The Possible Ameliorative Influence of Quercetin on Cardiac Muscle Changes induced by High Fat Diet in Adult Male Albino Rats: Light and Electron Microscopic Study

Original Article

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ABSTRACT

Introduction: Cardiovascular diseases is a primary health concern. Recent studies have demonstrated that high-fat diet (HFD)-induced obesity could lead to cardiac dysfunction, hypertrophy, fibrosis and even heart failure. Quercetin (Q) is a common nontoxic polyphenol having a broad range of pharmacological and biological activities. The present study aimed at evaluating the histological and ultrastructural effects of high fat diet on cardiac muscle of adult male albino rat and the possible protective role of quercetin.

Materials and Methods: Forty adult male albino rats were used and divided into four groups. Group I: Control group (10% of energy from fat). Group II: Quercetin group (10% of energy from fat + 60 mg/kg quercetin). Group III: HFD group (60% of energy from fat). Group IV: HFDQ group (60% of energy from fat + 60 mg/kg quercetin).

At the end of experiment after12 weeks, all rats were sacrificed, and hearts were taken and processed for light (H&E and Mallory's trichrome stains) and electron microscopic examination. Histological, morphometric and statistical studies were performed.

Results: Sections from rats of group III showed widely separated cardiomyocytes, some atrophic degenerated fibers, vacuoles in the cytoplasm, engorged capillaries and marked increase in collagen deposition. Electron microscopic examination showed disorganized fragmented myofibrils, few swollen mitochondria and bizarre indented nuclei. Sections from rats of group IV revealed obvious improvement of the previous alterations. There was highly statistically significant difference between group I and group III and between group II and group III as regard collagen area percent.

Conclusion: The results obtained from the current study confirmed the negative influence of high fat diet intake on cardiomyocytes. Concomitant administration of quercetin markedly improved myocardial damage so, it could be used as an effective cardioprotective agent against hyperlipidemia and aging insults.

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Key Words: Cardiac muscle, EM, HFD, quercetin.

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INTRODUCTION

In recent years, cardiovascular diseases (CVD) have emerged as a major public health issue around the world[1]. The combination of genetic and environmental factors comprising a sedentary lifestyle is a well-known cause of cardiovascular disease, which is worsened by metabolic conditions, for example hyperglycemia, hyperlipidemia, overweight, and obesity. The worldwide progressive increase in consumption of appetizing junk food rich in sugar and fat is yet representing an alarming sign and is adding to the disease liability. Physical lethargy is often related to a decline in health[2].

Lately, the onset of CVD pathology in children and adolescents is a well-known clinical problem and early biochemical determinations prevent numerous CVD[3].

Recent research has shown that many cardiac disorders for instance, myocardial hypertrophy[4], interstitial fibrosis[5], heart failure[6] and many other forms of cardiac dysfunction[7] are caused by obesity resulting from high-fat diet (HFD). Although the exact underlying complex mechanism is still not well grasped, these cardiac disorders are thought to be linked to the triggering of apoptosis, inflammation, and cellular oxidative stress[8].

A number of negative health concerns are observably related to the oxidative stress resulting from high consumption of fatty diet and subsequent obesity[9,10].

The disproportion between prooxidative and antioxidative dynamics resulted in oxidative stress phenomenon[11]. Oxidative impairment to cellular proteins and membranes often impairs cardiac function, leading to

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cardiomyocyte dysfunction and/or death via apoptosis and necrosis[12].

The myocardial contractility requires an increasing demands of adenosine triphosphate (ATP) obtained from oxidation of fatty acids which is aggravated by HFD[13].

Flavonoids have also been shown to have cardiovascular effects[14] such as reducing blood pressure[15] and stimulating lipid-lowering effects in the treatment of dyslipidaemia[16]. A wide scope of fruits and vegetables comprise quercetin which is one of the most popular flavonoid polyphenols consumed on a daily basis by the general public[17].

Quercetin is nontoxic and has anticarcinogenic, antioxidative, vasoprotective, antidiabetic and antiplatelet properties[18].

Previous researches have shown that quercetin has a significant clinical effect on a variety of CVD symptoms. Various research groups have looked into the possible systemic, cellular, and molecular pathways engaged in quercetin's cardiovascular safety in recent years. The majority of the findings suggested that quercetin has a pleiotropic effect, as it can reduce cardiac hypertrophy, infarct areas, oxidative strain, and inflammation in animal models of human cardiovascular diseases[19-21].

As most of the previous studies concerned with HFD impact on the heart and the possible protective role of quercetin were based on clinical and/or biochemical effects, our present study aimed at evaluating the same effect histologically and ultrastructurally on cardiac muscle of adult male albino rat.

MATERIALS AND METHODS

Materials

Quercetin (manufactured by Sigma-Aldrich, Egypt) (60 mg/kg/day) liquefied in olive oil and was introduced orally to rats by oral gavage[22].

Animals

In isolated cages, forty adult male albino rats (weight 150-200 gm) were kept in a constant temperature (22-24 °C) and light-controlled room on an alternating12:12 h light-dark.

