Cardio protective effect of *Coriandrum sativum* L. on isoproterenol induced myocardial necrosis in rats

Dipak K. Patel\(^a\), Swati N. Desai\(^a\), Hardik P. Gandhi\(^b\), Ranjitsinh V. Devkar\(^a,\ast\), A.V. Ramachandran\(^a\)

\(^a\) Division of Phytotherapeutics and Metabolic Endocrinology, Department of Zoology, Faculty of Science, The Maharaja Sayajirao University of Baroda, Vadodara-390002, Gujarat, India

1. Introduction

Epidemiological studies predict an ominous prevalence of cardiac vascular diseases globally as well as in India during next decade (Lopez and Murray, 1998; Gilski and Borkenhagen, 2005). Myocardial infarction, a highly prevalent ischemic condition characterized by tissue necrosis develops essentially due to an imbalance between oxygen need and actual supply (De Bono and Boon, 1992) and results in irreversible histopathological damages and subsequent cardiovascular complications (Gross and Auchampach, 2007).

Isoproteenol (IP), a synthetic catecholamine and \(\beta\)-adrenergic agonist increases heart rate and exhaust energy reservoir of cardiac myocytes leading to cell death. It induces myocardial necrosis via multiple modes of action in experimental animals. It is essentially manifest by its stimulation of sarcoplasmic adenylate cyclase and \(Na^+\) and \(Ca^{2+}\) channels resulting in exaggerated influx of \(Ca^{2+}\) and energy consumption and consequent cell death (Milei et al., 1978). Free radicals produced by IP initiate the peroxidation of membrane bound polyunsaturated fatty acids (PUFAs) leading to both structural and functional myocardial injury (Thompson and Hess, 1986). IP-induced myocardial necrosis serves as an excellent experimental model to study catecholamines induced cardiac dysfunction and also to evaluate the possible cardioprotective efficacy of various natural and synthetic agents.

*Coriandrum sativum* L. (Apiaceae) (CS) is an ubiquitous annual herb, the leaves and seeds of which form a key ingredient of Middle Eastern, Mediterranean, Indian, Latin American, African and South-East Asian cuisines. Apart from its usage as a condiment, decoction and tincture of powdered seeds of CS find usage either alone or in combination with other herbs in the treatment of cough, dysentery, sore throat, convulsion, insomnia and anxiety (Grieve, 1971). An extract of CS seeds is also reported to have therapeutic potential against diabetes, cardiovascular and urinary disorders (Eguale et al., 2007; Emamghoreishi et al., 2005). Phytochemical analysis of CS seeds has revealed the presence of polyphenols (rutin, ferulic acid, galic acid, chlorogenic acid and caffeic acid derivatives), flavonoids (queretin and isouqueretin) and \(\beta\)-carotenoids (Melo et al., 2003). The oil of CS seeds is rich in \(\alpha\) and \(\beta\)-pinene, camphor, citronellol, coriandrol, \(\beta\)-cymene, geraniol, geranyl acetate, limonene, linalool, myrcene, \(\alpha\) and \(\beta\) phellandrene and terpinene besides many water soluble compounds such as monoterpoid glycosides and their derivatives (Sergeeva, 1975; Ishikawa et al., 2003). The reported pharmacological actions of CS are many with its oil shown to possess antifungal (Garg and Siddiqui, 1992) and antimicrobial (Baratta et al., 1998) properties and seed extract shown to possess hypoglycemic (Gray and Flatt, 1999), hypolipidemic (Chithra and Leelamma, 1997; Chithra and Leelamma, 1999; Lal et al., 2004), hypocholesterolemic (Dhanapakiam et al., 2008),...
anti-insulin resistance activity (Patel et al., 2011), antihypertensive (Medhin et al., 1986) and antioxidant (Melo et al., 2003; Ramadan et al., 2003; Bajpai et al., 2005) competence.

