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ORIGINAL ARTICLE
ORİJİNAL ARAŞTIRMA

Evaluation of Cardiac Clues in Patients Admitted to Pediatric Cardiology Outpatient Clinic with Syncope

Çocuk Kardiyoloji Polikliniğine Senkop ile Başvuran Olgularda Kardiyak İpuçlarının Değerlendirilmesi

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ABSTRACT

Aim: In our study, we aimed to determine the incidence rates of children admitted to the pediatric cardiology outpatient clinic with syncope according to their etiologies; and especially to reveal the clues that distinguish cardiac syncope cases from other cases.

Material and Method: The study was conducted by obtaining the information of 795 children aged 5-17 years who were diagnosed with syncope in the pediatric cardiology outpatient clinic of our tertiary university hospital between 01.01.2021 and 01.06.2024 from the data in our hospital automation system. Medical history of the patient and his/her family, syncopal event, physical examination findings, 12-lead electrocardiography (ECG), echocardiography, 24-hour rhythm holter and exercise ECG records were analyzed.

Results: Cardiac syncope was 14.5% (10.8% arrhythmia and 3.7% structural heart disease) while noncardiac syncope was 85.5% (VVS 60.6%, OH 18.6%, situational 1% and 4.8% idiopathic). The most common conditions associated with cardiac syncope were a history of arrhythmia and heart disease, a family history of sudden death at a young age, and syncope associated with exercise-palpitations-chest pain. Rhythm holter test had a low diagnostic rate, while exercise ECG test had a high diagnostic rate.

Conclusion: It is important to diagnose cardiac syncope as it can cause sudden death. Standard 12-lead ECG, echocardiography and rhythm holter examination should be performed in all suspected patients; exercise ECG, head-up tilt test, genetic arrhythmia/cardiomyopathy investigations and electrophysiologic study should be performed in selected cases. In addition, simultaneous ECG monitoring during electroencephalography (EEG) examination in patients presenting with atonic seizures will be very useful in terms of clarifying the etiology.

Keywords: Syncope, cardiac syncope, children, arrhythmia

ÖZ

Amaç: Çalışmamızda çocuk kardiyoloji polikliniğine senkop nedeni ile başvuran çocukların etiyolojilerine göre görülme oranlarının; ve özellikle kardiyak senkoplu olguları diğer olgulardan ayıran ip uçlarının ortaya konulması amaçlanmıştır.

Gereç ve Yöntem: Çalışma üçüncü basamak üniversite hastanemiz çocuk kardiyoloji polikliniğinde 01.01.2021 ile 01.06.2024 tarihleri arasında senkop tanısı konulan 5-17 yaşlarındaki 795 çocuğun bilgilerinin hastanemiz kayıt sistemindeki verilerinden elde edilmesiyle gerçekleştirildi. Hastanın kendisi ve ailesinin tıbbi öyküsü, senkobun görülme şekli, fizik muayene bulguları, 12 kanallı elektrokardiyografi (EKG), ekokardiyografi, 24 saatlik ritim holter ve egzersiz EKG kayıtları incelendi.

Bulgular: Kardiyak senkop 14.5% oranında (10.8% aritmi ve 3.7% yapısal kalp hastalığı) iken nonkardiyak senkop 85.5% (VVS 60.6 %, OH 18.6%, situational 1% ve 4.8% idiyopatik) olarak tespit edildi. Kardiyak senkopla en çok ilişkilendirilen durumlar; hastanın kendisinde aritmi-kalp hastalığı öyküsü bulunması, ailesinde genç yaşta ani ölüm öyküsü bulunması ve senkobun egzersiz, çarpıntı ve göğüs ağrısı ile ilişkili olması idi. Ritim holter tetkikinin tanı koyma oranı düşük iken egzersiz EKG testinin ise yüksekti.

Sonuç: Ani ölüme neden olabildiği için kardiyak senkop tanısının konulması önem arz etmektedir. Şüphelenilen bütün hastalara standart 12 kanallı EKG, ekokardiyografi ve ritim holter tetkiki yapılmalı; seçilmiş olgulara da egzersiz EKG incelemesi, head-up tilt testi, genetik aritmi/kardiyomiyopati araştırmaları ve elektrofizyolojik çalışma uygulanmalıdır. Ayrıca atonik nöbet ile başvuran olgulara EEG tetkiki esnasında eş zamanlı olarak EKG monitörizasyonu yapılması etiyolojinin aydınlatılması açısından oldukça faydalı olacaktır.