In sanitary environment, the animals were kept, fed ad libitum, and had unlimited access to water. They were kept for one week before beginning the experiment for acclimatization. The experiment was carried out in the Animal House of Kasr Al Einy, Faculty of Medicine, Cairo University for 12 weeks. The rats were treated in accordance with guidelines approved by the Animal Use Committee of Cairo University. The rats were divided into four main groups (10 rats each):

Group I: Control group (10 percent of energy from fat).

Group II: Quercetin group (10 percent of energy from fat + 60 mg/kg quercetin) once orally daily until the end of experiment.

Group III: HFD group (60 percent of energy from fat). Every gram of its ingredient contains 5.24 kcal, including 232 mg cholesterol.

The control diet was comprised of 13.5 percent of fat, 61.3 percent of carbohydrate and 25.2 percent of protein (total energy content: 2830 kcal/kg),whilst HFD consisted of 60 percent of fat, 20 percent of carbohydrate, and 20 percent of protein based on total energy content of 5243 kcal/kg[23].

Group IV: HFDQ group (60 percent of energy from fat + 60 mg/kg quercetin) once orally daily until the end of experiment.

At the end of experiment, all rats were sacrificed by overdose of anesthesia (100 mg/kg ketamine-xylazaine IP[24].

Hearts were taken and processed for

Light microscopic examination

Specimens from the left ventricle were dissected then processed for general histological examination using Hematoxylin & Eosin (H&E)[25] and Mallory's trichrome stains[26].

Electron Microscopic examination (Transmission electron microscopy)

For semithin and ultrathin sections, small specimens from left ventricles were fixed in 2-3% Glutaraldehyde, kept in the fridge overnight then processed and post-fixed in osmium tetroxide. Semithin sections were cut, stained with toluidine blue stain, examined and photographed then used to select specific areas for preparing ultrathin sections of the electron microscope. In the Faculty of Agriculture, Cairo University, Electron Microscope unit, ultra-thin sections were cut and stained with uranyl acetate and lead citrate, then checked and photographed with a JEOL (JEM-100cx) transmission electron microscope[27].

Morphometric study

Measurement of the area percent of collagen stained with Mallory's trichrome of the four tested groups took place at the Beni-suef University, Faculty of Veterinary Medicine, using image analyzer computer system with Leica Qwin 500 software (Cambridge, England). At a magnification of 400, ten randomly chosen non-overlapping nominated fields were assessed for each section

Statistical analysis

Comparison between the different groups in morphometric results was calculated by analysis of variance (ANOVA) followed by post hoc tukey test using version 24 of the statistical package SPSS (Statistical Package for the Social Sciences). The differences were considered statistically significant if probability value (*P-value*) was less than 0.05 and highly significant if *P value* less than 0.001[28].

RESULTS

No deaths were observed in all rats.

A. Light microscope examination

1- H&E stained sections

Examination of group I (standard diet) and group II (standard diet + quercetin) showed normal architecture of cardiac muscle with striated branching cardiomyocytes, acidophilic cytoplasm, central oval vesicular nuclei, intercalated discs and intervening blood capillaries (Figures 1A,B). Sections from rats of group III (HFD) showed widely separated cardiomyocytes and the cytoplasm showed some vacuoles. Some fibers were atrophic and degenerated. Capillaries were markedly engorged (Figure 1C). Sections from rats of group IV (HFD + quercetin) showed apparently normal architecture of cardiac muscle with almost regular cardiomyocytes with central oval vesicular nuclei (Figure 1D).

2- Mallory's trichrome stained sections

Collagen deposition between cardiomyocytes and around blood capillaries were found to be minimal in group I and group II (Figures 2A,B). Collagen fiber deposition in between cardiomyocytes and around blood capillaries was significantly increased in group III (Figure 2C). Sections of group IV showed little collagen deposits in between cardiomyocytes and around blood capillaries (Figure 2D).

B. Electron microscope examination

Examination of ultrathin sections of control rats of group I (standard diet) and group II (standard diet + quercetin) showed cardiomyocytes with regularly arranged parallel myofibrils and numerous mitochondria in between. The myofibrils were formed of sarcomeres. Each sarcomere extended in between two successive Z lines with alternating dark bands and light bands. Z line bisected the light band. Euchromatic nuclei were seen with prominent nucleoli. The adjacent cardiomyocytes were connected by intercalated disc. Blood capillaries were detected (Figures 3A,B).

Sections of group III (HFD) showed cardiomyocytes with disorganized fragmented myofibrils which varied in thickness between thick and thin with areas of focal lysis. Mitochondria were fewer, malformed, disarranged and swollen. The sarcoplasm revealed many large vacuoles and areas of rarefaction. The sarcolemma was thickened in some points. Intercalated discs were interrupted and disfigured. A bizarre nucleus with indentation was detected. Thick collagen bundles were deposited in between cardiomyocytes. Blood capillaries lined by swollen endothelium were spotted (Figures 3C-i,ii) and (Figures 4C-i,ii,iii).

