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Protective Effect of Chlorogenic Acid on endoplasmic reticulum (ER) stress and Oxidative Stress Crosstalk in Mice

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ABSTRACT

Background & Objective: Cross-talk between endoplasmic reticulum (ER) stress and oxidative stress has been recognized as contributing to several pathological conditions, particularly those affecting the respiratory system. This study aims to investigate the potential protective role of chlorogenic acid (CA) against oxidative stress in a murine model of ER stress induced by tunicamycin.

Materials & Methods:

36 male C57BL/6 mice were divided into six groups: a control group (saline), a vehicle group (DMSO), a CA group, a tunicamycin group, a CA + tunicamycin group, and a CA + tunicamycin group (both with a 20 mg/kg CA dose). After 72 hours of treatment, protein levels were investigated by ELISA, mRNA levels by RT-PCR, and histopathological alterations were examined by H&E staining.

Results: We found that 20 mg/kg CA alleviated GRP78 mRNA levels, decreased MDA, and increased GSH levels. It also showed potent anti-inflammatory and anti-fibrotic effects in the lungs of ER-stress induced mice.

Conclusion: Chlorogenic acid administration decreased oxidative stress markers by alleviating ER stress. This indicates a mechanistic link between ER stress and oxidative stress.

Keywords: Chlrogenic Acid, Endoplasmic Reticulum Stress, Oxidative Stress, Lung

1. Introduction

The accumulation of unfolded or misfolded proteins, a hallmark of endoplasmic reticulum (ER) stress, significantly contributes to various lung pathologies. This aberrant cellular response triggers a cascade of events, including oxidative stress, inflammation, and apoptosis, ultimately leading to tissue damage and impaired lung function (1, 2). Existing research indicates that many plants, including those containing chlorogenic acid (CA), can be potent anti-inflammatory agents (3, 4). Chlorogenic acid (5-O-caffeoylquinic acid; CA), a naturally occurring polyphenol, exhibits many health-promoting properties (5). It is abundant in tomatoes (6), coffee beans (7), and tea (8), which contain a significant amount of chlorogenic acid (CA). Emerging evidence suggests that CA possesses cytoprotective effects against ER stress in other organs (9, 10). Recent studies have spotlighted its anti-inflammatory (3), potent antioxidant (11), anti-cancer (12), anti-fibrotic (13), and anti-apoptotic properties. Recent evidence indicates that CA has protective effects against endoplasmic reticulum (ER) stress in various organs (9, 14).

This study was designed to investigate the therapeutic potential of CA in ER stress induced by tunicamycin (TM). We hypothesized that CA could have a protective effect by mitigating oxidative stress, attenuating inflammation, and preserving lung tissue integrity in a mouse model of ER stress. The results provide valuable evidence that CA is effective in preventing and treating ER stress-related lung diseases.

2. Materials and Methods

2.1. Chemicals and Reagents

Chlorogenic acid (C3878, purity ≥ 95%) and tunicamycin (11089-65-9) were purchased from Sigma-Aldrich (St. Louis, MO, USA). Tunicamycin was dissolved in dimethyl sulfoxide (DMSO; Calbiochem, EMD Bioscience Inc., La Jolla, CA, USA).

2.2. Animal and Experimental Design

Male C57BL/6 mice weighing between 23 and 25 grams were obtained from the Animal Center of Pasteur Institute in Tehran, Iran. Each group in the experiment included six mice (n = 6 per group). The research was commenced following approval from the Institutional Animal Ethics Committee (Ethics Code: IR.MUQ.AEC.1401.031). The mice were maintained in standard laboratory settings, with a 12-hour light/dark cycle, a controlled temperature of 20–22 °C, and free access to water and standard rodent chow. (Pars Animal Feed Co., Tehran, Iran) (15). The animals were randomly assigned into six groups: saline (0.2 mL sterile normal saline intraperitoneal (i.p.) injection), CA (50 mg/kg chlorogenic acid i.p. injection) (16), vehicle (0.2 mL DMSO i.p. injection), TM (received a single i.p. dose of tunicamycin (2 µg/g body weight) to induce ER-stress) (17), CA 20-TM (pretreated with 20 mg/kg of CA 60 minutes prior to tunicamycin i.p. injection) (18), CA 50-TM (pretreated with 50 mg/kg of CA 60 minutes before tunicamycin i.p. injection). Thirty hours following Tunicamycin administration (19), the animals were anesthetized using sodium pentobarbital (20).

