Effect of Coenzyme Q10 on Acute Pulmonary Damage Following the Experimental Thoracic Trauma

Deneysel Toraks Travması Sonrası Oluşan Akciğer Hasarı Üzerine Koenzim Q10 Etkinliği

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• *Presented as poster in TATKON 2011 Congress*
• *This study was supported by Erciyes University BAP (Unit of Science Research Project)*

Özet

Amaç: Deneysel künt toraks travması sonrası oluşan pulmoner kontüzyondaki birincil ve ikincil hasar üzerine Koenzim Q10'un etkisinin biyokimyasal ve histopatolojik parametreler ile değerlendirilmesi amaçlanmıştır. Gereç ve Yöntem: Bu çalışmada Wistar Albino cinsi 16 haftalık, 205±45 gr. ağırlığında 56 adet dişi rat kullanıldı. Her gurupta sekiz rat olacak şekilde rastgele yedi guruba ayrıldı. Travma modeli için sabit bir platform ve alimünyum tüpten oluşan bir travma düzeneği hazırlandı. Pulmoner kontüzyonu oluşturmak için 2.45 joule'lük bir travma şiddeti oluşturuldu. Kontrol ve Çalışma gurupları sakrifiye edilme zamanlarına göre isimlendirildi. Şam grubuna travma ve/veya ilaç uygulanmazken Kontrol gruplarına sadece travma oluşturuldu. Çalışma gruplarına ise travma uygulandıktan sonra 0. - 24.- 48. saatlerde Koenzim Q10 intraperitoneal uygulandı. Şam gurubu hemen, Kontrol ve Çalışma gurupları travmadan 24, 48, 72 saat sonra sakrifiye edilerek kan ve akciğer doku örnekleri alınarak incelendi. Bulgular: Şam grubu ile Çalışma 72 arasında Yüksek Duyarlıklı C-Reaktif Protein değeri açısından anlamlı fark yoktu. Histopatolojik olarak incelendiğinde; Çalışma grupları ile Kontrol grupları arasında fark gözlenmedi. Şam grubu ile Çalışma grupları arasında fark gözlenmez iken, Şam grubu ile Kontrol grupları arasında fark gözlendi. Tartışma:Künt toraks travmasında sekonder hasarı azaltamak için Koenzim Q10 antioksidan bir ajan olarak kullanılabilir.

Anahtar Kelimeler

Pulmoner Kontüzyon; Koenzim Q10; Deneysel Çalışma

Abstract

Aim: Pulmonary contusion negatively affects prognosis in the case of damages following a trauma. Objective of this experimental study performed in Turkey was to evaluate effects of coenzyme Q10 on primary and secondary damages of pulmonary contusion following experimental thoracic blunt trauma using biochemical and histopathological parameters. Material and Method: A total of 56 Wistar Albino female rats with a mean weight of 205±45 g were included in this study. Rats were randomly divided into seven groups with each group having eight rats. A trauma device which consisted of a fixed platform, and an aluminium tube was prepared. Rats were administered 2.45 J of chest impact energy in order to generate pulmonary contusion. Control and Study groups were named according to the sacrificed time. No process (trauma and/or medication) was performed in the sham group, while only trauma was induced in the controls. On the other hand, after induced trauma, intraperitoneal Q10 (0. - 24. - 48. hours) was administered to study group. Rats were sacrificed at the end of the after trauma 24, 48 and 72 hours, and their blood and lung tissue samples were analyzed. Results: No significant difference was found between sham and Study-72 groups in terms of high-sensitivity C-reactive protein. On the histopathological examination, no significant difference was found between study and control groups. While no significant difference was found between the sham and study groups, significant difference was observed between sham and control groups. Discussion: Coenzyme Q10, an antioxidant agent, can be used as an antioxidant agent in order to reduce the secondary damage in blunt thoracic trauma.

Keywords

Thoracic Injuries; Coenzyme Q10; Animal Experimentation

DOI: 10.4328/JCAM.3718 Received: 01.07.2015 Accepted: 31.07.2015 Printed: 01.12.2015 J Clin Anal Med 2015;6(suppl 6): 811-6
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Introduction

Traumas are one of the most important public health problems in the world. Severe thoracic trauma cases are accounted for about one-third of the patients hospitalized due to trauma. Increasing number of thoracic injuries, mainly due to traffic accidents is encountered in our country [1, 2].

