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The relationship between troponin levels in pneumonia and the severity of pneumonia and the oxidant-antioxidant balance

Sevil Öner*[1], Ayşegül Karalezli[2]

[1]Department of Pulmonary Diseases, Yenimahalle Training and Research Hospital, Ankara, Turkiye
[2]Department of Pulmonary Diseases, Faculty of Medicine, Ankara Yıldırım Beyazıt University, Ankara, Turkiye

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***Corresponding Author:** Sevil Öner, sevilpempeci@gmail.com

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ABSTRACT

Aims: This study aimed to determine the association between serum troponin levels and pneumonia severity, and to evaluate the oxidant-antioxidant balance in patients with community-acquired pneumonia.

Methods: A total of 100 patients diagnosed with community-acquired pneumonia and 100 healthy volunteers were enrolled in this prospective, case-control study conducted at Ankara City Hospital between October and December 2019. Troponin, native thiol, total thiol, disulphide, ischemia-modified albumin, C-reactive protein, and procalcitonin levels were analyzed and compared between groups. Pneumonia severity was determined using the Pneumonia Severity Index.

Results: Troponin levels were significantly higher in the pneumonia group than in the control group ($p<0.05$), with a mean troponin of 48.03 ng/L in pneumonia patients and 3.15 ng/L in controls. Troponin levels correlated positively with c-reactive protein and procalcitonin values ($p<0.05$) and were significantly higher in patients with abnormal echocardiography and electrocardiogram findings. Native thiol and total thiol levels were significantly lower in pneumonia patients, while the disulphide/native thiol and disulphide/total thiol ratios were higher ($p<0.05$). Ischemia-modified albumin, levels were also higher in the pneumonia group ($p<0.05$). Troponin and ischemia-modified albümin levels increased significantly with pneumonia severity index stage.

Conclusion: Elevated serum troponin and ischemia-modified albümin levels, along with altered thiol-disulphide balance, may reflect increased oxidative stress and disease severity in pneumonia. These biomarkers may contribute to clinical assessment and prognosis in pneumonia cases.

Keywords: Pneumonia, cardiac troponin, thiol, disulfide, ischemia-modified albumin

INTRODUCTION

Pneumonia accounts for a significant proportion of physician visits, treatment costs, work and school days lost, and deaths worldwide.[1] It is the most common cause of death from infection.[2,3] Several inflammatory biomarkers are used to predict disease severity and monitor treatment in pneumonia cases.[4] At the same time, some objective criteria have been defined to assist physicians in deciding whether to hospitalise patients. The most commonly used index in this regard is the Pneumonia Severity Index (PSI). The selection of the appropriate empirical antibiotic and the severity of the patient's condition in pneumonia patients are determined according to the PSI.

Cardiac troponins (cTn) are highly sensitive and specific markers of myocardial damage. In acute coronary syndrome, elevated cTn levels are important for both prognosis and treatment guidance. However, elevated cTn levels may also be seen in conditions other than acute coronary syndrome, such as heart failure (acute and chronic), aortic dissection, aortic valve disease or hypertrophic cardiomyopathy, cardiac contusion, cardioversion, ablation, pacing, inflammatory diseases such as myocarditis and pericarditis, pulmonary embolism or severe pulmonary disease, hypothyroidism, and renal dysfunction.[5] There is no comprehensive study on the relationship between elevated troponin levels and the severity of pneumonia in children, except for one study conducted in children.

Antioxidant defence systems are responsible for preventing damage caused by reactive oxygen species in the body. Thiols are organic compounds containing a sulphydryl (-SH) group that play a critical role in preventing the formation of any oxidative stress conditions in cells. They play important roles in stabilising protein structures, regulating protein functions, regulating enzyme functions, and in receptors, carriers, Na-K channels, and transcription.[6]

There is considerable evidence indicating that abnormal thiol disulphide homeostasis plays a role in the pathogenesis of various diseases. In respiratory system diseases such as asthma, chronic obstructive pulmonary disease (COPD), and asthma-COPD overlap syndrome, community-acquired pneumonia and pulmonary thromboembolism.[7–9]