The thoracic cage was carefully excised, and the lungs were extracted. One section of the lung tissue was fixed in 10% formalin for histopathological analysis, while the other portion was set aside to assess antioxidant activity.

2.3. Histology

Lung tissues were harvested 30 hours after tunicamycin administration from each experimental group (n = 6). Samples were immediately fixed in 10% buffered formalin and subsequently processed following standard histological procedures. Following dehydration and paraffin embedding, tissue sections measuring 5 µm thick were prepared and stained with hematoxylin and eosin (H&E) (21). The stained sections were examined under an Olympus CX22 light microscope to evaluate histopathological changes. A descriptive analysis was performed by comparing representative microscopic fields across all experimental groups to assess the extent of tissue injury.

2.4. Antioxidant Assessment

Lung tissues from each experimental group (n = 6) were used to evaluate antioxidant levels. Approximately 100 mg of lung tissue was precisely weighed and homogenized in 1 mL of phosphate buffer, following the manufacturer's protocol (MDA: Cat. No. ZB-MDA-96A; GSH: Cat. No. ZB-GSH-96A). For each assay, 50 µL of either the standard or sample was transferred into labeled test tubes, followed by the addition of 50 µL of R4 reagent and thorough mixing. Subsequently, 1 mL of the ready chromogenic solution was added, and the mixtures were incubated in a boiling water bath for one hour.

Following incubation, the tubes were immediately cooled on ice and centrifuged at 3000–4000 rpm for 10 minutes. A 200 µL aliquot of the resulting pink-colored supernatant was then transferred to a microplate, and absorbance was recorded using an ELISA reader at 535 nm for malondialdehyde (MDA) and 405 nm for reduced glutathione (GSH) (22).

2.5. Real-time PCR analysis

Quantitative real-time PCR (qRT-PCR) was performed to measure GRP78 gene expression, using GAPDH as the internal control. Relative expression levels were determined using the comparative threshold cycle ($\Delta\Delta^{Ct}$) method. The primer sequences employed in this analysis are provided in Table 1 (23).

Table 1. Primer sequences for quantitative RT-PCR

Gene	primers sequences (Forward (top), Reverse (bottom))
GRP78	5′-*TGTGTGTGAGACCAGAACCG*-3′ 5′-*TAGGTGGTCCCCAAGTCGAT*-3′
GAPDH	Forward: 5’-TGGCCTTCCGTGTTCCTAC-3’ Reverse: 5’-GAGTTGCTGTTGAAGTCGCA-3’

Abbreviations are GRP78: Glucose-regulated protein 78, GAPDH: Glyceraldehyde 3-phosphate dehydrogenase.

2.6. Statistics analysis

Statistical analysis was conducted using one-way analysis of variance (ANOVA), followed by Tukey’s post hoc test to evaluate differences among different groups. All data were analyzed using SPSS software, version 18.0 (IBM Corp., Armonk, NY, USA). Results were presented as mean ± standard error of the mean (SEM), with statistical significance as $p < 0.05$.

3. Results

3.1. CA decreased TM-induced ER-stress in mice

Our results demonstrated that a single administration of TM effectively induced ER-stress, as evidenced by a marked elevation in GRP78 gene expression in the TM group (4.12 ± 0.35, $P < 0.05$) compared to the Saline and DMSO groups (1.00 ± 0.00 and 1.08 ± 0.021,

respectively; $P < 0.05$). In contrast, treatment with chlorogenic acid (CA) significantly attenuated GRP78 expression in both the CA 20–ER stress (2.61 ± 0.17, $P < 0.05$) and CA 50–ER stress groups (3.57 ± 0.38, $P < 0.05$) compared to the TM group (Figure 1).

