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EVALUATION OF CARDIOPROTECTIVE EFFECT OF *Actinidia deliciosa* FRESH FRUIT JUICE ON ISOPROTERENOL INDUCED MYOCARDIAL INJURY IN RATS

CHAKALI AYYANNA[1*], S.MAHABOOB BEE[2], SREE SUDHA TY[3], NAGA SUDHA B[4], LAKSHMI DURGA .V [5] AND P. ABDUL WAJAHATH ALI KHAN[6]

1, 2, 5, 6: Department of Pharmacology, CES College of Pharmacy, Kurnool, Andhra Pradesh, India

3: Department of Pharmacology, AIIMS, Raipur, Chhattisgarh, India

4: Department of Pharmaceutical Chemistry, CES College of Pharmacy, Andhra Pradesh, India

[*]**Corresponding Author: Chakali Ayyanna: ayyannac5@gmail.com**

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ABSTRACT

To investigate the cardioprotective effects of *Actnidia deliciosa* fruit juice [ADFFJ] against isoproterenol-induced myocardial infarction in rat myocardium. Wistar rats were pretreated with ADFFJ Cardioprotector, Isoproterenol, Myocardial Infarction, the imbalance between myocardial oxygen supply and demand. The increase produces toxic reactive oxygen species (ROS) such as O_2, H_2O_2, OH, etc. **[1, 2]**. *Actnidia deliciosa* fruit juice (100, 200 and 400 mg/kg) orally for 21 days followed by intoxication with isoproterenol [85 mg/kg/I.P] for 2 consecutive days. Biochemical markers of myocardial infarction such as CKMB, SGOT and SGPT have been identified. In addition, the antioxidant status of cardiac tissue was evaluated by examining the activity of antioxidant enzymes such as lipid peroxidation, superoxide dismutase, reduced glutathione and catalase. The results indicated that the pretreatment of *Actnidia deliciosa* fruit juice significantly inhibited the decrease in antioxidants and the slow increase in markers of heart damage in isoproterenol-treated rats. Furthermore, these findings were clearly supported by the remarkable protection revealed by histopathological studies. *Actnidia deliciosa* fruit juice has been found to help protect the heart.

Key words: *Actnidia deliciosa* fruit juice, Myocardial infarction, Isoproterenol, Antioxidants

INTRODUCTION

Cardiovascular disease is the most common cause of death worldwide. Myocardial infarction (MI), an insidious disease, plays an important role in this mortality **[1, 2, 3]**. MI is characterized by severe chest pain, which can spread to the neck, jaw, and arms, and can cause shortness of breath. Patients with metabolic and cardiovascular disorders such as hypertension, atherosclerosis, and diabetes have a higher risk of MI **[4, 5, 6]**. Myocardial infarction is the rapid development of myocardial necrosis caused by a severe imbalance between myocardial oxygen supply and demand. The increase produces toxic reactive oxygen species (ROS) such as O_2, H_2O_2, OH, etc. causes severe oxidative stress on -CVD -prone myocardium, such as ischemic heart disease, atherosclerosis, congestive heart failure, cardiomyopathy, and arrhythmias. Recent studies suggest that increased β-adrenergic receptors play a role in the induction of myocardial oxidative stress, inflammation, calcium overload and coronary vasospasm, followed by cell loss **[7, 8]**. Beta-adrenergic-mediated oxidative stress induces reactive oxygen species (ROS), leading to macrophage atheroma formation and as a result the risk of unstable angina, thrombosis, and acute MI **[9, 10]**. Isoproterenol, a synthetic adrenoreceptor agonist, has been shown to induce myocardial infarction in rats after disrupting the physiological balance between free radical production and the antioxidant defense system **[11, 12]**. It is acute myocardial necrosis accompanied by elevation of cardiac markers, alternating ischemia on electrocardiogram, accumulation of lipid peroxides and impaired cardiac function **[13, 14, 15, 16]**. In this study, this mouse model of myocardial infarction was used. to evaluate the efficacy of AD in protecting the heart against ISO and focus on correlated changes in function, biochemistry and histopathology. The scientific name for the kiwi is *Actinidia deliciosa* **[17]**. It is also known as Chinese gooseberry, this fruit is popular all over the world, due to its high nutritional value, high content of vitamin C, in addition to excellent organoleptic qualities, in connection with its adaptability. Several studies **[18–19]** suggest that the kiwi contains more nutrients than other commonly consumed fruits and highlights its therapeutic benefits in terms of healthy metabolism, iron content, digestive potential, antioxidant properties, immune function and also protective effects against coronary heart disease. . As a source of ascorbic acid and polyphenols, kiwi fruit helps reduce the risk

of hardening of the arteries, cardiovascular disease and some forms of cancer **[20]** in irritable bowel syndrome **[21]** and also protects cells in in vitro oxidative damage to DNA. .