Several pre-clinical and clinical studies involving pretreatment with vitamins and antioxidants have demonstrated their potential to prevent myocardial damage (Singh et al., 1994; Senthil et al., 2004). Previously Hashim et al. (2005) have investigated that hydro-methanolic extract of CS seed had strong antioxidant property and it had prevented oxidative damage induced by H₂O₂ to lymphocytes. The present study was designed to assess cardioprotective potential of hydro-methanolic extract of the customarily used spice CS seeds in IP induced multifocal myocardial necrosis in rats.

2. Materials and methods

2.1. Plant material and preparation of extract

CS plants were collected in the seedling months (February and March) and Dr. P.S. Nagar, Department of Botany, The M.S. University of Baroda identified the plant and a sample specimen was deposited in the herbarium of the Department of Botany. Hundred grams of powdered dry seeds soaked in methanol:water (80:20 v/v) at room temperature was allowed to stand for seven days. Resultant extract filtered through a muslin cloth was concentrated in a rotary evaporator under reduced pressure to obtain a thick semisolid brown paste (Qaiser et al., 2009). The final yield obtained was 8.3 g (w/w).

2.2. Experimental animals

Adult male Wistar rats (150–200 g; obtained from Zydus Cadila Research Centre, Ahmedabad, Gujarat, India) were housed under standard animal house conditions (23 ± 2 °C; LD 12:12 and 45–50% humidity) and provided with pellet diet (M/S Pranav agro, Ltd., Baroda, India) and water ad libitum. The animals were maintained as per the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (PCPSEA) India and the experimental protocol approved by the animal ethical committee of the Department of Zoology, The M.S. University of Baroda, Vadodara (Approval No.827/ac/04/PCPSEA).

2.3. Experiment design

Thirty animals were randomly divided into five groups of six animals each. Group I (NC) served as control and received 0.5% Carboxy methyl cellulose (CMC; p.o.) for 28 days and normal saline (s.c.) on days 29 and 30. Group II (IP) served as positive control rats and received 0.5 CMC (p.o.) for 28 days and isoproterenol (85 mg/kg body weight, s.c.) on days 29 and 30 while, the remaining groups [Group III (IP + CS100), Group IV (IP + CS200) and group V (IP + CS300)] received respectively 100, 200 and 300 mg/kg body weight of CS extract daily for 28 days (p.o.) and IP (85 mg/kg, s.c.) on days 29 and 30. The protocol for IP treatment schedule was as per the previous works from this laboratory (Jadeja et al., 2010; Thounaojam et al., 2011). At the end of the experimental period (i.e. 31st day), animals were fasted overnight (12 h) and blood samples were collected from retro-orbital sinus under mild ether anesthesia. Plasma was obtained by cold centrifugation of samples at 3000 rpm for 10 min. Later, animals were sacrificed by cervical dislocation under mild ether anesthesia. Plasma was used for the estimation of Na+, K+, Ca²⁺, Mg²⁺, total protein content (Lowry et al., 1951) and lipid peroxidation levels (LPO; Buege and Aust, 1978). Total ascorbic acid content (AA) was measured as per Roe and Kie- ther (1943) by preparing homogenates of fresh cardiac tissue in 6% Trichloro acetic acid.

2.4. Plasma markers of cardiac damage

Plasma levels of creatine phosphokinase- MB (CK-MB), lactate dehydrogenase (LDH), aspartate transaminase (AST), alanine transaminase (ALT) and uric acid were assayed by using commercially available kits (Recon Diagnostic, Ltd., Vadodara, India). Low density lipoprotein (LDL) and Very low density lipoprotein (VLDL) were calculated by Friedewald’s formula (Friedewald et al., 1972).

2.5. Plasma lipid profile

Triglyceride (TG), total cholesterol (TC) and high density lipoprotein (HDL) content were assayed by using commercially available kits (Recon Diagnostic, Ltd., Vadodara, India). Low density lipoprotein (LDL) and Very low density lipoprotein (VLDL) were calculated by Friedewald’s formula (Friedewald et al., 1972).

