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RESEARCH ARTICLE

Effectiveness of Low-Level Laser Therapy for Treatment of Cats with Thoracic Trauma

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ABSTRACT

Although the mechanisms by which non-invasive, non-toxic, and non-polluting Low-Level Laser Therapy (LLLT) accelerates tissue healing and reduces pain and inflammation have been identified, its use in patients with thoracic trauma is unknown. This study aimed to investigate the effectiveness of low-level laser therapy in cats with thoracic trauma. The study was conducted in 48 cats with thoracic trauma. Group I (24 cats) received the standard thoracic trauma protocol, while Group II (24 cats) received additional LLLT. In addition to clinical examination findings, complete blood counts, biochemistry, blood gases, and bronchoalveolar lavage (BAL) fluid cytokine, antioxidant, and cytologic analyses were performed. The study findings showed that vital signs in the trauma patient remained at expected levels and could be controlled with symptomatic treatment, while no significant differences were observed in complete blood counts and biochemical parameters. Clinically, a significantly faster recovery in GII compared to GI, especially after day 3, was attributed to LLLT. LLLT limited the elevation of proinflammatory cytokines and preserved anti-inflammatory cytokine and antioxidant parameters. These findings support the therapeutic potential of photobiomodulation in models of inflammatory and oxidative stress. Similarly, cytological results demonstrated that LLLT significantly accelerated inflammatory regeneration at the cellular level.

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INTRODUCTION

In general, head and chest trauma in traumatized animals always carries a higher mortality rate than trauma to other parts of the body. Therefore, the management of chest trauma remains a constant concern. Whether the source of chest trauma is blunt or penetrating, it is serious and potentially life-threatening (Sigrist *et al.,* 2004; Salci *et al.,* 2010). On the other hand, while clinicians focus more on orthopedic issues than trauma management, chest trauma can cause cardiovascular and respiratory problems, as well as shock. Because chest trauma carries a high morbidity and mortality rate, early diagnosis and treatment are crucial. Post-traumatic thoracic complications can include tracheal trauma, pneumothorax, hemothorax, pulmonary contusion, rib fractures, flail chest, myocardial contusion, or perforation. Although there are studies addressing many different issues such as the importance of adequate oxygenation, shock management and thoracocentesis after trauma in patients affected by the thorax, pneumothorax or effusion accumulation in the thoracic cavity still remains a serious problem (Sigrist *et al.,* 2004; Salci *et al.,* 2010; Swinbourne, 2020).

Laser therapy has three main effects on animal tissue: first, it reduces inflammation, second, it reduces pain, and third, it accelerates healing. Although numerous studies have been conducted using laser at different doses and durations on wound healing, peripheral nerve injury, arthritis, bone healing, traumatic brain injuries, laryngitis, edema, lung injuries, and lung fibrosis (Gunn, 2005; Aimbire *et al.,* 2008; de Lima *et al.,* 2011; Hentschke *et al.,* 2013; Lee *et al.,* 2013; Marinho *et al.,* 2013 Cury *et al.,* 2016; Mehani 2017; Ocal and Kumandas 2019; de Brito *et al.,* 2020; Fazza *et al.,* 2020; Fesseha 2020; Ehrenzweig 2022), clinical studies are limited. Although the use of LLLT in thoracic trauma is not well known, it has been proven to contribute to treatment by

accelerating lung recovery in rats with experimentally induced (non-traumatic) lung injury or in humans with COVID-19 (Sigman *et al.*, 2020; Moskvin *et al.*, 2021; Nejatifard *et al.*, 2021).

LLLT, which has a photobiomodulation mechanism, activates mitochondria without causing damage to tissues. This occurs when laser light is absorbed by cytochrome c oxidase, increasing ATP production, accelerating cellular metabolism, and promoting tissue repair. In addition, cytochrome c-dependent nitric oxide (NO) release occurs. This release results in vasodilation and an increase in microcirculation, thus contributing to tissue oxygenation. This, in turn, indirectly triggers healing. Maintaining controlled levels of reactive oxygen species (ROS) is essential because controllable ROS activate cellular signaling pathways, stimulating proliferation and healing genes and contributing to healing. In addition, LLLT increases fibroblast proliferation, collagen synthesis, and stimulates angiogenesis (Fig. 1). In summary, LLLT stimulates the mitochondria of cells, increasing energy production, reducing inflammation, improving circulation, and accelerating tissue healing (Ocal and Kumandas, 2019; Okur and Okumus, 2023).

The aim of this study was to investigate the effectiveness of low-level laser therapy in cats with thoracic trauma.

MATERIALS AND METHODS

Patient Selection: Cats with trauma were initially assessed for age, sex, weight, and breed. Information was also obtained regarding the time elapsed since the trauma and whether any interventions had been performed. Baseline heart rate (HR), respiratory rate (RR), thoracic auscultation results, signs of respiratory distress, perfusion status, thoracic radiography findings, presence of other injuries, and time to discharge were recorded. In this study, respiratory and perfusion assessments were performed according to the classification outlined by Sigrist *et al.* (2004). Accordingly, baseline heart rate in cats was classified as normal (120–180 bpm), moderately elevated (180–200 bpm), or severely elevated (>200 bpm). Similarly, following RR and/or analgesic administration, RR was graded as normal (20–44 breaths/min), moderately increased (44–64 breaths/min), and severely increased (>64 breaths/min). Panting cats were defined as cats showing signs of labored breathing. Baseline perfusion status was assessed based on mucous membrane color, capillary refill time, and HR. Additional injuries were categorized as head trauma, intra-abdominal trauma, skeletal trauma to the forelimbs or hindlimbs, isolated skin wounds, spinal disorders, and combinations thereof. Then, thoracic (TFAST) and abdominal (AFAST) ultrasonography was performed to investigate the presence of free fluid or other possible pathologies.

