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# Contents

## Original Article

*The role of uric acid and hscrp in predicting metabolic syndrome*
Hayriye Cankar Dal, Yasar Acar  
1-5

*Multislice computed tomography angiography imaging findings of pathologies that may mimic pulmonary embolism*
Seda Akyol, Nilgun Isiksalan Ozbulbul  
6-13

## Case Report

*As a different cause of kidney failure, liver failure, and fever: Leptospirosis*
Mehmet Gokhan Gok, Ahmet Melih Arslan  
14-5

*Membranous glomerulonephritis with positive serum PLA2R antibodies: A case report of replacing membranous nephropathy, massive proteinuria despite immunosuppressive therapies*
Ceren Cicek, Gozde Nur Sari  
16-9
INTRODUCTION
Metabolic syndrome, which is becoming increasingly common worldwide, is defined as the combination of all metabolic risk factors such as abdominal obesity that starts with insulin resistance, glucose intolerance or diabetes mellitus, dyslipidemia, hypertension, and systemic disorders such as coronary artery disease, and it has high morbidity and mortality rates with cardiometabolic complications (1). Despite significant advances in treatment approaches, atherosclerotic cardiovascular diseases remain the most common cause of death worldwide.

The role of uric acid and hscrp in predicting metabolic syndrome

Aim: Metabolic syndrome is a group of diseases characterized by the coexistence of cardiometabolic risk factors such as abdominal obesity, dyslipidemia, glucose intolerance, and high blood pressure. Metabolic syndrome is a common disease that can lead to high mortality with cardiovascular complications. Many studies are ongoing for laboratory parameters that can be used in the early diagnosis of this syndrome, but for daily clinical use and the diagnostic criteria, any parameter has not been revealed yet. We planned to carry out this study to predict the usability of uric acid and HSCRP in clinical practice, which have been reported to be associated with metabolic syndrome in the literature.

Material and Methods: Patients with metabolic syndrome who applied to Ankara Training and Research Hospital’s Internal Medicine department outpatient clinic for any reason within nine months were included in the study group. In the same period, completely healthy cases who applied to the outpatient clinic for any reason were included in the control group. Demographic data, physical examination findings, anthropometric measurement results, biochemical parameters, and HSCRP levels of the cases were recorded. Metabolic syndrome and the control group were analyzed for uric acid and HSCRP values. SPSS 25.0 was used for statistical analysis. The data were evaluated within the 95% confidence interval, and p<0.05 was considered significant.

Results: In the study conducted that with a total of 174 patients, including 95 patients with metabolic syndrome and 79 healthy patients, the mean uric acid level of the metabolic syndrome group (5.22±1.39mg/dl) was significantly higher than that of the control group (4.35±1.11mg/dl) (p<0.01). With the ROC curve, the uric acid cut-off value for detecting metabolic syndrome incidence was found to be 5.45mg/dl (specificity 89.9%; sensitivity 51.6%). Its positive predictive value was 86%, and its negative predictive value was 60.7%. The mean HSCRP levels of the metabolic syndrome group (4.72±3.54mg/L) were found to be significantly higher than the control group (3.30±3.21mg/L) (p<0.01).

Conclusion: Serum uric acid and HSCRP levels in the study group with metabolic syndrome were significantly higher than in the control group. This syndrome, which is very common today, can lead to various outcomes in terms of mortality and morbidity; when the early diagnosis is delayed, it constitutes an important part of health expenditures. We believe that early diagnosis with laboratory parameters that can be very practical in daily practice will prevent metabolic syndrome complications.

Keywords: Metabolic syndrome, obesity, systemic inflammation, uric acid, HSCRP
This pioneering status of cardiovascular diseases parallels the increasing epidemic of obesity and type 2 diabetes mellitus, the most common endocrine disease worldwide. The most important etiological factor that brings this trio together is the metabolic syndrome, which now affects so many regions in the world that it can be called a pandemic. This prevalent worldwide syndrome that cannot be diagnosed with a single parameter can lead to critical consequences with high morbidity and mortality when the diagnosis is delayed.

Metabolic syndrome can be mentioned as a result of specific anthropometric measurements, laboratory results, and clinical evaluation, starting with the syndrome being thought of first. In addition to the metabolic syndrome's first defined diagnostic criteria, which was published in 1998, various diagnostic criteria are defined by many organizations worldwide, and new parameters that are thought to be helpful in diagnosis are being studied (2). Most of the parameters investigated in patients with metabolic syndrome remained at the level of scientific studies. They could not take their place among the diagnostic criteria, and they were not used in daily clinical use due to the fact that scientific competence could not be achieved, and they were not financially cost-effective.

Uric acid and high-sensitivity C-reactive protein (HSCRP) are two parameters that have been reported in the literature to be associated with metabolic syndrome and its components such as hypertension, hyperglycemia, obesity, and dyslipidemia.

Chronic inflammation is the main factor in the development and progression of atherosclerosis and cardiovascular diseases. Many inflammatory markers have been studied in this regard; the most critical acute phase response marker associated with cardiovascular disease risk in different populations is CRP. Studies in the literature show that a high CRP level is one of the critical risk factors for developing chronic diseases with increasing prevalence, such as diabetes or hypertension (3,4). Upon the demonstration of the effectiveness of CRP in predicting subclinical inflammation, the HSCRP test, which can measure CRP levels at lower concentrations, and which is determined by high-sensitivity methods, was developed (5).

Obesity, the main component of metabolic syndrome, is associated with systemic inflammation originating from adipose tissue. That cytokines such as tumor necrosis factor-alpha and IL-6, which are released in large amounts from adipose tissue, trigger chronic subclinical inflammation by stimulating CRP production from the liver, which is thought to be the underlying mechanism of high HSCRP levels in obese individuals (6).

