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Preventive measures of CoQ10 against bromobenzene-induced toxicity in rats

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Abstract:

Background of The Study: Bromobenzene an environmental toxin and an industrial raw material. It is highly toxic cause's toxicity in rats when administered. CoQ10 a natural antioxidant found normally in the body at very low can scavenge the free radicals produced by bromobenzene and reduce the toxicity.

Aim: The main aim and purpose of this study are to evaluate the protective effects of CoQ10 against the toxicity induced by bromobenzene in rats.

Materials and Methods: Rats are randomly made into five different groups. Group1-control with normal food and water, Group2-bromobenzene treated rats (10mmol/kg), Group3-Bromobenzne (10mmol/kg) and CoQ10 (10mg/kg) treated rats, Group4 -bromobenzene (10mmol/kg and silymarin-100mg/kg treated rats, Group5-CoQ10-10mg/kg treated rats. Animals were sacrificed at the end of the experimental period. Antioxidant and Biochemical parameters and histopathological analysis are done.

Results: Bromobenzene treated rats showed elevated levels of biochemical parameters and decreased levels of antioxidants. CoQ10 can alter elevated and decreased levels of parameters and decreased toxicity.

Conclusion: CoQ10 can be used as an alternative to minimise bromobenzene induced toxicity.

Keywords: Bromobenzene, CoQ10, Antioxidant, Toxicity, Silymarin.

INTRODUCTION:

Bromobenzene is an industrial raw material used in the manufacturing of many medicines and chemicals that is a xenobiotic. It's a colorless, odorless liquid (Vedi et al., 2014). Bromobenzene is a well-known toxicant; when administered or ingested, it induces toxicity. During processing or during the production of phenyl magnesium bromide, bromobenzene is released into the atmosphere as well as used as a solvent and as an additive in motor oil (El-Sharaky et al., 2009). Several tests have shown that bromobenzene induces toxicity in rats at a rate of 0.45 g / kg or 10 mM / kg or 0.1 m / kg. Bromobenzene is commonly used with corn oil (0.1 m) or coconut oil (0.1 m) in rats (Zhao et al., 2018). Bromobenzene is generally given by intraperitoneal or intragastric intubation or oral gavages. Bromobenzene is biotransformed in the liver when administered in the rat. Bromobenzene is biologically activated through the bioactivation of a cytochrome P450 enzyme. CYP450 hydrolyses bromobenzene into the strongly electrophilic bromobenzene(Hamed et al., 2013). It causes toxicity when triggered by the the indicators of liver and kidney function such as lipid peroxidation, cytokines (TNF-alpha, VEGF, IL-1beta). The intracellular defense from reactive oxygen species (ROS) and xenobiotic metabolites has been lost at high doses of bromobenzene. This will result in certain defects such as lipid peroxidation, systemic inflammation, ATP depletion, mitochondrial dysfunction, energy deficiency, altered calcium levels intracellular(El-Sharaky et al., 2009). Bromobenzene administration also decreases antioxidant activity. Bromobenzene consists of two metabolites: primary bromobenzene-2, 3 oxides, bromobenzene-3, 4 oxides) and secondary bromophenoisomers, 4-bromocatechol, 2-bromohydroquinone, and benzoquinone(Vedi et al., 2014). Main metabolites of bromobenzene are hepatotoxic, while nephrotoxic are secondary metabolites. The primary bromobenzene metabolites that bind to glutathione and reduce the first line of defense in rats, resulting in ROS in rats. The highly toxic and reactive secondary metabolites of bromobenzene reduces the antioxidants present in rats. For rats, however, higher dose of bromobenzene causes more ROS, which in turn results in toxicity, in general intracellular defense against ROS.

CoQ10 is a compound that occurs naturally and is well known for its anti-inflammatory properties. It is also referred to as ubicinone(Fouad and Jresat, 2012). Due to its hydrophobicity and high molecular weight, CoQ10 is absorbed slowly and very limited. CoQ10 uptake is similar to vitamin uptake. Major sources of CoQ10 are present in fish, meat, nuts and certain oils6. It is found in low quantity in dairy products, vegetables, fruits, cereals. CoQ10 is taken as a food supplementary or dietary supplement. CoQ10 is also found in heart liver and skeletal muscle where energy is required in high amount(Prajapati et al., 2017). CoQ10 administration improves the activity of antioxidant enzymes and helps to lower levels of inflammatory markers such as TNF-alpha andIL-6. This reduces the production of pro-inflammatory cytokines such as factor of tumor necrosis (El-Sheikh et al., 2012). CoQ10 is also an effective antioxidant in which free radicals are scavenged. Because bromobenzene administration induces the free radicals that induce toxicity, CoQ10 can be used to prevent free radicals from being formed and thus mitigate toxicity in rats.