This study used a radiographic (XR) score (Sigrist *et al.*, 2004) based on abnormal thoracic radiograph findings (pneumothorax, pneumomediastinum, lung contusions, chest wall trauma, chronic disease, and/or other abnormalities) and categorized as normal (0), mild (1), moderate (2), or severe (3). Cats with chronic lung disease were excluded from the study. Additionally, Animal Trauma Triage (ATT) scores (as Sigrist *et al.*, 2004) were defined as mild trauma (ATT scores 1-3), moderate trauma (ATT scores 4-7), and severe trauma (ATT score 8). After evaluating cats with a history of trauma, a total of 48 cats with thoracic trauma, moderately elevated HR and RR, moderate ATT scores, and moderate radiographic scores were included in the study according to the criteria described above.

Trauma Management and Study Groups

Standard Treatment (Group I, n=24): Based on clinical examination findings, oxygen therapy was administered via mask to stabilize respiration. Venous catheterization was performed subsequently or simultaneously. All patients were monitored for HR, RR, systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), body temperature, and SpO_2. Lactated Ringer's solution and mannitol infusion were administered for fluid support. Meloxicam (0.5 mg/kg, IV) was used for pain control. Tranexamic acid (10 mg/kg, IV) was administered in cases of suspected intrathoracic bleeding and no more than 2 hours after trauma. Amoxicillin and clavulanic acid (8.75 mg/kg, IM) were used as antibiotics. Furosemide (2.5 mg/kg IM) was preferred to preserve renal function and reduce local edema. Thoracocentesis was performed in animals with suspected pneumothorax or hemothorax using a three-way stopcock under aseptic conditions to diagnose pleural pathology and restore negative intrapleural pressure. In cases where air or fluid accumulation persisted in the pleural space, a thoracostomy tube was placed under local anesthesia (intercostal block) (as recommended by Salci *et al.,* 2010), and continuous drainage (Heimlich valve) and intermittent aspiration were performed. The animals were monitored and stabilized, and radiographic and ultrasonographic examinations were completed.

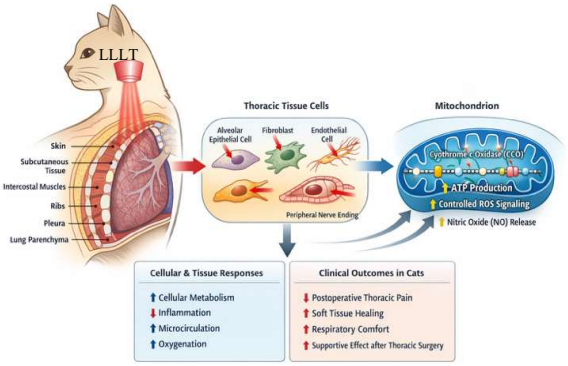

Fig. 1: Mitochondrial photobiomodulation mechanisms underlying the therapeutic effects of LLLT.

LLLT Application (Group II, n=24): In this group, in addition to standard treatment, LLLT was applied directly to the skin in the traumatized area. A portable infrared laser device (Lasermed 2200, Eme Phsio, Italy) with a wavelength of 904 nanometers (nm) (Gallium-Arsenic [GaAs]) and a power output of 25mW was used for the treatment. Following the reference of Arza (2017), the treatment was performed with a 904 nm wavelength laser at a dose of 3 J/cm², holding the LLLT beam at a right angle. This treatment was applied for 60 seconds for seven days. The laser probe was held at a 90° angle to the rib cage using the full contact technique. LLLT treatment was applied by the same person at the same time each day, and protective goggles were worn during the procedure.

In our study, blood and BAL fluid taken at baseline (0) and on days 3, 5 and 7 of treatment were used to determine blood count, biochemistry (such as AST, ALT, ALP, GGT, CK-MB, TP, albumin, cholesterol, triglyceride, glucose, urea, and creatinine), inflammatory response, antioxidant (MDA, NO, and SOD) and cytokine levels (TNF-α, IL-6, IL-8, and IL-10). BAL was performed blindly by inserting an endotracheal tube and flushing with 3-10 ml of warm sterile saline solution following a brief induction with propofol (2-4 mg/kg IV) as previously described by Crisi *et al.* (2019) (Fig 2).

Fig. 2: Images of BAL fluid collection.

For histopathological evaluation, BAL fluids were centrifuged at 800g for 7 minutes at +40°C. The supernatant was used for cytokine analysis at -80°C. The cell pellet remaining at the bottom of the tube was suspended in 1 ml of PBS. For inflammatory cell counting, 0.1 ml of the cell suspension was transferred to a slide, and the prepared slides were stained using May-Grunwald-Giemsa and Papanicolaou staining methods to determine the inflammatory index. Inflammatory cells were graded from 1 to 3 as previously described by Stanzel (2012). The distribution of inflammatory cells in BAL cytology was assessed at high magnification (×40 objective magnification) using a light microscope (E-400; Nikon, Tokyo, Japan) equipped with a DS-RI1 video camera (DS-U3, Nikon, Tokyo, Japan), and three different fields in each preparation were quantified by image analysis.