2.6. Cardiac antioxidants and Lipid peroxidation (LPO)

Cardiac tissue from control and treated groups was weighed and homogenized (10% w/v) in chilled Tris buffer (10 mM; pH 7.4) and centrifuged at 10,000 g for 20 min at 0 °C. Clear supernatant was used to assay superoxide dismutase (SOD; Marklund and Marklund, 1974), catalase (CAT; Aebi, 1984), glutathione peroxidase (GPx; Rotruck et al., 1973), glutathione s-transferase (GST; Habig et al., 1974), reduced glutathione (GSH; Beutler, 1963), vitamin E (Vit. E: Baker and Frank, 1968), total protein content (Lowry et al., 1951) and lipid peroxidation levels (LPO; Buege and Aust, 1978). Total ascorbic acid content (AA) was measured as per Roe and Kie- ther (1943) by preparing homogenates of fresh cardiac tissue in 6% Trichloro acetic acid.

2.7. Cardiac ATPases

Pellets obtained from tissue homogenate after centrifugation was re-suspended in ice-cold Tris buffer (10 mM, pH 7.4) to get a final concentration of 1% and was used for the estimation of Na⁺ K⁺ ATPase (Bontling et al., 1970), Ca²⁺ ATPase (Hjerken and Pan, 1983) and Mg²⁺ ATPase (Ohnishi et al., 1982). Protein was estimated according to the method of Lowry et al. (1951).

2.8. Macroscopic and microscopic evaluation of cardiac tissue

Heart tissue slices (approx. 2–3 mm thick) transversely cut across the ventricle were kept in a covered glass dish containing 1% TTC (2, 3, 5- triphenyltetrazolium chloride; Sigma, St. Louis, MO) solution and incubated at 37 °C for 20 min for differentiation of viable tissue from necrotic areas (Li et al., 2011).

Heart samples from control and treated rats were fixed in 4% buffered paraformaldehyde, dehydrated in graded alcohol series and embedded in paraffin wax. Five micrometer thick sections cut (by Leica RM2135 Microtome) and stained with haematoxylin-eosin, were photographed with Canon power shot 572 digital Camera (200 x) attached to a Leica microscope.

2.9. Statistical analysis

Statistical analysis of data was done by one way ANOVA followed by Bonferroni’s multiple comparison test and results were expressed as mean ± S.E.M (Using Graph Pad Prism version 3.0 for Windows, Graph Pad Software, San Diego California USA).

3. Results

3.1. Plasma markers of cardiac damage

IP treated rats showed significant (p < 0.005) increment in the plasma levels of CK-MB, LDH, AST, ALT and uric acid compared to NC rats. Pretreatment of IP rats with CS prevented the IP induced increase in the serum levels of these parameters in a dose dependent manner (Table 1).

3.2. Plasma lipid profile

IP treatment recorded significant (p < 0.005) increase in plasma TG, TC, LDL, and VLDL and decrement in HDL levels compared to the NC group. CS treatment showed dose dependent decrement

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NC</th>
<th>IP</th>
<th>IP + CS100</th>
<th>IP + CS200</th>
<th>IP + CS300</th>
</tr>
</thead>
<tbody>
<tr>
<td>CKMB^b</td>
<td>75.66 ± 6.91</td>
<td>218.20 ± 29.16c</td>
<td>171.20 ± 9.19c</td>
<td>133.10 ± 6.16c</td>
<td>80.10 ± 9.03c</td>
</tr>
<tr>
<td>LDH^b</td>
<td>82.71 ± 6.50</td>
<td>189.60 ± 7.36c</td>
<td>149.70 ± 4.32c</td>
<td>126.00 ± 4.15c</td>
<td>85.60 ± 6.35c</td>
</tr>
<tr>
<td>AST</td>
<td>50.33 ± 1.99</td>
<td>61.17 ± 2.24c</td>
<td>50.33 ± 1.76c</td>
<td>43.50 ± 1.91c</td>
<td>31.67 ± 1.02c</td>
</tr>
<tr>
<td>ALT</td>
<td>19.33 ± 1.11</td>
<td>44.83 ± 2.18c</td>
<td>36.67 ± 1.82c</td>
<td>31.00 ± 1.73c</td>
<td>22.17 ± 1.99c</td>
</tr>
<tr>
<td>Uric acid^c</td>
<td>1.91 ± 0.21</td>
<td>7.01 ± 0.47c</td>
<td>5.24 ± 0.41c</td>
<td>3.72 ± 0.19c</td>
<td>2.14 ± 0.21c</td>
</tr>
</tbody>
</table>