Elevated uric acid is thought to be an essential risk factor for developing cardiovascular events related to oxidative stress and endothelial dysfunction (7). It has been observed that insulin resistance underlying the metabolic syndrome is inversely related to renal uric acid clearance, and as a result, there is a decrease in renal excretion of uric acid in patients with hyperinsulinemia (8,9).

Although uric acid and HSCRP measurements are strongly associated with metabolic syndrome and its components, they are not included in the diagnostic criteria for metabolic syndrome defined by different organizations and in the recently published review of these definitions. In this study, we aimed to evaluate the usability of measurement of serum uric acid levels and HSCRP values, which can also be studied in our hospital laboratory, in clinical practice, in the prediction of metabolic syndrome, which can lead to serious outcomes in terms of mortality and morbidity when the diagnosis is delayed.

**MATERIAL AND METHODS**

The study included 95 patients with metabolic syndrome who applied to the Internal Medicine outpatient clinic of SBU Ankara Training and Research Hospital for any reason within nine months after the ethics committee's approval. Ethics committee approval was obtained from Ankara Training and Research Hospital, Education, Planning and Coordination Ethical Board (2012/02).

The diagnosis of metabolic syndrome was made according to the criteria of NCEP ATP III (The National Cholesterol Education Program's Adult Treatment Panel III report), which is the most accepted definition in practice due to its ease of application (10). Patients with known renal insufficiency, hepatic insufficiency, pregnancy, active malignancy, active infection, psoriasis, aspirin, diuretic, cyclosporine, pyrazinamide, ethambutol-containing drug users, alcohol, and cigarette users were excluded from the study. The control group includes 79 completely healthy volunteers who applied during the same period as the study group. Demographic data of the subjects included in the study, physical examination findings, anthropometric measurement results, fasting blood glucose (FBG), urea, creatinine, total cholesterol, HDL-C, LDL-C, TG, uric acid, insulin, biochemical including hemoglobin A1c (HbA1c) parameters and HSCRP levels were recorded.

SPSS 25.0 was used for statistical analysis. Independent sample t-test was used to compare parametric data, one-way analysis of variance (ANOVA) was used to compare more than two independent samples, chi-square test was used to compare categorical values, and correlation test was used to compare values with each other. Data were evaluated at a 95% confidence interval. p<0.05 was considered significant.

**RESULT**

The study was carried out with a total of 174 cases, including 95 patients with metabolic syndrome and 79 healthy patients. Of the patients in the metabolic syndrome group, 58.9% (56) were female, 41.1% (39) were male; 57% (45) of the control group were female, and 43% (34) were male, and the mean age of the study population was 51.25±11.60 years (Table 1). The mean body mass index of all subjects included in the study was 27.79±6.05, and the mean waist circumference was 96.24±16.05. Mean waist circumference and body mass indexes of patients...
with metabolic syndrome were statistically significantly higher than the control group (p<0.01). Fasting glucose, triglyceride, LDL, HbA1c, fasting insulin level, and insulin resistance were significantly higher in the metabolic syndrome group compared to the control group (p<0.01). There was no significant difference between serum creatinine levels of both groups (p:0.960). HDL levels of patients with metabolic syndrome were found to be lower than the control group (p<0.01) (Table 2).
The area under the curve (AUC) is 0.688

In a large-series study with 7399 cases, the relationship between hyperuricemia in patients with metabolic syndrome was investigated, and it was reported that the ROC curve for serum uric acid value in the prediction of metabolic syndrome was found to be 6.3 mg/dl in men and 4.9 mg/dl in women (13). In most laboratories, 7 mg/dl is taken as the upper limit of uric acid. In our study results, that similar to Zhang et al. study, the uric acid cut-off value was determined as 5.45 mg/dl in the prediction of metabolic syndrome, which shows that in association with serum uric acid levels and metabolic syndrome, in addition to overt hyperuricemia, uric acid levels at the upper limit of normal may also be significant.

HSCRP is an acute phase reactant, which has been supported by studies in which high levels are associated with an increased risk of cardiovascular disease and that it is found at higher levels in patients with metabolic syndrome compared to healthy adults (14). In a large series study comparing 3285 patients with metabolic syndrome with 3999 healthy individuals, it was reported that the increase in HSCRP level is an independent risk factor in showing the risk of cardiovascular diseases such as coronary artery disease and ischemic stroke. As the number of metabolic syndrome components increases, HSCRP levels increase in correlation with the number of components (15).

Zuliani et al., in the metabolic syndrome study performed by 1044 cases, it was reported that the rate of HSCRP elevation was found to be significantly higher in the metabolic syndrome group (54.5%) compared to healthy subjects (41.3%) (p < 0.001). It has also been reported that there is a positive correlation between the increase in waist circumference and HSCRP levels and that HSCRP values increase as the waist circumference increases, independent of confounding factors such as age, gender, and comorbidity (16).

In our study, according to the correlation analysis between waist circumference measurements and body mass index and HSCRP values, it was determined that there was a positive correlation between body mass index and waist circumference measurements and HSCRP levels.

CONCLUSION

In conclusion; in our study to evaluate the relationship of uric acid and HSCRP levels with the metabolic syndrome and its components, and its usability in predicting metabolic syndrome in daily practice, serum uric acid and HSCRP levels were found to be significantly higher in the metabolic syndrome patient group compared to the control group. In this study, we demonstrated that this syndrome, which is very common today, can lead to various outcomes in terms of mortality and morbidity; when the early diagnosis is delayed, it constitutes an important part of health expenditures. We believe that predicting it with laboratory parameters that can be easily used in daily practice will prevent complications that may develop.
Conflict of interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical approval
Ethics committee approval was obtained from Ankara Training and Research Hospital, Education, Planning and Coordination Ethical Board (2012/02).