Minitap 18 package program was used for statistical calculations in the study. Data were evaluated using the normality distribution test and analyzed using parametric or nonparametric tests (Mann Whitney U, Paired T) or Kruskall Wallis, one-way ANOVA). A P level of <0.05 was considered statistically significant.

RESULTS

The cause of trauma in the study was identified as falls from height in 41 (85%) cases, traffic accidents in 5 (20%) cases, and trauma of unknown cause in 1 (4%) case. Cat breeds were not taken into account. The physical conditions of the cats, their vital signs on initial clinical examination, and their respiratory and circulatory status are summarized in Tables 1 and 2. Additionally, the radiography (XR) score is given in Table 3, and photographs of some cases were given (Fig. 3, 4, 5).

Blood, biochemical, antioxidant and cytokine levels and histopathological data obtained from the study were summarized in tables (Table 4-7). The cellular composition of BAL fluid did not differ significantly between GI and GII, summarized in Table 7, Fig. 6,7.

Table 1: Distribution of age, gender, body weight and vital values of cats by groups

	GI (n=24)	GII (n=24)	Vital values	GI	GII
Age (years)	2.91±2.18[a]	3.20±2.02	HR	188.79±32.00[a]	194.63±30.67[a]
Gender	10 ♀, 14 ♂ [a]	16 ♀, 8 ♂	RR	54.25±15.86 [a]	56.08±14.79 [a]
Weight (kg)	3.12±0.99 [a]	4.04±1.48 [a]	SAP	151.46±21.71 [a]	160.58±16.56 [a]
ATT score*	5.62±0.87 [a]	5.58±0.92 [a]	DAP	97.33±6.74 [a]	104.08±5.26 [b]
			MAP	98.83±12.93 [a]	103.50±4.57 [a]
			VT	37.97±0.11 [a]	37.78±0.42 [a]
			SpO2	79.92±8.02 [a]	75.00±5.31 [a]

* ATT score results were defined as mild trauma (ATT score 1-3), moderate trauma (ATT score 4-7), and severe trauma (ATT score 8), and moderate trauma patients were included in the study. Animals were also excluded if they had already been stabilized by the referring veterinarian or if more than 48 hours had passed since the trauma.

a, b: shows the statistically significant difference in each row, P<0.05.
Pulse rate (HR), respiratory rate (RR), systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), body temperature (VT), and SpO2 values

Table 2: Categorization of respiration and circulation according to Sigrist *et al.*, (2004)

Groups	HR (beats/min)			RR (breath/min)		
	Normal 120-180	Moderate 180-200	Severe >200	Normal 20-44	Moderate 44-64	Severe >64
GI (n=24)	8 (33%)	6 (25%)	10 (41%)	5 (20%)	10 (41%)	9 (37.5%)
GII (n=24)	7 (29%)	8 (33%)	9 (37.5%)	4 (16%)	9 (37.5%)	11 (45%)
Additional Information						
	Normal	GI: 8 GII:6		<1 sec	-	
	Pale	GI:16 GII: 17		<2 sec	GI:18 GII: 14	
Color of mucous membranes	Hyperemic	-	Capillary refill time	>2 sec	GI: 6 GII: 7	
	Cyanotic	GI:0 GII:1				
Number of cats out of breath			GI	9		
			GII	11		
Those with additional injuries						
			GI	GII		
Head trauma			1 (4%)	1 (4%)		
Intra-abdominal trauma			2 (8%)	1 (4%)		
Forearm or hind limb trauma			5 (20%)	7 (29%)		
Skeletal trauma			4 (16%)	3 (12%)		
Isolated skin wounds			3 (12%)	2 (8%)		
Vertebral disorders			1 (4%)	3 (12%)		
Combinations of these			2 (8%)	1 (4%)		

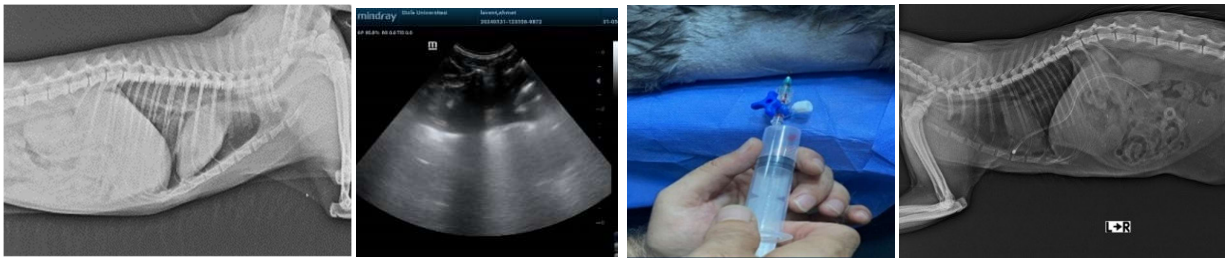

Fig. 3: Pneumothorax case; A: thorax radiograph, B: screenshot during TFAST, C: air aspiration by thoracocentesis, D: in another case, a chest tube was placed.