Pulmonary contusion (PC) negatively affects prognosis in the case of damages following a trauma. Blunt thoracic trauma is often accompanied by moderate blunt trauma and severe PC. Thus, resuscitation efforts and supportive interventions have a positive effect on the prognosis if performed early. Although the exact mechanism is not fully understood, PC may cause various pathophysiological changes in a wide spectrum [3, 4]. Currently, there is not any widely accepted and standardized pharmacologic treatment approach for PC. Available treatment methods are limited to the options which are originated from the empirical observations and clinical judgments. Supportive care therapies are applied such as oxygen, cardiopulmonary monitoring, analgesia and preventive care for infection [4].

Pulmonary contusion has been demonstrated to be concomitant with a progressive inflammatory response mediated by the local and systemic immunological changes [4, 5]. Therefore, efficiency of antioxidant agents for treatment of PC is investigated.

The aim of the present study was to evaluate the effects of coenzyme Q10 on primary and secondary damages of pulmonary contusion following experimental thoracic blunt trauma using biochemical and histopathological parameters.

Material and Method

This experimental study was conducted with the permission of Erciyes University Experimental Animals Ethics Committee. Fifty-six 16-week Wistar Albino female rats with a mean weight of 205±45g were used. The standards recommended by the Council of Europe (European Convention For the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposed: ETS 123) were followed in this experimental study. The rats were accommodated in cages meeting the requirements of cleanliness and nutrition with a room temperature of 20 to 26 °C before and during the study as to be four rats per cage with circadian rhythms as 12-hour day/night in Erciyes University Experimental Animals Research and Breeding Laboratory. The rats were fasted 12 hours before the experiment. The rats were randomly divided into seven groups with each group having 8 rats:

• Group 1 (Sham group): Lung tissue and blood samples were collected without creating trauma.

• Group 2 (Control-24): Blunt thoracic trauma was created. Lung tissue and blood samples were collected by thoracotomy performed at 24th hour of trauma.

• Group 3 (Control-48): Blunt thoracic trauma was created. Lung tissue and blood samples were collected by thoracotomy performed at 48th hour of trauma.

• Group 4 (Control-72): Blunt thoracic trauma was created. Lung tissue and blood samples were collected by thoracotomy performed at 72nd hour of trauma.

• Group 5 (Study-24): Blunt thoracic trauma was created. CoQ10 was administered intraperitoneally at the hour zero. Lung tissue and blood samples were collected by thoracotomy performed at 24th hour of trauma.

• Group 6 (Study-48): Blunt thoracic trauma was created. CoQ10 was administered intraperitoneally at the hour zero and 24. Lung tissue and blood samples were collected by thoracotomy performed at 48th hour of trauma.

• Group 7 (Study-72): Blunt thoracic trauma was created. CoQ10 was administered intraperitoneally at the hour zero, 24 and 48. Lung tissue and blood samples were collected by thoracotomy performed at 72nd hour of trauma.

A trauma device which consisted of a fixed platform, and an aluminum tube was prepared. Our model to create blunt thoracic trauma was prepared by examining the similar studies in the literature, considering features such as variability of the trauma impact, easily ensured standardization (weight and height of the weight dropping may be changed), reproducibility, ease of implementation and portability [3, 4, 6].

Previous studies have shown that a trauma of approximately 2.45 j is required in order to create a life compatible PC in rats [3, 4, 6]. Cylinder height and weight to be dropped are calculated with the formula of E=mgh, in which, E stands for energy to be applied (joule), m stands for weight to be dropped (kg), g stands for gravitational acceleration (9.8 m/s2) and h shows height of the weight to be dropped (m). The proper height was found as 50 cm, and the weight 500 g. by this way, the trauma to be created was standardized.

Using this mechanism, rats were administered anesthesia and analgesia, and laid on the foam surface platform in a supine position as to be 45 degrees toward the left side in order to decrease the risk for the heart contusion. Except for Sham group in this position, a metal weight of 500 g was dropped from height of 50 cm with free fall at a constant speed through a cylindrical aluminum tube on the right hemithorax. Rats were followed after trauma.