Where, S = (U/L), ^b = U/mL, ^c = KA Units/L, ^d = mg/dl, n = 6. Data were expressed as mean ± S.E.M. a (p < 0.05), b (p < 0.01), c (p < 0.001) when NC vs. IP and A (p < 0.05), B (p < 0.01), C (p < 0.001) when IP vs. IP + CS.
in TC, TG, LDL, VLDL and significant increment in HDL compared to IP treated rats (Table 2).

3.3. Cardiac anti-oxidants and LPO

IP treated group recorded significant ($p < 0.001$) increment in LPO level, as well as significant ($p < 0.001$) decrement in the activities of enzymatic antioxidants (SOD, CAT, GPx and GST) and content of non-enzymatic antioxidants (GSH, AA and Vit. E) compared to NC rats. Administration of CS (100, 200 and 300 mg/kg body weight, respectively) markedly prevented all the alterations with respect to antioxidants and LPO in IP treated rats and maintained them to the near normal levels (Table 3).

3.4. Cardiac ATPase

The cardiac tissue of IP treated rats depicted significant ($p < 0.005$) decrement in the activities of Na$^+$/K$^+$, Mg$^2+$ and Ca$^{2+}$-ATPases compared to that of NC rats while, IP + CS treated cardiac tissue recorded significant resistance (Table 4).

3.5. TTC and HE staining of cardiac tissue

TTC staining of heart of control rats showed brick red coloration indicative of more number of viable cells whereas, IP treated rats showed large area of pale yellow coloration suggestive of necrosis. However, IP rats pretreated with CS showed a protective effect with a minimal or no pale yellow coloration in a dose dependent manner (Fig. 1).

HE staining of cardiac tissue from NC rats showed histoarchitecture of myofibers that were characteristically multinucleated and intact. IP treatment resulted in focal myocardial necrosis (encircled area) and disrupted myofibers. However, IP + CS treated groups showed relatively less disruption of myofibers with IP + CS 300 showing maximum fiber integrity. (Fig. 2).

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### Table 2
Effect of CS seed extract on plasma lipid profile.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NC</th>
<th>IP</th>
<th>IP + CS100</th>
<th>IP + CS200</th>
<th>IP + CS300</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC$^a$</td>
<td>54.50 ± 1.72</td>
<td>90.83 ± 2.58$^c$</td>
<td>75.83 ± 2.16$^a$</td>
<td>65.33 ± 2.88$^c$</td>
<td>59.50 ± 2.23$^c$</td>
</tr>
<tr>
<td>TG$^a$</td>
<td>33.17 ± 2.37</td>
<td>53.00 ± 2.67$^c$</td>
<td>50.67 ± 1.82$^{NS}$</td>
<td>44.67 ± 2.60$^c$</td>
<td>35.00 ± 1.48$^c$</td>
</tr>
<tr>
<td>VLDL$^a$</td>
<td>6.63 ± 0.47</td>
<td>10.60 ± 0.53$^c$</td>
<td>10.13 ± 0.36$^{NS}$</td>
<td>8.93 ± 0.52$^a$</td>
<td>7.00 ± 0.29$^c$</td>
</tr>
<tr>
<td>LDL$^a$</td>
<td>34.30 ± 0.52</td>
<td>90.27 ± 2.83$^c$</td>
<td>70.97 ± 2.62$^a$</td>
<td>58.10 ± 3.92$^c$</td>
<td>43.67 ± 2.75$^c$</td>
</tr>
<tr>
<td>HDL$^a$</td>
<td>26.83 ± 0.98</td>
<td>11.17 ± 1.04$^a$</td>
<td>15.00 ± 0.73$^a$</td>
<td>16.17 ± 1.35$^a$</td>
<td>22.83 ± 0.79$^a$</td>
</tr>
</tbody>
</table>

