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# Efficacy of lipid, NT-proBNP and D-dimer biomarkers in the differential diagnosis of patients presenting to the emergency department with syncope

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## ABSTRACT

**Aims**: Syncope is defined as a temporary loss of consciousness in any disorder characterized by a self-limited loss of consciousness, whatever the mechanism. It accounts for 1% of emergency room admissions. NT-proBNP, D-dimer and lipids are important parameters in the diagnosis and differential diagnosis of patients with syncope. This study aims to determine the levels of these biomarkers during and after treatment in patients admitted to the emergency department(ED) due to syncope.

**Methods**: Forty-nine patients admitted to the emergency department due to syncope were included in this study. Forty-nine age- and sex-matched individuals without a history of syncope were taken as the control group. Blood samples were taken from the patient group three times, at the time of admission to the emergency department, 24 hours after admission, and on the day they were discharged from the hospital, and only once from the control group. The patient and control groups were compared in terms of NT-proBNP, D-dimer and lipids.

**Results**: The mean HDL level at discharge was 33.63±9.62 mg/dl, which was significantly lower than the mean HDL level in the control group (38.77±10.33 mg/dl) (t=2.14, p=0.012). Although the mean LDL levels at discharge (108.02±48.03 mg/dl) were higher than the control group (92.53±37.39 mg/dl), this increase was not statistically significant (t=1.78, p=0.078). However, the mean LDL levels during hospitalization and after 24 hours (126.08±51.88 mg/dl, 116.26±48.21 mg/dl, respectively) compared to the control group (92.53±7.39 mg/dl) were statistically significantly higher (t=3.67, p=0.001, t=2.73, p=0.008). NT proBNP and D-dimer median values at the time of admission to the emergency department (844.00 pg/ml, 616.50 mcg/L, respectively), after 24 hours (1985.00 pg/ml, 662.00 mcg/L, respectively) and at discharge (748.00 pg/ml, 702.50 mcg/L respectively) compared to the control group (85.00 pg/ml, 176.00 mcg/L, respectively), a statistically significant increase was detected (p=0.001).

**Conclusion**: In the patients admitted to the ED with a diagnosis of syncope, early treatment can be achieved getting the differential diagnosis of syncope in a short time with NT-pro BNP, D-dimer and lipid blood levels that can be worked easily. In addition, the need for serious interventional procedures and further investigations in the diagnostic process will be reduced.

**Keywords**: Syncope, NT-pro BNP, D-dimer, lipid profile

## INTRODUCTION

Syncope is defined as transient loss of consciousness, characterized by rapid onset, short duration, and spontaneous complete recovery. The cause of unconsciousness is transient global cerebral hypoperfusion. Any temporary loss of consciousness for no apparent reason should be considered syncope until proven otherwise. 12-48 % of young healthy adults experience syncope once in their lifetime. Syncope accounts for about 1% of admissions to the ED. About 40% of patients with syncope are admitted to the hospital.[1-3] According to recent studies, syncope is very common in the general population and has costs, both in terms of health expenditure and socioeconomically. Recurrent syncope has a serious impact on quality of life. The quality of daily life is impaired by 33% in patients with frequent recurrent syncope. In large-scale studies, the average hospital stay was 5.5.[4,5] days, and the length of hospital stay accounted for > 75% of total costs.[6-8]

Syncope is a common symptom that can result from a variety of etiological causes ranging from benign to life-threatening and is also one of the most common causes of ED admissions. Syncopes occur as an outcome of different etiologic causes, including vascular, cardiac, neurological, psychogenic, metabolic, and unexplained causes. The pathophysiological

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process of syncope, which is a common symptom of many etiological causes, is the same. Considering all these causes of syncope, the most important point is to determine the conditions that cause the pathophysiological process. Biochemical markers that change or emerge depending on the results of the pathophysiologic process will enable the detection of the etiologic causes of syncope and the initiation of treatment for the cause.

The aim of this study is to emphasize the efficacy of lipid, NT-proBNP and D-Dimer biomarkers in the diagnostic process of patients with syncope, simultaneous treatment process and prognostic prediction.

## METHODS

The study was carried out with the permission of Sivas Cumhuriyet University Ethics Committee (Date: 17.06.2011, Decision No: 171). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The patient group consisted of 49 randomly selected patients who were admitted to the ED due to fainting/syncope, had brain tomography and were hospitalized and followed up. As the control group, 49 age- and sex-matched healthy volunteers were included in the study. Blood samples of 49 individuals in the patient group were taken at the time of admission to the ED, at the 24th hour and at discharge. Only one blood sample was taken from the control group.