All the rats were administered xylazine at a dose of 10 mg/kg and ketamine hydrochloride of 75 mg/kg through intraperitoneal route before the surgical processes. The doses not exceeding 20% of the initial doses were intermittently repeated when it was deemed necessary. Analgesia was provided with intraperitoneal morphine sulfate administration of 0.05 mg/kg before and after the trauma and surgical processes.

Coenzyme Q10 was dissolved in soybean oil. Soybean oil was sterilized at 135 °C in a gravity autoclave for 5 minutes. It was intraperitoneally administered at a dose of 30 mg/kg/day for anti-inflammatory efficiency of CoQ10 once a day. Then thoracotomy was performed using midsternotomy technique following the analgesia and anesthesia, and 5 ml of blood was drawn from the heart.

Blood samples sent for biochemical analysis were centrifuged at 3000 rpm for 10 minutes and separated to their serums. High - sensitivity C - reactive protein (Hs - CRP) was studied blindly with ELISA method. The results were evaluated as µg/mL.

Following the blood collecting, right and left main bronchi were clamped and euthanasia was applied. Lung tissues were rapidly removed. The samples collected for histopathological examination were put into 10% formaldehyde and fixed.

Sections of 5 mm thickness were obtained from the paraffin

blocks prepared from the lung tissues removed from the rats. These sections were stained with Hematoxylin and eosin (H & E) and studied blindly by a single pathologist under a light microscope at 40 and 100 magnification (Table 1) [3, 4, 6, 7].

Table 1. Evaluation of the histopathologic examination

		Grade 0	Grade 1	Grade 2	Grade 3
Atelectasis, congestion and intraalveolar hemoorhage		No	Focal	Patched (small, but multifocal foci from 5 magnification areas)	Diffused (along 5 magnification areas)
Parenchymal inflammation	According to cell density	No inflammation	Mild inflammation/ Individual cells	Moderate inflammation/ Aggregations forming groups of 2-4 cells	Severe inflammation/ aggregations > 5 cells
	According to cell type	No inflammation	Neutrophil weighed	Neutrophil and mononuclear cells weighed	Mononuclear cells weighed
Perivascular mononuclear inflammation		No	Mild (individual cells)	Moderate (cell groups)	Severe (aggregation forming cuff around the vessel)
Bronchial damage		No	Damage to focal epithelium	Destruction of focal wall	Intense damage in bronchiole with abscessing

Statistical analysis: Statistical analysis was performed with SPSS 15.0 (Statistical Package for Social Sciences, SPSS Inc, Chicago, USA) software. Evaluation of the differences between the groups was carried out with "Chi-Square" test. Mean levels of Hs-CRP were compared with "One-Way Analysis of Variance (ANOVA)" and histopathological evaluations were compared with Kruskal Wallis test. Holm-Sidak Test from the "Post - Hoc" test was applied. Compliance of the data with normal distribution was evaluated with "Shapiro-Wilk" and the data were found to comply with normal distribution. Values of P<0.05 were considered statistically significant.

Results

Hs-CRP: Mean level of Hs-CRP was found at the lowest level in the sham group, while this value was observed to be high in the control and study groups that were exposed to trauma. ANOVA results showed that groups were statistically different in terms of Hs-CRP (P=0,000). Holm-Sidak test to see statistically different groups indicated sham and Study-72 groups did not differ significantly (P>0.05), while other control and study groups had significantly higher Hs-CRP levels than the sham group (P<0.05). No statistically significant difference was found between control and study groups at 24, 48, and 72 hours (P>0.05) (Table 2) (Figure 1).

Histopathological Evaluation: Kruskal Wallis Test results showed that groups were statistically different in terms of histopathological evaluation terms (P=0.002 for atelectasis, congestion and intraalveolar hemorrhage, P=0.001 for cell density of parenchymal inflammation, P=0.000 for parenchymal inflammation according to cell type, P=0.000 for perivascular mononuclear inflammation and P=0.045 for bronchial damage) (Table 3).

Atelectasis, congestion and intraalveolar hemorrhage: Significant differences were observed between sham and control groups at 24, 48, and 72 hours in terms of atelectasis, congestion and intraalveolar hemorrhage (P<0.05), while no significant difference was observed between sham and study groups at 24, 48, and 72 hours (P>0.05). No statistically significant difference was found between control and study groups at 24, 48, and 72 hours (P>0.05) (Figure 2).