MATERIALS AND METHODS

Drugs and chemicals: Isoproterenol, propranolol, DTNB (5,5-dithiobis(2- nitro benzoic acid), sodium pyrophosphate buffer, Phenazine methosulphate sodium, Nitro blue tetrazolium, Nicotinamide adenine dinucleotide reduced disodium salt (NADH), Glacial acetic acid, Tris –Hcl buffer, n-butanol, pyridine, Thio barbituric acid (TBA), H_2O_2

Collection of fruit:

The Actinidia deliciosa, were purchased from the local market of Kurnool. The fruit was authenticated by the botanist from Govt. Degree College of for men, Kurnool.

Preparation of *Actinidia deliciosa* juice*:*

The fresh fruit of *Actindia deliciousa* was prepared the help of juicer without addition of water. The fresh fruit was hopped into a small pieces and juice was collated juice was filtered with sterile cloth and the resultant filtrate was used and oral dosing to animal. Fresh juice was subject the weight of Petridis containing dry residue of juice was taken and equivalent dose of 50ml juice was calculated by subtracin initial weight of dried Petridis. The same procedure was repeated for six times at different days. It was clear from the mean that, 50ml of juice gives 8.680mg f total solid residue in dried juice, which is equivalent to 143 gm of fresh juice of kiwi. The dose of fresh juice of kiwi (ml) equivalent to 100, 200, &400m was administered orally to rat's mg/kg/day for 21 days **[22]**.

Animals

36 Wistar rats weighing 150-200 g were used in the present study. They were housed in individual polypropylene cages under standard laboratory conditions of light, temperature and relative humidity. Animals were provided standard rat pellets (Pranav Argo's ltd.) and drinking water *ad libitum*. The experimental protocol was approved by the Institutional Animal Ethical Committee of Creative Educational Society College of Pharmacy (IAEC/CESCOP/2019-OCT-11).

Experimental design

Isoproterenol Induced Myocardial Infarction [22]

Myocardial infarction was induced in laboratory rats by injection of isoproterenol hydrochloride (ISO, 85 mg/kg/I.P body weight, dissolved in physiological saline, for two consecutive days)

Thirty-six animals after acclimatization, the animals were divided into six groups with

six animals each. Group I animals were given a normal saline solution and were referred to as the control group (1 ml). /kg/day p.o) for 21 days. Group II animals were treated with isoproterenol at 85 mg/kg/i.p. daily for the last two consecutive days. Group III animals were treated with standard propranolol (10 mg/kg, p.o.) for one week following the saline treatment interval. Group IV, group V and group VI animals were pre-treated with *Actinidia* at 100 mg/kg, 200 mg/kg and 400 mg/kg body weight orally for 21 days. Animals from groups II to V received isoproterenol (ISO) 85 mg/kg, intraperitoneally on days 20 and 21 with a 24-hour interval. All animals were sacrificed by cervical dislocation on the twenty-first day of the trial. The hearts were removed, washed in cold saline solution and stored in the refrigerator for biochemical and antioxidant studies.

Estimation of Biochemical parameters

Preparation of serum from blood

The serum was separated by centrifugation at 3000 rpm at 30◦C for 15 min and it was used for the estimation of cardiac marker enzymes like creatine kinase myoglobin (CK-MB), serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), total protein (TP)

Preparation of heart homogenate

The heart was dissected out, washed with ice-cold saline and a 10% homogenate was prepared in phosphate buffer (50 mm, pH 7.4). The homogenate was centrifuged at 7000 rpm for 15 min and the supernatant was used for the assay of superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH), glutathione peroxidase (GPx) and lipid peroxidation (MDA).