The blood samples of both groups were taken into 2 ml red-capped gel tubes for lipid profiles, 1.5 ml green-capped heparin tubes for NT-proBNP, and 1.5 ml capped tubes containing Na3-Citrate for d-dimer. Lipids, HDL; With the "direct HDL, Immunoseparation" method, LDL; by the method of "direct LDL, Immunoseparation", triglyceride; NT-proBNP with "lipase /GK UV. No correction" method; Radiometer AQT90 Flex device was studied with immunoassay technology and time-dependent fluorometric detection method. D-dimer was studied with the immuno-turbidimetric method in the STA compact device. The blood values of the patient group at the time of admission, at the 24th hour and at discharge were compared among themselves and with the control group.

Patients with a prediagnosis of cardiac or neurological syncope who underwent brain tomography, blood glucose measurement, electrocardiography, vital signs were recorded and hospitalized were included in the study.

Patients with a diagnosis of vasovagal syncope, conversion-related syncope, patients who did not have brain tomography, blood glucose, measurements were not made, electrocardiography was not taken, and vital signs were not recorded from the study.

### Statistical Analysis

The data of our study was loaded into the SPSS (ver: 14.0) program and the significance test between the two means was applied when parametric assumptions were fulfilled in the evaluation of the data. When parametric assumptions could not be fulfilled, Mann Whitney U test and Chi-Square test were used. According to these test results, the median, minimum and maximum values of some comparisons are given. Our data were stated in the tables as arithmetic mean±standard deviation, number of individuals and percentage, and the error level was taken as 0.05.

## RESULTS

Patients admitted to the emergency department with a prediagnosis of syncope within a 6-month period were included in our study. The mean age of individuals in the patient group was 59.16±11.46 years and the mean age of the control group was 55.02±12.12 years. The difference between the groups in terms of age was not statistically significant (t=1.73; p= 0.085; p > 0.05). Both patient and control groups consisted of 26 (53.1%) women and 23 (46.9%) men. There was no statistical difference between the groups in terms of gender.

HDL levels at discharge were statistically significantly lower than in the control group, whereas LDL levels at admission and 24 hours later were statistically significantly higher than in the control group. No statistically significant difference was found between the groups in terms of TG values (**Table 1**).

**Table 1. Comparison of measured parameters between patient and control group**

| Measurements | Patient Mean±SD (mg/dl) | Control Mean±SD (mg/dl) | P value |
|---|---|---|---|
| HDL[1] | 39.38±10.81 | 38.77±10.33 | t=0.28, p=0.750 |
| HDL[2] | 38.44±11.06 | 38.77±10.33 | t=0.15, p=0.880 |
| HDL[3] | 33.63±9.62 | 38.77±10.33 | t=2.14, p=0.012* |
| LDL[1] | 126.08±51.88 | 92.53±37.39 | t=3.67, p=0.001* |
| LDL[2] | 116.26±48.21 | 92.53±37.39 | t=2.73, p=0.008* |
| LDL[3] | 108.02±48.03 | 92.53±37.39 | t=1.78, p=0.078 |
| TG[1] | 128.87±83.54 | 104.44±114.72 | t=1.20, p=0.231 |
| TG[2] | 114.44±54.44 | 104.44±114.72 | t=0.55, p=0.583 |
| TG[3] | 123.53±63.87 | 104.44±114.72 | t=1.01, p=0.312 |

1: Admission, 2: 24th hour, 3: Discharge, * : p<0.05 significant

NT-proBNP and D-dimer values at the time of presentation to the ED, at 24 hours and at discharge were statistically significantly higher compared to the control group (**Table 2**).