Cell density of parenchymal inflammation: Significant differences were observed between sham and Study-48 groups in terms of cell density of parenchymal inflammation (P<0.05), while no significant difference was observed among sham, Study-24 and Study-72 groups (P>0.05). No statistically significant difference was found between control and study groups at 24, 48, and 72 hours (P>0.05) (Figure 2).

Parenchymal inflammation according to cell type: Significant differences were observed among sham, control and Study-48 groups in terms of the cell type (P<0.05), while no significant difference was found between sham and Study-72 groups (P>0.05). No statistically significant difference was found between control and study groups at 24, 48, and 72 hours (P>0.05) (Figure 2).

Table 2. ANOVA results for Hs-CRP.

Groups	n	Mean	Std. Dev.	F	p	Homogenous Subsets
Sham	8	28.80	13.99	6.110	0.000	a
Control-24	8	52.80	9.44			b
Control-48	8	61.75	16.32			b
Control-72	8	56.24	14.21			b
Study-24	8	69.29	15.26			b
Study-48	8	52.75	8.12			b
Study-72	8	50.08	19.84			ab

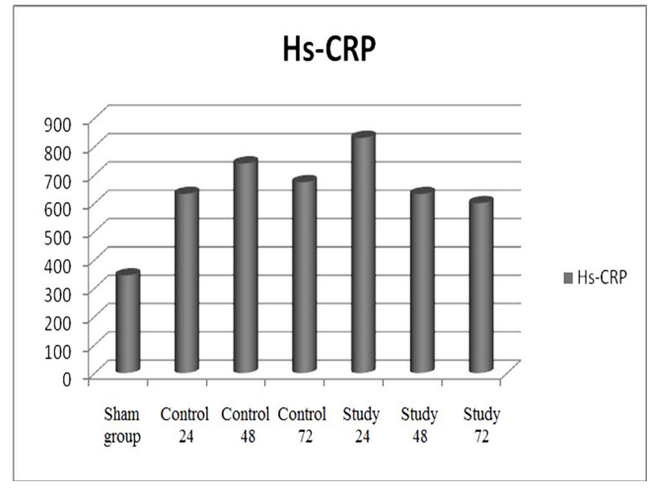


Figure 1. Mean value of Hs-CRP variations of the groups.

Table 3. Kruskal Wallis test results for histopathological evaluation

Histopathological Evaluation	Groups	n	Median	IQR	p	Homogenous Subsets
Atelectasis, congestion and intraalveolar hemorrhage	Sham	8	0.00	1.00	0.002	a
	Control-24	8	1.00	1.00		b
	Control-48	8	2.00	1.75		b
	Control-72	8	1.00	0.75		b
	Study-24	8	1.00	1.00		ab
	Study-48	8	1.00	0.75		ab
	Study-72	8	1.00	0.00		ab
Parenchymal inflammation	Sham	8	0.00	0.75	0.001	a
	Control-24	8	1.00	0.00		ab
	Control-48	8	2.00	1.00		ab
	Control-72	8	1.00	0.00		ab
	Study-24	8	1.00	1.00		ab
	Study-48	8	1.00	0.00		b
	Study-72	8	0.50	1.00		ab
Cell type	Sham	8	0.00	0.75	0.000	a
	Control-24	8	1.00	0.75		b
	Control-48	8	2.00	0.00		b
	Control-72	8	3.00	0.00		b
	Study-24	8	1.00	1.00		b
	Study-48	8	1.00	0.00		b
	Study-72	8	0.50	1.00		ab
Perivascular mononuclear inflammation	Sham	8	0.00	0.00	0.000	a
	Control-24	8	1.00	1.50		b
	Control-48	8	2.50	1.75		b
	Control-72	8	1.00	0.00		b
	Study-24	8	0.00	0.75		ab
	Study-48	8	0.00	0.75		ab
	Study-72	8	0.00	1.00		ab
Bronchial damage	Sham	8	0.00	0.00	0.045	a
	Control-24	8	-	-		-
	Control-48	8	0.50	1.00		b
	Control-72	8	0.00	0.75		ab
	Study-24	8	-	-		-
	Study-48	8	0.00	0.00		ab
	Study-72	8	0.00	0.00		ab

Perivascular mononuclear inflammation: Significant differences were observed between sham and control groups in terms of perivascular mononuclear grades (P<0.05), while no significant difference was found between sham and study groups (P>0.05). No statistically significant difference was found between control and study groups at 24, 48, and 72 hours (P>0.05) (Figure 2).