Estimation of serum cardioprotective parameters:

Estimation of creatine kinase myoglobin (CK-MB) activity by using *Agappe kits*

SGOT&SGPT by using *Agappe kits*

***In- vitro* anti-oxidant studies**

Lipid peroxidation [23]

Cardiac tissues were homogenized in 10% trichloroacetic acid (TCA) buffer in ice. Pipette 0.2 ml of homogenate into the test tube. Add 2 ml of 8.1% sodium dodecyl sulfate (SDS), 1.5 ml of etic acid (pH 3.5) and 1.5 ml of 0.8% thiobarbituric acid (TBA). The tubes were boiled at 95 ° C for 60 min then allowed to cool. 1.0 ml of double-distilled water and 5.0 ml of nbutanol: pyridine (15: 1, v / v) were added to the tubes and centrifuged at 5000 rpm for 10 min. The absorbance of the organic layer was measured at 540 nm. Malonyl dialdehyde (MDA), an end product of lipid peroxidation, forms pink byproducts with

TBARS. The degree of lipid peroxidation was expressed as µM MDA/g of cardiac tissue.

Glutathione [23]

Cardiac tissues homogenized with 10% TCA buffer were centrifuged at 3000 rpm for 10 min at 4 °C. The reaction mixture contained 0.1 mL supernatant, 2.0 mL phosphate buffer 0. 3M (pH 8.4), 0.4 mL double distilled water and 0.5 mL DTNB [5,5dithiobis (2nitrobenzoic acid)]. The reaction mixture was incubated for 10 min and absorbance was measured at 412 nm for 15 min. GSH concentration is expressed as µg/g of cardiac tissue.

SOD [23]

Cardiac tissues were homogenized in 0.25 M Tris sucrose buffer, pH 7.4 and centrifuged at 10,000 rpm for 15 min at 4 ° C), 0.1 mL of phenazine metosulfate solution (186 µM) and 0.3 mL of nitro green tetrazolium solution (300 µM). The reaction was initiated by adding 0.2 ml of nicotineamidadenine dinucleotide (NADH) induced disodium saline (NADH) (780 µM). The reaction mixture was incubated for 90 s at room temperature then stopped by adding 1 mL of glacial acetic acid. The absorbance of the reaction mixture was read spectro-photometrically at 560 nm. SOD activity is expressed in U / mg of protein.

Catalase [24]

The tissue was homogenized in isotonic buffer (pH 7.4) and centrifuged at 1000 × g for 10 min. 20 µl of tissue diluted 100 times. The supernatant was added to 980 L of the test mixture; the test mixture consisted of (900 µl 10 mmol / L H2O2. 50 l of Tris HCl buffer (pH 8) and 30 l of water). The degree of decomposition of H2O2 was followed spectrophotometrically at 240 nm.

Gutathione peroxidise (GP_x) [25]

A volume of 0.1 ml of the diluted tissue was incubated at 37 ° C with a reaction mixture consisting of 0.2 ml each of EDTA, sodium azide and H_2O_2. 0.5 ml of TCA were added to this mixture to stop the reaction and then centrifuged at 2000 rpm. 4 ml of disodium hydrogen phosphate and 0.5 ml of 5.5 'dithiobis nitro benzoic acid (DTNB) was added to 0.5 ml of the supernatant, and the color formation was recorded at 420 nm in an optical spectrophotometer.

Histopathological study

Paraffin fragments of the heart sample fixed in buffered formalin were stained with hematoxylin and eosin. The cross-sections were examined under light microscopy and microphotography.

Statistical Analysis:

The data were expressed as mean ± S.E.M from 6 animals [n=6]. The results were

subjected to statistical analysis by using Unpaired t-test to calculate the significance difference if any among the groups. $P<0.05$ was considered as statistical significance using Graph Pad Prism Software.

RESULTS AND DISCUSSION

Preliminary phytochemical screening: (Table 1)

Acute toxicity study: (Table 2)

In the acute toxicity study, no mortality or signs of behavioral change were observed during the 14 days following the single oral administration of *Actinidia deliciosa* juice up to a dose of 2000 mg / kg. Dose levels were chosen such that the dose was approximately one-half (100 mg / kg) one-tenth (200 mg / kg) and one-tenth (400 mg / kg) of the maximum dose used in the Acute toxicity studies, i.e. 2000 mg / kg body weight.