**Table 2. Comparison of patient and control group D-dimer and NT-proBNP values**

| Measurements | Patient, n=49 | Control, n=49 | P value |
|---|---|---|---|
| | Median (pg/ml) | Median (pg/ml) | |
| NT-proBNP[1] | 844.00 | | |
| NT-proBNP[2] | 1985.00 | 85.00 | p=0.001* |
| NT-proBNP[3] | 748.00 | | |
| | Median (mcg/L) | Median (mcg/L) | |
| D-dimer[1] | 616.50 | | |
| D-dimer[2] | 662.00 | 176.00 | p=0.001* |
| D-dimer[3] | 702.50 | | |

1: Admission, 2: 24th hour, 3: Discharge, * : p<0.05 level of significance

Brain tomography was unremarkable in 26 (53%) patients presenting with syncope. Cerebrovascular disease (CVD) was detected in 23 (37%) patients. In patients with CVD, brain tomography revealed infarction in 16 (32.7%) patients and hemorrhage in 7 (14.3%) patients. All patients presenting to the ED with syncope had comorbidities. CAD was the most common comorbidity in 23 (46.9%) patients and HT was the second most common comorbidity in 13 (26.5%) patients. DM was detected as comorbidity in 4 (8.2%) patients. Hypoglycemia was detected in only one (2%) syncope patient. Hyperglycemia was detected in 18 (36.7%) patients. ECG showed normal sinus rhythm in 28 (57.1%) patients, while pathology was recorded in 21 patients. Atrial fibrillation was the most common ECG pathology in 12 (24.52%) patients.

The blood pressure values of the individuals in the patient group were above normal. Systolic blood pressure mean±standard deviation (SD)=157.10±30.06 and diastolic blood pressure

mean±SD=92.85±18.02. There was a mean increase of "5.79±31.07" in systolic blood pressure and "3.77±16.02" in diastolic blood pressure compared to normal blood pressure values. Blood pressure was statistically significantly higher in patients with syncope compared to the control group (p<0.05).

When comparing the patients' HDL and LDL levels at the time of admission and on the day of discharge, it was found that both levels were lower at discharge. This decrease in HDL and LDL levels was statistically significant (p < 0.05). Although lower values were observed at discharge compared to the time of admission at the TG level, this was not statistically significant. For D-dimer and NT-proBNP levels, although the values at discharge were higher than the values at admission, this increase was not statistically significant (**Table 3**).

**Table 3. Comparison of the parameters measured in the patient group at the time of admission and discharge**

| Patient n=49 | | Mean±SD (mg/dl) | P Value |
|---|---|---|---|
| 1 | HDL1<br>HDL3 | 39.38±10.81<br>33.63±9.62 | t= 3.90<br>p=0.001 * |
| 2 | LDL1<br>LDL3 | 126.08±51.88<br>108.02±48.03 | t= 3.37<br>p=0.002 * |
| 3 | TG1<br>TG3 | 128.87±83.54<br>123.53±63.87 | t= 0.54<br>p=0.584 |
| | | **Median (mcg/L)** | |
| 4 | D-Dimer1<br>D-Dimer3 | 616.50<br>702.50 | p=0.932 |
| | | **Median (pg/ml)** | |
| 5 | NT-proBNP1<br>NT-proBNP3 | 844.00<br>748.00 | p=0.831 |

1: Admission, 3: Discharge, * : p<0.05 level of significance

In all comorbid conditions, the difference between NT-proBNP levels at admission, after 24 hours and at discharge was found to be statistically significant (p<0.05). While the difference between D-dimer levels at admission and after 24 hours was statistically significant, the difference between D-dimer values at discharge was not statistically significant (**Table 4**).

## DISCUSSION

Syncope is a temporary loss of consciousness due to transient global cerebral hypoperfusion, characterised by sudden onset, short-term and spontaneous complete recovery.[9] Syncope is a very common symptom, occurring in 3-37% of the general population and 6% of the elderly.[10]

Our body, which perceives life-threatening situations and serious illnesses as stress factors, gives various hormonal and metabolic responses for adaptation. During stress, cortisol secretion increases. An increase in cortisol increases glucose levels, which is the most important fuel for vital organs. Other counter-regulatory hormones such as epinephrine, norepinephrine, and glucagon also play an important role in adaptation. In addition, epinephrine and norepinephrine cause early hyperglycemia mainly through glycogenolysis in the liver and skeletal muscle.[11] The hyperglycemia detected in 18 patients in our study supports the conclusion that hyperglycemia occurs due to the activation of some hormonal and metabolic mechanisms during stress in the light of literature.

In the presence of a history and evidence of central nervous system dysfunction, imaging may be required based on clinical neurologic evaluation. Brain tomography (CT) is indicated for the detection of the etiologic cause of syncope if seizures or focal neurologic findings are present. Apart from this, they reported that nonselective use of CT would not be helpful in diagnosis.[12] In our study, it is seen that brain tomography has a high diagnostic value of 47% (in 23 patients). We think that the reason for this high rate is the inclusion of patients who underwent brain tomography in our study.