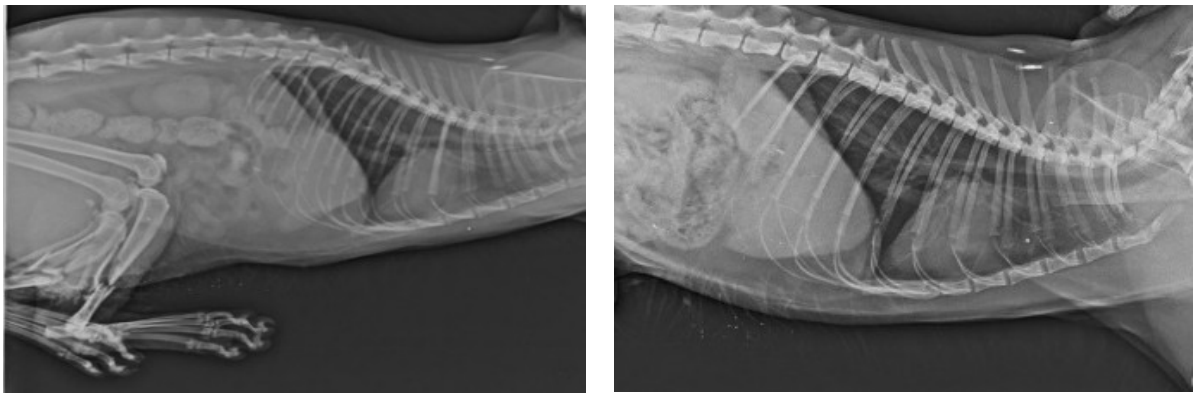

Fig. 4: Images from the 3rd day of the trauma; A. Pleural effusion cranial to the heart on the thoracic radiograph (L-R laterolateral view). C. post-treatment thoracic radiograph in GII (L-R laterolateral view). A significant decrease in the effusion is striking.

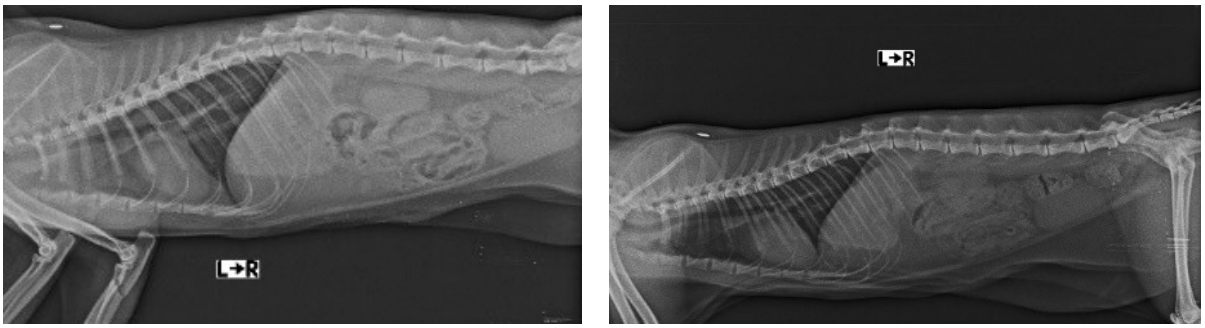

Fig. 5: Chest radiograph on day 3 of LLLT application. A clear difference is seen in the cranial aspect of the heart.

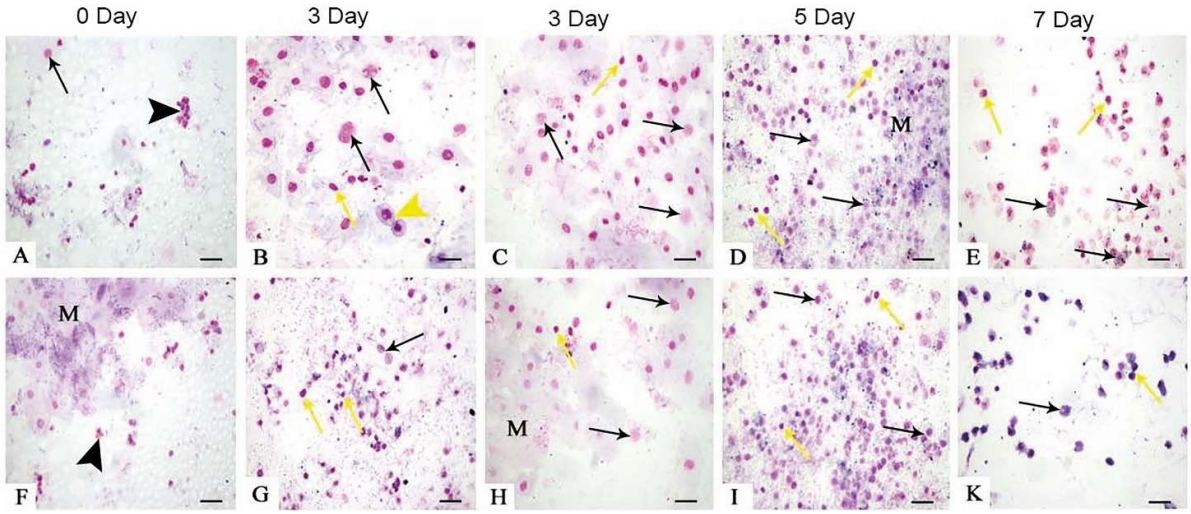

Fig. 6: Appearance of erythrocytes, macrophages, lymphocytes, epithelial cells, and mucus in the Treatment (A-E) and Laser groups (F-K) according to application days. Black arrowhead; erythrocyte clusters, yellow arrowhead; epithelial cells, black arrow; macrophages, yellow arrow; lymphocytes, and M; mucus. Wright-Giemsa staining. Bar: 25 µm.