Sections of group IV (HFD + quercetin) showed cardiomyocytes with regular parallel myofibrils and regular sarcomeres in most areas, but in other areas, the myofibrils appeared thin atrophic and separated.

Apparently normally looking mitochondria in between the myofibrils were seen with few encountered swollen ones. The sarcoplasm showed small vacuoles. The nuclei appeared normal and euchromatic. (Figure 3D) and (Figures 4D-i,ii).

C-Morphometric results

Statistical analysis of the data

There was highly statistically significant difference between four groups as regard collagen area percent which was more significantly increased in group III followed by group IV (Histogram 1, Table 1).

There was highly statistically significant difference between group I and group III and between group II and group III as regard collagen area percent, also there were highly statistically significant differences between group IV and each of group I, II and group III as regard collagen area percent (Histogram 1, Table 2).

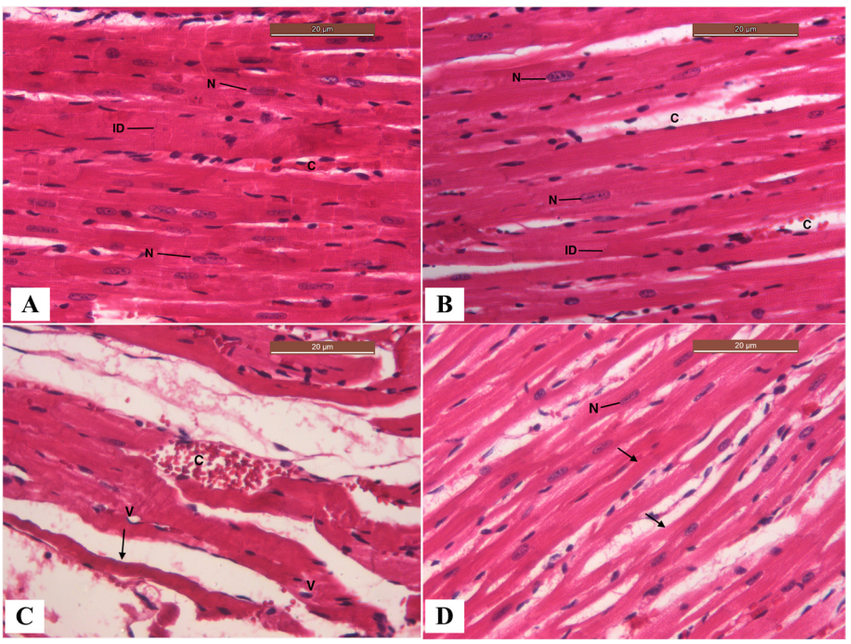


Fig. 1: Photomicrographs of the left ventricle in the different studied groups. (A) Section from a control rat group I (standard diet) and (B) Section from a rat of group II (standard diet + quercetin) showing normal architecture of cardiac muscles with striated branching cardiomyocytes with acidophilic cytoplasm, central oval vesicular nuclei (N), intercalated discs (ID) and intervening blood capillaries (C). (C) Section from a rat of group III (HFD) showing widely separated cardiomyocytes, cytoplasm shows some vacuoles (V). Some fibers are atrophic and degenerated (arrow). Capillaries are markedly engorged (C). (D) Section from a rat of group IV (HFD + quercetin) showing apparently normal architecture of cardiac muscles with almost regular cardiomyocytes (arrow) with central oval vesicular nucleus (N). (H&E X 400)

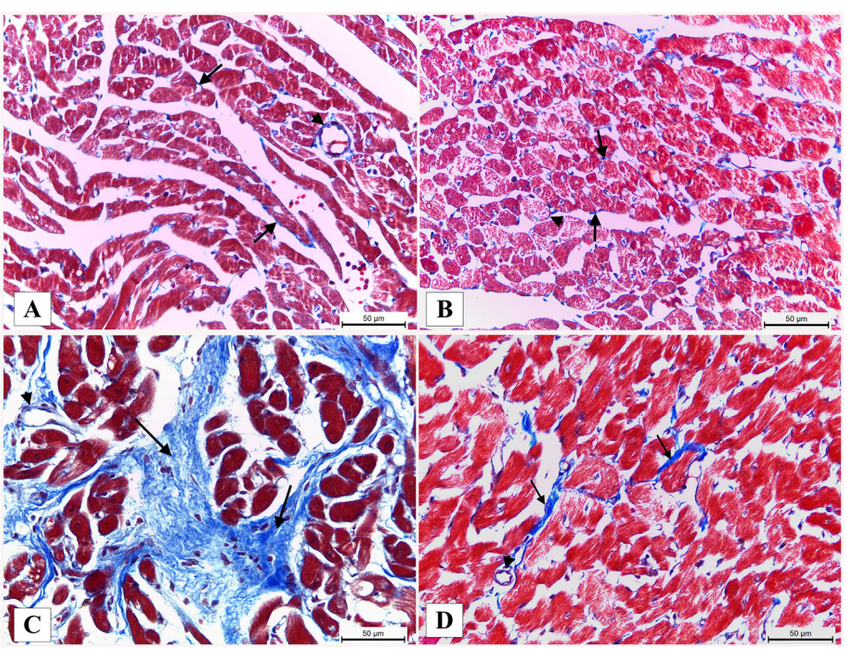


Fig. 2: Photomicrographs of the left ventricle in the different studied groups. (A) Section from a control rat (group I) and (B) Section from a rat of group II showing minimal amount of collagen in between cardiomyocytes (arrows) and around blood capillaries (arrowheads). (C) Section from a rat of group III showing marked increase in collagen fibers deposition in between cardiomyocytes (arrows) and around blood capillaries (arrowhead). (D) Section from a rat of group IV showing little collagen deposits in between cardiomyocytes (arrows) and around blood capillaries (arrowhead) (Mallory's trichrome X 400)