Cardiovascular diseases are one of the conditions that cause syncope. Due to increased structural heart disease in older age, syncope is more likely to be of cardiac origin. If syncope occurs in the background of underlying heart disease, the sudden death rate is approximately 25%.[4]

HT is the most important risk factor for haemorrhagic strokes, thromboembolic stroke and lacunar infarctions, as well as for TIA, which has a high prevalence in the population.[13] Syncope may develop as a result of decreased cerebral blood flow due to cardiac and cerebral vasoconstriction in hypertension. In our study, the blood pressure values of the individuals in the patient group were: systolic mean±SD=157.10±30.06 mmHg and diastolic mean±SD=92.85±18.02 mmHg, and the increase in blood pressure in the patient group was found to be significant. Systolic blood pressure was found to increase more than diastolic blood pressure. Cardiac or cerebral vasoconstriction developing after hypertension is thought to contribute to the pathophysiology of syncope in the patients included in the study.

**Table 4. Comparison of comorbidities of individuals in patient groups and NT-proBNP, D-dimer values at admission, 24th hour and discharge**

| | | | CAD, n=23 | DM, n=4 | HT, n=13 | Other, n=9 | P value |
|---|---|---|---|---|---|---|---|
| NT-proBNP[1] | pg/ml | Median<br>Minimum<br>Maximum | 1480.00<br>12.00<br>35000.00 | 8252.00<br>84.00<br>35000.00 | 509.00<br>26.00<br>35000.00 | 662.00<br>31.00<br>9810.00 | KW=14.41<br>p=0.001 |
| NT-proBNP[2] | | Median<br>Minimum<br>Maximum | 2945.00<br>117.00<br>35000.00 | 17544.00<br>42.00<br>35000.00 | 627.00<br>12.00<br>22800.00 | 897.00<br>13.00<br>9160.00 | KW=25.45<br>p=0.001 |
| NT-proBNP[3] | | Median<br>Minimum<br>Maximum | 2180.00<br>35.00<br>35000.00 | 17558.00<br>15.00<br>35000.00 | 423.00<br>22.00<br>35000.00 | 903.00<br>16.00<br>4945.00 | KW=26.90<br>p= 0.001 |
| D-dimer[1] | mcg/L | Median<br>Minimum<br>Maximum | 647.00 –<br>168.00 –<br>1768.00 | 1705.00<br>120.00<br>3701.00 | 537.00<br>109.00<br>15982.00 | 622.00<br>129.00<br>2904.00 | KW=6.95<br>p=0.031 |
| D-dimer[2] | | Median<br>Minimum<br>Maximum | 662.00 –<br>177.00 –<br>9110.00 | 1694.50<br>157.00<br>64880.00 | 742.00<br>105.00<br>15735.00 | 813.00<br>146.00<br>4145.00 | KW=7.36<br>p=0.025 |
| D-dimer[3] | | Median<br>Minimum<br>Maximum | 732.00<br>176.00<br>7673.00 | 749.00<br>116.00<br>73380.00 | 673.00<br>243.00<br>6455.00 | 538.50<br>137.00<br>5860.00 | KW=4.46<br>p=0.107 |

NT- pro BNP[1]: Admission, NT- pro BNP[2]: 24th hour, NT- pro BNP[3]: Discharge
D-dimer[1]: Admission, D-dimer[2]: 24th hour, D-dimer[3]: Discharge

Coronary artery disease and MI are the leading risk factors for stroke. One of the most feared acute myocardial infarction (MI) complications is ischemic stroke. Stroke after AMI is an important cause of morbidity and mortality. The incidence of stroke in the first month following acute MI has been reported to be 0.9–3.7%.[14] About 87% of strokes are ischemic infarcts; atherosclerosis is one of the most important factors in ischemic strokes. Cardiac or cerebral thrombi constitute the majority of syncope cases, and the share of coronary artery disease and atherosclerosis is quite high in these cases.[15] The history of CAD and higher lipid values in 23 (46.9%) of the patients included in our study compared to the control group are consistent with the literature.