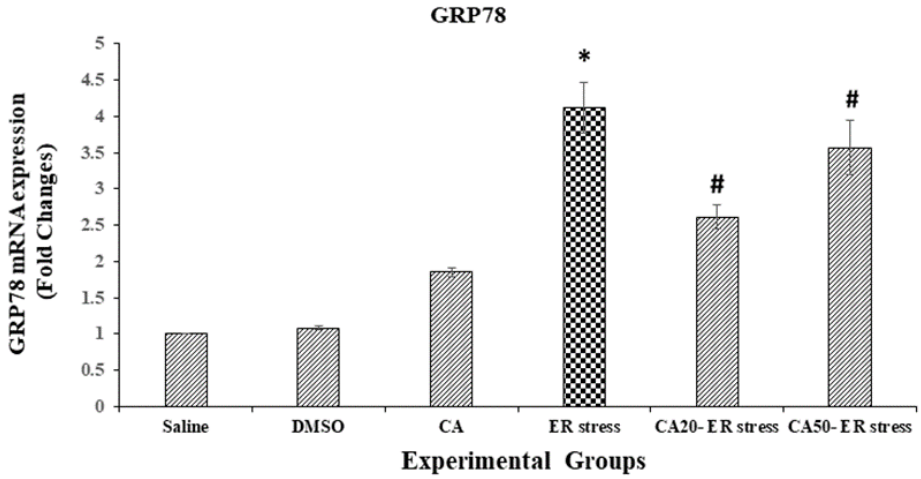

Figure 1. Relative mRNA expression levels of GRP78, an ER-stress marker, across the experimental groups. Data are expressed as mean ± SEM (n = 6 per group). *p < 0.05 vs. Saline group; #p < 0.05 vs. ER-stress group.

Abbreviations: CA, chlorogenic acid; GRP78, glucose-regulated protein 78.

3.2. CA enhanced lung antioxidant defense in the TM induced ER-stress in mice

The ER-stress exhibited a markedly elevated level of MDA (51.18 ± 11.1), an indicator of lipid oxidation, compared to the Saline and DMSO groups (23.54 ± 5.56 and 21.85 ± 5.9, P < 0.05, respectively). While, pretreatment with CA resulted in normal levels of MDA in the treated groups (31.25 ± 4.2 and 42.3 ± 3.82 P < 0.01, respectively), as illustrated in Fig. 2A.

However, GSH tissue levels were significantly decreased in the ER-stress group (0.024 ± 0.0053 P < 0.05, respectively) in comparison to the Saline and DMSO groups (0.071 0.0051 and 0.067 ± 0.0087, P < 0.05) and pretreatment with CA significantly increased GSH tissue levels compared with the ER-stress group (0.051 ± 0.0067 and 0.036 ± 0.0061, P < 0.05, respectively) as indicated in Figure 2B.

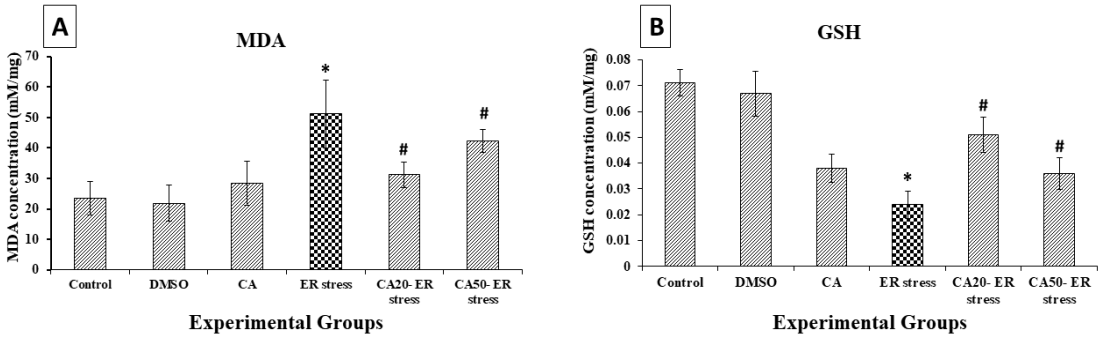

Figure 2. Levels of MDA and GSH between the experimental groups. Data are presented as mean ± SEM (n = 6 per group). *p < 0.05 indicates a significant difference compared to the Saline group; #p < 0.05 indicates a significant difference compared to the Tunicamycin (TM) group.

Abbreviations: CA, chlorogenic acid; MDA, malondialdehyde; GSH, glutathione peroxidase.