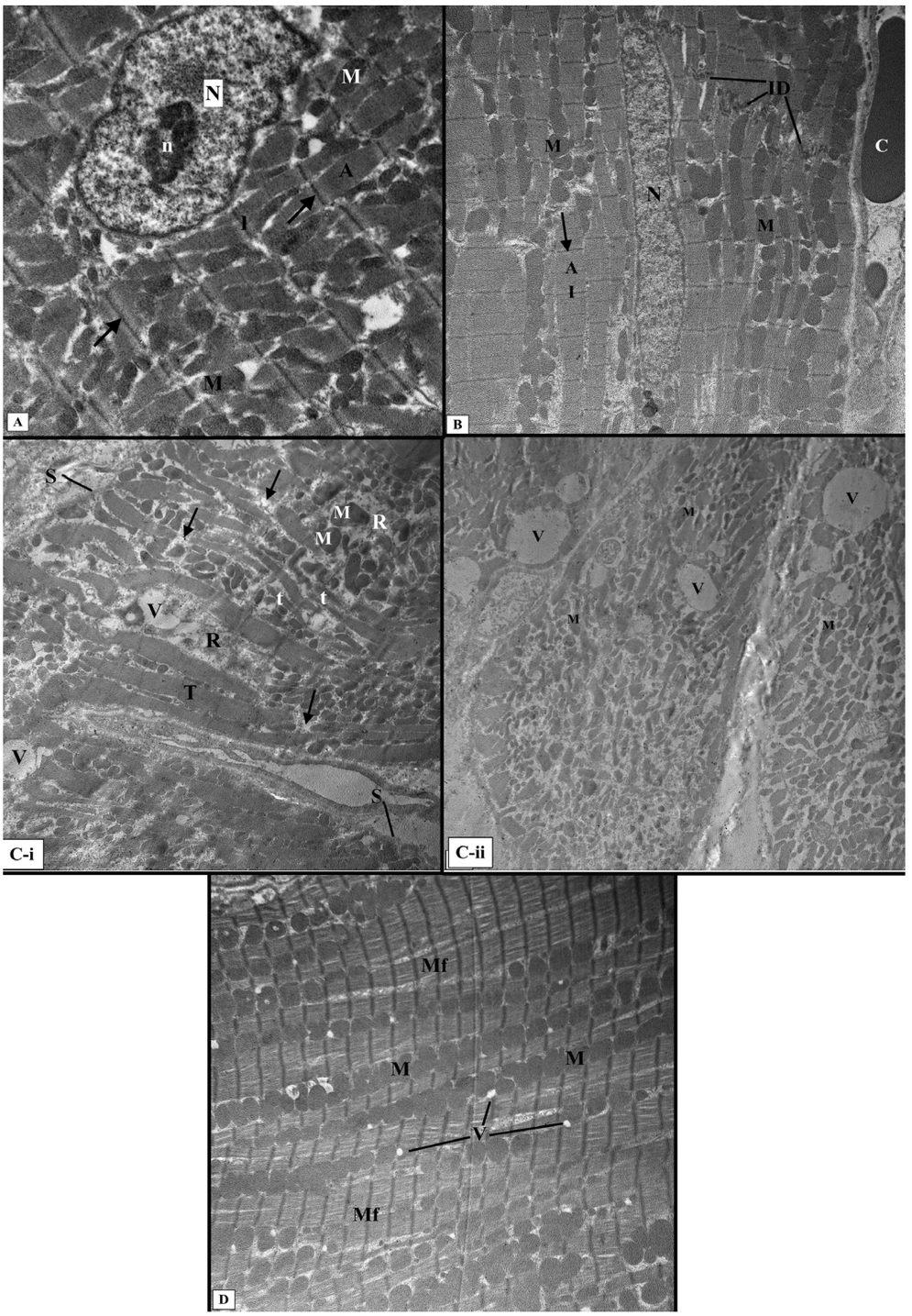


Fig 3: TEM photomicrographs revealing: (A) Section from a control rat group I showing cardiomyocytes with regularly arranged myofibrils and numerous mitochondria in between (M). Myofibrils are formed of sarcomeres. Each sarcomere extends in between two successive Z lines (arrows) with alternating dark (A) bands and light (I) bands. Z line bisects the light (I) band. A single euchromatic nucleus is seen (N) with prominent nucleolus (n) (X 8000). (B) Section from a rat of group II showing the same ultrastructural features of control group in the form of cardiomyocytes with regular myofibrils and numerous mitochondria in between (M). Sarcomeres are seen with alternating dark (A) band and light (I) bands bisected by Z line (arrow). Elongated euchromatic nucleus with extended chromatin (N). The adjacent cardiomyocytes are connected by intercalated disc (ID). A blood capillary (C) can be seen (X 5000). (C-i) Section from a rat of group III showing cardiomyocytes with disorganized fragmented myofibrils which vary in thickness between thick (T) and thin (t) ones with areas of focal lysis (arrows). Mitochondria are malformed and swollen (M). The sarcoplasm shows many large vacuoles (V) and areas of rarefaction (R). The sarcolemma is thickened in some points (S) (X 5000). (C-ii) Section from a rat of group III showing cardiomyocytes with severely disorganized myofibrils, malformed disarranged mitochondria (M) and many large sarcoplasmic vacuoles (V) (X 3000). (D) Section from a rat of group IV showing cardiomyocytes with regular parallel myofibrils (Mf) and regular sarcomeres. Apparently normal mitochondria in between the myofibrils (M). The sarcoplasm shows multiple small vacuoles (V). (X 4000)