Anahtar Kelimeler: Senkop, kardiyak senkop, çocuk, aritmi

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INTRODUCTION

Loss of consciousness is a cognitive state in which a person is not aware of himself/herself and his/her condition and is unable to respond to stimuli. Transient loss of consciousness is defined as a real or apparent loss of consciousness with loss of awareness, characterized by abnormal motor control, loss of responsiveness and short-term amnesia. Syncope is defined as transient loss of consciousness due to cerebral hypoperfusion; it is characterized by rapid onset, short duration and spontaneous full recovery. The pathophysiology of syncope involves a fall in systemic blood pressure and cerebral hypoperfusion. Systemic blood pressure is determined by cardiac output and peripheral vascular resistance; low levels of both are frequently involved in syncope. Therefore, epilepsy, hypoxic ischemic attack, intracerebral or subarachnoid hemorrhage, psychogenic pseudosyncope and post-traumatic loss of consciousness that do not have this pathophysiology are not defined as syncope (1).

The most common causes of syncope in children include noncardiac causes such as vasovagal (VVS) and orthostatic hypotension (OH). However, since syncope cases with cardiac causes such as arrhythmias and structural heart diseases including hypertrophic cardiomyopathy (HCMP) may cause sudden death, it is very important to make a rapid and accurate differential diagnosis (1). Patients with cardiac syncope have unique features that distinguish them from other syncope etiologies, such as a family history of sudden death at a young age and cardiac disease in the child, abnormal cardiovascular system examination and association with exercise-palpitations-chest pain.

In our study, we aimed to make the etiologic differentiation of patients consulted to the pediatric cardiology department with syncope by utilizing the anamnesis-physical examination-12 lead standard electrocardiography (ECG)-24-hour rhythm holter-echocardiography and exercise ECG records in the automation system of our hospital; and to reveal clues that may help in the diagnosis of syncope cases with cardiac causes.

MATERIAL AND METHOD

On May 29, 2024, the Clinical Research Ethics Committee of our hospital granted approval for our study under decision number 2024-9/4. In this study, the Declaration of Helsinki's tenets were adhered to.

Our study was conducted by obtaining the information of children aged 5-17 years who presented to the pediatric cardiology outpatient clinic of our tertiary university hospital with transient loss of consciousness between 01.01.2021 and 01.06.2024 and were diagnosed with syncope from the data on the automation system. The history of systemic disease-arrhythmia-congenital heart disease (with or without surgery), family history of sudden death-arrhythmia-cardiomyopathy, prodromal symptoms before syncope (such as pallor, sweating and nausea) and syncopal event syncope (prolonged standing, sudden standing up from a sitting position, bloodletting and exercise-related syncope) were recorded. All patients underwent a detailed physical examination including cardiac examination, standard 12-lead ECG and echocardiography examination. Patients with a life-threatening syncope (e.g. head trauma), three or more episodes of syncope, symptoms suspicious for cardiac syncope (e.g. syncope accompanied by palpitations and/or chest pain, syncope during exercise, family history of sudden death, and a history of arrhythmia-congenital heart disease-cardiac surgery) or arrhythmia detected during a standard 12-lead ECG underwent rhythm holter examination. In addition, rhythm holter examination was performed in patients who presented with atonic seizures and were found to have arrhythmia as a result of simultaneous ECG monitoring during electroencephalography (EEG) examination. Exercise ECG test was performed in patients with syncope during exercise who could not be diagnosed by 12-lead ECG-echocardiography-rhythm holter examinations. Genetic examination was performed in all patients with long QT. Ajmaline stimulation test was performed in patients with a family history of Brugada syndrome that could not be detected by standard ECG-rhythm holter examination.

According to the causes of syncope, patients were divided into cardiac and noncardiac syncope. Cardiac causes of syncope were divided into two groups as arrhythmia and structural heart disease, while noncardiac causes were divided into three groups as vasovagal, situational and orthostatic hypotension.

Vasovagal syncope was diagnosed in patients who had prodromal symptoms including pallor, sweating and nausea in the anamnesis and who developed short-term transient loss of consciousness in prolonged standing or sitting, pain-fear, crowded-hot-airless environments (2).

Situational syncope was diagnosed in cases of transient loss of consciousness in situations such as urination, defecation, retching, swallowing, coughing, sneezing and after exercise (2).

A diagnosis of syncope due to orthostatic hypotension was made in the case of transient loss of consciousness within three minutes (blood pressure drop of more than 20 mmHg systolic and 10 mmHg diastolic) when the patient suddenly stood up from the bed or toilet during morning awakening after the use of blood pressure-lowering drugs such as antidepressants and diuretics for autonomic reasons such as diarrhea-vomiting-bleeding, diabetes and chronic renal failure (2).