2. MATERIALS AND METHODS:

2.1. Drugs:

CoQ10 which is commercially available was purchased from Sigma Aldrich Ltd. India and dissolved in olive oil. Silymarin was purchased from Quality pharmaceuticals Ltd. India and dissolved in sterile distilled water. The effective dosage of Bromobenzene and Silymarin to be used was obtained from previous studies(Vedi et al.,

2014). Bromobenzene was purchased from sigma chemical co. and dissolved in coconut oil. CoQ10 at a rate of 10mg/kg.b.wt was found to be effective. All other standard reagents were obtained locally and are of analytical grade.

2.2. Animals:

Female Wistar albino rats which are 150-250 g body weight were purchased from the animal house in VIT University, Vellore, India. All the animals purchased were maintained under the guidelines of CPSCEA (Committee for supervision and control of experiment on animals) Government of India, Chennai, Tamilnadu. Animals were acclimatized for a week in a specialised Temperature and humidity control room which must be pathogen-free with a 12hr light/dark cycle. The feed for the animals is commercial pelleted feed from Hindustan lever Ltd... The experimental procedure on the rats was approved by the Ethical committee of VIT.

2.3. Experimental Design:

Animals were randomly allocated into five groups;

Group I: The normal control, received 1.5 mL of coconut oil on day 1 and normal food and water until the end of the experiment.

Group II: A single oral dose of Bromobenzene (10 mmol/kg b.w) on day 8 to induces toxicity before which normal food and water were given until day 8.

Group III: A single oral dose of Bromobenzene (10 mmol/kg b.w) was given on day 8 after a daily dosage of COQ10 (10 mg/kg b.w).

Group IV: Received a single oral dose of Bromobenzene (10 mmol/kg b.w) on day 8 after a daily dosage of the standard drug Silymarin (100 mg/kg b.w).

Group V: Received no Bromobenzene, and a daily dosage of COQ10 (10 mg/kg b.w).

In the end, all the experimental animals were sacrificed by using ether anaesthesia. Blood samples of the rats were collected, and samples of kidney and liver tissues (approximately 0.05–0.1 g) was collected and homogenized using phosphate buffer (pH 7.4) to give 20 % w/v homogenate, which is centrifuged at 3000g at 4 °C for 10 min; Until analysis, the supernatant was stored at −20 °C. Also, urine samples (24 hr) were collected by using a metabolic cage.

2.4. Biochemical Parameters:

The activities of AST (Aspartate aminotransferase), ALT (Alanine aminotransferase), ALP (Alkaline phosphatase), Albumin and total bilirubin were determined based on the protocol given by the manufacturer of the kits (Auto span diagnostics, Bangalore, India) in the serum of both controls and experimental rats. Levels of Urea, Uric acid, Acid phosphatase and Creatinine were determined in the serum and 24-h urine sample control and experimental mice using respective kits (Auto span diagnostics, Bangalore, India) according to the manufacturer's protocol. The levels of Triglycerides, Cholesterol and High-density lipoprotein (HDL) in the serum were measured by using the diagnostic kits bought from Auto span diagnostics, Bangalore, India. Friedewald's equation (1972) is used to calculate the VLDL (Very low-density lipoprotein) and LDL (Low-density lipoprotein) fractions by using the formula: VLDL=TG/5 and LDL=total cholesterol− (HDL+VLDL) respectively. All the values calculated were expressed in terms of a milligram per decilitre. Commercially obtained kits from Auto span diagnostics Bangalore, India was used for calculating the level of Total protein.

2.5. Antioxidant Assays:

The activities of superoxide dismutase (SOD) (MARKLUND and MARKLUND, 1974)Catalase (CAT) (Sinha, 1972)and reduced glutathione(Moron et al., 1979) were also measured in the liver and kidney tissues of the mice.

2.6. Histopathology:

Soon after the sacrifice, liver and kidney portions were fixed in formalin (10 %), washed, and dehydrated in descending grades of isopropanol and xylene is used to rinse finally. Molten paraffin wax was used to embed the tissues. Sections were cut at a thickness of 5µm, stained with hematoxylin and eosin, and observed microscopically for histopathological changes**.**

2.7 Statistical Analysis:

The obtained data from each group are combined and its difference between different set of groups is determined. They are statistically expressed by mean±standard deviation (SD). ANOVA was done. Newman-keul's test is used for the comparison between the groups. *$p<0.05$ is used to denote the statistically significant in results.