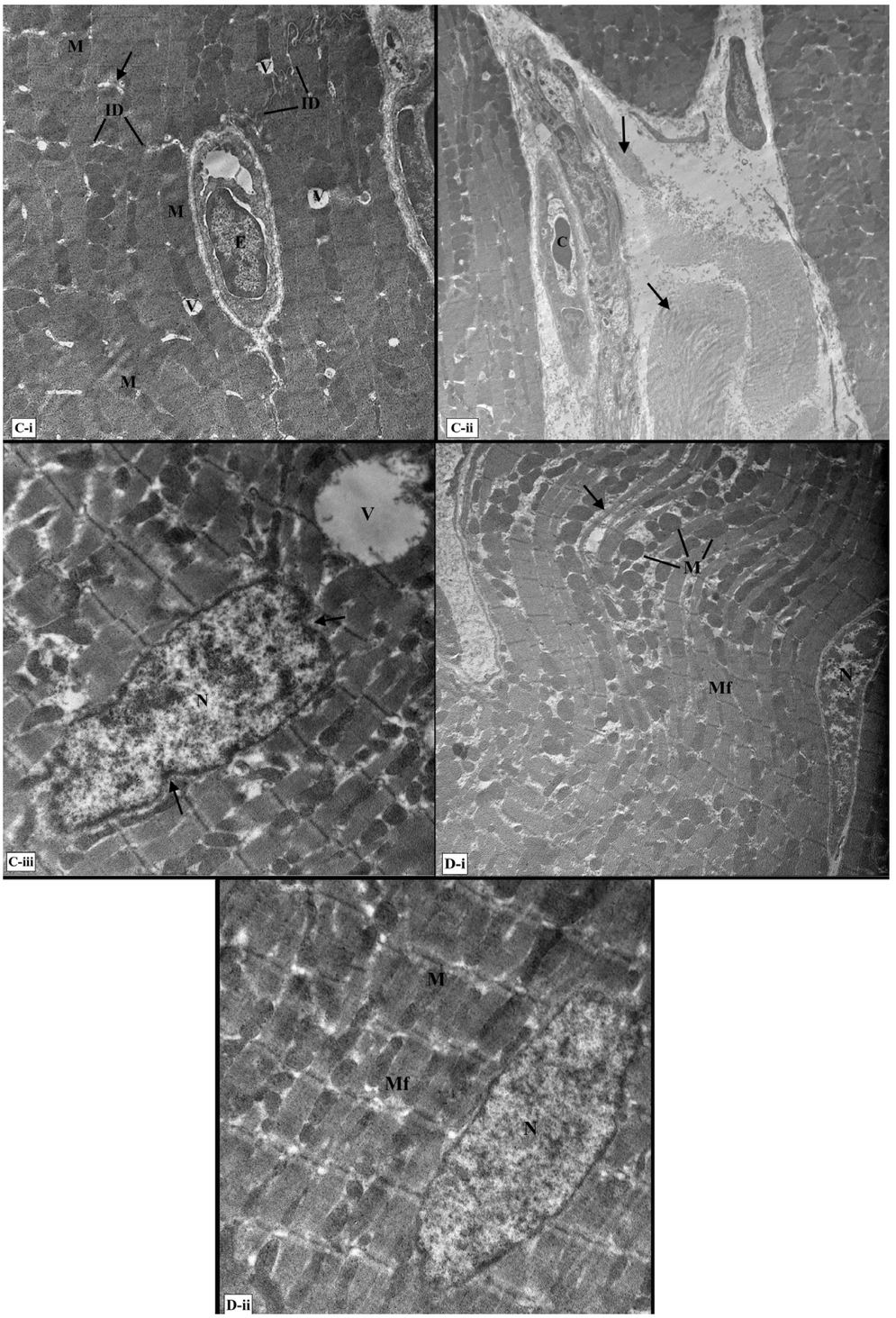


Fig 4: TEM photomicrographs revealing: (C-i) Section from a rat of group III showing cardiomyocytes with decreased number and swollen mitochondria (M). Sarcoplasm shows many vacuoles (V). Intercalated discs (ID) are interrupted and disfigured. Focal areas of sarcoplasmic rarefication can be seen (arrow). A blood capillary is present in between cardiomyocytes and lined by a swollen endothelial cell (E) (X 6000). (C-ii) Section from a rat of group III showing thick collagen bundles in between cardiomyocytes (arrows). A blood capillary can be seen (C) (X 3000). (C-iii) Section from a rat of group III showing cardiomyocytes with a large sarcoplasmic vacuole (V) and bizarre nucleus (N) with indentation (arrows) (X 8000). (D-i) Section form a rat of group IV showing cardiomyocytes with apparently normal nucleus (N). The myofibrils appear regular in some areas (Mf) and thin atrophic and separated in others (arrow). Mitochondria are swollen (M) (X 5000). (D-ii) Section from a rat of group IV showing cardiomyocytes with regularly arranged myofibrils (Mf) and normally looking mitochondria in between (M). The nucleus is oval and euchromatic (N) (X 8000).

Table 1: Comparison between the studied groups according to the collagen area percent ± Standard deviation

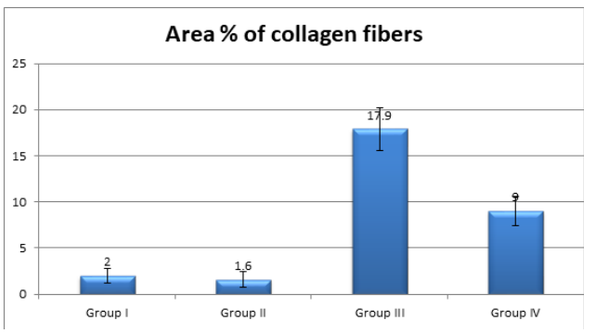
	Group I (n = 10)	Group II (n = 10)	Group III (n = 10)	Group IV (n = 10)
Area%	2 ± 0.8	1.6 ± 0.8	17.9#$ ± 2.3	9#$@ ± 1.6

#: Significant with Group I $: Significant with Group II
@: Significant with Group III *: Statistically significant at $p \leq 0.05$
Group I: Control group Group II: Quercetin group
Group III: HFD group Group IV: HFDQ group

Table 2: Pairwise comparison between each 2 groups was done using Post Hoc Test (Tukey)

The collagen area percent	Group I (n = 10)	Group II (n = 10)	Group III (n = 10)	Group IV (n = 10)
Group I		0.948	<0.001*	<0.001*
Group II			<0.001*	<0.001*
Group III				<0.001*
Group IV				

*: Statistically significant at $p \leq 0.05$.



Histogram 1: Histogram showing collagen area percent among studied groups.

DISCUSSION

Cardiovascular disease is the leading cause of death in low and middle-income countries, putting public health systems and developed economies under stress[29]. Even when followed for a short period of time, studies have found a strong connection between HFD intake and metabolic and cardiovascular diseases[30].