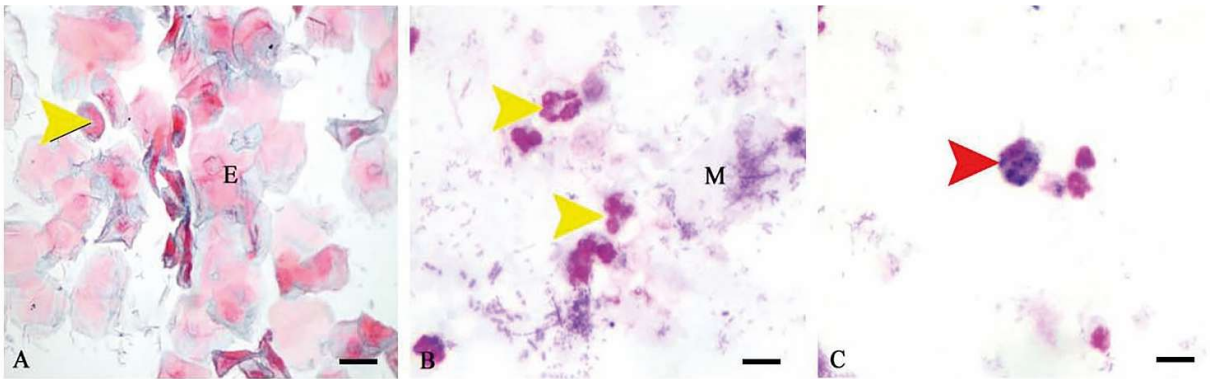

Fig. 7: Appearance of epithelial cells (A), neutrophils (B, yellow arrowhead), eosinophils (A, yellow arrowhead), and basophils (C, red arrowhead) in both GI and GII groups. E; epithelial cells, M; mucus. Papanicolaou's stain. Bar: 25 µm.

Table 3: General status of the cases according to XR scoring*

Lesions (Point)		GI	GII
Pneumothorax	Normal (0)	11 (45%)	12 (50)
	Mild (1)	11 (45%)	8 (33%)
	Moderate (2)	2 (8%)	4 (16%)
	Severe (3)	-	-
Contusion	Normal (0)	21 (87.5%)	19 (79%)
	Mild (1)	3 (12.5%)	5 (20%)
	Unilateral (2)	-	-
	Bilateral (3)	-	-
Chest wall trauma	Normal (0)	21 (87.5%)	20 (83%)
	Mild (1)	2 (8%)	3 (12.5%)
	Moderate (2)	1 (4%)	1 (8%)
	Severe (3)	-	-
Chronic disease and/or other abnormalities	Normal (0)		
	Mild (1)	-	-
	Moderate (2)	-	-
	Severe (3)	-	-

* XR score is a score composed of points given according to the severity of pneumothorax, lung contusions, chest wall trauma and chronic disease.

Table 4: Biochemistry, complete blood and blood gas results of the groups

Biochemical parameters	GI	GII	Complete blood count findings	GI	GII
AST (IU/L)	129.38±24.38 [a]	184.17±48.18 [b]	RBC (10^6/ µL)	5.03±0.85	5.08±0.49
ALT (IU/L)	158.50±65.70 [a]	220.30±64.90 [b]	WBC (µL)	16500±1995	18521±926
ALP (IU/L)	68.75±24.19 [a]	90.00±12.51 [b]	Neutrophil (µL)	13542±1058	14500±896
GGT (IU/L)	11.08±3.72 [a]	16.08±3.13 [b]	Lymphocyte (µL)	966.90±96.0	917.10±103.10
CK-MB (IU/L)	8146±2744 [a]	9729±3501 [a]	Platelet (µL)	250000.101±	201012±98.20
TP g/dL	6.19±0.95 [a]	6.67±0.84 [a]	Hct (%)	27.62±1.52	26.79±0.83
Albumin g/dL	2.27±0.19 [a]	2.64±0.27 [b]	Blood gases	GI	GII
Cholesterol mg/dL	83.33±15.23 [a]	114.17±32.43 [b]	pH	7.28±0.97	7.35±0.08
Triglycerides mg/dL	95.00±20.22 [a]	115.83±20.62 [b]	PvCO2 mmHg	40.33±2.51	40.45±2.87
Glucose mg/dL	186.25±42.41 [a]	202.75±38.03 [a]	PvO2 mmHg	34.54±2.94	36.16±2.35
Urea (BUN) mg/dL	44.17±7.09 [a]	49.33±7.17 [a]	HCO3 mmol/L	18.00±0.28	18.06±0.18
Creatinine mg/dL	1.40±0.45 [a]	2.36±0.28 [b]	BE mmol/L	-3.75±0.46	-4.12±0.26
Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma glutamyl transferase (GGT), CK-MB, total protein (TP).			Lactate mmol/L	3.80±0.34	4.01±0.84
			Na mmol/L	148.25±18.32	162.80±44.17
			K mmol/L	4.80±0.18	5.01±0.22
			Cl mmol/L	118.80±12.24	126.40±23.36

a,b: shows the statistically significant difference in each row, P<0.05.