Effect of ADFJ On SGPT, SGOT, CK-MB And GPX Levels In Isoproterenol induced Rats Serum (Table 2)

Effect of ADFJ ON LPO, SOD, CAT and GSH Levels in Isoproterenol induced Rat Brain (Table 3)

Table 1: Preliminary photochemical screening

S. No.	Name of the test	Results
1	Alkaloids	+
2	Tannins	-
3	Glycosides	+
4	Flavonoids	-
5	Phenolics	-
6	Terpenoids	-

Table 2: Effect of ADFJ on SGPT, SGOT, CK-MB and GPX Levels in Isoproterenol induced Rats Serum

Group	Drug treatment	SGPT (U/L)	SGOT (U/L)	CK-MB (IU/mg of protein)	Gpx (µ/mg protein)
I	Normal	35.50 ±2.89	51.33±2.16	82.17 ± 3.21	249.8 ± 6.96
II	Isoproterenol 85 mg/kg/I.P	75.67±0.0225###	117.8±3.38###	166.2 ± 4.98###	133.5 ± 5.33###
III	Propranolol 10 mg/kg/P.O + Isoproterenol 85 mg/kg/I.P	39.67±0.0169***	56.67±2.53***	88.83 ± 3.71***	259.8 ± 7.67***
IV	*ADFJ* 100 mg/kg + Isoproterenol 85 mg/kg/I.P	55.33±0.0155*	76.67±2.35*	130.8 ± 2.46*	212.7 ± 5.40*
V	*ADFJ* 200 mg/kg + Isoproterenol 85 mg/kg/I.P	48.83±0.0148**	67.83±2.40**	114.3 ± 3.30**	241.3 ± 6.60**
VI	*ADFJ* 400 mg/kg + Isoproterenol 85 mg/kg/I.P	42.67±0.0142***	61.50±2.16***	97.17 ± 2.35***	264.2 ± 5.25***

All values are expressed as Mean±SEM, n=6. Data were analyzed by one- way ANOVA followed by post Tukey's multiple comparison test (p<0.05 when compared to normal; p<0.05 when compared to negative control; p<0.05 when compared to positive control; p<0.05 when compared to ADFJ 200; p<0.05 when compared to ADFJ 400)

Table 3: Effect of ADFJ ON LPO, SOD, CAT and GSH Levels in induced Administered Rat Brain

Group	Drug treatment	LPO (nmoles/mg protein)	SOD (µ/mgprotein)	CATALASE (µ/mg protein)	GSH (µ/mg protein)
I	Normal	0.297 ± 0.0098	11.04 ± 0.251	6.252± 0.249	7.23± 0.200
II	Isoproterenol 85 mg/kg/I.P	0.529 ± 0.0225###	6.02 ± 0.311###	2.485 ± 0.139###	3.34 ± 0.198###
III	Propranolol 10 mg/kg/P.O + Isoproterenol 85 mg/kg/I.P	0.341 ± 0.0169***	11.41 ± 0.246***	6.577 ± 0.166***	7.91 ± 0.291***
IV	*ADFJ* 100 mg/kg + Isoproterenol 85 mg/kg/I.P	0.439 ± 0.0155*	8.28 ± 0.302*	5.038 ± 0.138*	6.09 ± 0.193*
V	*ADFJ* 200 mg/kg + Isoproterenol 85 mg/kg/I.P	0.395 ± 0.0148**	9.38 ± 0.276**	6.033 ± 0.140**	.08 ± 0.271**
VI	*ADFJ* 400 mg/kg + Isoproterenol 85 mg/kg/I.P	0.358 ± 0.0142***	10.50 ± 0.256***	7.043 ± 0.153***	8.54 ± 0.212***

All values are expressed as Mean±SEM, n=6. Data were analyzed by one- way ANOVA followed by post Tukey's multiple comparison test (p<0.05 when compared to normal; p<0.05 when compared to negative control; p<0.05 when compared to positive control; p<0.05 when compared to ADFJ 200; p<0.05 when compared to ADFJ 400)

Histopathological study:

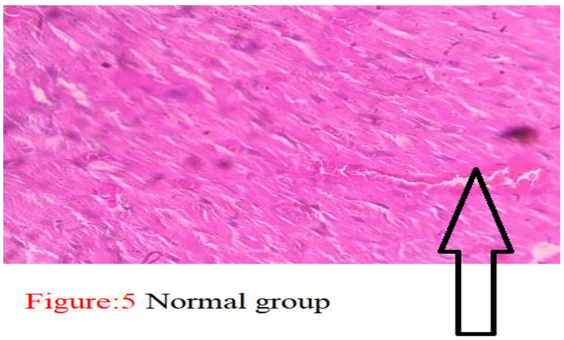

Figure:5 Normal group

Arrow shows intact integrity of myocardial cell membrane, myofibrillar structure with striations and continuity with adjacent myofibrils. The interstitial space appeared intact.