The HFD caused a rise in blood pressure, pulse pressure, vascular sympathetic function, and dyslipidemia, all of which are risk factors for CVD and death[31].

In the current study, effects of high fat diet on cardiac muscle of adult male albino rat and the possible protective role of quercetin are studied histologically.

The results of the current study revealed that on light microscopic examination, sections from group I and group II showed normal architecture of cardiac muscles with striated branching cardiomyocytes, acidophilic cytoplasm, central oval vesicular nuclei, intercalated discs and intervening blood capillaries. The results of current study are in concurrence with Dawood and Hareedy[32] who observed that the normal pattern of the control group revealed individual cylindrical cardiac myocytes that branch and anastomose forming three-dimensional network. Their sarcoplasm was highly acidophilic and crossly striated with a single, central and oval vesicular nucleus. Intercalated discs appeared as deeply stained transverse lines among myocytes.

In the current study, sections from rats of group III showed widely separated cardiomyocytes with some vacuoles in their cytoplasm. Some fibers were atrophic and degenerated. Capillaries were markedly engorged. These results are in accordance with Dawood and Hareedy[32] who that many muscle fibers were lost with subsequent marked widening of intercellular spaces. Some inflammatory cell infiltrate and congestion of blood vessels were noticed. Some myocytes showed focal degeneration while some others had wavy corrugated appearance. Sarcoplasm was pale, disintegrated and lost its cross striations and nuclei were dense and pyknotic. The observed cardiac muscle damage could be attributed to serious effects of hyperlipidemia that can be evoked at molecular and cellular levels.

The cellular oxidative impairment is highly facilitated by consumption of high-cholesterol diet with the resultant lipid peroxidation and production of large quantities of reactive oxygen species[33].

Hyperlipidemic rats have a disrupted antioxidant enzyme functions resulting in an antioxidant/oxidant imbalance[34].