Where, $^a$ = mg/dl. $n = 6$. Data were expressed as mean ± S.E.M. $a(p < 0.05)$, $b(p < 0.01)$, $c(p < 0.001)$ when NC vs. IP and $A(p < 0.05)$, $B(p < 0.01)$, $C(p < 0.001)$ when IP vs. IP + CS.

### Table 3
Effect of CS seed extract on Cardiac LPO levels and enzymatic and non-enzymatic anti-oxidant.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NC</th>
<th>IP</th>
<th>IP + CS100</th>
<th>IP + CS200</th>
<th>IP + CS300</th>
</tr>
</thead>
<tbody>
<tr>
<td>LPO$^a$</td>
<td>0.96 ± 0.53</td>
<td>3.22 ± 0.27$^c$</td>
<td>2.05 ± 0.73$^a$</td>
<td>1.61 ± 0.28$^c$</td>
<td>1.27 ± 0.57$^c$</td>
</tr>
<tr>
<td>SOD</td>
<td>8.06 ± 0.63</td>
<td>3.46 ± 0.60$^c$</td>
<td>6.12 ± 0.37$^a$</td>
<td>6.54 ± 0.19$^c$</td>
<td>6.98 ± 0.17$^c$</td>
</tr>
<tr>
<td>CAT$^a$</td>
<td>5.15 ± 0.43</td>
<td>1.92 ± 0.31$^c$</td>
<td>2.21 ± 0.14$^{NS}$</td>
<td>3.06 ± 0.29$^c$</td>
<td>4.40 ± 0.34$^c$</td>
</tr>
<tr>
<td>GPx$^a$</td>
<td>3.08 ± 0.05</td>
<td>1.03 ± 0.06$^c$</td>
<td>1.22 ± 0.06$^a$</td>
<td>1.81 ± 0.07$^a$</td>
<td>2.80 ± 0.04$^c$</td>
</tr>
<tr>
<td>GST$^a$</td>
<td>787.4 ± 14.41</td>
<td>423.6 ± 12.63$^c$</td>
<td>541.4 ± 10.19$^c$</td>
<td>625.7 ± 11.34$^c$</td>
<td>760.7 ± 16.06$^c$</td>
</tr>
<tr>
<td>GSH$^a$</td>
<td>9.37 ± 0.17</td>
<td>3.81 ± 0.22$^c$</td>
<td>4.79 ± 0.26$^a$</td>
<td>5.87 ± 0.31$^a$</td>
<td>7.81 ± 0.18$^a$</td>
</tr>
<tr>
<td>Vit E$^c$</td>
<td>5.29 ± 0.23</td>
<td>1.33 ± 0.31$^a$</td>
<td>2.65 ± 0.36$^{NS}$</td>
<td>3.32 ± 0.21$^c$</td>
<td>4.91 ± 0.29$^c$</td>
</tr>
</tbody>
</table>

Where, $^a$ = μmol/mg protein, $^c$ = nmol/mg protein, $^d$ = unit/mg protein, $^e$ = μmol/min/mg protein, $^f$ = mg/100 g tissue Where, $n = 6$. Data were expressed as mean ± S.E.M. $a(p < 0.05)$, $b(p < 0.01)$, $c(p < 0.001)$ when NC vs. IP and $A(p < 0.05)$, $B(p < 0.01)$, $C(p < 0.001)$ when IP vs. IP + CS.