Bronchial damage: Because there was not any branchial damage observed on histopathologic examination of all the subjects in the sham, Control-24 and Study-24 groups, the differences between them could not be calculated. Significant differences were found between sham and Control-48 groups (P<0.05), while no significant difference was found among sham, Control-72, Study-48 and Study-72 groups (P>0.05). No statistically significant difference was found between control and study groups at 24, 48, and 72 hours (P>0.05) (Figure 2).

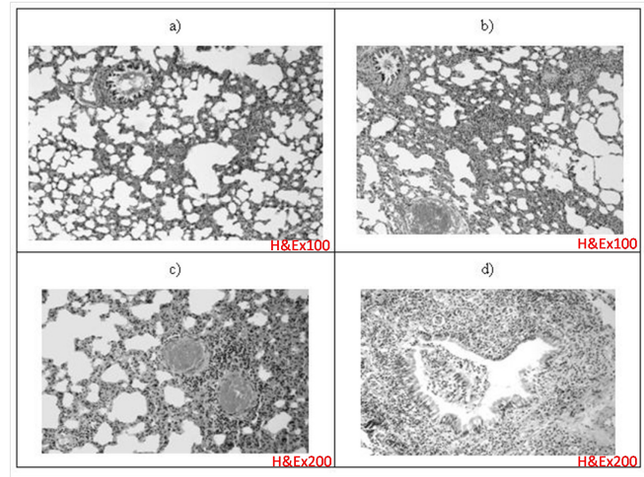


Figure 2. Appearances of histopathologic examination with Hematoxylin and Eosin (H&E) staining. a) Histopathological appearance of hemorrhage area in the parenchyma in the lung tissue of the rat at examination with H&E x 100. b) Histopathological appearance of inflammation area in the parenchyma in the lung tissue of rat at examination with H&E x 100. c) Histopathological appearance of lymphoplasmacytic cell infiltration around vessel in the lung tissue of the rat at examination with H&E x 200. d) Histopathological appearance of inflammation and damage in bronchial epithelium in the lung tissue of the rat at examination with H&E x 200.

Discussion

Blunt thoracic trauma may cause to PC in lung parenchyma. PC radiologically begins to be seen about six hours after trauma. Edema and interstitial hemorrhage begin to develop in the lung one or two hours after the injury. Blood and proteins begin to fill into intrapulmonary airways about 24 hours after the trauma. Contusion reaches to a maximum within 48 hours. Radiologically abnormal increase of opacity in a patch form and air bubbles develop. Lesions may be single or diffused. Lesions may be located in the perihilar region and areas adjacent to vertebra, sternum and ribs [8]. It is clear that understanding of the pathophysiology of blunt thoracic injury and demonstrating of the correlation between this pathophysiology and duration will elucidate the diagnosis and treatment processes, reducing morbidity and mortality [9].

Alpha-fetoproteins are known to elevate in traumas. Hs-CRP is an alpha-fetoprotein group. It is highly sensitive and can be detected even in the low levels, providing an advantage. It is more valuable than CRP in detection of slight elevations [10]. In their study, Is et al. stated that Hs-CRP elevates to very high levels in serum and cerebrospinal fluid samples of the patients with severe head traumas, thus might be used as an inflammatory index in traumas [11]. In a study by Turkyilmaz et al., level of PO2/FiO2 in arterial blood gas was the most sensitive parameter in determination of the grade of lung injury due to thoracotomy, which was followed by CRP, white blood cell, erythrocyte sedimentation rate, D - Dimer and fibrinogen [12]. In this study, mean values of Hs-CRP were found significantly higher in the groups exposed to trauma, consistently with the above-mentioned studies. In our study, no significant difference was found between the control and study groups in terms of Hs-CRP values (P>0.05). However, there was not a significant difference between the sham group and study 72 groups, which received more CoQ10 (three times; at the 0, 24 and 48th hours)

than the other study groups in terms of Hs-CRP values (P>0.05). We believed that this might increase the antiinflammatory efficacy of CoQ10.