Cardiac syncope was suspected in the presence of palpitations and/or chest pain with syncope, development of syncope during exercise, a family history of sudden death without cause or arrhythmia, a history of arrhythmia, congenital heart disease or cardiac operation in the patient, or pathologic findings on cardiovascular examination. Cardiac syncope was diagnosed in patients with structural heart diseases that may cause syncope such as HCMP, dilated CMP, valve lesions with stenosis, myocarditis, pulmonary hypertension (PHT), and rhythm disorders that may cause syncope such as long QT, Brugada syndrome, ventricular tachycardia (VT), supraventricular tachycardia (SVT) and severe atrioventricular block on electrocardiography (2).

Patients who were thought to have transient loss of consciousness with neurologic cause were evaluated by a pediatric neurology specialist.

Exclusion criteria were as follows: Generalized seizures, complex partial seizures, absence epilepsy, falls without transient loss of consciousness, posttraumatic transient loss of consciousness, psychogenic pseudosyncope, intracerebral or subarachnoid hemorrhage, transient ischemic attack, hypoglycemia, hypoxia, hypocapnia, metabolic disorders including hyperventilation with hypocapnia, intoxication and coma (1-2).

In the design of our study, we utilized the algorithm for classification of patients presenting with loss of consciousness given in the ESC guideline (**Figure 1**).

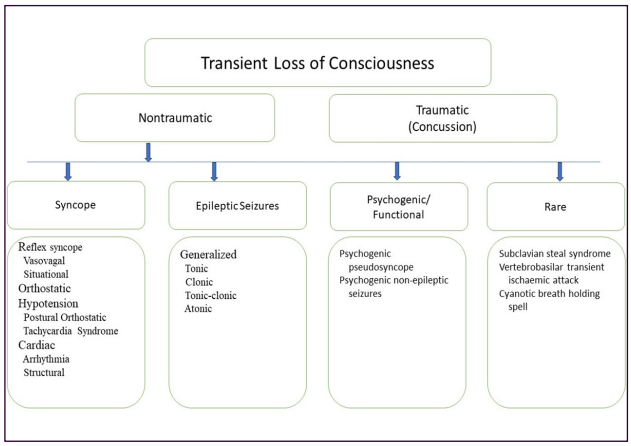

Figure 1. Classification of patients presenting with loss of consciousness (1)

Statistical Analysis

Statistical analysis of the study was performed using the Statistical Package for the Social Sciences for Windows ver. 26.0 package program was used. For categorical variables, n (%) was used, for continuous variables, mean ± SD (standard deviation) was used in case of conformity to normal distribution, and median and IQR (Inter Quantile Range) values were used when conformity was not achieved. Descriptive analyses were employed to assess the distribution and frequency of the data. Normality analysis was performed using the Kolmogorov–Smirnov test. In intergroup comparisons, independent t test was used for those with normal distribution and Mann Whitney U test was used for those without normal distribution.

RESULTS

Our study consisted of 795 children aged between 5-17 years [mean 13 (5-17)], 494 of whom were girls (62.1%). Children presenting with cardiac syncope accounted for 14.5% (115 cases) of the cases, while those presenting with non-cardiac causes accounted for 85.5% (680 cases).

Among patients with cardiac syncope, 86 (10.8% of total syncope cases and 74.8% of cardiac syncope cases) were due to arrhythmia and 29 (3.7% of total syncope cases and 25.2% of cardiac syncope cases) were due to structural heart disease. Among the non-cardiac causes of syncope, most of the patients (60.6%) were in the VVS group, followed by OH (18.6%) and situational (1%). Thirty-eight (4.8%) patients were accepted idiopathic. The proportion of female patients was high in the noncardiac syncope group (noncardiac 65.4% - cardiac 49.6%, p<0.001). The highest proportion of female patients (67.3%) was found in the VVS group. The mean age of patients in the noncardiac syncope group was higher [noncardiac 14 years (8-17) vs cardiac 11 years (5-17), p<0.001]. The highest mean age was found in the VVS group [14 years (9-17)] and the lowest in the structural heart disease group [10 years (5-16)]. The number of syncope episodes was 1.4±0.3 in the cardiac syncope group and 2±0.4 in the noncardiac syncope group (p<0.001). The number of syncope episodes was highest in the VVS group (2.1±0.3) and lowest in the structural heart disease group (1.3±0.3) (**Table 1**).