3. RESULTS:

3.1. Liver Functional Markers:

Table 1 shows the observed results in rats which are pre-treated with CoQ10 and Silymarin. A single oral dosage of bromobenzene caused liver damage which is indicated by the elevated levels of liver function markers such as AST, ALP, ALT, total bilirubin and direct bilirubin in group 2. Rats pretreated with CoQ10 in group 3 has significantly reduced the elevated levels of liver markers AST, ALP, ALT, total bilirubin and direct bilirubin (Table 1). Standard drug Silymarin also decreased the elevated levels of liver markers which can be seen in group4. CoQ10 treated rats in group 5 has no side effects on liver markers.

3.2. Body Weight:

Body weights of the experimental animals in different groups were measured in day 1, 3, 6 and 9. Average body weight of rats is 200g.Body weight of the rats in group 2 at day 9 is reduced due to the toxicity induced by bromobenzene on day 8.CoQ10 and silymarin has made significant difference on body weight on day 9 which can be seen in group 3 and 4(Fig.1).

3.3. Renal Markers:

Table 2 indicates rats treated with bromobenzeneshowed significant increase in the levels of urea, uric acid, Creatinine and acid phosphatase Group 2.CoQ10 significantly reduced the high levels of urea, uric acid, Creatinine and acid phosphates thus minimising the toxicity induced by bromobenzeneGroup 3. The standard drug silymarin also reduce the high levels of urea, uric acid,Creatinine and Acid phosphatase Group 4). CoQ10

has showed no side effects on the experimental animals in Group 5.

3.4. Lipid Profile Markers:

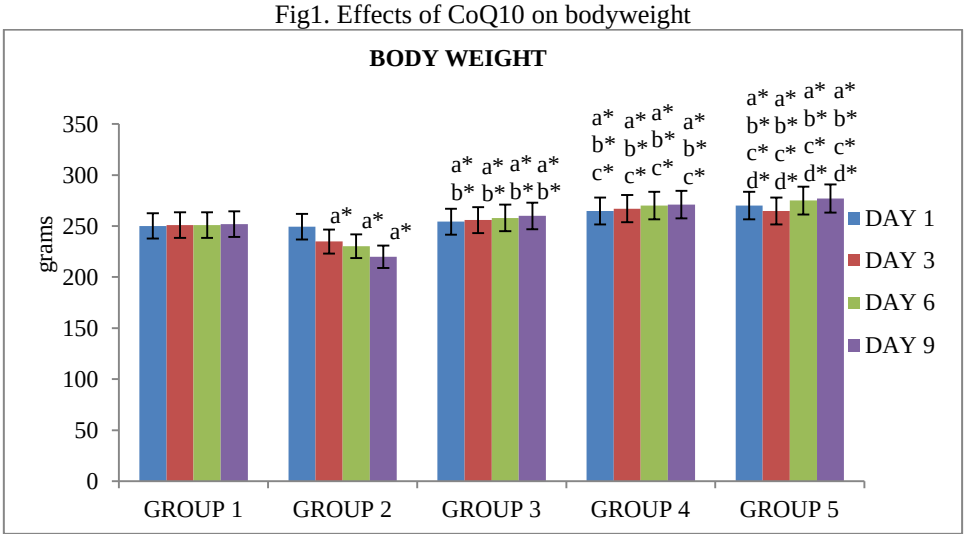
Table 3 shows the levels of cholesterol and HDL which seems to be decreased in bromobenzene treated rats Group 2, whereas in group 3 rats treated with CoQ10 have altered levels of cholesterol and HDL to normal range.Elevated levels of Triglyceride is seen in group 2 rats treated with bromobenzene due to toxicity is cured and decreased by CoQ10 in Group 3.

Table 1 Effects of CoQ10 on liver functional marker

Parameters	Group1 (control)	Group2 (Bromobenzene	Group3 (Bromobenzene+CoQ10)	Group4 (Bromobenzene+Silymarin)	Group5(CoQ10)
AST(U/L)	89.67±0.03	208.47±0.02^{a*}	108.01±0.02^{a*b*}	109.62±0.02^{a*b*c*}	103.78±0.03^{a*b*c*d*}
ALP(U/L)	98.86±0.03	364.11±0.04^{a*}	142.36±0.03^{a*b*}	179.96±0.03^{a*b*c*}	103.86±0.02^{a*b*c*d*}
ALT(U/L)	47.52±0.04	125.57±0.03^{a*}	60.92±0.05^{a*b*}	78.73±0.03^{a*b*c*}	61.76±0.04^{a*b*c*d*}
Total bilirubin (mg/dL)	0.75±0.18	3.70±0.01^{a*}	0.98±0.03^{a*b*}	1.59±0.02^{a*b*c*}	1.19±0.02^{a*b*c*d*}
Direct bilirubin (mg/dL)	0.06±0.01	1.9±0.18^{a*}	0.05±0.01^{b*}	0.08±0.01^{b*}	0.68±0.14^{a*b*c*d*}
Total protein(g/dL)	8.49±0.01	6.15±0.03^{a*}	7.30±0.02^{a*b*}	7.51±0.04^{a*b*c*}	8.92±0.05^{a*b*c*d*}
Albumin (g/dL)	5.31±0.11	3.01±0.02^{a*}	5.23±0.04^{b*}	4.73±0.04^{a*b*c*}	5.69±0.06^{a*b*c*d*}