3.3. Histological finding

Hematoxylin and eosin (H&E) staining was performed to assess structural alterations in lung tissue. As illustrated in Figure 3, lung sections from the Saline and DMSO groups exhibited well-preserved architecture, characterized by clearly defined alveolar sacs, numerous alveoli with thin septa, and minimal presence of alveolar macrophages. These control groups exhibited no histological signs of fibrosis or inflammatory responses. In contrast, the ER-stress group displayed pronounced histopathological alterations, including marked thickening of the alveolar septa due to

dense cellular infiltration, increased numbers of interstitial alveolar macrophages, and widespread collapse or obliteration of alveolar spaces. Additionally, interstitial hemorrhage was frequently noted. These changes confirm the successful induction of ER stress–mediated pulmonary fibrosis in this model.

Lung tissues from the CA20-ER stress and CA50-ER stress treatment groups showed partial improvement in tissue architecture. Although interstitial hemorrhage and scattered alveolar macrophages were still evident, structural damage was noticeably reduced. Among the two treatment groups, the 20 mg/kg dose of chlorogenic acid demonstrated more pronounced protective effects, as evidenced by diminished fibrotic and inflammatory changes.

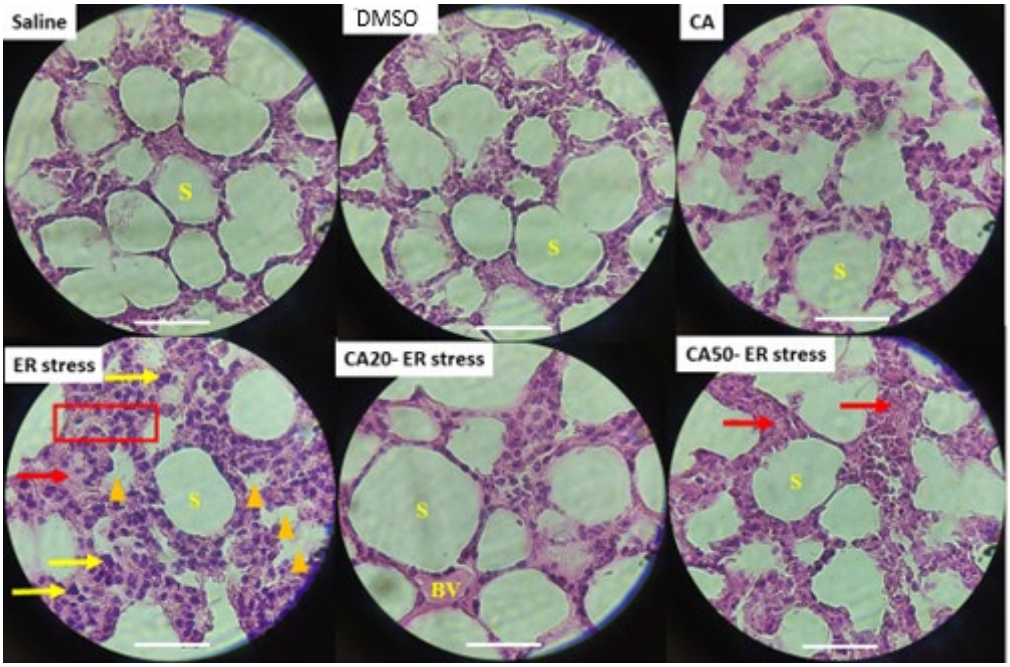

Figure 3. Representative high-magnification images of lung tissue stained with hematoxylin and eosin (×400 magnification; scale bar = 100μm). Yellow arrows indicate the presence of alveolar macrophages within the interstitial space and alveolar lumen. The red box highlights interalveolar septa infiltrated with inflammatory cells. Orange triangles denote areas of alveolar collapse or obliteration observed in the ER-stress group. Red arrows point to regions of interstitial hemorrhage. BV: blood vessels; S: alveolar sacs.

4. Discussion

Tunicamycin is a well-known pharmacological inducer of endoplasmic reticulum (ER) stress that has been shown to trigger ER-stress responses in lung tissue, ultimately contributing to the development of pulmonary fibrosis (10, 24). In this study, we evaluated GRP78, a key molecular marker of ER stress. As anticipated, GRP78 mRNA expression was significantly upregulated in the ER-stress group. Administration of chlorogenic acid (CA) at a dose of 20 mg/kg significantly resulted in a notable downregulation of GRP78 expression.