Table 5: Serum and BAL fluid cytokine results (ng/L)

Group /Day		Serum				BAL fluid			
		TNFα	Il 6	IL 8	IL 10	TNFα	Il 6	IL 8	IL 10
G I	0	43.75±22.02[a]	33.79±28.13	87.20±113.23	584.67±311.47	84.4±68.83	28.62±15.70	53.75±19.59	656.33±350.30
	3	77.93±31.15[ab]	27.35±17.82	121.34±115.78	676.33±282.39	56.76±21.87	28.87±12.77	51.88±22.39	874.67±413.40
	5	86.17±26.56[b]	19.42±15.43	94.04±73.69	411.33±51.54	102.01±27.13	29.68±15.08	56.45±24.10	618.00±171.44
	7	65.38±10.88[ab]	37.31±13.46	42.78±14.52	739.67±164.40	87.76±52.95	31.05±17.81	38.61±22.68	828.00±157.83
a,b: shows the statistically significant difference in each column, P<0.05.									
G II	0	36.71±19.16	28.28±12.28	89.87±101.20	668.00±99.35[ab]	79.52±57.19	93.09±41.51	73.55±11,89	939.67±284.30
	3	59.87±34.83	23.48±12.21	87.95±119.78	678.00±68.34[b]	40.79±44.16	81.46±11.48	77.52±6,57	1078.00±311.18
	5	66.78±39.51	18.84±10.41	29.88±10.15	481.33±153.81[a]	117.23±20.94	87.68±49.74	64.64±40,00	1016.33±535.34
	7	53.81±19.62	29.05±15.58	69.93±55.20	738.00±127.23[b]	98.61±65.45	49.79±31.53	42.98±11,93	1198.00±369.91

Table 6: Plasma and BAL antioxidant and oxidative stress parameter results

Group	Days	Plasma			BAL fluid		
		MDA (nmol/mL)	SOD (ng/mL)	NO (µmol/mL)	MDA (nmol/mL)	SOD (ng/mL)	NO (µmol/mL)
GI	0	7.70±3.09^{a}	3.10±3.80	38.62±23.12	7.35±1.99^{a}	4.50±2.30	20.77±14.47
	3	10.81±0.99^{ab}	1.04±1.62	41.08±36.13	9.55±1.42^{ab}	1.51±1.27	20.74±9.41
	5	8.03±1.09^{a}	1.57±1.94	25.88±11.62	7.15±1.19^{a}	4.35±3.34	15.70±10.66
	7	11.87±1.89^{b}	2.66±2.61	30.81±13.31	10.58±2.78^{b}	2.26±1.58	18.69±15.62
a,b: shows the statistically significant difference in each column, P<0.05.							
GII	0	6.87±2.73^{a}	3.46±2.29	34.63±8.04^{ab}	8.26±3.16	9.10±6.72^{b}	56.26±7.13^{bc}
	3	9.19±1.38^{ab}	1.00±0.81	31.71±7.99^{ab}	11.29±3.79	3.83±0.44^{ab}	68.31±7.68^{c}
	5	6.14±0.41^{a}	3.14±5.01	22.44±10.27^{a}	8.16±2.20	2.78±1.66^{a}	40.98±29.40^{ab}
	7	10.43±2.81^{b}	2.60±0.92	38.83±9.05^{b}	11.39±2.40	3.04±1.12^{a}	25.84±12.19^{a}

a,b: shows the statistically significant difference in each column, P<0.05.

Table 7: Differential cell density in BAL fluid in cats.

Group	Days	Cell types				
		Macrophage	Lymphocyte	Neutrophil	Eosinophil	Basophils
GI	0	1.0±0.63	0.50±0.55	0.50±0.55	0.33±0.52	0.17±0.41
	3	2.33±0.82	2.0±0.63	0.83±0.75	0.50±0.55	0.17±0.41
	5	1.67±0.82	0.92±0.57	0.85±0.52	0.47±0.51	0.33±0.51
	7	1.33±0.52	0.84±0.63	0.50±0.55	0.17±0.41	0.17±0.41
GII	0	0.83±0.40	0.67±0.52	0.33±0.52	0.33±0.52	0.33±0.52
	3	2.17±0.98	1.83±0.41	1.0±0.63	0.33±0.52	0.16±0.41
	5	1.50±0.84	1.16±0.40	0.83±0.41	0.50±0.54	0.16±0.41
	7	1.50±0.55	1.16±0.75	0.33±0.52	0.16±0.40	0.16±0.40

Results are presented as mean ± SD. 1: inflammatory cells representing less than 10% of the cell population observed in the BAL fluid, 2: inflammatory cells representing 10% to 50% of the cell population observed in the BAL fluid, 3: inflammatory cells representing more than 50% of the cell population observed in the BAL fluid.

DISCUSSION

Chest trauma is clinically significant, particularly in terms of mortality. During falls from height, which are common in cats, the cat's first contact with the ground occurs when it lands on its forearms and sternum, affecting the rib cage. Because the rib cage houses vital organs such as the trachea, aorta, heart, and lungs, conscious and professional management of chest trauma will directly impact survival. While numerous studies, including human studies, have been reported on chest trauma, studies on possible post-traumatic pathologies, particularly effusion and pneumothorax, remain relevant (Özaydın 2004a; Özaydın 2004b; Sigrist *et al.,* 2004; Salci *et al.,* 2010; Swinbourne, 2020; Özaydın and Erdikmen, 2023). Although the mechanisms of action of LLLT in accelerating tissue healing by reducing inflammation are known (Arza, 2017; Rogatko *et al.,* 2017; Okur and Okumus, 2023), its use in thoracic trauma is quite limited. Therefore, this study aimed to investigate the effectiveness of LLLT in the treatment of cats with thoracic trauma.