The electrocardiogram (ECG), although normal in most patients with syncope, is an essential diagnostic tool with the potential to identify patients with a high probability of cardiac syncope due to arrhythmic or cardiopulmonary disorder. Although a number of diagnostic procedures are routinely performed in patients with suspected syncope, a 12-lead electrocardiogram should be obtained for the initial evaluation of these patients. ECG may reveal an arrhythmia with a high probability of syncope in 7% of patients referred to the ED with the diagnosis of syncope and allows specific treatment without further evaluation.[16]

The risk of stroke and systemic embolism in patients with atrial fibrillation (AF) is associated with many underlying pathophysiological mechanisms. The most common cause (>90%) in these patients is left atrial appendage embolism due to non-valvular AF (NVAF). The incidence of NVAF increases with age.[17] In our study, atrial fibrillation was the most common ECG finding in 12 patients (24.5%) in patients with syncope. Cardiac or cerebral thromboembolism is frequently seen in AF as in the literature. It is supported by the literature that the most common ECG finding in our study was AF.

Elevated cholesterol levels increase the risk of both coronary heart disease and thromboembolic stroke.[18] Syncope also occurs in a significant proportion of patients who have had a stroke. Decreased high-density lipoprotein (HDL) levels increase the risk of coronary heart disease. Elevated total cholesterol and low-density lipoprotein (LDL) levels are also associated with the development of atherosclerosis. The development of coronary atherosclerosis at the abnormal coronary artery is more common and manifests clinically as myocardial infarction (MI), syncope, arrhythmia, angina pectoris or sudden death at a young age.[4]

Demir et al.[19] found a significant correlation between the change in serum lipid profile after acute myocardial infarction (AMI) and baseline values. They reported that the decrease in total cholesterol and LDL levels at day 10 and month 1 was significant compared to baseline, while there was a significant increase in triglyceride (TG) levels at day 10 after AMI and a significant decrease at month 1 compared to baseline and day 10 values, and the changes in HDL levels were not significant.

Rosenson et al.[20] reported that there was a change in serum levels of plasma proteins as an acute phase response after AMI, and the decrease in lipid and lipoprotein levels started in 24-48 hours and reached a maximum in 4-7 days. Again, in the same study, they claimed that lipid and lipoprotein levels returned to their true values within 2 months after infarction.

In our study, the mean HDL level at discharge was 33.63±9.62 mg/dl, which was significantly lower than the mean HDL level in the control group (38.77±10.33 mg/dl) (t=2.14, p=0.012). Although the mean LDL levels at discharge (108.02±48.03 mg/dl) were higher than the control group (92.53±37.39 mg/dl), this increase was not statistically significant (t=1.78, p=0.078). However, the mean LDL levels during hospitalization and after 24 hours (126.08±51.88 mg/dl, 116.26±48.21 mg/dl, respectively) compared to the control group (92.53±7.39 mg/dl) were statistically significantly higher (t=3.67, p=0.001, t=2.73, p=0.008). It was observed that HDL and LDL values started to decrease after syncope in diseases that are among the etiological causes of syncope, but TG levels remained stable. HDL level was observed to be normal at the time of admission and the 24$^{th}$ hour, but a significant decrease was detected on the average 10$^{th}$ day of hospitalization. On the other hand, it was observed that the high LDL values decreased to normal values on the 10$^{th}$ day of the hospitalization period. In our study, we could not observe the decrease in TG levels and the process of returning to normal, as the average discharge time was 10 days. In this regard, it is recommended to conduct studies with a longer follow-up period.

N terminal B type natriuretic peptide (NT-proBNP) is a powerful and independent diagnostic method in patients hospitalized for syncope. NT-proBNP levels were found to be higher in cardiac syncopes compared to non-cardiac syncopes. However, NT-proBNP, which is a cardiac marker in syncope patients, is not used sufficiently.[21] Talwar et al.[22] found that NT-proBNP values were higher in survivors 6 weeks after acute myocardial infarction. In our study, NT proBNP median values were found at the time of admission to the emergency department (844.00 pg/ml), after 24 hours (1985.00 pg/ml), and at discharge (748.00 pg/ml) compared to the control group (85.00 pg/ml). ml) was found to be statistically significantly higher (p=0.001). In addition, NT-proBNP values were higher in patients with a history of CAD, consistent with the literature. It was found that the NT-proBNP value at the time of admission was higher than the values at discharge, but it was not statistically significant (p>0.05). The reason why NT-proBNP values are high in the acute period but still higher than normal values even during the discharge period can be explained by the presence of the current clinical situation. The fact that the NT-proBNP values did not increase further can be attributed to the stable clinical course of the patients and the fact that the destruction process has begun because the half-life of NT-proBNP is approximately 120 minutes.[23]

