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Acute Coronary Syndrome

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Summary

Cardiovascular events related to ischemic coronary diseases are among the leading causes of death in the world. The most common of these diseases are in the diagnosis group called acute coronary syndromes (ACS). According to the 2012 data of the World Health Organization (WHO), Ischemic Heart Disease, stroke with 7.4 million people, and 6.7 million people and 3.1 people with Chronic Obstructive Pulmonary Disease (COPD). Cardiac Troponins (cTn) are very sensitive and specific indicators of myocardial damage in cardiac markers.It is included in the group of Cardiac Troponins, Troponin T (cTnT) and Troponin I (cTnI). In ACS, increased cTn levels are important in terms of both prognosis and treatment. In international algorithms, they are accepted as standard markers in the diagnosis and treatment of ACS. In many studies, cTn elevation was found to have negative prognostic value in the short-term, with or without myocardial infarction (MI), in patients hospitalized in the hospital intensive care unit. However, the level of cTn in the blood can increase even due to ischemic coronary diseases and even for non-cardiac reasons. Acute Coronary Syndrome occurs as a result of impaired integrity of the atherosclerotic plaque in the coronary vessel. The clot formed on the plaque impairs various degrees of coronary blood flow. In addition to the clot, different degrees of coronary spasm may accompany the picture. As a result of these changes, acute elevation myocardial infarction (STEMI), ST elevation acute myocardial infarction (NSTEMI) or unstable angina pectoris (Unstable Angina Pectoris, UAP) may occur in the clinic.

Keywords: *Troponin; Heart failure; Unstable angina*

Introduction

Cardiovascular events related to ischemic coronary diseases are the leading causes of death in the world [1]. Those who complain about ischemic coronary disease often apply to emergency departments for initial diagnosis and treatments. The most common of these diseases are in the diagnosis group called acute coronary syndromes (ACS) [2]. An approach principle consisting of clinical history, ECG and cardiac markers is used in the diagnosis of ACS's emergency department. Although the history and ECG elements of the clinical approach have the same characteristics since the past, cardiac markers have been frequently changed in recent years, and they continue to take place in diagnostic approaches by updating and updating them.

Among the cardiac markers, cardiac Troponins (cTn) are highly sensitive and specific indicators of myocardial damage. It is included in the group of Cardiac Troponins, Troponin T (cTnT) and Troponin I (cTnI). In ACS, increased cTn levels are important in terms of both prognosis and treatment [3]. In international algorithms, they are accepted as standard markers in the diagnosis and treatment of ACS [4,5]. In many studies, the height of cTn was found to have negative prognostic value in the short-term, with or without myocardial infarction (MI), in patients hospitalized in the hospital intensive care unit. However, the level of cTn in the blood can increase even without ischemic coronary diseases [6]. The number of clinical studies conducted on the increase of the level of cTn in the blood when it is not related to ischemic coronary diseases is low.

Definition

Acute coronary syndrome (ACS) is a condition characterized by symptoms and clinical manifestations associated with acute myocardial ischemia. According to 2012 data of the World Health Organization (WHO), the top three causes of death in the world are Ischemic Heart Disease with 7.4 million people, Stroke with 6.7 million people and Chronic Obstructive Pulmonary Disease (COPD with 3.1 people)). AKS is the most common one among Ischemic Heart Diseases [7]. Acute Coronary Syndrome occurs as a result of impaired integrity of the atherosclerotic plaque in the coronary vessel [8]. The clot formed on the plaque disrupts coronary blood flow to various degrees. In addition to the clot, different degrees of coronary spasm may accompany the picture [9]. As a result of these changes, acute elevation myocardial infarction (STEMI), ST elevation acute myocardial infarction (NSTEMI) or unstable angina pectoris (Unstable Angina Pectoris, UAP) may occur in the

clinic [10]. Patients with ischemic complaints may or may not have ST segment elevation in Electrocardiography (ECG). STEMI may be in patients with ST segment elevation and UAP or NSTEMI in patients without ST segment elevation. The distinction between these two conditions can only be made with cardiac enzymes. If there is an increase in cardiac enzymes, NSTEMI is UAP if there is no increase [11].