In the evaluation of trauma patients, ATT scores adapted from human medicine are defined as mild trauma (ATT scores 1-3), moderate trauma (ATT scores 4-7), and severe trauma (ATT scores 8) (Sigrist *et al.,* 2004). For standardization purposes, patients with moderate trauma were included in this study. This minimized the impact of trauma severity on both treatment outcomes and survival rates. Initial examination findings were found to be quite similar between the study groups. This was attributed to the selection of patients from the same group for the ATT scoring. Therefore, the use of the ATT score in trauma management will contribute to the process. In addition to the vital values checked at the first examination of the cases, there was a great deal of similarity between the groups in all parameters such as biochemistry and blood gases, and there was a statistically significant difference in some values, but what was most striking was that there was an increase in both vital values and other blood results that could be considered moderate compared to the cat reference values. The cause of tachycardia was attributed to pain, stress, hypovolemia or an expected condition that developed after trauma, while the increase in respiratory rate was associated with thoracic trauma. Partial hypothermia seen in all groups suggests vasoconstriction. A careful fluid selection along with quickly started oxygen therapy is effective at this stage. One of the important issues is the speed of administration of the fluid, and it should never be given too quickly in case of pulmonary edema.

While no significant increase in biochemical parameters was observed, there was an increase compared to reference values. This can be explained entirely by the lack of oxygen and muscle breakdown in the trauma patient. Increased ALT, AST, and CK-MB may be due to muscle breakdown, while increased lactate, in particular, is due to anaerobic respiration or decreased oxygen levels. Changes in BUN and creatinine ratios suggest hypovolemia. Another important value is a decrease in albumin levels; bleeding should not be ignored in this situation. Among biochemical parameters, lactate change is an important consideration, and a lack of sustained lactate decrease despite treatment (especially at or above 6 mmol/L) indicates a poor prognosis (Turgut 2000; Özaydın 2004a; Özaydın 2004b; Swinbourne 2020; Özaydın and Erdikmen 2023; Eris *et al.,* 2025).

Although there was no statistically significant difference when whole blood data were compared between groups in the study, increases or decreases were observed in both groups. The decrease in RBC and HCT values may be due to bleeding or anemia. WBC, which is considered important for sepsis, was close to the upper limit, and antibiotic use is sometimes recommended in thoracic trauma (Turgut 2000; Özaydın 2004a). In our study, sepsis and related problems were not encountered without the need for a second antibiotic.

In patients exposed to trauma, tissue hyperperfusion should be taken into consideration as it will provide important information about whether adequate oxygenation and ventilation are provided, shock, acid-base balance and the current metabolic state. In this study, changes in blood gases were observed in both groups. The reason for the pH change is hypoperfusion and metabolic acidosis that develop with the decrease in oxygenation, as shown by the increase in lactate. The changes in all blood gases in this study are consistent with a metabolic acidosis due to early shock or hypoperfusion for both GI and GII. This condition can be managed by oxygenation, regulation

of ventilation and appropriate fluid therapy, but in more serious increases the clinical picture may not be so favorable. Another reason why our cases are not very severe is that they are moderately severe trauma patients according to the ATT score. On the other hand, pneumothorax was confirmed in almost half of our cases in both groups, and pneumothorax alone would be sufficient to disrupt ventilation (Sigrist *et al.,* 2004; Salci *et al.,* 2010; Swinbourne 2020).

In cases where ventilation is affected, such as pnuomothorax, effusion or lung contusion, hypoperfusion should be prevented as well as rapid initiation of oxygen therapy, but solutions should be considered for the underlying cause. In such cases, thoracentesis is performed with a three-way tap, which is extremely easy and economical. However, if the problem persists despite the application, a thoracic tube may be required (Salci *et al.,* 2010). In this study, thoraconcetesis was performed in many patients and effusion or air was evacuated from the thorax. A tube was placed in only two cases. We believe that thoracocentesis should be performed without waiting in suspicious cases.

Parameters measured in this study included proinflammatory cytokines (TNF-α, IL-6, IL-8), anti-inflammatory cytokines (IL-10), and oxidative stress markers (MDA, SOD, NO). TNF-α, in particular, stands out as one of the primary proinflammatory mediators that triggers immune cell infiltration, increased endothelial permeability, and alveolar cell death in lung tissue. IL-6, a critical mediator of the acute phase response, promotes both local and systemic inflammation and can exacerbate tissue damage by increasing neutrophil activation. IL-8, a potent chemokine, plays a central role in the progression of inflammation, particularly by inducing neutrophil migration into lung tissue. These findings are consistent with the literature, as it has been previously shown that TNF-α and IL-6 levels are increased in experimental lung injury models and that these parameters are directly related to the degree of injury (Matute-Bello *et al.,* 2008; Fanelli and Ranieri, 2015). Similarly, IL-8 has been reported to be highly associated with alveolar neutrophil accumulation and tissue destruction, particularly in models of acute respiratory distress syndrome (ARDS). Therefore, these cytokines are considered important tools not only for understanding the pathophysiological process but also as biomarkers of lung injury in both clinical and experimental research. In serum analyses, TNF-α and IL-8 levels peak on day 5 of GI and then decline, indicating rapid activation of inflammation and partial activation of homeostatic control. This dynamic confirms that the cytokine response in acute lung injury is both rapid and regulated.