Ischemia-modified albumin (IMA) is a test that has received food and drug administration (FDA) approval among newly investigated cardiac markers.[10] The principle of the test is based on the reduction in albumin's cobalt-binding capacity due to chemical changes in albumin caused by oxidative free radicals formed during ischaemia, hypoxia and acidosis. This new albumin molecule is also called ischaemia-modified albumin. The formation of this new albumin molecule, which has lost its cobalt-binding ability, is one of the earliest markers of ischaemia.[11] Recent studies show that IMA, which has come to the fore as a cardiac ischaemia marker, may also increase in different pathologies.[12,13] In today's conditions, where we are constantly exposed to oxidative stress, there are insufficient studies evaluating the relationship between pneumonia and oxidative stress. This study aimed to investigate the relationship between troponin levels in pneumonia and the severity of pneumonia, and to assess the oxidant-antioxidant balance, thereby exploring its potential use as a biomarker for pneumonia and for grading the severity of pneumonia. Although individual associations between pneumonia and cardiac troponin, thiol-disulfide homeostasis, or ischemia-modified albumin have been previously reported, there is limited evidence regarding the co-assessment of these biomarkers in adult patients with community-acquired pneumonia, particularly in relation to disease severity. Co-assessment of these biomarkers could potentially improve the assessment of pneumonia severity by providing a more comprehensive perspective on the interaction between myocardial damage, oxidative stress, and inflammatory load in pneumonia. In our study, we showed that serum troponin and ischemia-modified albumin levels were increased in patients diagnosed with pneumonia and that, as assessed by the PSI, they changed in parallel with pneumonia severity. We share our study to contribute to the literature.

METHODS

This study was conducted at the Ankara City Hospital Chest Diseases Clinic between October and December 2019. Ethical approval for this prospective, case-control study was obtained from the Clinical Researches Ethics Committee of Yıldırım Beyazıt University Faculty of Medicine (Date: 09.10.2019, Decision No: 102). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. The study population included outpatients and inpatients who presented to the Chest Diseases Clinic of Ankara City Hospital with a diagnosis of community-acquired pneumonia during the study period. Patients were classified according to the PSI as follows: outpatients with PSI I and II, inpatients with PSI III, IV, and V, and patients admitted to intensive care based on intensive care admission criteria.

In our study, 100 patients diagnosed with pneumonia and 100 healthy volunteers were compared based on troponin, native thiol, total thiol, disulphide, IMA, haemogram, biochemistry, postero-anterior (PA) chest X-ray, echocardiography (ECHO), electrocardiography (ECG), and chest computed tomography (CT) when necessary. The medical history, comorbidities, and physical examination findings of the pneumonia patients were recorded. Pneumonia patients were grouped according to whether they received outpatient treatment or inpatient treatment in the ward or intensive care unit. Patients were also assessed according to the PSI.

In pneumonia patients, a hemogram, biochemistry, arterial blood gas analysis, and PA chest X-ray were routinely performed to determine the severity of the disease. Chest CT was performed when necessary. The medical history, comorbidities, and physical examination findings of pneumonia patients and control group patients were recorded. Cardiovascular assessment of pneumonia patients was performed by a cardiologist using ECG and ECHO with cardiology consultation.