Pathophysiology

Acute coronary syndromes share a common physiopathological basis that is very common with different clinical manifestations. This common mechanism is seen as the rupture of the atherosclerotic plaque. In fact, atherosclerotic vascular disease develops gradually since childhood with the contribution of risk factors and proceeds insidiously for many years without symptoms [12]. The occurrence of symptoms occurs when the atheroma plaque becomes too large and prevents blood flow in the lumen, which is called clinically stable angina pectoris (SAP). Or AKSs occur with plaque rupture and a different degree of thrombus formation on it. It follows a process with atherosclerosis continuity that includes stages of stability and instability rather than a straight course. Sudden and unpredictable changes in symptoms are associated with plaque rupture. Plaques that are more prone to rupture and complication have a larger lipid nucleus, more inflammatory cells, less smooth muscle cells, and a thinner fibrous capsule that covers the lipid nucleus than stable plaques [13]. Plaque fragility may also depend on the size and placement of the plaque, environmental wall stress, the effect of current on the luminal plaque surface. In addition to plaque rupture, plaque erosion is another underlying mechanism of acute coronary syndrome. When plaque erosion develops, thrombus adheres to the surface of the plaque, while plaque rupture proceeds to deeper layers, to the lipid core. If the process of remodeling does not occur in the thrombus, this process can lead to growth and rapid progression of the plaque. In these patients, clinical findings may progress from unstable angina to MI depending on the degree of occlusion of the thrombus. In NSTEMI and UAP, thrombus narrows the lumen but does not clog up. This thrombus is rich in platelets. In STEMI, the thrombus lumen is plugged and rich in fibrin [14,15].

Diagnosis

The result of the ECG and blood cardiac markers in patients with chest pain determines the type of ACS:

NSTEMI: Defined by ST segment depression or prominent T wave reversal and / or positive necrosis biomarkers such as troponin in the ECG without ST segment elevation and in the presence of a compatible clinical picture. The most common reason is that a thrombus that develops in a ruptured atherosclerotic plaque, generally not completely occlusive, increases coronary artery stenosis and reduces myocardial perfusion. UAP: Unstable coronary artery disease is a heterogeneous clinical syndrome. Unstable angina and non-ST -elevated myocardial infarction are distinguished precisely only when the results of cardiac markers are obtained. UAP is an acute coronary syndrome without myocardial necrosis. The key point in estimating how to treat the hospitalized patients and their prognosis in and after the hospital is the determination of the patient's risk status. The determination of the need for coronary revascularization and determination of the intensity of treatment in the early period is decided after the risk is determined [16]. According to the "Braunwald" classification proposed by Braunwald shown in **Table 1** in 1989 and still valid, unstable angina pectoris is subdivided according to the severity of the disease, etiopathogenesis and intensity of antiischemic therapy administered [17].

Table 1: Braunwald risk classification Clinical conditions.

severity	A. There is a non-cardiac cause that increases myocardial ischemia (secondary)	B. No non-cardiac causes that increase myocardial ischemia (primary)	c. Non-cardiac causes that increase myocardial ischemiaNo angina (postinfarction) within two weeks of myocardial infarction (primary)
1. Angina or accelerated angina to beginner, no resting pain	1A	1B	1C
2. Subacute resting angina (no pain in the last 48 hours)	2A	2B	2C
3. Resting angina in the last 48 hours	3A	3B-T negatif 3B-T pozitif	3C

Apart from this classification, TIMI risk scoring is also used clinically. TIMI risk score criteria have been established based on TIMI 11B and ESSENCE studies.