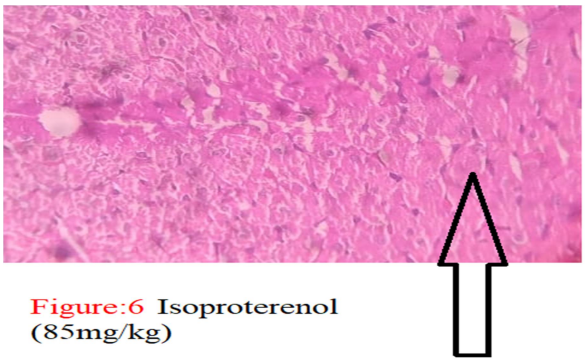

Figure:6 Isoproterenol (85mg/kg)

Arrow shows loss of integrity of myocardial cell membrane, myofibrillar structure with loss of striations and loss of continuity with adjacent myofibrils. The interstitial space at few areas appeared increased.

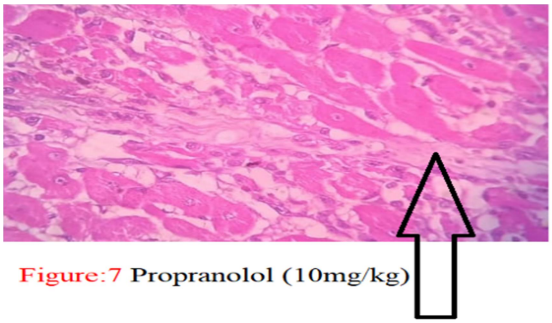

Figure:7 Propranolol (10mg/kg)

Arrow shows integrity of myocardial cell membrane, intact myofibrillar structure with striations and continuity with adjacent myofibrils and scattered inflammatory infiltration

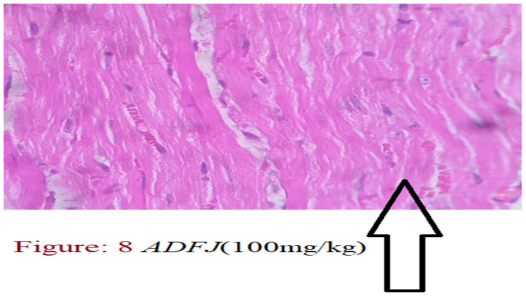

Figure: 8 *ADFJ*(100mg/kg)

Arrow shows cardiac muscle fibers show loss of integrity of myocardial cell membrane, myofibrillar structure with loss of striations and loss of continuity with adjacent myofibrils. The interstitial space at focal areas appeared increas

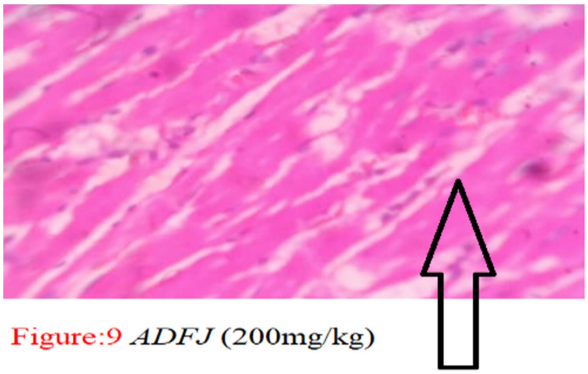

Figure:9 *ADFJ* (200mg/kg)

Arrow shows cardiac muscle fibers show loss of integrity of myocardial cell membrane, myofibrillar structure with loss of striations and loss of continuity with adjacent myofibrils. The interstitial space at focal areas appeared increas

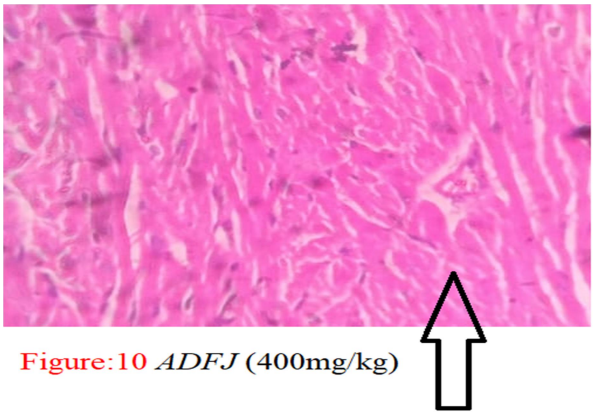

Figure:10 *ADFJ* (400mg/kg)