D-dimer is one of the important parameters determining the etiology of syncope. sensitivity and specificity of the D-dimer test; Many factors play a key role, such as the extent of thrombosis and fibrinolytic activity, duration of symptoms, anticoagulant therapy, comorbid conditions, inflammatory diseases, cancer, old age, pregnancy and the postpartum period, and previous venous thrombosis embolism. Many previous studies have shown that the D-dimer test is highly sensitive (>95%) in acute deep venous thrombosis or pulmonary embolism.[24,25] One of the etiological causes of syncope is pulmonary thromboembolism. Syncope is a rare presenting finding for pulmonary embolism and occurs in 9-13% of patients with acute PE.[26,27] The presence of syncope in these patients indicates a poor prognosis.[28,29] A significant percentage of the patients included in our study consisted of syncopes as a result of cardiac or cerebral thromboembolism. D-dimer values at the time of admission to the emergency department (616.50 mcg/L), after 24 hours (662.00 mcg/L) and at discharge (702.50 mcg/L) were found to be higher

than the control group (176.00 mcg/L). Although pulmonary thromboembolism was not diagnosed among the etiologic causes of syncope in our study, it is an expected result that D dimer values were found to be high due to other thrombotic events.

It has been reported that D-dimer levels rise acutely in transient ischemic attacks (TIA) and approach normal levels towards the end of the first month.[30] In our study, it was found that the D-dimer levels of patients with pathological brain tomography (infarction, bleeding) and syncope due to other etiological reasons were higher at discharge than D-dimer levels at the time of admission. However, this height was not statistically significant ($p>0.05$). Similar results were obtained in all other disease groups that we took as the etiological causes of syncope. The high D dimer values in patients with cerebrovascular infarction in our study are supported by the literature. The reason for the elevation in cerebrovascular haemorrhage patients may be due to disseminated intravascular coagulation or bleeding into cerebral infarction. Since the discharge time was 10 days on average, unfortunately, we could not detect that D-dimer levels approached normal since the mean discharge time of the patients included in the study was 10 days. In this respect, we recommend longer follow-up studies.

Danesh et al.[31] showed that even in individuals without prior known vascular disease, plasma D-dimer levels above physiological limits pose a 70% higher risk for coronary heart disease. In previous studies, when patients with chest pain presenting to the ED were grouped as AMI, unstable angina, and non-ischemic pain, D-dimer levels were found to be higher in the ischemic group.[32] In our study, In all comorbid conditions, the difference between NT-proBNP levels at admission, after 24 hours and at discharge was found to be statistically significant ($p<0.05$). While the difference between D-dimer levels at admission and after 24 hours was statistically significant, the difference between D-dimer values at discharge was not statistically significant. It is thought that the change in D-dimer levels cannot be observed completely due to the short follow-up period or the insufficient number of cases. In this respect, it is recommended to conduct studies with a larger number of cases and longer follow-up periods. All these results show that the acute thrombotic process plays an important role in many other comorbid diseases such as CAD, DM and HT.

In patients presenting to the emergency department with syncope, ECG, blood pressure measurement, blood glucose level and CT are indispensable tests in the first stage of the diagnosis and simultaneous treatment process. In addition to symptomatic treatment, additional biochemical markers such as lipid, NT-proBNP and D-Dimer levels should be used in order to quickly formulate a treatment plan for the cause.

## CONCLUSION

NT-proBNP, D-dimer and lipid blood values, which can be accessed quickly and easily in the emergency department, can be used to determine the etiologic causes of syncope. It is also possible to evaluate the efficacy of treatment by monitoring these parameters. More importantly, we believe that the management of patients presenting to the emergency department with a prediagnosis of syncope can be effectively performed with NT-proBNP, D-dimer and lipid levels without the need for more serious interventional procedures and investigations.

## ETHICAL DECLARATIONS

**Ethics Committee Approval:** The study was carried out with the permission of Sivas Cumhuriyet University Ethics Committee (Date: 17.06.2011, Decision No: 171).

**Informed Consent:** All patients signed the free and informed consent form.

**Referee Evaluation Process:** Externally peer-reviewed.

**Conflict of Interest Statement:** The authors have no conflicts of interest to declare.

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**Author Contributions:** All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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