TIMI risk scoring criteria:

a. Age> 65
b. 3 cardiovascular risk factors
c. Coronary artery disease with narrowing of 50% or more in coronary arteries
d. ST segment change in ECG
e. The presence of more than two angina atagas in the past 24 hours
f. Using aspirin in the past week
g. Increased serum cardiac biomarkers

Apart from this classification, the presence of each risk factor in TIMI risk TIMI risk scoring is considered as one point. The high risk score is associated with an increased risk of death, new or recurrent myocardial infarction and ischemia requiring revascularization [16].

STEMI: It is the group with the highest mortality among acute coronary syndromes. Patients are characterized by chest pain lasting more than 30 minutes, dynamic changes in sequential electrocardiograms, and increase and decrease of cardiac biomarkers showing myocardial damage [18].

Clinic

The factors to be considered in the first evaluation of the patient are given below in order of importance.

a. The nature of anginal symptoms
b. Known coronary artery disease
c. Male sex
d. Old age

Increase in traditional risk factors [13].

Angina pectoris, the most common symptom of acute coronary syndrome, is similar to chronic stable angina pectoris, but is generally more severe and longer lasting. The classic clinical presentation is retrosternal pressure (angina) or a feeling of heaviness that can be limited or permanent for a few minutes and spread to the left arm, neck or chin. These symptoms may be accompanied by symptoms such as diaphoresis, nausea, abdominal pain, syncope, dyspnea. However, atypical presentations of acute coronary syndrome are also frequently seen. These include

epigastric pain, new-onset indigestion, chest pain in the stinging style, chest pain with pleurotic features. Atypical complaints are frequently observed in women, young patients (25–40 years), in the geriatric group, in patients with diabetes mellitus, dementia and chronic kidney failure [19,20].

ECG

The first thing to be done for the purpose of diagnosis in patients suspected of ACS is to perform a standard 12 lead ECG at rest. ECG recording must be taken within the first 10 minutes after the patient's evaluation. Detection of ST segment elevation lasting longer than [21] 20 minutes reveals STEMI and requires a different treatment approach [22]. In cases where there is no ST elevation and the patient is symptomatic, recurrent ECG recordings should be taken and compared with those taken in an asymptomatic state. Comparison with a previous ECG, if possible, can provide valuable information, especially in cases of concomitant cardiac disease, such as previous myocardial infarction or left ventricular hypertrophy. ST segment shifts and T wave changes are ECG indicators of unstable coronary artery disease [23]. While the number of leads showing ST depression and the extent of ST depression provide information about the severity and extent of ischemia, it is also associated with prognosis [24]. In the presence of the appropriate clinical picture, the ST segment depression NSTEMI greater than 0.5 mm (0.5 mV) in two consecutive leads is an indication of acute coronary syndrome and is associated with prognosis [25]. It should be remembered that the NSTEMI of a completely normal ECG does not remove the possibility of acute coronary syndrome. Many studies have shown that 5% of patients discharged from the emergency department due to normal ECG have either acute myocardial infarction or unstable angina pectoris [26,27]. If a patient shows signs and symptoms of a potential ACS, the clinician uses the ECG findings to insert the patient into one of three groups:

i. ST-segment elevation or predicted new LBBB: classified as ST-segment elevation MI (STEMI) and characterized by ST-segment elevation in two or more successive leads. Threshold values for ST segment elevation compatible with STEMI; 0.2 mV (2mm) in leads V2 and V3 and 0.1 mV (1mm) in all other leads (male ≥40 years); 0.25 mV (2.5mm) in leads V2 and V3 and 0.1 mV (1mm) in all other leads (male <40 years); 0.15 mV (1.5mm) in leads V2 and V3 and 0.1 mV (1mm) in all other leads (female).