All the above mentioned values are expressed in terms of mean±S.D. ANOVA is used to carry out the statistical analysis. Above mentioned symbols represent significance of statistics *p<0.05. Comparisons: group 1 vs. group II, III, IV and V,(a)group II vs. III, IV and V, (b)group III vs. IV and V, (c)group IV vs. group V.

Fig1. Effects of CoQ10 on bodyweight



All the above mentioned values are expressed in terms of mean±S.D. ANOVA is used to carry out the statistical analysis. Above mentioned symbols represent significance of statistics *p<0.05. Comparisons: group 1 vs. group II, III, IV and V,(a)group II vs. III, IV and V, (b)group III vs. IV and V, (c)group IV vs. group V.

Table 2 Effects of CoQ10 on renal functional markers.

Parameters	Group 1 (control)	Group 2 (Bromobenzene)	Group3(Bromobenzene +CoQ10)	Group 4 (Bromobenzene +Silymarin)	Group 5 (CoQ10)
Urea	13.32±0.40	44.06±0.11a*	15.42±0.15a*b*	18.25±0.07a*b*c*	12.25±0.06a*b*c*d*
Uric acid	7.20±0.11	27.16±0.12a*	8.61±0.12a*b*	8.13±0.14a*b*c*	7.15±0.13b*c*d*
Creatinine	2.60±4.64	9.55±15.28	2.87±5.52	3.94±6.30	2.29±4.38
Acidphosphatase	1.44±0.06	3.31±0.06a*	2.17±0.03a*b*	2.19±0.03a*b*c*	2.02±0.04a*b*c*d*

All the above mentioned values are expressed in terms of mean±S.D. ANOVA is used to carry out the statistical analysis. Above mentioned symbols represent significance of statistics *p<0.05. Comparisons: group 1 vs. group II, III, IV and V,(a)group II vs. III, IV and V, (b)group III vs. IV and V, (c)group IV vs. group V.

Table 3 Effect of CoQ10 on lipid profile markers:

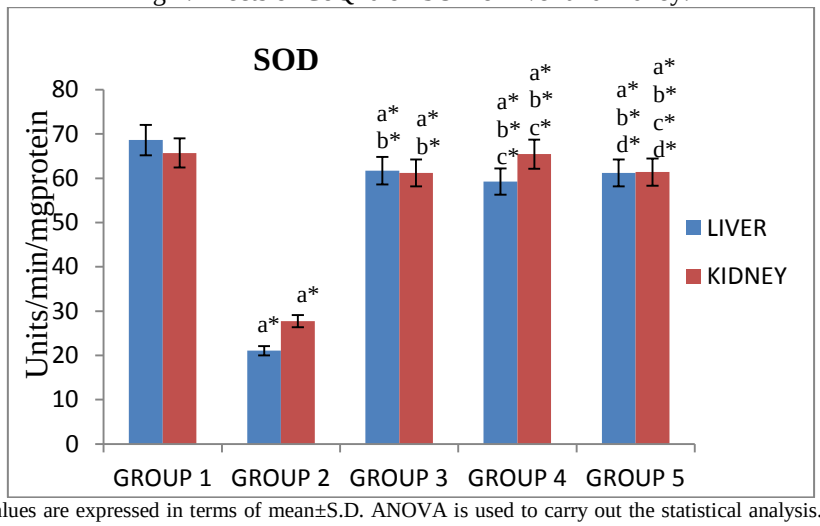
Parameters	Group 1 (control)	Group 2(Bromobenzene)	Group 3 (Bromobenzene+CoQ10)	Group 4 (Bromobenzene +Silymarin)	Group 5 (CoQ10)
Cholesterol	104.76±0.02	43.57±0.02a*	89.74±0.01a*b*	89.05±0.02a*b*c*	91.84±0.04a*b*c*d*
Triglyceride	118.37±0.43	247.73±0.22a*	180.81±0.09a*b*	168.05±0.03a*b*c*	125.63±0.21a*b*c*d*
HDL	61.77±0.02	25.77±0.03a*	57.04±0.02a*b*	50.05±0.03a*b*c*	59.35±0.02a*b*c*d*
LDL	80.26±0.54	271.50±0.029a*	159.93±0.11a*b*	151.61±0.05a*b*c*	91.410±0.25a*b*c*d*
VLDL	23.67±0.08	49.54±0.04a*	36.16±0.01a*b*	33.61±0.006a*b*c*	25.12±0.04a*b*c*d*