### Table 4
Effect of CS seed extract on Cardiac ATPases.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NC</th>
<th>IP</th>
<th>IP + CS100</th>
<th>IP + CS200</th>
<th>IP + CS300</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na$^+$/K$^+$ATPase$^a$</td>
<td>5.00 ± 0.50</td>
<td>2.11 ± 0.17$^c$</td>
<td>2.51 ± 0.14$^{NS}$</td>
<td>2.96 ± 0.33$^a$</td>
<td>4.40 ± 0.28$^c$</td>
</tr>
<tr>
<td>Mg$^2+$ ATPase$^a$</td>
<td>2.74 ± 0.02</td>
<td>0.45 ± 0.03$^c$</td>
<td>0.84 ± 0.05$^{NS}$</td>
<td>1.02 ± 0.08$^a$</td>
<td>2.09 ± 0.09$^c$</td>
</tr>
<tr>
<td>Ca$^{2+}$ ATPase$^a$</td>
<td>2.03 ± 0.18</td>
<td>0.98 ± 0.02$^c$</td>
<td>1.29 ± 0.02$^a$</td>
<td>1.46 ± 0.04$^c$</td>
<td>1.83 ± 0.04$^c$</td>
</tr>
</tbody>
</table>

Where, $^a$ = μmol phosphate liberated/mg protein. $n = 6$. Data were expressed as mean ± S.E.M. $a(p < 0.05)$, $b(p < 0.01)$, $c(p < 0.001)$ when NC vs. IP and $A(p < 0.05)$, $B(p < 0.01)$, $C(p < 0.001)$ when IP vs. IP + CS.

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Fig. 1. Effect of CS seed extract on triphenyltetrazolium chloride (TTC) stained cardiac tissue slices. Arrows indicate necrotic tissue.
4. Discussion

Administration of higher doses of IP to rats induces increment in heart rate, systolic and diastolic irregularities and abnormal ECG pattern (Rona, 1985; Karthick and Prince, 2006). These events epitomized by hypoxia, calcium over load and increased production of reactive oxygen species (ROS) lead to degenerative changes in cardiac tissue that culminate in necrosis.

Accordingly, IP treated rats herein recorded significant increment in plasma levels of CK-MB, LDH, AST, ALT and uric acid, which is in keeping with the known IP induced deficiency of oxygen supply and increased sarcolemmal permeability and consequent leaching of CK-MB and LDH into the blood stream along with increased plasma levels of AST, ALT and uric acid (Mathew et al., 1985; Weir et al., 2003). The recorded ability of CS to effectively prevent these alterations clearly points towards its cardio-protective competence and maintenance of sarcolemmal integrity.

Also the activity levels of 3-hydroxy-3-methyl-glutaryl-CoA (HMG CoA) reductase and Lechitin-cholesterol acyltransferase (LCAT) have been reported to undergo significant alterations following IP treatment which resulted in altered lipid and lipoprotein profiles (Rajadurai et al., 2006). Hence, the observed decrement in lipid profile in IP + CS treated groups indicates a possible modulatory influence of CS on activity levels of HMG CoA and LCAT that requires further investigations.