ii. Ischemic ST-segment depression is classified as> 0.5 mm (0.05 mV) or dynamic T-wave inversion (box) NSTEMI with pain or discomfort. Non-chronic or temporary ST-segment elevation (<0.5mm for <20 minutes) is also included in this category. Threshold values for ST-segment depression compatible with ischemia; It is 0.05 mV (-.5mm) in leads V2 and V3 and -0.1mV (-1mm) in all other leads.

iii. Non-diagnostic ECG either normal or minimally abnormal (for example, nonspecific ST-segment or T wave changes): This ECG is not diagnostic and decisive for ischemia requires further risk classification. This classification includes patients with normal ECG and <0.5 mm (0.05 mV) ST segment deviation or dalga0.2 mV T wave inversion. The ECG in this category is called non-diagnostic [28].

The newly developed left bundle branch block is always pathological and may be a sign of myocardial infarction. Three criteria are used to diagnose infarction in patients with left bundle branch block:

a. In the leads with positive QRS complex> 1 mm concordant (same directional) ST elevation (5 points).

b. ST collapse from concordant> 1 mm in V1-3 (3 points).

c. In elevations with negative QRS complex> 5mm excessive discordant ST elevation (2 points)

It has 90% specificity to diagnose myocardial infarction ard 3 points in total [29].

Cardiac Biomarkers

Cardiac markers complement the risk classification, triage, diagnosis and management of patients with suspected acute coronary syndrome, 12-lead electrocardiography (ECG), clinical evaluation [30]. Aspartate Transaminase (AST), Lactate Dehydrogenase (LDH) and Lactate Dehydrogenase subforms are not used today due to low cardiac specificity and late elevations [31]. **Table 2** shows the most used biomarkers in acute coronary syndrome [32].

Table 2: Characteristics of cardiac markers in acute myocardial infarction.

Serum cardiac marker	The first positive time	Peak level	sensitivity (%)	Specificite (%)	Positive predictive value	Negative predictive value
CK						
Single measurement	3-8	12-24	35	80	20	90
Series measurement			95	68	30	99
CK-MB						
Single measurement	4-6	12-24	35	85	25	90
Series measurement			95	95	73	99
Troponin I and T						
4 hours after the onset of chest pain	4-10		35	96	56	91
10 hours after the onset of chest pain		8-28	89	95	72	98

Creatine Kinase (CK)

Creatine kinase (CK) is an enzyme found in striated muscle, brain, kidney, lung tissues and gastrointestinal tract. This commonly used marker has low sensitivity and specificity in demonstrating cardiac damage. In addition, CK levels may increase in a number of non-cardiac conditions, such as trauma, seizures, kidney failure, hyperthermia, and hyperthyroidism. CK levels rise 3-8 hours after myocardial injury, reaches peak level 12-24 hours and goes down to old levels in 3-4 days [32]. Serum CK levels can be used as screening tests for the need for more specific tests. Although creatine Kinase-Muscle Brain (CK-MB) was hospitalized and was measured frequently after 6-12 hours, cardiac troponin and CK-MB replaced it.

CK-MB Isoenzyme: It is more specific than CK alone and is an earlier marker for myocardial infarction [31].

CK-MB Subfoms. It can be customized to CK-MB subforms. CK-MB is present in 1 myocardium and CK-MB 2 in the placenta. Measured in about 25 minutes [33]. In a large study involving one hundred and ten chest pain patients [34], the sensitivity of CK-MB subforms studied 6 hours after the onset of symptoms was 96% and the specificity was 94%

Myoglobin

It is a low molecular weight protein found in skeleton and heart muscle. It reaches the level measured 2 hours after the onset of myocardial necrosis [31]. It has low cardiac specificity but high sensitivity. It is more useful if the level is normal 4-8 hours after the onset of the symptom. It should be used in conjunction with other markers because its level drops rapidly [35].