REFERENCES
INTRODUCTION

Pulmonary thromboembolism (PTE) is a disease with high morbidity and mortality, which refers to the migration of clots or clots from the systemic deep veins to the pulmonary vascular bed (1,2). About 2/3 of the patients who have undergone, and survived PTE cannot be diagnosed. Diagnosis is delayed in a short time because common symptoms such as dyspnea, tachypnea, tachycardia, chest pain, cough, pleuritic flank pain, hemoptysis, and physical examination findings are not specific to this disease (3,4). The mortality rate in these patients reaches 30%. This clinical entity, which can be confused with many diseases, decreases below 10% when other diseases are eliminated quickly and accurately, and appropriate treatment is performed (1). Today, Multislice Computed Tomography
Angiography (MSCTA) has come to the forefront because of its advantage in providing information about pleura, parenchyma, and mediastinal pathologies, enabling us to quickly diagnose embolism (4,5).

In this study, we aimed to detect lesions that may mimic this clinical picture, except for embolism, in patients with a pre-diagnosis of pulmonary embolism and no embolism in MSCTA.

MATERIAL AND METHODS

Turkey Yuksek Iltisas Hospital Radiology Department in our CT unit; Between January 2007 and July 2008, from the emergency service and other clinics; 180 cases who were referred to our clinic with suspicion of PTE based on history, physical examination, chest X-ray, and laboratory findings and who underwent Pulmonary Angiography (PCTA) with Multislice Computed Tomography (MCT) were analyzed. Of the patients, 86 were male, and 94 were female, with a mean age of 55 (91-16).

Age, history of surgical operation, and additional signs of cardiac failure were recorded for all patients.

All MSCTPAs of the patients were evaluated in terms of the presence or absence of intraluminal filling defects in the pulmonary arteries presence or absence of pulmonary effusion (2,15,16).

Parenchymal abnormalities examined in parenchymal window settings; emphysema (increased aeration), atelectasis (loss of volume), consolidation (non-wedge-shaped bright area that hides bronchovascular signs), ground-glass appearance (parenchymal shadow that does not obscure bronchovascular signs), interstitial fibrosis (structures in the form of thin bright lines or bands), ascending aortic aneurysm (diameter exceeding 40mm in measurements made in the widest part of the ascending aorta), aortic aneurysm in the arcus (cases in diameter exceeding 30mm in measurements made in the widest part of the aortic arch) cases exceeding (4,17,18).

The presence of any parenchymal abnormality mentioned above and the number of cases in total was recorded in cases with and without PE.

RESULT

A total of 180 cases, 86 male (47.7%) and 94 female (52.2%), aged between 16 and 91 years (mean age: 55 years) who were referred to our clinic with the preliminary diagnosis of pulmonary embolism were evaluated. In the cases, the diagnosis of pulmonary thromboembolism (PTE) was made with the joint decision of the two evaluators, who saw a filling defect in the pulmonary artery in MCPBTA. In 11 (6.1%) of these cases, vascular enhancement, which could lead to a misconception in the diagnosis of PTE, was not optimal, and in 4 (2.2%) cases, it could not be evaluated optimally due to intense motion artifacts.

PTE was detected in 37 (20.5%) of 180 cases included in the study, and PTE was not found in 143 (79.4%) cases. At least one parenchymal abnormality was found in 37 of 37 patients with pulmonary embolism and in 125 of 143 patients without
pulmonary embolism.

The most common parenchymal finding was interstitial fibrosis in cases with or without PTE. In cases without PTE, emphysema, atelectasis, ground-glass appearance, pleural effusion, and consolidation were followed, respectively. Pericardial effusion, thoracic aortic aneurysm, and mass were seen much less frequently (4,19).

The most common parenchymal findings in patients without pulmonary embolism are summarized in Figure 1

Figure 1. The frequency of parenchymal findings in pulmonary emboli negative (-) case

Eighteen (12.5%) of 143 patients presented with pulmonary embolism, and no PTE had normal tomography. Interstitial fibrosis in 94 (65.7%) cases, atelectasis in 53 cases (37.06%), emphysema in 53 cases (37.06%), ground-glass opacity in 38 cases (26.5%), pleural effusion in 34 cases (23.7%), consolidation in 22 cases (15.3%), Pericardial effusion in 14 cases (9.7%), ascending aortic aneurysm in 12 cases (8.3%), aneurysm in the aortic arch in 11 cases (7.6%), mass in the lung in 10 cases (7.6%), and aneurysm in the descending aorta in 9 cases (6.9%).

The frequency of parenchymal findings in patients with and without pulmonary embolism is summarized in Table 1.

<table>
<thead>
<tr>
<th>PTE (-)</th>
<th>PTE (+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>18</td>
</tr>
<tr>
<td>Emphysema</td>
<td>53</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>53</td>
</tr>
<tr>
<td>Consolidation</td>
<td>22</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>34</td>
</tr>
<tr>
<td>Frosted glass</td>
<td>38</td>
</tr>
<tr>
<td>Interstitial fibrosis</td>
<td>94</td>
</tr>
<tr>
<td>AC operation</td>
<td>0</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>14</td>
</tr>
<tr>
<td>Ascending aortic aneurysm</td>
<td>12</td>
</tr>
<tr>
<td>Arch aortic aneurysm</td>
<td>11</td>
</tr>
<tr>
<td>Dessenden aortic aneurysm</td>
<td>9</td>
</tr>
<tr>
<td>AC mass</td>
<td>10</td>
</tr>
</tbody>
</table>

There was no significant relationship between the probability of having PTE and the frequency of parenchymal findings. However, in cases without PTE, the most common parenchymal finding was interstitial fibrosis, which may also manifest interstitial lung disease. Interstitial fibrosis was detected in 94 patients who did not have PE. Of these patients, 23 (24.4%) had lung infection, 17 (18%) cardiac findings, 6 (6.3%) masses, 1 (1.06%) Pneumothorax (20,21). Interstitial fibrosis was common in those with pulmonary infection and underlying cardiac disease.