SOD and CAT are enzymatic antioxidants that act as the first line of cellular defense and help in scavenging free radicals. Therefore, a decrement in their activity levels results in free radical induced cellular damage. Other enzymatic antioxidants GPx and GST and non-enzymatic GSH also help maintain healthy cell functions by scavenging free radicals like peroxy radicals, superoxide ions and singlet oxygen formed by toxicants (Rathore et al., 1998). AA is a water soluble vitamin that acts as an antioxidant and scavenger of superoxide and other free radicals, getting transformed in the process to dehydroascorbate (Frei et al., 1986; Packer et al., 1979). Vitamin E is a lipid soluble antioxidant that protects membrane polyunsaturated fatty acids and other components from oxidation by free radicals (Tappel, 1972). Presently, we have observed increased LPO and decreased endogenous antioxidants (both enzymatic and non-enzymatic) in IP treated rats. Apparently, IP causes heightened oxidative damage of cellular macromolecules marked by elevated level of LPO by way of increased generation of free radicals as has also been inferred by Gokkusu and Mostafazadeh (2003). However, pretreatment of IP animals with CS prevented the decrease in antioxidant levels and increase in LPO significantly in a dose dependent manner. Plant based extracts that are rich in polyphenols and flavonoids are supposedly strong antioxidants and CS seed extract has been reported to be rich in flavonoids, terpenoids (Wangensteen et al., 2004) and polyphenols (Hashim et al., 2005). The latter workers have opined that alcoholic extract of CS has maximal content of the said antioxidants compared to other types of extract. The currently observed effects of CS may be attributable to the presence of these secondary metabolites.

ATPases, by maintaining differential levels of ions play important roles in the regulation of contraction–relaxation cycles of cardiac muscles and consequently, peroxidation of sarcolemmal lipids can result in their inactivation as suggested by Kako et al. (1988). Reduced activity of Na+/K+ ATPase with compromised Na+ efflux can result in altered membrane permeability (Finotti and Palatini, 1986). A decrement in Ca2+ATPase expectantly would decrease sarcoplasmic Ca2+ concentration and weaken the contractility of heart. Hence, loss of ATPase activity in the ischemic state could contribute to myofibrillar necrosis and functional damage. Even Chernysheva et al. (1980) have reported IP induced decrement in the activity levels of Na+/K+, Mg2+ and Ca2+ ATPase in rats. However, IP + CS treated rats show a dose dependent significant up keep of these ATPases, essentially attributable to the membrane stabilizing aproperty of CS extract that protects the sarcolemma and intracellular membranes from the deleterious effect of IP and consequent myocardial damage (Hashim et al., 2005).

TTC is a redox indicator that is commonly used to differentiate between metabolically active and inactive cells and tissues (Altman, 1976). Staining of cardiac tissue slices with TTC provides...
insight regarding the infarct size and is a well accepted method to assess necrosis of myocardial tissue (Prabhu et al., 2006). TTC is enzymatically reduced to brick red precipitates of formazan dye or TPF (1,3,5-triphenylformazan). Active mitochondrial respiration generating reduced coenzymes is responsible for the reduction of TTC to TPF in all tissues including the cardiac tissue (Ramkisson, 1996). Hence, appearance of patches of pale white color in cardiac tissue slices of IP treated rats indicates areas of focal necrosis due to non-reduction of TTC as observed in the present study in IP treated rats. The IP + CS rats (especially CS300) depicted minimal pale yellow patches suggestive of normal myocardial structure. Histological observations further confirm the IP induced necrotic changes affecting myofiber disruption and fraying of fibers. These deleterious changes seem ably resisted by pretreatment with CS with the highest dose affording maximal protection. These observations provide compelling macroscopic and microscopic evidences regarding the cardioprotective potential of CS seed extract.

Parameters investigated here in indicate that hydro-methanolic extract of CS is potent in mitigating IP induced myocardial necrosis. The same is evidenced in form of CS induced favourable alterations in biochemical and histo-morphological parameters. Although the observed results have been attributed to high content of polyphenols in hydro-methanolic extract of CS. Our further studies are aimed that isolating the active component of CS and to reassess its cardioprotective potential in more appropriate experimental model (coronary ligation) and using gold standard marker enzyme such as cardiac Troponin I that underlying mechanism of CS induced cardioprotection.

5. Conclusion

It can be concluded from the present study that hydro-methanolic extract of CS seeds has cardioprotective potential. The same is attributable to high polyphenol content in CS seeds.

Conflict of Interest

The authors declare that there are no conflicts of interest.

Acknowledgement

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References