Troponins

Troponins (T, I, C) are found in the skeleton and heart muscle. The troponin complex, consisting of Troponin T, I and C, enables the interaction of actin and myosin through calcium and takes place in thin filaments [4,36]. Isoforms of Troponin T and I in the heart and skeletal muscle are known as "cardiac troponins", unlike C. Preferred biomarkers in myocardial injury [37]. The high level of their clinical sensitivity depends on their high level of presence in the heart tissue compared to other markers, and in healthy people, their circulation levels are very low [4,38]. Their high specificity results from heart-specific cardiac Troponin T (cTnT) and cardiac Troponin I (cTnI) isoforms. For this reason, problems arising due to high values seen in creatine kinase (CK) and CK-MB due to skeletal muscle damage are not an issue for cardiac troponins (cTn) [4,36]. The sensitivity and specificity of Troponin T and I are similar. Unlike Troponin I, Troponin T levels may be elevated in patients with kidney disease, polymyocytes or dermatomyositis.

Cardiac troponins come to the emergency room and are measured again after 6-12 hours [39]. Acute myocardial infarction with a high altitude of Troponin and CK-MB is considered, but ongoing minor myocardial damage or micro-infarction should be considered in patients with high Troponin value and normal CK-MB value. Cardiac troponins can remain high in the blood for about 2 weeks [31]. The high circulation levels of cardiac troponins for a long period such as 7-14 days allows them to be used in the diagnosis of acute MI as well as in the diagnosis of subacute MI and eliminates the need for LDH isoenzymes. High Troponin T or I levels are useful for identifying patients with an increased risk of death and developing myocardial infarction [32].

The increased risk relates to the quantitative value of Troponin. In a study including acute chest pain to the emergency room (AS), 773 patients with normal or near normal ECG values with the first and sixth normal troponin values, the risk of major cardiac events at [40], 0-30 days was found to be very low. The head-to-bed Troponin values have also improved considerably. AMI has been identified jointly by the European Cardiology Association, the American Heart Association, the American College of Cardiology, and the World Heart Federation, and since 2000, CTN has been reported to be 99% effective in diagnosing AMI in healthy individuals [41,42]. Early normal Troponin values can cause false negative diagnosis in patients with small or large AMI. To measure cardiomyocyte damage, research has recently been carried out on cTn measurement methods. It has been shown to be more sensitive than traditional tropon [43,44]. However, the high sensitivity test designed to detect minimal myocardial damage and reduce the number of unidentified axle patients showed low specificity. Indeed, an increase has been associated with emergency care and hypertensive attack, congestive heart failure, pulmonary embolism, sepsis, and high-intensity exercise, including various cardiac and non-cardiac conditions [45,46]. **Table 3** shows the clinical conditions in which CTN is high.

Table 3: Clinical Conditions Other Than Acute Coronary Syndrome with the Height of Troponin.

Heart failure (acute and chronic)
Aortic dissection, aortic valve diseases or hypertrophic cardiomyopathy
Cardiac contusion, cardioversion, ablation, pacing, endomyocardial biopsy
Inflammatory diseases such as myocarditis and pericarditis
Hypertensive crisis
Tachyarrhythmia and bradyarrhythmias
Pulmonary embolism or serious pulmonary disease
hypothyroidism
Takatsubo syndrome
Acute neurological events (stroke, bleeding, etc.)
Chronic or acute renal failure
Infiltrative diseases (hemochromatosis, amyloidosis, etc.)
Drug toxicity (adriamycin, 5-fluorouracil, snake venom, etc.)
rhabdomyolysis
Burn that covers more than 30% of the body
Serious general condition disorder (sepsis, respiratory failure, etc.)

