techniques for cardiovascular events—cardiac ischemia followed by reperfusion still poses a significant risk [10].

This study examined the impact of EMPA on ischaemia reperfusion injury in male albino rats with and without diabetes. EMPA was given either acutely, an hour prior to ischemia reperfusion, or chronically, a week prior to ischemia reperfusion.

In the present study, EMPA didn't make any significant changes in Insulin level, fasting blood sugar or HOMA IR, while in diabetic control group there were significant increase in these parameter and EMPA decreased these parameters significantly in chronic EMPA group than acute EMPA group.

The results of this study demonstrate the cardioprotective qualities of EMPA, since EMPA infusion significantly reduced cardiac troponin (cTn), a biomarker with strong diagnostic value that also offers reliable prognostic information in cases of acute myocardial infarction (AMI).1. Due to its increased specificity and sensitivity as well as decreased levels of the well-known biomarker CK-MB, which indicates myocardial damage in which the cardiac cell membrane becomes permeable or bursts, allowing these enzymes to leak out, it is still the most recommended biomarker for detecting myocardial injury [11]. Therefore, EMPA may lessen heart damage, as seen by the chronic EMPA group's decreased fraction of necrotic tissue to LV mass Zhou et al. [12] demonstrated that EMPA can reduce cardiac damage by anti-apoptotic, anti-inflammatory, anti-oxidant effects.

Another conclusion of the current study is that EMPA significantly increased the oxidative stress indicators SOD and MDA significantly decreased MDA [13].

A landmark study by Farías et al. [13] demonstrated that strong oxidant radicals, including peroxynitrite, hydroxyl radical, and superoxide anion, are generated in the initial few minutes of reflow and are essential to the development of reperfusion damage. These free radicals are important in the pathophysiology of acute myocardial infarction (AMI) because they can react with unsaturated lipids to start a self-replicating chain reaction of lipid peroxidation, which results in the production of MDA, a hallmark of oxidative damage [14].

As an oxygen radical scavenger, SOD is found in mitochondria and is induced in response to moderately harmful stimuli, indicating that it is involved in cellular defense. By converting superoxide anion radicals that arise in the upper stream of the reactive oxygen metabolism cascade, SOD can shield cells from oxidative damage [15]. This enzyme is crucial for the oxidative stress that occurs during I/R injury [16].

In addition, Li et al. [16] discovered that DM mice had much higher levels of oxidative stress. Diabetes, on the other hand, causes oxidative stress. Intracellular hyperglycemia increases the development of intracellular advanced glycation end products and mitochondrial reactive oxygen species (ROS). Insulin resistance, the production of oxidized low density lipoprotein, and the expression of inflammatory and adhesion factors are all directly increased by ROS [17].

Yang & Zhang [18] found that in patients with diabetes and AMI, EMPA can enhance cardiac function, decrease the extent of the myocardial infarct, and improve the prognosis for the cardiovascular system. For the therapeutic therapy of myocardial I/R damage in diabetes with AMI, SGLT2i are anticipated to be efficacious medications.

Another study Mizuno et al. [19] demonstrated that the mechanism of empagliflozin-induced prevention of the excessive reduction in mitochondrial size in hearts from a rat model of T2D following AMI required ROS suppression and autophagy restoration.

However, Empa decreased treponin, CK.MB and oxidative stress in acute administration but these effects were not sufficient to decrease infarct size.

Conclusions: Empagliflozin has a strong role in reducing infarct size and improving oxidative stress in ischemia and reperfusion injury in induced type ll diabetes and non-diabetic rats.

Conflict of interest: None

Funding source: None

REFERENNCES

1. Abouzid MR, Ali K, Elkhawas I, Elshafei SM. An overview of diabetes mellitus in Egypt and the significance of integrating preventive cardiology in diabetes management. Cureus, 2022; 14(7).