The results of the current study are consistent with previous research by Zhang et al (25), who demonstrated that CA attenuated the expression of GRP78, along with other ER stress markers such as PERK and IRE1, thereby alleviating bleomycin-induced pulmonary fibrosis (25). Another study showed that CA ameliorated paraquat-induced inflammatory, fibrotic, and oxidative injuries to the lungs in rats (26). These findings are consistent with our results and further spotlight the protective effect of chlorogenic acid in alleviating ER stress in the lungs of mice exposed to tunicamycin. However, in our study, the 50 mg/kg CA dose raised the ER-stress index and upregulated GRP78 expression (27). Thus, it appears that 50 mg/kg CA was toxic for lung tissue and increased this marker. Building on previous research in the field, it can be suggested that the influence of CA on gene expression occurs in a dose-dependent manner. (18).

Histopathological analysis revealed significant structural alterations in the lung tissue of ER stress-induced mice. These changes included substantial thickening of the alveolar septa due to dense cellular infiltration, a marked increase in the number of interstitial alveolar macrophages, and widespread alveolar collapse or obliteration. Additionally, interstitial hemorrhage was observed throughout the affected lung tissue, which is consistent with previous studies (10, 26). Among the treatment groups, administering chlorogenic acid (CA) at a dose of 20

mg/kg showed the most significant anti-fibrotic effect, preserving lung architecture in the ER-stress model.

In this study, the histological findings were consistent with the gene expression data for ER-stress markers, further supporting the protective role of chlorogenic acid (CA) in mitigating ER stress-induced lung injury. Earlier research has documented the anti-apoptotic and anti-fibrotic effects of chlorogenic acid (CA), indicating its potential as a therapeutic candidate for managing various pathological conditions **(5, 26, 27)**. However, in the present study, treatment with a higher dose of CA (50 mg/kg) did not lead to any significant improvement in lung tissue architecture in mice subjected to ER stress. This suggests that at higher doses, CA may be less effective in suppressing ER stress, potentially due to a diminished capacity to modulate ER stress pathways.

In this study, malondialdehyde (MDA) protein levels increased, while glutathione (GSH) levels decreased in mice challenged with TM. Prior research has demonstrated that MDA protein levels are upregulated in the ER-stress model in lung tissue **(28)** while GSH levels decrease in lung diseases **(29)**. In contrast, in this study, although 20mg/kg CA effectively decreased MDA, increased GSH levels in lung tissue, and attenuated TM-induced lung injury, the 50 mg/kg dose did not alter these markers, as it could not mitigate ER-stress. These findings suggest that chlorogenic acid, at lower concentrations, may provide a protective effect against tunicamycin-induced ER stress in lung tissue.

5. Conclusion

In conclusion, administering tunicamycin effectively induced endoplasmic reticulum stress in lung tissue. However, treatment with a low dose of chlorogenic acid significantly reduced this response. The alleviation of ER stress was accompanied by a corresponding decrease in oxidative stress markers, suggesting a relationship between ER stress and oxidative stress pathways. These findings highlight the potential therapeutic value of low-dose chlorogenic acid in mitigating ER-stress-related pulmonary injury.

6. Declarations

6.1 Acknowledgments

The authors gratefully acknowledge the financial support provided by Qom University of Medical Sciences.

6.2 Ethical Considerations

The research was commenced following approval from the Institutional Animal Ethics Committee. The medical ethics committee of Qom University of Medical Sciences (Ethics Code: IR.MUQ.AEC.1401.031) approved the study protocol.

6.3 Authors' Contributions

AM designed the experiments, TKM performed experiments and collected data, FH discussed the results and strategy, AM, TKM and FH final approved of the version to be published.

6.4 Conflict of Interest

The authors declare that there are no conflicts of interest.

6.5 Fund or Financial Support

The fund of this research was provided by the authors.

6.6 Using Artificial Intelligence Tools (AI Tools)

The authors were not utilized AI Tools.

6.7 Mechanistic approach and the limitation of present study

Due to the limitation in Funds, it was not possible to conduct more studies.

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