Serious general condition disorder (sepsis)

In a study, the level of cTnT level (toplum0.1µg / l) was found to be 7% in society, and it was shown to be associated with left ventricular dysfunction, diabetes mellitus, left ventricular hypertrophy, and moderate renal impairment [47,48]. In men, African-American concentrations are high. It is known that cTn levels can be measured high not only in healthy individuals, but also in patients who are admitted to or hospitalized for any reason. In a study conducted by Alcalai et al. [49], it was found that cTnT level was measured in all patients who applied to the hospital for various reasons within a 10-month period, and 53% of 635 patients with cTnT level> 0.1 µg / l were diagnosed with AKS and 41% did not have thrombotic causes. In 6%, no reason was found. The results of this study show that although the majority of patients admitted to the hospital have high cTn, this is not caused by coronary artery disease. In the aforementioned study, patients without troponin height were investigated, non-ischemic cardiac events such as myocarditis or arrhythmia in 5%, surgical conditions in 8% (trauma, intense gastrointestinal bleeding, intestinal obstruction, etc.), Renal failure was observed in 2%, and cardiopulmonary resuscitation (CPR) was performed in 2%. Another important finding obtained in the same study is that in patients diagnosed with acute coronary syndrome, the mean cTnI level is 1.5 ± 2.4 µg / l, while patients with troponin height without thrombotic cause have 0.6 ± 0.9 µg / l ($p < 0.01$). Patients with a cTnI level <1.0µg / l are relatively unlikely to have ACS. In this case, applying antiaggregant or antithrombotic therapy is of no use, and may even affect the course of the underlying disease [50]. Treatment should aim at correcting the pathology that causes this condition in non-thrombotic troponin elevation, i.e. in clinical situations where there is no ischemia in the coronary arteries but elevation of Troponin in peripheral blood sampling [51].

lack of um, etc.)

CRP and hs-CRP

C-Reactive Protein (CRP) and standardized high sensitivity CRP (hs-CRP) are the most clinically researched inflammatory biomarkers to prove the location of inflammation in cardiovascular diseases. CRP is an acute phase protein that is produced in response to cytokines, such as IL-6 and tumor necrotizing factor

alpha (TNF-α) in liver cells [52]. The increase of hs-CRP levels in acute coronary syndromes shows not only localized plaque rupture, but also excessive response of the common vascular inflammation and inflammatory system. High hs-CRP values at the time of admission and before discharge are associated with short-term and long-term prognosis in NSTEMI patients [53].

Fatty Acid Binding Protein (h-FAB)

It was first discovered in 1972 by Ockner during a study examining the absorption of fatty acids from the intestines [54]. The heart muscle is rapidly released into the circulation after cell damage and rises in the first 1-3 hours. 6-8 of damage to the peak value. It reaches its normal level and returns to its normal level in 24-30 hours [55]. Although it is thought to be theoretically elevated in the early period and to be useful in making the diagnosis of AMI earlier, there is insufficient evidence to enter daily use [56].

SolubleCD40Ligand (sCD40L)

Much of the circulating sCD40L is secreted from activated platelets and triggers the initiation of an inflammatory reaction in the vascular endothelial cells by cytokine and chemokine release [57]. In the OPUS-TIMI16 study, sCD40L concentrations were found to be significantly higher in patients with acute coronary syndrome than in control patients, and increased sCD40L levels were associated with a higher risk of future cardiovascular events. In the same study, it was reported that when combined with cardiac troponin I it provides a better risk assessment for death and nonfatal myocardial infarction [58].

Pregnancy-related Plasma Protein (PAPP-A)

PAPP-A is considered as a marker of unstable plaque rupture in patients with sudden cardiac death. PAPP-A increase in patients with suspected ACS may be an independent marker of future ischemic events [59].

Myeloperoxidase (MPO)

It is suggested that myeloperoxidase, which is formed by the degranulation of leukocyte cells, may be useful in the period before irreversible damage occurs because it reflects activation of hemostasis or inflammatory response after plaque rupture. It is not specific for ACS; Infection related to neutrophil activation also increases in inflammatory and infiltrative diseases [60].