All the above mentioned values are expressed in terms of mean±S.D. ANOVA is used to carry out the statistical analysis. Above mentioned symbols represent significance of statistics *p<0.05. Comparisons: group 1 vs. group II, III, IV and V,(a)group II vs. III, IV and V, (b)group III vs. IV and V, (c)group IV vs. group V.

3.5. Antioxidants:

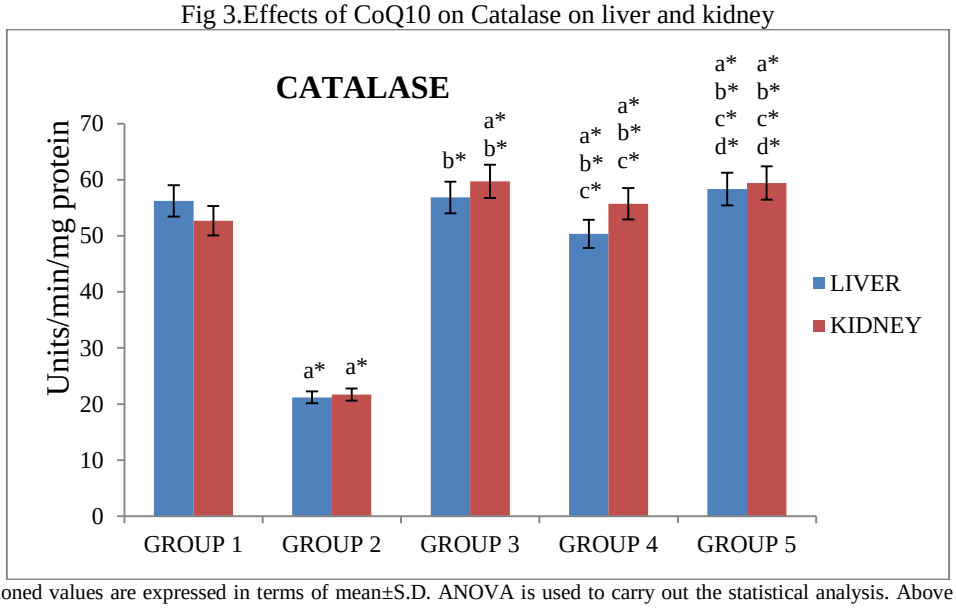
Rats treated with bromobenzene in group 2 has decreased level of antioxidants such as Superoxide dismutase, Catalase, Reduced glutathione which are present in the body to protect from toxicity to cause liver damage. Rats pre-treated with CoQ10 and silymarin in group 3 and 4 has increased the antioxidants and minimised the toxicity induced by bromobenzene.

Fig 2.Effects of CoQ10 on SOD of liver and kidney:



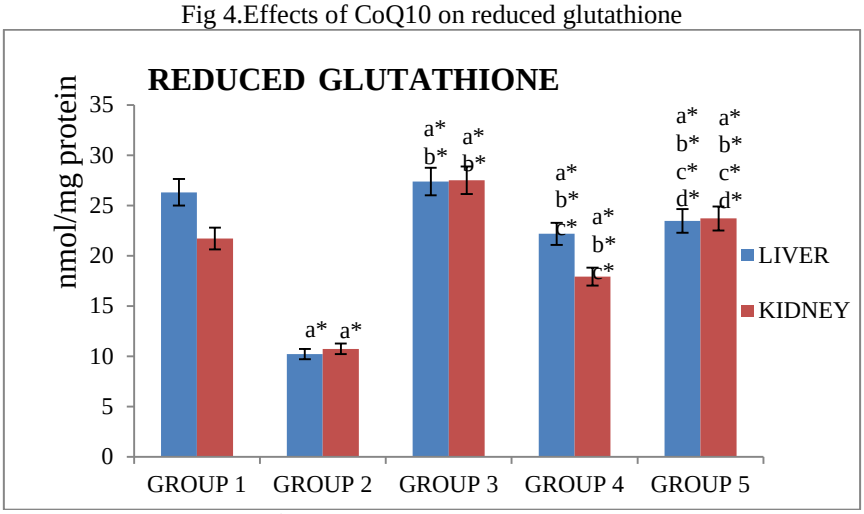
All the above mentioned values are expressed in terms of mean±S.D. ANOVA is used to carry out the statistical analysis. Above mentioned symbols represent significance of statistics *p<0.05. Comparisons: group 1 vs. group II, III, IV and V,(a)group II vs. III, IV and V, (b)group III vs. IV and V, (c)group IV vs. group V.