Saturated fats are the heart's primary metabolic fuel[35-38], and an excess of lipid can, paradoxically, induce mitochondrial overload and trigger cardiac remodelling molecular mechanisms[37]. So, cardiac remodeling is a consequence of lipotoxicity[37] and/or oxidative stress[38]. Ceramide and diacylglycerol, which are potential substrates mediating intracellular molecular pathways of injurious non-oxidative mechanisms such as apoptosis, interstitial myocardial fibrosis and hypertrophy of cardiomyocytes, are mainly caused by the accretion of the products of lipid metabolization[37].

Furthermore, oxidative injury to DNA and cell's proteins by oxygen free radicals commonly occurs in the course of lipid oxidation. They were also linked to pathways that regulate myocardial hypertrophy and interstitial space remodelling[38].

The present study revealed that sections from rats of group IV showed apparently normal architecture of cardiac muscles with almost regular cardiomyocytes with central oval vesicular nuclei.

These findings are in line with Takizawa *et al.*,[39] who stated that quercetin is a natural antioxidant that works by inhibiting lipid peroxidation via the xanthine oxidase enzyme and directly scavenging cytotoxic free radicals. Quercetin has a potent anti-inflammatory activity as it inhibits inducible nitric oxide synthase expression[40].

Because of its ability to increase antioxidant enzyme levels and scavenge lipid peroxides, quercetin's beneficial effects can be attributed to its antioxidant properties. As a result, it was considered that quercetin is a promising natural cardioprotective agent[41].

In the present study, examination of Mallory's trichrome staining in group II showed minimal amount of collagen deposition in between cardiomyocytes and around blood capillaries. Sections from rats of group III showed marked increase in collagen fibers deposition in between cardiomyocytes and around blood capillaries. Sections from rats of group IV showed little collagen deposits in between cardiomyocytes and around blood capillaries.

It was stated that myocardial hypertrophy, fibrosis and many forms of cardiac dysfunction manifesting the lipotoxic myocardial pathology is greatly aggravated by HFD that can cause disproportion in myocardial fatty acid absorption and utilization with the resultant accretion of cardiotoxic lipid species[42].

Due to the fact that both fibrosis and collagen deposition affect the mechanical criteria of the myocardium, Mallory's trichrome stain was used to identify the fibrotic areas. High fat diet aggravated interstitial and perivascular fibrosis within the myocardium. This was linked to enhanced expression of myocardial gene of collagen type 1 (Col1A1) and 3 (Col3A1) pro-1 chains[43].

Another study in rabbits confirmed that a high-fat diet for 12 weeks caused fibrosis in coronary vessels as well as collagen accretion in the interstitial tissue of myocardium[44].

This is in accordance with a previous study which postulated that myocardial fibrosis is the resultant of increased levels of endothelin, cytokines, and renin-angiotensin-aldosterone that occur in cases of obesity[45].

Wang *et al*.,[46] highlighted the effect of quercetin dihydrate that could appreciably prevent cardiac fibrosis by hindering the proliferating and migrating fibroblasts in *vitro*. In addition, quercetin could inhibit the expression of Collagen I and Collagen III, which are the markers of fibroblast differentiation. Furthermore, it was postulated that quercetin dihydrate prevent cardiac oxidative stress, inflammation, and cardiac diastolic dysfunction brought by Angiotensin II. Consequently, quercetin should be considered as a propitious new therapy for myocardial dysfunction and fibrosis depending upon these outcomes.

In our current study, these results are confirmed by highly statistically significant difference between the four groups as regard collagen area percent which was more significantly increased in group III compared with group IV.

On ultra-structural examination, the results of current study revealed that the section from rats of group III showed cardiomyocytes with disorganized fragmented myofibrils which varied in thickness with areas of focal lysis. Mitochondria were few, disarranged, malformed and swollen. The sarcoplasm revealed many large vacuoles and areas of rarefaction. The sarcolemma was thickened in some points. Intercalated discs were interrupted and disfigured. Blood capillaries in between cardiomyocytes were lined by swollen endothelium. Thick collagen bundles were deposited in between the cardiomyocytes.

This is in agreement with Leopoldo *et al*.,[47] who recorded in a previous study that the myocardium of rats fed on a high-fat diet had significant ultrastructural abnormalities in cardiomyocytes, including the existence of large quantities of lipid droplets within the sarcoplasm between myofibrils, distended sarcoplasmic reticulum vesicles, the absence and/or disordered myofilaments, and mitochondrial changes.

It was revealed in a previous study that the nuclei of most hepatocytes in rats receiving high fat diet showed, condensed chromatin, irregularity, indentation, and widening of the perinuclear space[48]. Many degenerative changes in mitochondria within cardiac sections induced by high-fat diet, including cristolysis, matrix dilution, and mitochondria-associated lamellar bodies, but these morphological changes were not linked to cardiac activity[49].

Sections from rats of group IV showed cardiomyocytes with regular parallel myofibrils and regular sarcomeres but in few areas, the myofibrils were thin, atrophic and separated. Apparently, the majority of mitochondria were normal however, few of them were swollen. The sarcoplasm showed occasional small vacuoles. The nuclei appeared normal, oval and euchromatic without nuclear membrane indentation.