Relationship of troponin elevation with events other than acute coronary syndrome

Heart Failure

In approximately 50% of patients, the height of cTn can be observed both in the acute decompensation phase and in the chronic compensation period [61,62]. Although the pathophysiology is not fully known, the level of cTn in patients with heart failure is a sign that myocyte integrity is impaired. Although a limited number of patients may have concurrent coronary ischemia, the high level of cTn in the heart failure process is independent of coronary ischemia in the majority of patients [63]. In heart failure, high cTn level has been reported to be associated with lower ejection fraction, impaired hemodynamic parameters and increased risk of mortality. In fact, increased cTn level in compensated heart failure has been shown to be associated with an increased risk of death or hospitalization [64]. An important point is that the level of cTn level is low in heart failure. In one study, mean cTn-T level was found to be 0.181 ± 0.53 µg / l in 265 patients (mean EF = 30 ± 0.01%) with heart failure [65].

Pulmonary Embolism

An increase in cTn levels is observed in 20-40% of patients diagnosed with moderate or severe pulmonary embolism [66,67]. It is thought that the reason for the increase in cTn may be accompanying hypotension, right ventricular infarction or hypoxemia [68,69]. Studies show that the height of cTnT in the course of pulmonary embolism varies between 0.24-0.66µg / l, which values are very low compared to the cTnT heights during ACS. Unlike acute coronary syndrome, these values rise within 6-8 hours and peak within 10 hours, decline to normal levels within 40 hours [67,70]. In pulmonary embolism, the level of cTn is directly proportional to the severity of the clinical condition. In-hospital death has increased 30 times and more aggressive treatment is needed in these patients. Pulmonary embolism is a clinical condition that may be similar to acute coronary syndrome in terms of its symptoms, and similarly, cTn elevation can be monitored. Therefore, it should be considered in the differential diagnosis of ACS, especially in patients with mild cTn elevation.

Cardioversion

Increased cTn after elective electrical cardioversion is rare, and if there is an increase, this is at low levels [71,72]. An increase in cTn is frequently observed in patients resuscitated after cardiac arrest [73].

Sepsis

An increase in cTn levels is a common finding in patients with sepsis, and a significant proportion of these patients have been shown to have no coronary artery disease in coronary angiography or autopsy examinations [74,75]. Although the reason for the increase of cTn in sepsis is not known precisely, factors such as endotoxins with cytotoxic effects, inflammatory mediators (IL, TNF, heat shock protein, etc.), septic microembolics, vasoactive drugs, concomitant hypotension and myocarditis are thought to be responsible [76,77].

Kidney Failure

In 16-94% of asymptomatic patients with end-stage renal failure, cTn elevation is seen [78,79]. In hemodialysis patients, cTn elevation is associated with atherosclerotic risk factors such as hypercholesterolemia, left ventricular hypertrophy, severe coronary calcification and diabetes mellitus [80].

Myocarditis

Patients with myocarditis diagnosed have high cTn levels. Studies have reported that cTn levels in myocarditis and myopericarditis may increase at similar levels with acute myocardial infarction [81,82].

Supraventricular Tachycardia

It is known that there may be an increase in Troponin values in patients with supraventricular tachycardia since 2003. High troponin values can be seen in these patients with normal angiography [83]. The increase in cardiac conditions at the high sensitivity Troponin (hs-cTn) level does not cause confusion due to the presence of specific treatment guidelines. However, the lack of a definitive guide for the increase of hs-cTn in non-cardiac conditions leads to confusion in these cases in terms of the need for additional diagnosis and treatment [84,85]. To resolve this diagnostic dilemma, 'A Consensus Statement on Redefining Myocardial Infarction', patients with typical AKS with elevation of troponin (type 1 MI), and patients with primarily

heterogeneous clinics and non-ACS (non-ACS conditions). It has classified. The prevalence of non-ACS cases with high cTn remains uncertain compared to high cTn patients with ACS, due to limited observational studies in the health system [86]. In a study, a significant difference in mortality was not found in a group of patients discharged from the emergency department with high and normal cTn [87].

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