Fig 3.Effects of CoQ10 on Catalase on liver and kidney



All the above mentioned values are expressed in terms of mean±S.D. ANOVA is used to carry out the statistical analysis. Above mentioned symbols represent significance of statistics *p<0.05. Comparisons: group 1 vs. group II, III, IV and V,(a)group II vs. III, IV and V, (b)group III vs. IV and V, (c)group IV vs. group V.

Fig 4.Effects of CoQ10 on reduced glutathione



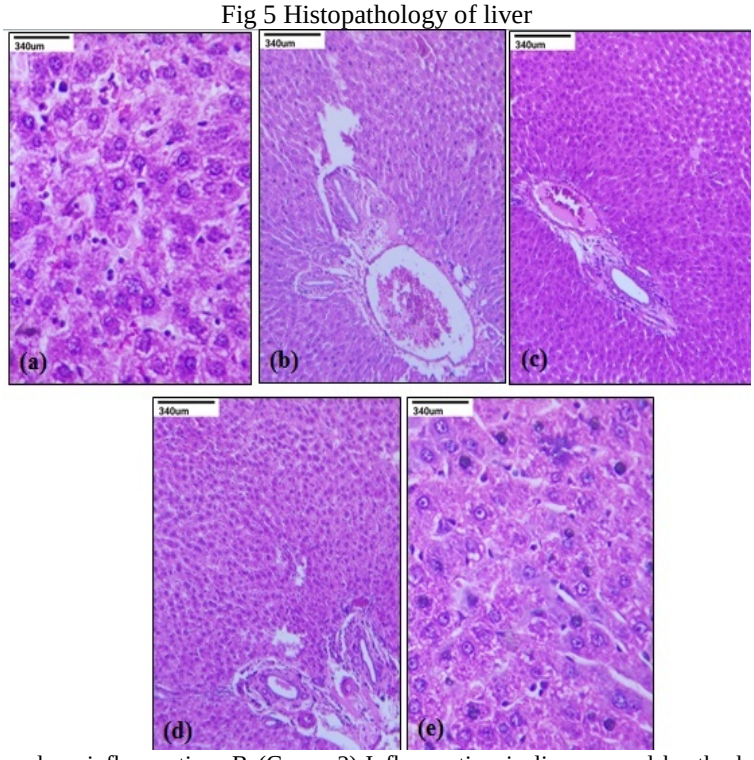
All the above mentioned values are expressed in terms of mean±S.D. ANOVA is used to carry out the statistical analysis. Above mentioned symbols represent significance of statistics *p<0.05. Comparisons: group 1 vs. group II, III, IV and V,(a)group II vs. III, IV and V, (b)group III vs. IV and V, (c)group IV vs. group V.

.3. 6. Liver and Kidney Histopathology:

Group1a shows normal liver with normal hepatocytes and central vein and has no inflammation. Inflammation and infiltration periportal in Group 2b is caused due to the bromobenzenetreatment.CoQ10 has significantly worked on the inflammation caused by bromobenzene and cured it which can be viewed in Group 3(c).Standard drug silymarin which is used in Group 4(d) also cured the inflammation caused.No visible side effect has been caused by the Drug CoQ10 in Group 5(e).