The relationship of interstitial fibrosis in cases without pulmonary embolism and underlying diseases are summarized in Table 2.

Table 2. The relationship between interstitial fibrosis and underlying diseases in patients without Pulmonary Embolism

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interstitial fibrosis</td>
<td>94</td>
<td>65.7%</td>
</tr>
<tr>
<td>Lung infection</td>
<td>23</td>
<td>24.4</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>Mass</td>
<td>6</td>
<td>6.3</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>1</td>
<td>1.06</td>
</tr>
</tbody>
</table>

Atelectasis was found in 53 cases in which no pulmonary embolism was detected. In 15 (28.3%) of these patients, external compression with cardiac pathologies, lung infection in 6 (11.3%), mass in 5 (9.4%), pneumothorax in 1 (1.8%), and mucus plug in 1 (1.8%) were detected (22). Atelectasis can be observed for obstructive or non-obstructive reasons. Atelectasis is frequently observed in patients with cardiac pathologies and masses.

The relationship between atelectasis and underlying diseases in patients without pulmonary embolism is summarized in Table 3.

Table 3. Relationship between atelectasis and underlying diseases in cases without Pulmonary Embolism

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atelectasis</td>
<td>15</td>
<td>28.3</td>
</tr>
<tr>
<td>Lung infection</td>
<td>6</td>
<td>11.3</td>
</tr>
<tr>
<td>Mass</td>
<td>5</td>
<td>9.4</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>1</td>
<td>1.8</td>
</tr>
<tr>
<td>Mucus plug</td>
<td>1</td>
<td>1.8</td>
</tr>
</tbody>
</table>

A frosted glass appearance was observed in 38 patients who presented with a preliminary diagnosis of pulmonary embolism, and no embolism was detected. Pulmonary edema was found in 17 (44.7%) of these patients, lung infection in 5 (13.1%), and bronchial cancer in 2 (5.2%) patients. No pathology was found in the CT of 14 patients (36.8%). It is a non-specific finding and can be observed in many unrelated diseases. In our study, the frosted glass appearance was most frequently observed with pulmonary
edema and lung infection (22).

The relationship between frosted glass appearance and underlying diseases in cases without PE is summarized in Table 4.

Table 4. The relationship between the frosted glass appearance and the underlying diseases in cases without pulmonary embolism

<table>
<thead>
<tr>
<th>PTE (-)</th>
<th>Frosted glass appearance</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>AC edema</td>
<td>17</td>
<td></td>
<td>44.7</td>
</tr>
<tr>
<td>AC infection</td>
<td>5</td>
<td></td>
<td>13.1</td>
</tr>
<tr>
<td>Mass</td>
<td>2</td>
<td></td>
<td>5.2</td>
</tr>
</tbody>
</table>

Pleural effusion was observed in 34 cases in whom pulmonary embolism was not detected. Congestive heart failure in 13 (38.2%) of these patients, pneumonia in 10 (29.4%), cardiac operation in 6 (17.6%), mass in 3 (8.8%), viral diseases in 2 (5.8%) detected. Pleural effusion was most frequently detected in patients with congestive heart failure and lung infection (21).

The relationship between pleural effusion and underlying diseases in patients without pulmonary embolism is summarized in Table 5.

Table 5. The relationship of pleural effusion with underlying diseases in patients without pulmonary embolism

<table>
<thead>
<tr>
<th>PTE (-)</th>
<th>Pleural Effusion</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>13</td>
<td>38.2</td>
<td></td>
</tr>
<tr>
<td>Ac infection</td>
<td>10</td>
<td></td>
<td>29.4</td>
</tr>
<tr>
<td>Operation</td>
<td>6</td>
<td></td>
<td>17.6</td>
</tr>
<tr>
<td>Mass</td>
<td>3</td>
<td></td>
<td>8.8</td>
</tr>
</tbody>
</table>

Consolidation was observed in 22 patients in whom pulmonary embolism was not detected. Mass in 2 (9%) patients, pulmonary edema in 6 (27%), atelectasis in 2 (9%) and pneumonia in 12 (54.5%) patients (13). Consolidation was most frequently seen with a lung infection.

The relationship between consolidation and underlying diseases in patients without pulmonary embolism is summarized in Table 6.

Table 6. The relationship of consolidation with underlying diseases in cases without pulmonary embolism

<table>
<thead>
<tr>
<th>PTE (-)</th>
<th>Consolidation</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass</td>
<td>2</td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>AC edema</td>
<td>6</td>
<td></td>
<td>27</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>2</td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>12</td>
<td></td>
<td>54.5</td>
</tr>
</tbody>
</table>

Pericardial effusion, one of the most frequently confused clinical entities with pulmonary embolism clinic, was detected in 14 cases without embolism. Underlying cardiac diseases were found in 5 (35.7%) and mass lesions in 2 (14.2%) (13,23). Although there was no parenchymal finding besides pericardial effusion in the remaining seven patients (50%), it imitated embolism alone.

The relationship between pericardial effusion and underlying diseases in patients without pulmonary embolism is summarized in Table 7.