In a study by Raghavendran et al., PC was studied by creating an experimental blunt thoracic trauma [13]. They found hemorrhagic damage beginning at 8th minute of the trauma, being observed at 4th and 8th minutes, involving perihilar areas and extending to the surface of the visceral pleura and concomitant impairment in alveoli with areas of diffuse intra-alveolar hemorrhage. They observed atelectasis to become prominent at 24th hour of the trauma and leukocyte count to increase with neutrophil predominant in alveolar cavity and interstitial area. They found at 48th hour of the trauma neutrophilic infiltration to maintenance to be predominant and intraalveolar edema to be observed. In their study, Turut et al. found intraalveolar hemorrhage, congestion and alveolar edema to be predominant with fragmented alveoli in the post-traumatic early period and leucocytic infiltration and atelectasis to be obvious at 24th hour of the trauma [3]. In this study, atelectasis, congestion and intraalveolar hemorrhage in a focal appearance, neutrophil weighed mild parenchymal inflammation and perivascular inflammation in the form of individual cells were observed, while no bronchial damage was found at the 24th hour. Atelectasis, congestion and intraalveolar hemorrhage were found to be increased; diffused and mononuclear cell weighed severe inflammation, perivascular mononuclear inflammation forming a cuff around the vessels and mild bronchial damage were observed at 48th hour. Atelectasis, congestion and intraalveolar hemorrhage in a focal appearance, mononuclear cell weighed mild parenchymal inflammation, and mild perivascular mononuclear cell inflammation were observed and bronchial damage was found to quite decrease at 72nd hour of the trauma. These results were similar to those of Raghavendran et al. and Turut et al. [3, 13].

Relative ischemia emerging with injury is followed by reperfusion. However, injury becomes more serious due to formation of free oxygen radicals during reperfusion [14]. In a healthy organism, there is a balance between formation and environmental accumulation rates of free radicals and removal or deactivation of these by antioxidants [15]. This is known as oxidative balance. Organism is not influenced by free radicals as long as the oxidative balance is maintained. Cells encounter with attack of the oxidant agents after a trauma. If the systems fail to neutralize this attack, irreversible destruction emerges [16].

Today, generally fluid restriction, high-dose steroids, antibiotics, diuretics, oxygen and mucolytic are used for treatment of PC due to blunt thoracic trauma. In addition, increasingly more understanding of the role of oxidant in development of PC and ARDS leads to increase in the number of the studies on the treatment to be managed through antioxidants. Aribogan et al. administered pentoxifylline as an antioxidant agent in adult patients with PC in the early periods following the injury; while Maxwell et al. used albumin in the pigs in which they created blunt thoracic injury and demonstrated it was effective as an antioxidant [17, 18]. In their study on rats with experimentally produced PC, Turut et al. used dexamethasone (DXM), N - acetylcysteine (NAC) and aprotinin (APR), and observed histopathological and biochemical improvement in lung damage [3]. In our study, CoQ10 was used as an antioxidant agent.

CoQ10 is a strong antioxidant which is a part of the mitochondrial electron transport chain, playing a role in membrane stabilization [19]. Decrease of CoQ10 indicates the occurrence of a secondary damage [20]. In their study, Kaikkonen et al. [21] demonstrated CoQ10 to decrease oxidative stress and to increase antioxidant capacity at in vivo settings. Furthermore, CoQ10 supplementation was seen to reduce lipid peroxidation and increase antioxidant capacity of plasma. In their study on rats with experimentally produced spinal trauma, Kerimoglu et al. demonstrated that methylprednisolone administered with CoQ10 is effective in spinal cord damage [7]. These studies indicate that CoQ10 can be used as an antioxidant. Numerous studies are conducted about many antioxidant agents in lung injuries [3, 17-20]. However, no study was found conducted using CoQ10.

In their study on rats with experimentally produced PC, Turut et al. used DXM, NAC and APR, and found a slight improvement both in PaO2 and PaCO2 values [3]. In addition, they demonstrated in the patients that level of MDA in lung tissue were decreased. Examining bronchoalveolar lavage fluid, they found neutrophil count of DXM to decrease. They demonstrated significant improvement on histopathologic examination of rat lungs received APR and DXM. Consequently, they observed that administration of DXM, NAC and APR in the early period results in desired improvement in lung tissue, which is affected from PC implementation.