CRP levels were measured using a BNII Nephelometer Analyser (Siemens, Munich, Germany) with the CardioPhase hsCRP kit (Siemens Healthcare Diagnostics Products, Marburg, Germany) by turbidimetry. PMNLs were counted using the Sysmex XE-2100 automated haematology system (Sysmex, Kobe, Hyogo, Japan). Troponin T levels were measured using the cobas e411 device with the elecsys troponin T hs kit. IMA measurement was performed using a rapid, colorimetric method developed by Bar-Or et al. to determine cobalt reduced to albumin-binding capacity (IMA level). Briefly, 200 µL of patient serum was transferred to glass tubes and 50 µL of 0.1% $CoCl_2$ x $6H_2O$ (Sigma Aldrich Lot: S38901-248; Sigma Aldrich, St. Louis, MO, USA) was added. After gentle shaking, the mixture was incubated for 10 minutes to ensure sufficient cobalt albumin binding. Next, 50 µL of 1.5 mg/ml dithiothreitol (DTT) (Sigma-Aldrich Lot: D5545-1G; Sigma-Aldrich) was added as a decolourising agent. After 2 minutes, 1 ml of 0.9% NaCl was added to stop the cobalt-albumin binding. A blank was prepared for each sample. At the DTT addition step, 50 µL of distilled water was used instead of 50 µL of 1.5 mg/ml DTT to obtain a DTT-free blank. Absorbances were recorded at 470 nm using a spectrophotometer. Colour formation in DTT-containing samples was compared to colour formation in blank tubes, and results were expressed in absorbance units (ABSU) (Bar-Or D L. E., 2000).

The thiol disulphide balance measurement was performed using the automated spectrophotometric method described by Erel & Neselioglu.[6] In this method, disulphide bonds were reduced with sodium borohydride to form free functional thiol groups. Unused reducing sodium borohydride was removed by consumption with formaldehyde to prevent the reduction of 5,5'-dithiobis-(2-nitrobenzoic) acid (DTNB). All thiol groups, including reduced and native thiol groups, were determined after reaction with DTNB. The amount of dynamic disulphide was obtained as half the difference between total thiols and native thiols. After determining native and total thiols, disulphide amounts were calculated. The disulphide/native thiol, disulphide/total thiol, and native thiol/total thiol ratios were then calculated as percentages.

The inclusion criteria for the study group were: agreeing to participate in the study, signing the consent form and being

a volunteer, having a diagnosis of pneumonia in accordance with the American Thoracic Society (ATS) criteria, and being over 18 years of age. Those with respiratory complaints who were found to have no disease following examination and testing, those with a history of lung disease, radiological sequelae control, screening examinations, etc., having agreed to participate in the study, having signed the consent form, being a volunteer, and being over 18 years of age were determined as the criteria for inclusion in the control group. Patients under 18 years of age, pregnant women, those who did not sign a written document indicating their acceptance of the study, those with mental disabilities, those with acute coronary syndrome, those with renal failure, those with a history of trauma, those with pulmonary thromboembolism, and those with a recent history of coronary intervention were not included in the study.

RESULTS

The mean age of the 100 patients diagnosed with pneumonia was 65.8±15.1, while the mean age of the control group was 59.0±16.0. Thirty-eight per cent (n=38) of the pneumonia group were female and 62 per cent (n=62) were male, while 54 per cent (n=54) of the control group were female and 46 per cent (n=46) were male (p values, respectively; p=0.071, p=0.102) (**Table 1**).

Table 1. Demographic characteristics of patients

Pneumonia group			Control group	
	Mean±SD	Median (min-max)	Mean±SD	Median (min-max)
Age (years) (mean±SD)	65.8±15.1	66 (19 -92)	59.0±16.0	59 (19 -86)
	Pneumonia group	Control group	p-value	
	n	n		
Gender				
Female	38	54	p<0.05	
Male	62	46	p<0.05	
Total	100	100		
Smoking history				
Smoker	16	18	p>0.05	
Ex-smoker	50	31	p<0.05	
Non-smoker	34	51	p<0.05	
Total	100	100		

SD: Standard deviation, Min: Minimum, Max: Maximum

When troponin levels were assessed in pneumonia, troponin values were found to be significantly higher in the pneumonia group compared to the control group (p<0.05) (**Table 2, Figure 1**).