Table 7. The relationship of pericardial effusion with underlying diseases in cases without pulmonary embolism

<table>
<thead>
<tr>
<th>PTE (-)</th>
<th>Pericardial effusion</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease</td>
<td>5</td>
<td></td>
<td>35.7</td>
</tr>
<tr>
<td>Mass</td>
<td>2</td>
<td></td>
<td>14.2</td>
</tr>
</tbody>
</table>

CASE EXAMPLES

Case 1: 43-year-old male patient on axial CT sections; (a) A mass lesion of soft tissue density with a spiculated contour in the right lung middle lobe lateral segment in the parenchymal window. (b) A mass lesion of soft tissue density with spiculated contours in the lateral segment of the right lung middle lobe in the mediastinal window and subcarinal, hilar multiple conglomerated lymph nodes. (c) Several paratracheal lymphadenopathy in the mediastinal.
Case 2: Axial CT scans of a 32-year-old female patient. (a) Consolidation area in the left lung upper lobe apicoposterior segment in the parenchymal window and a slight ground glass area around it. (b) Consolidation area in the left lung upper lobe apicoposterior segment in the mediastinal window and several lymph nodes in the prevascular area. (c) Ground-glass area of the lower zone of the left lung in the parenchymal window.

Case 3: Axial CT scans of a 68-year-old male patient. (a) Linear opacities and bilateral pleural effusion in both lung lower lobe posterobasal segments in the parenchymal window. (b) Bilateral pleural effusion in the mediastinal window and adjacent linear compression atelectasis. (c) Bilateral pleural effusion in the mediastinal window and linear compression atelectasis adjacent to the right and left ventricles.

Case 4: A 55-year-old male patient on axial CT scans. (a) Pleural effusion in the left hemithorax in the mediastinal window, compression atelectasis adjacent to the effusion. (b) Ground-glass appearance in the lower zones of both lungs in the parenchymal window and pleural effusion in the left lung. (c) Pleural effusion and paratracheal millimetric lymph nodes in the left hemithorax in the mediastinal window.
DISCUSSION

MSCTPA accelerated the diagnosis process in patients with suspected pulmonary embolism by reducing it to a single breath-hold period (7,8,24). Today, MCBTA has become the first preferred diagnostic method because it is non-invasive as well as provides information about concomitant parenchymal findings and intrathoracic formations, as well as being fast (6,24,25). However, in routine practice, the focus is more on the presence or absence of PE in MCBTA. There are many studies dealing with the prevalence of parenchymal and pleural findings in cases with PE, but as far as we know, there are not many studies dealing with concomitant parenchymal findings in cases with no embolism.

Pulmonary embolism is a disease with high mortality if not diagnosed early. Its clinical findings are diverse and can mimic many diseases (1,3). In this study, we tried to determine the parenchymal findings of the patients who applied to our clinic with a preliminary diagnosis of pulmonary embolism and underwent PCTA in cases without embolism.

In our study, atelectasis was found to be the second most common parenchymal abnormality with 37.6%. Pulmonary atelectasis is a common alternative diagnosis in patients undergoing CTPA with a pre-diagnosis of PTE (20).

In our study, atelectasis was found in 53 patients with negative emboli, 15 (28.3%) external compression, 6 (11.3%) lung infection, 5 (9.4%) mass, 1 (1.8%) pneumothorax, 1 (1.8%) mucus plug has been detected. In the remaining 25 patients (47.1%), no obvious pathology was detected on CT.

In our study, ground-glass opacity was determined as the second most common parenchymal finding, and this rate was recorded as 26.5%. In cases without pulmonary embolism, the reason for the ground-glass opacity may be due to partial filling of the alveoli with fluid or cells or early atelectasis. In our study, 38 embolism-negative patients with frosted glass appearance, 17

<table>
<thead>
<tr>
<th>Study</th>
<th>Collimation</th>
<th>Available study</th>
<th>Any parenchymal abnormality</th>
<th>Atelectasis</th>
<th>Ground glass opacity</th>
<th>Consolidation</th>
<th>Linear opacity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2,5 mm</td>
<td>143 (%79.4)</td>
<td>125 (%87.4)</td>
<td>53 (%37)</td>
<td>38 (%26.5)</td>
<td>22 (%15.3)</td>
<td>94 (%65.7)</td>
</tr>
<tr>
<td>Coche et al.</td>
<td>3 mm</td>
<td>62 (%70.4)</td>
<td>N</td>
<td>17 (%27)</td>
<td>N</td>
<td>15 (%24)</td>
<td>13 (%21)</td>
</tr>
<tr>
<td>Shah et al.</td>
<td>3mm,5mm</td>
<td>64 (%69.5)</td>
<td>56 (%88)</td>
<td>41 (%64)</td>
<td>16 (%25)</td>
<td>14 (%22)</td>
<td>25 (%29)</td>
</tr>
<tr>
<td>Ozer et al.</td>
<td>3mm</td>
<td>71 (%70.3)</td>
<td>48 (%67.6)</td>
<td>16 (%22.5)</td>
<td>16 (%21.1)</td>
<td>19 (%26.7)</td>
<td>N</td>
</tr>
<tr>
<td>Total</td>
<td>340</td>
<td>229/278 (82.3)</td>
<td>127 (37.3)</td>
<td>69/278 (24.8)</td>
<td>70 (20.5)</td>
<td>132/269 (49)</td>
<td></td>
</tr>
</tbody>
</table>

PE: Pulmonary Embolism, N: Not reported
(44.7%) pulmonary edema, 5 (13.1%) lung infection, 2 (5.2%) bronchial cancer was detected. No pathology was found in the CT of 14 patients (36.8%).