2. Gunata M, Parlakpinar H. A review of myocardial ischaemia/reperfusion injury: pathophysiology, experimental models, biomarkers, genetics and pharmacological treatment. Cell Biochem. Funct., 2021; 39(2), 190-217.
3. Hu Z, Ju F, Du L, Abbott GW. Empagliflozin protects the heart against ischemia/reperfusion-induced sudden cardiac death. Cardiovasc. Diabetol., 2021; 20(1), 1-13.
4. Wiviott SD, Raz I, Bonaca MP, Mosenzon O, Kato ET, Cahn A, et al. Dapagliflozin and cardiovascular outcomes in type 2 diabetes. N Engl J Med., 2019; 380(4), 347-357.
5. Bertero E, Prates Roma L, Ameri P, Maack C. Cardiac effects of SGLT2 inhibitors: the sodium hypothesis. Cardiovasc Res., 2018; 114(1), 12-18.
6. Lu Q, Liu J, Li X, Sun X, Zhang J, Ren D, et al. Empagliflozin attenuates ischemia and reperfusion injury through LKB1/AMPK signaling pathway. Mol Cell Endocrinol, 2020; 501, 110642.
7. Gheibi S, Kashfi K, Ghasemi A. A practical guide for induction of type-2 diabetes in rat: Incorporating a high-fat diet and streptozotocin. Biomed Pharmacother, 2017; 95, 605-613.
8. CHMP. Assessment report, Jardiance, International nonproprietary name: empagliflozin. Procedure No. 2014. EMEA/ H/C/002677/0000.: European Medicines Agency; 2014 [Available from: https://www.ema.europa.eu/en/documents/assessment- report/jardiance-epar-public-assessment-report_en.pdf.
9. Khoubnasabjafari M, Ansarin K, Jouyban A. Reliability of malondialdehyde as a biomarker of oxidative stress in psychological disorders. BioImpacts: BI, 2015; 5(3), 123.
10. Hausenloy DJ, Yellon DM. Myocardial ischemia-reperfusion injury: a neglected therapeutic target. J Clin Invest, 2013; 123(1), 92-100.
11. Hodrea J, Saeed A, Molnar A, Fintha A, Barczi A, Wagner LJ, et al. SGLT2 inhibitor dapagliflozin prevents atherosclerotic and cardiac complications in experimental type 1 diabetes. PLoS One, 2022; 17(2), e0263285.
12. Zhou H, Wang S, Zhu P, Hu S, Chen Y, Ren J. Empagliflozin rescues diabetic myocardial microvascular injury via AMPK-mediated inhibition of mitochondrial fission. Redox Biol., 2018; 15, 335-346.
13. Farías JG, Molina VM, Carrasco RA, Zepeda AB, Figueroa E, Letelier P, et al. Antioxidant therapeutic strategies for cardiovascular conditions associated with oxidative stress. Nutr., 2017; 9(9), 966.
14. Cadenas S. ROS and redox signaling in myocardial ischemia-reperfusion injury and cardioprotection. Free Radic Biol Med. 2018;117:76–89.
15. Tawfik, M., Al-Aleem, A., Ibrahim, R., & Abaza, R. Effect of nesfatin-1 on ischaemia-reperfusion injury in isolated heart of albino rats. ZUMJ, 2016, 22(6), 1-11.
16. Li C, Zhang J, Xue M, Li X, Han F, Liu X, et al. SGLT2 inhibition with empagliflozin attenuates myocardial oxidative stress and fibrosis in diabetic mice heart. Cardiovasc. Diabetol., 2019; 18, 1-13.
17. Yuan T, Yang T, Chen H, Fu D, Hu Y, Wang J, et al. New insights into oxidative stress and inflammation during diabetes mellitus-accelerated atherosclerosis. Redox Biol. 2019; 20:247–60.
18. Yang T, Zhang D. Research progress on the effects of novel hypoglycemic drugs in diabetes combined with myocardial ischemia/reperfusion injury. Ageing Res. Rev., 2023; 86, 101884.
19. Mizuno M, Kuno A, Yano T, Miki T, Oshima H, Sato T, et al. Empagliflozin normalizes the size and number of mitochondria and prevents reduction in mitochondrial size after myocardial infarction in diabetic hearts. Physiol. Rep., 2018; 6(12),e13741.

Citation

Nermeen A Zaitoun[1], Salah M. Ibrahim[1], Reham H. Ibrahim[1], Ashraf S. Abdalsalam[1*], Ahmed Algazeery[2] Study on the Effects of Sodium-glucose Cotransporter 2 Inhibitors on Myocardial Ischemia-Reperfusion in type ll Diabetic Male Rats. . Zagazig University Medical Journal, 2025; (4923-4930): 10.21608/zumj.2024.292573.3412.