Table 2. Pneumonia-control group troponin value rank average

Troponin	n	Rank average	Sum of the series	U	Z	p
Pneumonia group	100	135.58	13558	1492	-9.584	.000
Control group	100	65.42	6542			

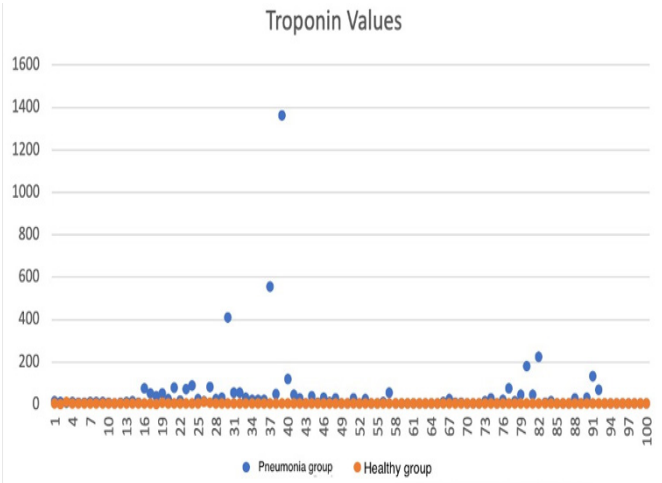

Figure 1. Pneumonia-distribution of troponin values in the control group

According to the parametric test results, the mean troponin level in the pneumonia group was 48.03 ng/L, while the mean in the control group was 3.15 ng/L; troponin levels were statistically higher in the pneumonia group. In the pneumonia group, troponin levels were statistically higher in those with pathological ECHO and ECG results compared to those without pathology (p<0.05). The troponin levels of pneumonia patients with normal ECHO were within normal limits when compared to the control group (**Table 3**).

Table 3. Average troponin values in the pneumonia group according to ECHO and ECG

Average troponin value	Normal/median (min-max)	Pathological/median (min-max)
ECHO	3 ng/L (3-75.03)	32.9 ng/L (3,13-1360)
ECG	5.56 ng/L (3-409.2)	42.95 ng/L (19-66)

Min: Minimum, Max: Maximum, ECHO: Echocardiography, ECG: Electrocardiography

When examining the relationship between C-reactive protein (CRP) and procalcitonin (PCT), which are useful in the diagnosis and follow-up of pneumonia, and troponin, a moderate, significant positive correlation was found between troponin and CRP values in the pneumonia group (p<0.05). A high and significant positive correlation was found between troponin and PCT values in the pneumonia group (p<0.05).

When examining whether there was a significant difference in troponin values between the pneumonia groups according to the PSI stage, a statistically significant difference was observed between troponin values in at least two stages (p<0.05). A significant difference was observed between stage 1 and stages 3, 4, 5, and between stage 2 and stages 3, 4, 5 (**Table 4, Figure 2**).

When evaluating thiol disulphide levels in the pneumonia group, native thiol and total thiol levels were found to be significantly lower compared to the control group (p<0.05). There was no statistical difference between the disulphide values of the pneumonia group and the control group (p>0.05). The disulphide/native thiol ratio and the disulphide/total thiol ratio in the pneumonia group were statistically higher than in the control group (p<0.05). The native thiol/

Table 4. Troponin values according to the PSI stage in the pneumonia group

Groups	n	Rank average	Degree of liberty	p	Significant difference
PSI 1	18	16.06	4	.000	PSI 1-PSI 3 PSI 1-PSI 4 PSI 1-PSI 5 PSI 2-PSI 3 PSI 2-PSI 4 PSI 2-PSI 5
PSI 2	15	21.47			
PSI 3	14	57.07			
PSI 4	39	68.00			
PSI 5	14	70.57			

n: Number of patients, PSI: Pneumonia Severity Index

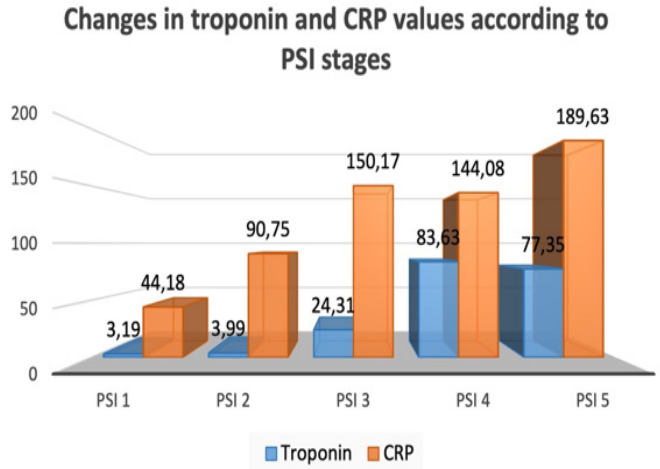

Figure 2. Change in troponin and CRP values according to PSI stages
CRP: C-reactive protein, PSI: Pneumonia Severity Index

total thiol ratio in the pneumonia group was statistically lower than in the control group (p<0.05) (**Table 5**).