In our study, consolidation was also among the most common parenchymal abnormalities, with a rate of 15.3%. In our study group, out of 22 patients without pulmonary embolism, mass was found in 2 (9%), pulmonary edema in 6 (27%), atelectasis in 2 (9%), pneumonia in 12 (54.5%).

Pleural effusion may occur due to various lung diseases and systemic diseases. In our study, pleural effusion was detected in 34 (23.7%) of the cases in which no embolism was detected. The most frequent causes of pleural effusion are congestive heart failure, pneumonia, and cancer. In our study, out of 34 patients with pleural effusion, 13 (38.2%) had congestive heart failure, 10 (29.4%) pneumonia, 6 (17.6%) cardiac operation, 3 (8.8%) mass, 2 (5.8%) viral diseases were detected.

Parenchymal abnormalities such as a mass are extremely rare. In our study, a mass was found in 10 (7.6%) of 143 patients without embolism. Among other studies evaluating the pleural and parenchymal findings on MSCTs of patients without pulmonary embolism, only Shah et al. (13) investigated the presence of a mass, and this rate was found to be 3% with two patients. The higher percentage of mass in our results may be related to the different CT devices and CT acquisition techniques (such as fine collimation).

**CONCLUSION**

In conclusion, we believe that most of the patients who were referred to our clinic with a pre-diagnosis of PE and underwent MSCT have pleuroparenchymal abnormalities (17). Pulmonary embolism clinical can be confused with many diseases, and many patients receive unnecessary treatment despite the absence of PE (26). Today, in patients with suspected PE and who can hold their breath, MSCTA is an increasingly important method in the diagnosis of pulmonary embolism since it directly shows emboli and provides additional information about the mediastinum and lung parenchymal, as well as vascular structures (19,26). Interstitial Lung Disease and Chronic Obstructive Pulmonary (COPD) Diseases are the group of diseases that should be considered primarily in the differential diagnosis in patients who do not have a pulmonary embolism.

**Conflict of interests**

The authors declare that there is no conflict of interest in the study.

**Financial Disclosure**

The authors declare that they have received no financial support for the study.

**Ethical approval**

Ethics committee approval was received from Ankara Turkey Yüksek İhtisas Hospital / 2009.

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CASE REPORT
At the age of 60, a male patient who has no previous systemic disease other than hypertension, coronary artery disease, and diabetes mellitus applied to the emergency department about one week ago with complaints of fever, nausea, vomiting, and dizziness. In the laboratory tests, requested after routine examination, leukocytosis, hyperbilirubinemia, kidney function tests, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) values detected elevated. Initial evaluation of the patient; general condition was moderate to good; tachycardia, tachypnea, fever, icterus, splenomegaly, and petechiae were examined; there were no unusual symptoms in other systemic examinations. The patient's hemogram had leukocytosis and thrombocytopenia. Also, the peripheral blood smear was compatible with the hemogram. In the biochemical examination, hyperbilirubinemia with direct dominance and creatinine values were elevated. There was metabolic acidosis with wide anion gap in blood gas. Then, empirical ceftriaxone treatment was started. The patient was on dialysis twice due to uremic complications. After the fourth day of her clinical follow-up, the patient did not have a fever, Bun-Creatine values decreased, thrombocytopenia resolved, and ESR and CRP values decreased to normal ranges. No microorganisms were grown in the culture. The patient, who was followed up in our clinic with a prediagnosis of Weil's disease, was discharged after 14 days of ceftriaxone treatment.

DISCUSSION
Leptospirosis is a common bacterial zoonosis in the world. It is seen in farmers, sewer workers, hunters, and swimmers in streams and lakes. The route of infection to humans occurs in direct contact with the urine of the sick animals' contact with the environment contaminated with urine. The clinical spectrum of leptospirosis is quite broad. It is subclinical in 90% of cases. Multiple organ failure, especially kidney, liver, and lung, can be seen in 5-10% of cases [1-2]. Weil's disease is the most severe form of leptospirosis. It progresses with liver dysfunction, acute renal failure, thrombocytopenia, and fever; If left untreated, it can be fatal at 1-5% [3]. In our case, a 60-year-old patient with fever, hyperbilirubinemia, acute renal failure, and thrombocytopenia will be discussed.

Keywords: Liver dysfunction, acute renal failure, thrombocytopenia, fever
varies between 0.1-1/100,000 in regions with low rainfall and 10-100/100,000 in tropical regions. The incidence is well above 100/100,000 during the epidemic after natural disasters and in risky groups [4]. In leptospirosis, leukocytosis and left shift of the oxygen dissociation curve is frequently observed as laboratory findings; thrombocytopenia is also commonly seen [5]. Some studies reported that thrombocytopenia was found in 75% of surviving patients and 83.3% of cases resulting in death [6]. Thrombocytopenia is transient and independent from disseminated intravascular coagulation. During infection, plasma 11-dehydro-thromboxane B2 (11-DH-TXB2) level increases. 11-DH-TXB2; induces activation-aggregation of platelets and phagocytosis by Kupfer cells. That is an essential mechanism in the pathogenesis of thrombocytopenia [7]. Again, In some studies, the relationship between thrombocytopenia and Weil's disease has been tried to be explained by disseminated intravascular coagulation [8]. In our case, platelet values were below 50,000. Platelet values increased from the fourth day of antibiotic treatment and remained within normal ranges. The definitive diagnosis of leptospirosis is made by producing and demonstrating the agent in culture. However, a bacterial culture is not practical in routine laboratories since the growth period of the bacteria is quite long (2 weeks-6 months) and requires special media [1]. It can be seen with Dark-field microscopy in clinical samples. However, the sensitivity of Dark-field microscopy is low. In order to evaluate spirochetes in Dark-field microscopy, 10^4 bacteria per milliliter should be present in the sample. Other methods used for diagnosis are the Microscopic agglutination test (MAT), the Complement Fixation Test (CFT), Enzyme-Linked Immunosorbent Assay (ELISA), Radioimmunoassay (RIA), and Polymerase Chain Reaction (PCR) [5]. Generally, oral Doxycycline (200 mg/day, one week), Ampicillin (4x500-750 mg), or Amoxicillin (3x500 mg) is recommended in mild-moderate cases. In severe cases, parenteral Penicillin (4x1.5 million units) or Ceftriaxone (1x1 gr/day) is recommended [1]. We also gave our patient 2*1 gr Ceftriaxone parenteral antibiotic treatment.