Aribogan et al. found that pentoxifylline administration in early period following the injury, might be effective in acute inflammatory response and controlling of lung damage in the patients with PC identified [17]. In their study with pigs in which they created blunt thoracic trauma, Maxwell et al. used albumin as an anti-inflammatory agent [18]. They found albumin to decrease the damage mediated by oxidants that occurred in pulmonary alveolar capillary membrane, which plays a role in secondary damage.

In our study, on histopathologic examination significant difference was found between the Sham and control groups in terms of atelectasis, congestion and intraalveolar hemorrhage, while no significant difference was found between the sham and study groups. This indicates that CoQ10 decreases atelectasis, congestion and intraalveolar hemorrhage which occurred in PC. On evaluation in terms of parenchymal inflammation, a significant difference was found between sham and control groups, while no significant difference was found between Study-24 and Study-72 groups. This is consistent with other studies in the literature [3, 7, 17, 18, 20] and indicates CoQ10 to decrease inflammation and to have anti-inflammatory efficiency. However, in our study, no significant difference was found between Sham and Study-48 groups in terms of parenchymal inflammation. CoQ10 is seen not to have an impact on parenchymal inflammation at 48th hour. However, efficiency of CoQ10 compared to the other parameters is seen clearly. No difference was found between sham and Study-48 groups, suggesting it may be resulted from the inflammation to be maximal at 48th hour. When the environment of blood vessels was examined in terms of the grade of mononucleate inflammation, a significant difference was found between Sham group and Control groups,

while no difference was found between the study groups. This result is consistent with the other studies in the literature [3, 7, 17, 18, 20] and CoQ10 decreases the inflammation which occurred in PC.

This is caused by damaged areas and could not be clearly detected on histopathologic sections. Surface of weigh used in trauma mechanism covered all the surface of the rat lung. Thus, PC did not occur diffusely in all areas of rat lung tissue. A new trauma mechanism is needed to produce equally or proportionally distributed contusion in all areas of rat lung tissue.

In conclusion, although no significant difference was observed between the control and study groups; also no significant difference was found between the sham and study groups, suggesting CoQ10 that is an antioxidant agent may be used in order to reduce effects of oxidants, which are responsible for the secondary damage in blunt thoracic trauma. It is believed that increasing the frequency and dose of CoQ10 may increase anti-inflammatory efficiency. Further studies with a greater number of subjects, longer follow-up duration and increased frequency of implementation are needed in order to demonstrate that CoQ10 can be useful in PC treatment and increasing of its frequency and dosage to increase its efficiency.

Limitations: Trauma device is not completely involved the chest wall of a rat. In this case; same contusion is not developed in both sides of the lung. On the histopathological examination; encountering the affected cells due to trauma is completely depended to the macroscopic examination. Probability of the lung tissue being to be less or more affected from the trauma might negatively affect the study. A device would create an equal trauma severity on all the lung tissue is needed in order to prevent this condition.

Competing interests

The authors declare that they have no competing interests.