When examining the relationship between native thiol values, total thiol values, disulphide values, and PSI stages, a significant difference was found between native thiol values and PSI stages between stage 1 and stage 4.5, and between stage 2 and stage 4.5, and between total thiol values and PSI stages, a significant difference was found between stage 1 and stage 4.5 and between stage 2 and stage 4 (p<0.05). No statistical difference was found between disulphide values and PSI stages in the pneumonia group (p>0.05) (**Table 6, Figure 3**).

When IMA levels were evaluated in pneumonia, the IMA level was found to be significantly higher in the pneumonia group compared to the control group (p<0.05). The mean IMA value in the pneumonia group was 0.71 ABSU, while the mean IMA in the control group was 0.65 ABSU (**Table 7**).

In the pneumonia group, IMA and troponin levels were significantly increased compared to the control group, and a weak but significant positive correlation was found between them (p<0.05) (**Figure 4**).

Table 6. Relationship between native thiol, total thiol, disulfide values and PSI phases

Rank average				
Groups	**Native thiol**	**Native thiol**	**Total thiol**	**Disulfide**
PSI1	18	69.72	67.89	51.8
PSI2	15	67.47	67.80	48.7
PSI3	14	43.21	45.79	50.9
PSI4	39	43.32	40.94	52.5
PSI5	14	34.89	40.96	47.07

PSI: Pneumonia Severity Index

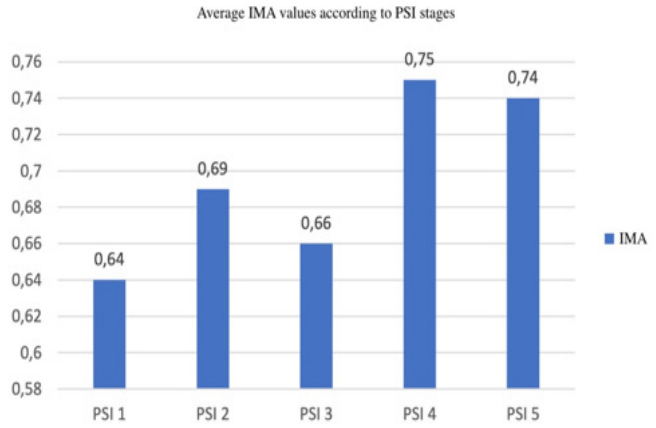

Figure 3. Average IMA values according to PSI stages
IMA: Ischemia-modified albumin, PSI: Pneumonia Severity Index

Table 7. IMA values for the pneumonia group and control group

IMA	n	Rank average	Total of rows	U	Z	p
Pneumonia	100	116.32	11632.00	3418	-3.866	.000
Control	100	84.68	8468.00			

IMA: Ischemia-modified albumin

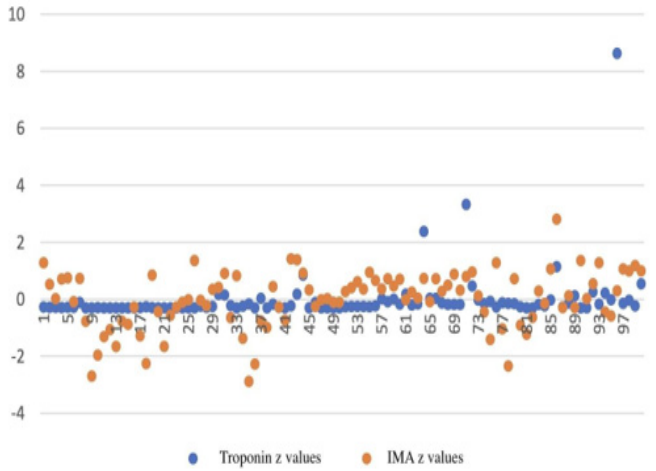

Figure 4. Distribution of IMA-troponin values in the pneumonia group
IMA: Ischemia-modified albumin