CONCLUSION

In conclusion, Weil's disease should be considered in the differential diagnosis of patients with severe thrombocytopenia, hepatic dysfunction, and renal failure who applied with the complaints of fever, muscle pain, and icterus both in our region and in our country.

Conflict of interests

The authors declare that they have no competing interests.

Financial Disclosure

All authors declare no financial support.

Informed Consent

Written consent was obtained from the patient and his parents.

REFERENCES

INTRODUCTION

Membranous nephropathy (MN) is among the most common causes of the nephrotic syndrome in non-diabetic adults, accounting for up to one-third of biopsied cases of nephrotic syndrome.

The term MN reflects the primary histologic change noted on light microscopy: glomerular basement membrane (GBM) thickening with little or no cellular proliferation or infiltration. MN is most often primary (previously called idiopathic), although it has been associated with hepatitis B antigen, autoimmune diseases, thyroiditis, malignancies (1,2).

We present a case of a male patient, showing features of membranous nephropathy, such as global glomerulosclerosis, fibrosis, mononuclear infiltration, thick basement membrane, and deposition of immunoglobulin IgG on immunofluorescence positive, PLA2R antibodies, weight loss without malignancy, processes are directed against endogenous antigens such as circulating cationic or low-molecular-weight antigens that have crossed the anionic charge barrier in the GBM [3,4].

Genetic: The major antigen was identified as the PLA2R (phospholipase A2 receptor) [5].

Malignancy: Up to %5-%20 of adults, particularly those over the age of 65 years, with MN, have been reported to have a malignancy, most commonly a solid tumor (principally carcinoma of the prostate, lung, breast, bladder, or gastrointestinal tract) [2,6,7]. MN diagnosis preceding that of malignancy is more likely in older adults with weight loss.

We present a case of the male patient, showing features of membranous nephropathy, such as global glomerulosclerosis, fibrosis, mononuclear infiltration, thick basement membrane, and deposition of immunoglobulin IgG on immunofluorescence positive, PLA2R antibodies, weight loss without malignancy,
and massive proteinuria, approximately 20 gr/day.

**CASE REPORT**

The patient who applied to the outpatient clinic on 28.02.2022 with complaints of nocturia and swelling in the legs since 2017 and was diagnosed with membranous glomerulonephritis after renal biopsy. He was admitted to the nephrology service due to swelling in the legs and 19180 mg/day proteinuria in his examinations during his routine controls, and a recent involuntary weight loss.

**Known diseases:**


**Drugs used:**

- Olmesartan + Amlodipine 40/5/12.5 gr 1x1
- Cyclosporine 25 mg 1x1
- Carvedilol 12.5 mg 1x1
- Vitamin D 10000 IU uses every other day
- Duloxetine 30 mg 1x1

**Physical Examination:**

- Head and Neck: No lymphadenopathy, normal
- Cardiovascular System: S1+ S2+ no additional sound/murmur
- Respiratory System: No ral/rhonchi, respiratory rate normal
- Abdomen: Normal inspection, percutaneous, liver palpation normal. No guarding/rebound, no painful focus.
- Lower Extremity: Peripheral pulses on, pretibial edema -/

The hospital information of the patient dated 02.03.2017 was reached. The patient had complaints of itching, redness, and edema in the legs, starting from the distal feet and arms and spreading to the chest and head; it has been around for 30 days. The patient applied to the dermatology department several times due to these complaints. After the physical examination in the internal medicine outpatient clinic revealed stage 3 hypertension and pretibial edema, albumin: 2 gr/dl, complete urinalysis: protein 3 positive (+). LDL: 340 mg/dl, the patient was referred to nephrology outpatient clinic, considering nephrotic syndrome. The results were urea: 43 mg/dl complete urinalysis: protein 3 positive (+), spot urine protein: 700 mg/dl. The patient was hospitalized in terms of proteinuria etiology.

Abdomen ultrasonography report; both kidneys were normal in size and localization, contours were regular, bilateral parenchyma thickness was normal, and grade I increased in echoes. The bilateral pelvicalyceal system was in normal shape and width, and there was no cystic or solid mass lesion or stone echo in both kidneys.

Protein electrophoresis was within normal limits, immunofixation monoclonal gammopathy not detected, kappa normal, lambda normal, antinuclear antibodies (ANA) negative, antibodies to double-stranded (AntiDs DNA) negative, serum complement 3 (C3) and complement 4 (C4) were within normal limits. Protein in 24-hour urine from the patient: 1007 mg/day. In 2017 biopsy was planned for the etiology of proteinuria, but when the patient did not accept the biopsy, ramipril 2.5 mg was added in terms of proteinuria. Later, he agreed on the procedure of renal biopsy. Biopsy resulted as membranous nephropathy.

On 26.04.2017 proteinuria in 24-hour urine result; 6885 mg/day; cyclosporine 2*100 mg, methylprednisolone 64 mg was started.