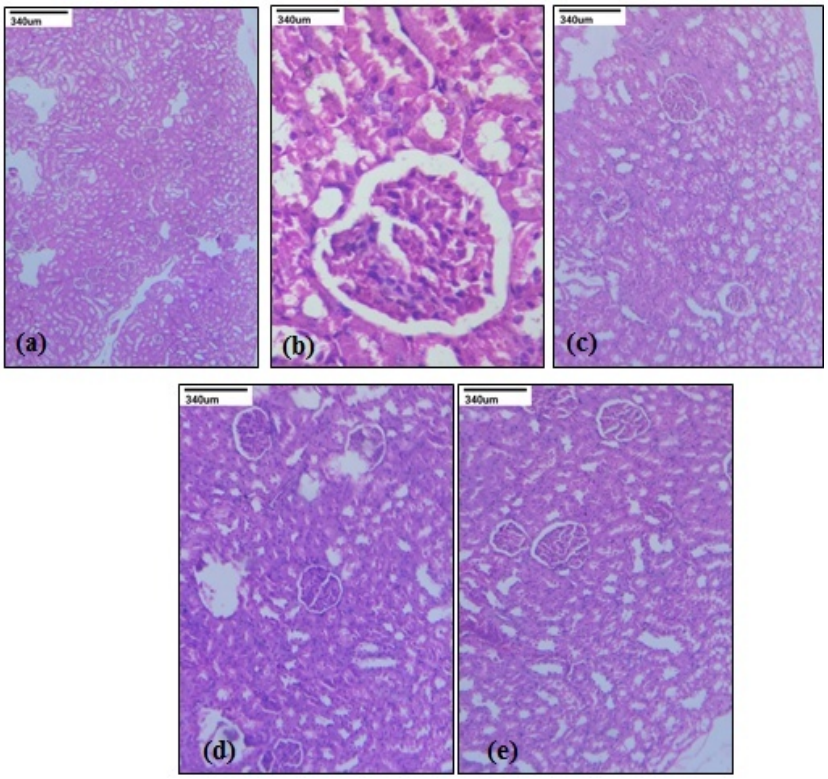
Group 1a had normal glomerular and tubular and had no changes.Bromobenzene treated rats in Group 2b had massive glomerular and tubular damage. Renal damage caused by bromobenzene had significantly reduced by the CoQ10 in rats of Group 3c. Group 4d treated with silymarin showed nearly the normal kidney histology.CoQ10 had no side effects on the kidney Group5e.

Fig 5 Histopathology of liver



A (Group 1) -Normal liver and no inflammation. B (Group 2)-Inflammation in liver caused by the bromobenzene. C (Group 3)-Inflammation caused and cured by the CoQ10. D (Group4)-Inflammation caused and cured by the standard silymarin. E (Group 5)-No side effects caused on liver by CoQ10.

Fig 6.Histopathology of kidney:



A (Group 1) - Normal Glomerular and tubular sections. B (Group 2)-Damage in Kidney glomerular and tubular caused by the bromobenzene. C (Group 3) - Damage in glomerular and tubular caused by bromobenzene which is cured by CoQ10. D (Group4) - Damage caused and cured by the standard silymarin. E (Group 5) - No side effects caused on Kidney by CoQ10.

4. DISCUSSION:

Our study has proved that bromobenzene caused liver damage and renal damage which causes the increase in biochemical parameters and a decrease in antioxidants. CoQ10 has made a possible effect on the toxicity in rats and minimised it through decreasing the biochemical parameters and increasing the antioxidants.

Bromobenzene is an environmentally toxic substance known to induce toxicity in rats. It causes toxicity by developing Reactive oxygen species as CoQ10 has the ability to scavenge against the ROS used to treat bromobenzene-induced toxicity in rats. Many other studies on bromobenzene toxicity has done and treated with various compounds such as Withaferin A, aged garlic extract, Oenanthejavanica, ginger extract, zinc, Hispidulin, Withaniasomnifera, Triphala, beta carotene, black seed oil, Apisceranafablicius honey, Allium jesdianum, Kampo formula are used.A steroidal lactone occurs in Withaniasomnifera's leaves and roots called withaferin A. It has properties such as anti-inflammatory, anti-inflammatory, anti-angiogenic, anti-proliferative, and antipyretic activity. Several previous studies using withaferin A have dismissed bromobenzene as having a protective effect. Hence withaferin A is used against the bromobenzene toxicity(Vedi and Sabina, 2016).Beta carotene is also used against the toxicity caused by bromobenzene in rats, which is a carotenoid and a potent antioxidant. It is a natural compound that appears to be present in human colostrums and mature milk. Beta-carotene bioavailability is directly proportional to that of the fats in the meal(Akkara and Sabina, 2019).Most natural products are now being used against toxicity for a day, because artificial compounds have side effects. Black seed oil, known as Nigella Sativa seeds, is one of the natural compounds used to avoid bromobenzene's hepatotoxicity. It is a natural product used in many diseases. Nigella Sativa seeds have anti-cancer, antibacterial, antioxidant and antihistaminic effects. It also can protect the organs from certain damages. Thymoquinone an active compound of N.S seeds is responsible for the pharmacological properties. It also possesses healing and immunostimulatory properties. One of the major reasons for toxicity is lipid peroxidation; black seed oil acts as an antioxidant and prevents the lipid peroxidation in membranes(Hamed et al., 2013). Another natural product used against the toxicity of bromobenzene is the Apisceranafabricius honey found in enormous quantities in the Middle East African Mountains. This sweetheart is a rich polyphenol source. The other types of honey are known to be the superior quality honey. It possesses anti-inflammatory, antimicrobial and hepatoprotective effect. Hence it is used against the bromobenzene toxicity in rats(Zhao et al., 2018). *Allium jesdianum*, an important source of phenolic compounds and dietary flavanoids, coming from the Middle East. The antioxidant properties of phenolic compounds present in this plant. This plant belongs to the Liliaceae family, growing well in altitudes. Since it is conventional, it is used against digestive discomfort. Since it has antioxidant properties in its compounds. The plant's hydroalcoholic extract is used for bromobenzene toxicity (Kalantari et al.,