Table 5. Thiol disulphide balance values for the pneumonia group and control group

		Rank average					
Groups	**n**	**Native tiyol**	**Total tiyol**	**Disulfit**	**Disülfit/native tiyol**	**Disülfit/total tiyol**	**Native tiyol/total tiyol**
Pneumonia	100	278.25	336.27	23.76	120.01	118.03	89.97
Control	100	377.83	427.41	23.98	80.99	82.98	111.03

DISCUSSION

In this study, serum troponin and IMA levels were found to be significantly higher in patients with pneumonia compared to the control group. Native thiol, total thiol levels, and the native thiol/total thiol ratio were lower than in the control group, while the disulphide/total thiol and disulphide/native thiol ratios were statistically higher than in the control group.

Elevated cTn levels can be observed in approximately 50% of patients with heart failure, both during the acute decompensation phase and in the chronic compensation period. Increased cTn levels are a common finding in patients with sepsis. Although the reason for the increase in cTn in sepsis is not precisely known, factors such as cytotoxic endotoxins, inflammatory mediators (IL, TNF, heat shock protein, etc.), septic microemboli, vasoactive drugs, accompanying hypotension, and myocarditis are thought to be responsible. Elevated cTn levels are reported more frequently in patients with severe left ventricular systolic dysfunction accompanying sepsis.[5] In patients hospitalized with sepsis in the intensive care unit, cardiac troponin T levels are independently associated with length of hospital stay and short-term mortality.[14] Although elevated troponin levels are used as a marker in acute coronary syndromes, they are also elevated in some patients presenting with causes other than acute coronary syndrome.[15] This situation can lead to misdiagnosis and unnecessary interventions. Troponin release from the myocardium indicates temporary or permanent myocardial damage. This damage may be due to many causes, such as ischaemia, inflammation, infection, toxins, or increased ventricular wall tension. Elevated troponin levels may be associated with pulmonary embolism, central nervous system diseases (intracranial haemorrhage, increased intracranial pressure, ischaemic stroke), aortic dissection, pneumothorax, acute cholecystitis, acute or chronic renal failure, pancreatitis, etc.[16,17]

Early deaths due to myocardial injury occur in sepsis patients, and this condition can be used independently of rehabilitation morbidity after discharge.[18,19] To our knowledge, there are no studies on cTn elevation in pneumonia. We thought that cTn elevation in pneumonia could be due to cytokines released due to inflammation, accompanied by hypotension, hypoxia, and cardiac pathologies other than acute coronary syndrome. Specifically, in our study, cTn levels were found to be higher in patients with left ventricular systolic dysfunction during cardiac evaluation in pneumonia compared to patients with normal cardiac function. Therefore, it is considered necessary to perform a cardiac evaluation if elevated cTn is detected in a patient with pneumonia.

In our study, patients with renal dysfunction and those suspected of having acute coronary syndrome were excluded from the pneumonia group; however, as with patients with the same renal dysfunction, a cut-off value for cTn can be determined in the pneumonia patient group, and it can help us distinguish whether the elevated cTn is due to pneumonia or ischaemia. Further studies are needed in this area.

There are many biological markers that can be used in the diagnosis and follow-up of pneumonia. CRP and PCT are some of these. These markers indicate the severity of pneumonia, and CRP is frequently used. PCT, which is used less frequently, can also be a guide in distinguishing between bacterial and viral infections.[20] In our study, when examining the relationship between cTn levels in pneumonia and CRP and PCT, which are useful in diagnosis and follow-up, a positive correlation was found between cTn and CRP values in the pneumonia group and between PCT values. It has been suggested that cTn levels could be a biomarker used in the diagnosis and follow-up of infectious processes such as pneumonia, similar to CRP and PCT.