Arrow shows intact integrity of myocardial cell membrane, myofibrillar structure with striations and continuity with adjacent myofibrils.The interstitial space appeared intact and amidst these

DISCUSSION

Estimation of Biochemical Parameters

The activities of enzymes CKMB, SGOT and SGPT were found to be significantly increased in the isoproterenol treatment group compared with the control group **(Table 3)**, the standard group showed a marked improvement in SGOT, SGPT compared with the control group **(Table 3)**. treatment with ADFJ. The treatment groups showed significantly reduced enzyme levels compared with the isoproterenol-treated group. Significant elevations in serum biochemical enzymes are used as a sensitive indicator of cardiac injury. Elevations of these biomarkers in serum suggest leakage of enzymes from mitochondria due to myocardial injury. This therefore suggests that the degree of cardiomyocyte injury in the pre-treated groups was significantly less. Thus, demonstrating the cardio protective activity of the tested drugs

Estimation of Antioxidant Activity

In the case of oxidative stress for myocardial cells by accumulation of lipid peroxide radicals, the increase in LPO increases in cardiac tissue. Isoproteenol has been significantly increased at the LPO of the induced group. The ADFJ pretreatment group has significantly reduced the LPO level. The standard propranolol has considerably removed the increase in LPO. These results showed that the pre-treated test drug group had a significant impact on LPO inhibition, reducing oxidative stress and maintaining the integrity of the membrane **(Table 4)**.

Isoproterenol induced group showed significant decrease in the SOD, CAT, and GSH levels when compared to the control group. The pre- treated groups of Propranolol, *ADFJ* significantly increased the levels compared to control group. This shows that the trial drug has significant effect on reducing the oxidative stress due to free radicals.

The result of present study revealed an increase in free radical mediated oxidative stress leading to cardiac necrosis and ADFJ showed a significant cardio protective effect against such oxidative stress. Myocardial necrosis can result indirectly due to disturbance in the blood supply to the heart or directly by any chemical insult to the myocyte. When the injury to myocyte is severe the enzymes present in lysosomes leak out of it and enter the cytoplasm. Thus, a promising approach to detect cardiac injury involves monitoring and estimation of certain cytoplasmic enzymes, as they can be detected in blood serum **[26]**. CK-MB, SGPT and SGOT, are some of the diagnostic cardiac

markers of myocardial infarction. According to Robbins, the mitochondrial membrane, plasma membrane and lysosomal membrane are more prone to attack in the event of cell injury. Damage to mitochondrial membrane leads to decline in production of ATP resulting in necrosis **[27]**.

Similarly, an injury to lysosomes leads to emptying of its enzymes in to intracellular fluid. Creatine kinase is a cytoplasmic enzyme which is a dimer with two subunits namely M and B. One of its isoenzymes, CK-MB (creatine kinase).The result of present study revealed an increase in free radical dative stress leading to cardiac necrosis and ADFJ showed a significant cardioprotective effect against such oxidative stress. Myocardial necrosis can result indirectly due to disturbance in the blood supply to the heart or directly by any chemical insult to the myocyte. Similarly, an injury to lysosomes leads to emptying of its enzymes in to intracellular fluid. Creatine incase is a cytoplasmic enzyme which is a dimer with two subunits MB (creatine kinase-muscle brain) is abundant in myocardium. Any elevation of the ck-mb fraction in serum is a reliable indication of myocardial infarction scrupulous study on the effect of isoproterenol induced myocardial damage has reported elevated levels of this marker enzyme inserum **[28]**.

However, pretreatment with ADFJ groups recorded a decrease in activity levels of enzyme CK-MB. The accordance with the previous reports since an increase in SGPT and SGOT enzymes are observed in isoproterenol treated groups. However, pretreatment with ADFJ groups recorded a decrease in activity (SGOT, SGPT) suggesting the cardio protective potential of the ADFJ. Additionally, in 2014, Khan et al reported that methanol extract of leaves ofits kupchan fractions exhibited better cytoto stabilizing and thrombolytic activities **[29]**. ADFJ consisted of array of phytoconstituents such as triterpenes [beta-hydroxyfriedalanol], phenols [3 methoxy stilbene], steroids [sitosterol muscle brain) is abundant in myocardium. Any elevation of the ckmb fraction in serum is a reliable indication of myocardial infarction. Scrupulous study on the effect of isoproterenol induced myocardial damage has reported elevated levels of this marker enzyme inserum **[28]**. Since ADFJof consisted of array of phytoconstituents such hydroxyfriedalanol], phenols[3-hydroxy-5methoxystilbene],steroids[sitosterol-(6-O-palmitoyl)-3-O-ß-Dglucopyranoside and sitosterol-3-O-ß-D glucopyranoside],

sesquiterpenes [2,3-dihydroxy isodrimeninol] etc., administration of this juice might prevent the leakage of biomarkers in to bloodstream by stabilizing the membranes **[30]**.