2018). Japanese herbal medicine called JTX (Juzen-taiho-to) which is known as Kampo formula is used for many diseases such as rheumatoid arthritis, cancer and atopic dermatitis. It consists of nearly ten medicinal herbs in it. Bromobenzene toxicity is caused by minimising the GSH first-line defence this JTX can increase the GSH and minimise the toxicity. It is first against the CCl4 induced toxicity which worked well. Since the CCl4 and bromobenzenehave a mechanism for inducing toxicity it is then tested against the bromobenzene(Yoshioka et al., 2016b). Rosa rugosa plant roots and a compound contained in it called Rosamultin have also been shown to treat the toxicity caused by the administration of bromobenzene in rats. On administration of bromobenzene, enzymes such as Aniline hydroxylase and N-demethylase aniline hydroxylase, which this plant reduces, will increase. It also causes an increase in the enzyme epoxide hydrolase. Among rats, bromobenzene poisoning causes lipid peroxidation that is avoided by extracting rosamultin and methanol. Nevertheless, it is not involved in increasing the rate of GSH, it has been shown to prevent bromobenzene-induced hepatotoxicity(Park et al., 1996). Zinc sulphate, in which MT (metallothionin) is caused by Zinc. MT plays a role as a free radicals endogenous scavenger. MT is a low molecular weight protein that works against oxidative stress. We are immune to radiation when MT is present in high cells. MT processing requires not only zinc or cadmium, but also some non-metallic compounds. Pre-treated rats with zinc or cadmium at low doses have been shown to inhibit bromobenzene toxicity 16. CoQ10 not only cures the toxicity of bromobenzene but also many toxic agents such as Acetaminophen, Isoniazid, rifampicin, 6-OHDA, Bisphenol A, Cadmium, and Oxytetracycline and used in diabetic rats too. Acetaminophen known analgesic and antipyretic which is usually safe at relatively low doses. However, at high doses, it leads to hepatotoxicity. Acetaminophen at rats of 100mg/kg body weight is proved to cause toxicity. Acetaminophen is usually metabolised by CYP450 enzyme. Glutathione that is present in the rats is decreased by the metabolised acetaminophen to initiates inflammation. CoQ10 which is a good source of antioxidants is used to decrease the toxicity(Fouad and Jresat, 2012). Those implicated in causing toxicity are certain antitubular medications such as isoniazid and rifampicin. Antitubular medications are used against tuberculosis; almost 99% of TB bacilli are destroyed. But it had some side effects in 1-2 percent of patients, such as peripheral neuritis and hepatotoxicity. CYP21 conducts metabolisms of these drugs as soon as it metabolizes it causes the inflammation-induced ROS. CoQ10 which can scavenge the free radicals it is used to decrease the toxicity(Baskaran et al., 2015). 6- Hydroxyl dopamine causes dopaminergic toxicity in rats. Administration of 6-OHDA cause symptoms similar to Parkinson's disease. Parkinson's disease is a neurodegenerative disease. CoQ10 is also proved prevent the mitochondrial dysfunction which is caused by the 6-OHDA(Prajapati et al., 2017). Cadmium in rats is responsible for reproductive toxicity. Heavy metal cadmium for various industrial uses. Cadmium toxicity can be caused by tobacco, smoke, and inhalation, intake of contaminated water or food. Cadmium releases huge amounts of ROS that contribute to oxidative stress and lipid peroxidation. CoQ10 along with Cadmium can improve the motility, sperm concentration, DNA integrity(Saha et al., 2019).

5. CONCLUSION:

This current study is done to evaluate the protective measures of an antioxidant CoQ10 against the environmental toxin Bromobenzene. Due to the inflammation caused in the liver, the enzymes in the liver are leaked into the blood which is used for the measurement. Bromobenzene increased the parameters such as ALP, AST, ALT, VLDL, LDL, Total and direct bilirubin, Triglyceride, Urea, Creatinine and Acid phosphatase and decreases antioxidants such as SOD, Catalase and reduced glutathione. CoQ10 increases the Antioxidants that are decreased by bromobenzene and decreases the parameters that are increased and reduce the toxicity.

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Conflicts of interest

Authors declare that they do not have any conflicts of interests.

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