Previous studies have disclosed that the favorable effects of quercetin included the launch of mitochondrial biogenesis through PGC-1α (Peroxisome proliferator-activated receptor gamma coactivator1-alpha), which is a transcriptional co-activator of genes linked with oxidative phosphorylation and mtDNA replication[50,51].

It was proposed that pre-neonatal treatment of primary cardiomyocytes with quercetin before anoxia/reoxygenation had dramatically lessened the subsequent apoptosis in injury through many mechanisms for instance, reduction of ROS generation, amending the rate of cell survival, preventing the opening of mitochondrial permeability transition pores (mPTP) and eluding the collapse of the mitochondria membrane potential. Moreover, in the same study, the authors assumed that the expression of protein kinase C (PKC) and the activity of downstream mediators of its pathway can be enhanced and improved by quercetin implying its cardioprotective effects[52].

The beneficial effects of quercetin in inhibiting cardiac dysfunction and several pathologies in animal models and various cell culture were comprehensively studied by Ferenczyova *et al*[53]. So, regarding its proven role in avoiding and treatment of various cardiac disorders, many

authors proposed that quercetin and its byproducts may be auspicious cardioprotective agents.

CONCLUSION

Quercetin, on histological and ultrastructural levels, showed positive cardiovascular beneficial effect by ameliorating and improving the harmful consequences of high fat diet on cardiac muscle. So, quercetin could be a promising natural cardioprotective factor but clinical researches on a greater number of patients are still required.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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الملخص العربى

التأثير المحسن المحتمل للكيرسيتين علي تغيرات عضلة القلب الناجمة عن النظام الغذائي عالي الدهون في ذكور الجرذان البيضاء البالغة: دراسة مجهرية ضوئية و إلكترونية

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الخلفية والأهداف: تعتبر أمراض القلب والأوعية الدموية من الشواغل الصحية الأولية. أظهرت الدراسات الحديثة أن السمنة الناتجة عن اتباع نظام غذائي عالي الدهون يمكن أن تؤدي إلى خلل في وظائف القلب وتضخم وتليف وحتى قصور في القلب. كيرسيتين بوليفينول شائع الاستخدام غير سام وله مجموعة واسعة من الأنشطة الدوائية والبيولوجية. هدفت الدراسة الحالية إلى تقييم التأثيرات النسيجية والتركيبية للنظام الغذائي عالي الدهون على عضلة القلب لدى ذكور الجرذان البيضاء البالغة والدور الوقائي المحتمل للكيرسيتين.

المواد والطرق: تم استخدام أربعين من ذكور الجرذان البالغة وتم تقسيمها إلى أربع مجموعات المجموعة الأولى: المجموعة الضابطة (١٠٪ من الطاقة من الدهون) المجموعة الثانية: مجموعة كيرسيتين (١٠٪ من الطاقة من الدهون + ٦٠ مجم / كجم كيرسيتين يوميا) المجموعة الثالثة:HFD مجموعة النظام الغذائي عالي الدهون (٦٠٪ من الطاقه من الدهون) المجموعة الرابعة: HFDQمجموعة (٦٠٪ من الطاقه من الدهون + ٦٠ مجم / كجم كيرسيتين يوميا) في نهاية التجربة بعد ١٢ أسبوعًا، تم التضحية بجميع الجرذان ، وتم أخذ القلوب ومعالجتها للفحص المجهري الضوئى (بصبغة الهيماتوكسيلين و الايوسين و المالوري ترايكروم) (H&E and Mallory's trichrome) أجريت الدراسات النسيجية والمورفومترية والإحصائية

النتائج: أظهرت المقاطع المأخوذة من جرذان المجموعة الثالثة وجود خلايا عضلية للقلب منفصلة على نطاق واسع ، وبعض الألياف الضامرة المتحللة ، وفجوات في السيتوبلازم ، وشعيرات دموية محتقنة ، وزيادة ملحوظة في ترسب الكولاجين. أظهر الفحص المجهري الإلكتروني ليفات عضلية مجزأة غير منظمة ، وعدد قليل من الميتوكوندريا المتضخمة ونواة غريبة مسننة. كشفت أقسام جرذان المجموعة الرابعة عن تحسن واضح في التعديلات السابقة.

كان هناك فرق ذو دلالة إحصائية عالية بين المجموعة الأولى والمجموعة الثالثة وبين المجموعة الثانية والمجموعة الثالثة فيما يتعلق بنسبة مساحة الكولاجين.

الاستنتاج: أكدت النتائج التي تم الحصول عليها من الدراسة الحالية التأثير السلبي للحمية الغذائية عالية الدهون على خلايا عضلة القلب. أدى تناول الكيرسيتين في نفس الوقت إلى تحسين تلف عضلة القلب بشكل ملحوظ، لذا يمكن استخدامه كعامل فعال في وقاية القلب ضد فرط شحميات الدم وتأثير الشيخوخة.