On 28.02.2022 proteinuria in 24-hour urine result; 19180 mg/day; Rituximab was started. cyclosporine 2*50 mg, methylprednisolone 40 mg treatment was continued.

By the time of his routine outpatient controls, see the results of the proteinuria under the immunosuppressive treatment (Table 1).

<table>
<thead>
<tr>
<th>Date</th>
<th>Creatine mg/dl</th>
<th>eGFR ml/minute</th>
<th>Proteinuria mg/day</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>26.04.2017</td>
<td>1.1</td>
<td>71</td>
<td>6885</td>
<td>Cyclosporine 2x100 mg, methylprednisolone morning: 48 mg night: 16 mg</td>
</tr>
<tr>
<td>25.11.2018</td>
<td>1.22</td>
<td>46</td>
<td>3680</td>
<td>Cyclosporine 2x125 mg, Methylprednisolone morning: 32 mg night: 16 mg</td>
</tr>
<tr>
<td>09.04.2019</td>
<td>1.13</td>
<td>40</td>
<td>1404</td>
<td>Cyclosporine 2x50 mg, methylprednisolone morning: 8 mg night: 4 mg</td>
</tr>
<tr>
<td>07.11.2019</td>
<td>1.16</td>
<td>68</td>
<td>875</td>
<td>Methylprednisolone morning: 8 mg night: 4 mg</td>
</tr>
<tr>
<td>02.01.2020</td>
<td>1.12</td>
<td>71</td>
<td>1885</td>
<td>Cyclosporine morning: 75 mg night: 50 mg, methylprednisolone 1x4 mg</td>
</tr>
<tr>
<td>20.02.2020</td>
<td>1.05</td>
<td>77</td>
<td>762</td>
<td>Cyclosporine morning: 50 mg night: 25 mg, methylprednisolone 2x4 mg</td>
</tr>
<tr>
<td>10.08.2021</td>
<td>1.34</td>
<td>58</td>
<td>262</td>
<td>Cyclosporine morning: 50 mg night: 25 mg, methylprednisolone 2x4 mg</td>
</tr>
<tr>
<td>28.02.2022</td>
<td>1.76</td>
<td>40</td>
<td>19180</td>
<td>cyclosporine 2x50 mg, methylprednisolone morning: 24 mg night: 16 mg, rituximab</td>
</tr>
</tbody>
</table>
Abdomen ultrasonography result (30.12.2021); no solid mass lesions were detected (including liver, gall bladder, pancreas, spleen, and kidneys).

Laboratory results; total prostate-specific antigen (PSA), cancer antigen-15.5, cancer antigen-125, alpha-fetoprotein (AFP), carcinoembryonic antigen, cancer antigen-19.9 were in normal ranges.

Table 2. PLA2R Antibodies (AB) serum levels of patient

<table>
<thead>
<tr>
<th>Date</th>
<th>Transaction name</th>
<th>Result</th>
<th>Result Unit</th>
<th>Reference Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>03.03.2022</td>
<td>PLA2R AB</td>
<td>774.95 (positive)</td>
<td>RU/ml</td>
<td>negative &lt;14  positive &gt;=20</td>
</tr>
</tbody>
</table>

Colonoscopy result (08/03/2022); colon and rectum are normal; Peripheral grade-1 hemorrhoids were observed in the anal canal.

No thoracoabdominal mass or pathology detected in the thoracic and abdominal scanning of the patient on 08/03/2022.

DISCUSSION

Membranous nephropathy occurs when the small blood vessels in the kidney (glomeruli), which filter wastes from the blood, become damaged and thickened.

As a result, proteins leak from the damaged blood vessels into the urine (proteinuria). For many, loss of these proteins eventually causes signs and symptoms known as nephrotic syndrome. Typically, loss of protein from the blood causes swelling in the legs and ankles and weight gain due to the extravasation of fluid. Many patients have much swelling from the onset of the disease, but some may not have any severe symptoms until they have advanced kidney disease.

In mild cases, membranous nephropathy may get better on its own, without any treatment. As protein leakage increases, the risk of long-term kidney damage increases, and the disease ultimately leads to kidney failure.

There is no absolute cure for membranous nephropathy, but successful treatment can lead to remission of proteinuria and a good long-term outlook [8-10].

Up to %5-%20 of adults, particularly those over the age of 65 years, with MN, have been reported to have a malignancy, most commonly a solid tumor (principally carcinoma of the prostate, lung, breast, bladder, or gastrointestinal tract) [2,6,7]. MN diagnosis preceding that of malignancy is more likely in older adults with weight loss.

CONCLUSION

In our case, the trigger factor of the disease is unknown, and the patient's altered response to therapy and the weight loss without accompanying malignancy is also unknown. The answers to these questions remain unknown as well. It is certainly possible that there were several different stimuli to the patient's immune system that caused the disease and different responses to therapy.

Future research may determine the nature of the stimuli that induce the response (development of antibodies to the known antigens and perhaps to more ). Until then, patients with MN will continue to be treated with powerful, frequently effective but
non-specific treatments.

Univariate analysis of the comparisons has shown that the low ejection fraction in the pre-operative period (P-value 0.010) and cardiogenic shock requiring the IABP installation (P-value 0.031) are the risk factors that determine surgical mortality. Having analyzed both these risk factors, it was found out that only cardiogenic shock was an independent risk factor for operative mortality with an odds ratio of 2.17. As to the low ejection fraction, it turned out a concomitant factor for operative mortality. Additional revascularization of coronary arteries had no impact on the survival rate.

Conflict of interests
The authors declare that they have no competing interests.

Financial Disclosure
All authors declare no financial support.

Informed Consent
Written consent was obtained from the patient and his parents.

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