Differences in the levels of the anti-inflammatory cytokine IL-10 are an important indicator of the pro-inflammatory-anti-inflammatory balance inherent in the immune response. The significant increase in serum IL-10 levels in the GI tract on day 7 suggests a compensatory mechanism activated to control excessive immune activation (Moore *et al.,* 2001). Conversely, low IL-10 levels suggest that inflammation is insufficient to suppress inflammation and contribute to the progression of tissue damage (Standiford *et al.*, 1995). In intense inflammatory processes such as acute lung injury, overproduction of proinflammatory cytokines such as TNF-α, IL-1β, and IL-6 exacerbates tissue damage, while IL-10 production is activated as a homeostatic response and acts to suppress inflammation (Moore *et al.*, 2001). Therefore, the increase in IL-10 levels reflects compensatory mechanisms activated to control excessive immune activity. On the other hand, low IL-10 levels appear to be insufficient to suppress inflammatory processes and contribute to the exacerbation of histopathological damage. The literature reports that IL-10 deficiency exacerbates the inflammatory response in lung injury, increasing neutrophil infiltration and worsening tissue damage (Standiford *et al.*, 1995). Conversely, IL-10 overexpression can increase susceptibility to infections by oversuppressing the immune response (O'Garra and Vieira, 2007). Therefore, the IL-10 differences obtained in this study highlight the importance of individual and group-level immune response differences in the regulation of inflammation and support the notion that IL-10 is a critical biomarker in the pathophysiology of lung injury (Saraiva and O'Garra, 2010).

The increase in MDA detected in this study demonstrates that oxidative stress, which develops as a result of excessive production of reactive oxygen species (ROS) and inadequate antioxidant defense mechanisms, plays a significant role in the pathogenesis of lung injury. ROS accumulation not only leads to lipid peroxidation; it also causes multiple cellular damages such as DNA damage, protein oxidation, and mitochondrial dysfunction. Thus, oxidative stress interacts with the inflammatory response, creating a cycle that increases the severity of tissue damage. Similarly, previous studies emphasize the critical importance of oxidative stress biomarkers, particularly lipid peroxidation products such as MDA, in the processes of lung injury and acute respiratory distress syndrome (ARDS) (Rahman and MacNee, 2000; Crimi *et al.*, 2006; Carvalho *et al.*, 2014). The increase in MDA observed in this study demonstrates that lung injury is associated not only with inflammatory mechanisms but also with cellular destruction mediated by oxidative stress.

Examination of BAL fluid is widely used to assess disease progression and response to treatment in lung infections. May-Grünwald Giemsa staining is considered the gold standard in BAL cytology (Haley *et al.*, 1989). In this study, BAL fluid cytology was evaluated with May-Grünwald Giemsa and Papanicolaou staining, and many visual and cytological findings were similar in both groups. Furthermore, the presence of macrophages, lymphocytes, neutrophils, eosinophils, basophils, erythrocytes, and epithelial cells suggested that lung trauma from falls from height in cats is similar to the cellular consequences reported in infectious, inflammatory, and parasitic diseases (Crisi *et al.,* 2019).

During tissue damage, platelets aggregate at the site of injury, limiting blood loss by adhering to damaged vessels and regulating other aspects of tissue repair, including cell migration, extracellular matrix (ECM) remodeling, cell proliferation and differentiation, and the angiogenesis/neovascularization process. Neutrophils, monocytes, and mature macrophages then migrate to the damaged organ/tissue within a few hours. This constitutes the "inflammatory" phase of tissue repair. Macrophages that settle in the damaged tissue contribute significantly to the resolution of inflammation, the recruitment and stimulation of stromal cells, and the remodeling of newly

produced ECM (Bouchery and Harris 2019; Tao *et al*., 2023). Macrophages in the lung play a crucial role in the innate and adaptive immune responses of organ systems against particles and pathogens. Macrophages contribute to the development and progression of acute or chronic inflammatory responses through the secretion of inflammatory cytokines/chemokines and the activation of transcription factors in the pathogenesis of inflammatory lung diseases (Lee *et al.,* 2021). In our study, macrophages were the predominant cell type in BAL cytology in both groups, followed by lymphocytes, neutrophils, eosinophils, and basophils. As mentioned above, these cells (especially macrophages) may play a direct or indirect role in the regeneration of lung trauma-related injury.

Conclusions: This study once again demonstrated that the clinical picture following trauma is consistent with changes in vital signs, biochemical parameters, and blood gases, and that normalization of this condition is possible with recommended standard treatments. However, LLLT, particularly applied to the GII, appears to have yielded a clinically significant difference when added to standard treatment on the third day after trauma. LLLT, when used in conjunction with standard treatment, was observed to limit the increase in proinflammatory cytokines and preserve anti-inflammatory cytokine and antioxidant parameters. These findings support the therapeutic potential of photobiomodulation in inflammatory and oxidative stress models. BAL cytology markers showed that the predominant cell type in both groups was macrophages, followed by lymphocytes, neutrophils, eosinophils, and basophils, suggesting that these cells (especially macrophages) may play a direct or indirect role in the regeneration of thoracic trauma-related injury. In conclusion, this study demonstrates that LLLT is a supportive treatment for thoracic trauma.

Ethical approval: This study was carried out with the permission of Dicle University HADYEK dated 12/07/2023 and numbered E-14891685-020-528274. In addition, patient owners were informed, and consent was obtained.

Author contribution: SY designed the study. EÇ, BEK, NS collected the retrospective data and interpreted the results. FA measured cytokines and antioxidants, HS and UT performed cytology. SY wrote the article. All authors interpreted the data, revised the manuscript critically for important intellectual content, and approved the final version.

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