References

1. Çobanoğlu U, Melek M, Kara V, Mergan D. Isolated hemothorax following thoracic trauma: Analysis of 57 zases. J Clin Anal Med 2012;3(1):41-5.
2. Kaya H, Kafalı ME, Aydın K, Şahin M, Duran A, Bayır A et al. A Novel Experimental Bilateral Blunt Chest Trauma Model on Rabbits and its Effects in Lung. Journal of Academic Emergency Medicine 2011;10(3):103-9.
3. Turut H, Ciralik H, Kilinc M, Ozbag D, Imrek SS. Effects of early administration of dexamethasone, N-acetylcysteine and aprotinin on inflammatory and oxidant-antioxidant status after lung contusion in rats. Injury 2009;40(5):521-7.
4. Raghavendran K, Davidson BA, Helinski JD, Marschke CJ, Manderscheid P, Woytash JA, Notter RH et al. A rat model for isolated bilateral lung contusion from blunt chest trauma. Anesth Analg 2005;101(5):1482-9.
5. Hoth JJ, Stitzel JD, Gayzik FS, Brownlee NA, Miller PR, Yoza BK et al. The pathogenesis of pulmonary contusion: an open chest model in the rat. J Trauma 2006;61(1):32-44.
6. Yücel O, Genç O, Özcan A, Çaylak H, Gözübüyük A, Gürkök S et al. The blunt thoracic trauma model on rat lungs: an experimental study. Gulhane Medical Journal 2008;50(4):249-52.
7. Kerimoglu A, Pasaoglu O, Kanbak G, Hanci V, Ozdemir F, Atasoy MA. Efficiency of coenzyme Q(10) at experimental spinal cord injury. Ulus Travma Acil Cerrahi Derg 2007;13(2):85-93.
8. Marti de Gracia M, Artigas Martin JM, Soto JA. Evaluation of thoracic vascular trauma with multidetector computed tomography. Semin Roentgenol 2012;47(4):342-351.
9. Bhullar IS, Wagner S. Management of cardiac tamponade secondary to pneumomediastinum after blunt thoracic trauma. Am Surg 2010;76(6):50-1.
10. Neumaier M, Metak G, Scherer MA. C-reactive protein as a parameter of surgical trauma: CRP response after different types of surgery in 349 hip fractures. Acta Orthop 2006;77(5):788-90.
11. Is M, Coskun A, Sanus GZ, Tanriverdi T, Kafadar AM, Hanimoglu H et al. High-sensitivity C-reactive protein levels in cerebrospinal fluid and serum in severe head injury: relationship to tumor necrosis factor-alpha and interleukin-6. J Clin Neurosci 2007;14(12):1163-71.
12. Türkyılmaz A, Yurt ZK, Eroğlu A. Biological Markers of Acute Lung Injury After Thoracotomy. The Eurasian Journal of Medicine 2006;38(3):107-12.
13. Raghavendran K, Davidson BA, Woytash JA, Helinski JD, Marschke CJ, Manderscheid PA et al. The evolution of isolated bilateral lung contusion from blunt chest trauma in rats: cellular and cytokine responses. Shock 2005;24(2):132-8.
14. Juranek I, Bezek S. Controversy of free radical hypothesis: reactive oxygen species--cause or consequence of tissue injury? Gen Physiol Biophys 2005;24(3):263-78.
15. Mohseni S, Emtenani S, Emtenani S, Asoodeh A. Antioxidant properties of a human neuropeptide and its protective effect on free radical-induced DNA damage. J Pept Sci 2014;20(6):429-37.
16. Li W, Wu Y, Ren C, Lu Y, Gao Y, Zheng X et al. The activity of recombinant human neuroglobin as an antioxidant and free radical scavenger. Proteins 2011;79(1):115-25.
17. Arıboğan A, Özbek U, Reyhan E, Bilgin E, Oral U, Akman H. Künt Göğüs Travması Olgularında Pentoksifilin Uygulamasının Serum TNF-alfa düzeyi ve Akciğer hasarı Üzerine Etkileri. Çukurova Üniversitesi Tıp Fakultesi Dergisi 1999;24:32-41.
18. Maxwell RA, Gibson JB, Fabian TC, Proctor KG. Effects of a novel antioxidant during resuscitation from severe blunt chest trauma. Shock 2000;14(6):646-51.
19. Brandmeyer EA, Shen Q, Thimmesch AR, Pierce JD. Using coenzyme Q10 in clinical practice. Nursing 2014;44(3):63-6.
20. Garrido-Maraver J, Cordero MD, Oropesa-Avila M, Vega AF, de la Mata M, Pavon AD et al. Clinical applications of coenzyme Q10. Front Biosci (Landmark Ed) 2014;19:619-33.
21. Kaikkonen J, Nyyssonen K, Tomasi A, Iannone A, Tuomainen TP, Porkkala-Sarataho E et al. Antioxidative efficacy of parallel and combined supplementation with coenzyme Q10 and d-alpha-tocopherol in mildly hypercholesterolemic subjects: a randomized placebo-controlled clinical study. Free Radic Res 2000;33(3):329-40.

How to cite this article:
Koyuncu M, Avsarogulları Ö.L, Duman A, Denız K, Saraymen R, Ozkan S, Durukan P, Akdur O, Ikızcelı İ. Effect of Coenzyme Q10 on Acute Pulmonary Damage Following the Experimental Thoracic Trauma. J Clin Anal Med 2015;6(suppl 6): 811-6.