The quantity and composition of lipids in the myocardium is also a determinative factor for development of cardiovascular disease. Isoproterenol is a synthetic catecholamine that stimulates beta adrenergic receptors and adenylate cyclase which results in theelevation of cAMP **[31]**. Also, free radicals produced from catecholamines activates cAMP-dependent protein kinases, I and II (PKAI and -II,) resulting in lipid accumulation in the myocardium. Free radicals also alter the structure of PK (phosphokinase enzyme) enzymes by causing a loss of a tryptophan residue from site A of phosphokinase enzyme **[32]**.

Catalase is a peroxisomal enzyme present in mitochondria of heart. It is one of the antioxidant defense enzymes which play a indispensable role in the oxidation of hydrogen peroxide to oxygen and water. Previous studies reported that cardiotoxicity was reduced in transgenic mice by 60-100-fold over expression of catalase citing the contribution of catalase in detoxification of hydrogen peroxide **[33**]. Similarly, SOD is another ROS defense enzyme present exclusively in the mitochondrial matrix and its prime function is to assist in dismutation of superoxide radicals. In the current study, isoproterenol treated groups exhibited a depletion or decrease in the amount of catalase and SOD resulting in intense myocardial necrosis **[34]**.

Administration of ADFJ to isoproterenol challenged rats reinstated the antioxidant enzyme levels to normalcy. This enhancement in antioxidant support system of myocardium might be due to the scavenging of free radicals ADFJ.

The glutathione antioxidant system including GSH, and GPx plays a crucial role in cellular defense against oxidative stress by upholding a cascade of reactions. Glutathione peroxidase (GPx) is a selenoprotein that oxidizes two molecules of glutathione (GSH) into oxidized glutathione (GSSG). This oxidation is favoured by the formation of a disulphiram bond. Glutathione reductase catalase the reduction of GSSG in to glutathione. Thus, glutathione is rapidly oxidized and regenerated in the cell **[35]**. The reduction in non-enzymatic GSH and enzymatic GPx in isoproterenol treated rats might be due to the influence of free radicals on antioxidant system. In present study, glutathione levels depleted by isoproterenol were significantly elevated by ADFJ. It was

understood that increased levels of GSH, GPx could be because of presence of flavonoids in ADFJ.

In the present study, elevation of lipid peroxidation in the heart ofrats treated with isoproterenol was observed. The increase in malondialdehyde levels in heart indicates excessive lipidperoxidation **[36]**. Free radicals produced from isoproterenol led to irreversible tissue damage in heart by activation of lipidperoxidation. Treatment with ADFJ significantly decreased the lipid peroxidation induced membrane damage. The cardioprotective effect of the ADFJ was further approved by histopathological examinations. ADFJ furnished cardio protection at different dose levels. Altogether, morphological and histological changes such asdecrease in swelling, lesser accumulation of fatty acids and lesser degree of karyolysis rendered considerable evidence for the cardio protective activity of ADFJ.

CONCLUSION

The results of this study concluded that *Actinidia deliciosa* (ADFJ) fruit juice had cardio protective activity against isoproterenol induced myocardial infarction in albino rats. The data obtained from the study are consistent with the concept that free radicals formed from isoproterenol play a major role in the induction of myocardial infarction. ADFJ restores the levels of heart enzymes (CKMB, SGOT, SGPT) and antioxidant parameters (CAT, SOD, GSH, GPx and LPO) to normal levels. Therefore, it can be concluded that 400 mg / kg showed significant cardio protective activity compared to 100 mg / kg and 200 mg / kg, probably because the active component of the flavonoids present in ADFJ could be the cause of the positive response against oxidative stress of myocytes. Histopathological results also confirmed the protective effect of *Actinidia deliciosa*. Further studies are recommended to elucidate the mechanism of cardio protective action of this fruit juice and to identify its active agents.

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