Prediction rules (CURB-65, PSI) have been developed for the classification of patients with pneumonia based on mortality risk prediction. As the CURB-65 and PSI scoring systems are only moderately sensitive and specific in determining risk in patients with pneumonia, additional risk factors and prognostic markers are needed to improve the prognostic performance of risk scores.[21] In our study, there was a significant difference in troponin values according to PSI stage, and cTn may be a marker for deciding on follow-up between the outpatient (PSI 1-2) patient group and the ward and intensive care unit (PSI 3-4-5) groups.

Studies have been conducted showing impaired thiol disulphide balance in cardiovascular diseases and pulmonary thromboembolism.[7,22] Studies have also been conducted showing changes in the dynamic thiol-disulphide balance in community-acquired pneumonia.[8,23] In all of these studies, native thiol and total thiol levels in the thiol-disulphide balance were reduced compared to the control group. Another study conducted on paediatric community-acquired pneumonia cases also found that disulphide levels were low in pneumonia cases. The disulphide/native thiol, disulphide/total thiol, and native thiol/total thiol ratios were significantly higher in the pneumonia group. In our study, native thiol and total thiol levels were also significantly lower than in the control group, while the disulphide/native thiol ratio and disulphide/total thiol ratio were higher. It has been stated that the thiol-disulphide balance shifts towards disulphide bond formation, and that oxidative stress therefore increases in community-acquired pneumonia.[24]

In our study, when IMA levels were evaluated, they were higher in the pneumonia group compared to the control group. Similarly, in a prospective case-control study conducted by Bolatkale and colleagues,[25] serum IMA levels were shown to be significantly increased in patients with pneumonia compared to healthy control subjects. To our knowledge, this study is the first to investigate serum IMA levels in adult patients presenting to the emergency department with TGP and has demonstrated that IMA may be a sensitive and specific new biomarker for the diagnosis of pneumonia in emergency department patients.

When the correlation between cTn values, an acute coronary syndrome marker, and IMA was evaluated in the pneumonia group, a weak but significant positive correlation was found. The evaluation of IMA and cTn in acute coronary syndrome, as in pneumonia, may guide us in the differential diagnosis of pneumonia and ischaemia.

Limitations

Our findings should be interpreted with certain limitations in mind. Firstly, this is a single-centre, small-scale study. It is the first study to evaluate cardiac troponin levels in adults with pneumonia. As cardiac troponin levels can increase for many reasons, it is difficult to establish specificity for pneumonia alone, and increases due to other causes must be considered. No threshold value has been established between myocardial ischaemia and other causes of elevation.

CONCLUSION

Consequently, cTn levels may be elevated for different reasons. The results of this study suggest that, when other causes are excluded, elevated levels in pneumonia indicate the need to evaluate cases for cardiac pathology and may also provide an important contribution as an indicator of pneumonia severity. It is thought that, particularly in cases of pneumonia with pre-existing cardiac pathologies, evaluating serum IMA levels alongside other factors could be useful in making decisions regarding hospital admission. Further studies could evaluate its potential use as a biomarker for the early detection of complications such as myocarditis that may develop during pneumonia. Furthermore, additional studies with larger sample sizes could determine a threshold value for cTn in pneumonia cases. It is therefore concluded that it could be more useful in practical application. However, a cost-effectiveness study is required.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study has been approved by the Clinical Researches Ethics Committee of Yıldırım Beyazıt University Faculty of Medicine (Date: 09.10.2019, Decision No: 102).

Informed Consent

Written informed consent was obtained from all individual participants prior to their inclusion in the study. Participants were fully informed about the study's aims, procedures, potential risks and benefits, and their rights—including the right to withdraw at any time without consequence. All participants voluntarily signed a written informed consent form.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: S.Ö., A.K.; Design: S.Ö., A.K.; Control: S.Ö., A.K.; Data Collection and/or Processing: S.Ö., A.K.; Analysis and/or Interpretation: S.Ö., A.K.; Literature Review: S.Ö., A.K.; Article Writing: S.Ö., A.K.; Critical Review: S.Ö., A.K.

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