Table 1. Distribution of children diagnosed with syncope

	Reflex syncope and OH, N=638 (80.2%)			Cardiac, N=115 (14.5%)		Idiopathic, N=42 (5.3)	Total, N=795
	VVS	Situational	OH	Arrhythmia	Structural		
N (%)	482 (60.6)	8 (1)	148 (18.6)	86 (10.8)	29 (3.7)	42 (5.3)	795 (100)
F (%)	338 (67.3)	4 (50)	75 (62.5)	42 (48.8)	15 (51.7)	20 (52.6)	494 (62.1)
Age	14 (9-17)	12 (9-16)	13 (8-17)	11 (5-17)	10 (5-16)	14 (10-17)	13 (5-17)
Number of episode	2.1±0.3	1.2±0.2	1.8±0.3	1.4±0.3	1.3±0.3	2±0.3	1.9±0.3

Cardiac syncope was diagnosed in 40 of 72 patients (55.6%) with palpitations and/or chest pain during syncope. Of these 40 patients; 19 had arrhythmia [nine paroxysmal SVT (four on standard 12 lead ECG and five on rhythm holter)], [five sustained VT (three on standard 12 lead ECG and two on rhythm holter)], [two had catecholaminergic polymorphic ventricular tachycardia (CPVT) (on exercise ECG], [three had Wolff Parkinson White syndrome (all three on 12 lead ECG)] and 21 had structural cardiac pathology (on echocardiography: five HCMP, five myocarditis, four dilated CMP, three aortic valve stenosis, two anomalous origin of coronary artery and two PHT).

Of 73 patients with syncope during exercise 39 (53.4%) were diagnosed with cardiac syncope; seven had long QT (three on 12 lead ECG and four on exercise ECG), four had grade 2 AV block (three on 12 lead ECG and one rhythm holter), one complete AV block (12 lead ECG), two catecholaminergic polymorphic ventricular tachycardia (exercise ECG), two sinus bradycardia (rhythm holter), four aortic valve stenosis (AS), two pulmonary valve stenosis (PS), five HCMP, four dilated CMP, two anomalous origin of coronary artery, two pulmonary hypertension (PHT) and four acute myocarditis.

Cardiac syncope was diagnosed in eight of 18 patients (44.4%) with family history of sudden death; three of these eight patients had long QT, two had Brugada syndrome and three had HCMP.

Thirty (25.6%) of the patients presenting with cardiac syncope had a known history of arrhythmia/structural heart disease/heart surgery. Of these patients, 14 (12.2%) had arrhythmia (three Wolff Parkinson White syndrome, three 2nd degree AV block, two long QT, two SVT, one VT, one sinus bradycardia, one sinus pause lasting longer than 3 seconds and one complete AV block), 12 (10. 4%) had structural heart disease (five AS, three dilated CMP, two HCMP, one PS and one PHT) and four patients (3%) had a history of cardiac surgery (three for tetralogy of Fallot and one for subvalvular and valvular aortic stenosis).

Of the 86 arrhythmia cases we detected in our study, 30 were detected in standard 12 lead ECG at the first admission, 40 were detected in rhythm holter when the admission ECG was normal and six were detected in exercise ECG when 12 lead ECG and rhythm holter were normal. In addition, 10 arrhythmias were detected of arrhythmia during EEG in neurology clinic.

Detection of arrhythmia during EEG in neurology clinic six of the 10 patients had grade 2 AV block, two had long QT, one had pause lasting longer than 3 seconds and one had sinus bradycardia.

In 80 (17.6 %) of a total of 455 patients who underwent rhythm holter examination, rhythm disturbances that could cause syncope were detected. However, when 40 patients who were known to have arrhythmia before rhythm holter examination were excluded (10 patients with arrhythmia detected of during EEG in neurology clinic and 30 patients with arrhythmia detected on standard 12 lead ECG in order to fully reveal the arrhythmia pattern), the rate of arrhythmia detection in rhythm holter examination was 9.6% (40/415). In 7 patients with arrhythmia that did not cause syncope on standard 12-lead ECG (two patients with 1st AV block, two patients with ventricular extrasystole, two patients with mild sinus bradycardia, one patient with premature atrial beat), rhythm holter examination revealed arrhythmia that could cause syncope (2nd degree AV block in two patients, VT in two patients, marked sinus bradycardia in two patients and supraventricular tachycardia in one patient).

Exercise ECG testing was performed in 14 patients; and cardiac syncope was diagnosed in eight of them (57.1%). Arrhythmias were detected in six of these eight patients (long QT in four and CPVT in two), and an abnormal origin of the coronary artery was suspected in two of them because ischemic changes were observed (diagnosis confirmed by diagnostic cardiac catheterization).

Genetic analysis was performed in all 15 patients with long QT and genetic mutations were found in 8 (53.3%). All patients with Brugada syndrome had a positive family history; three of the five patients had a classic pattern on 12-lead ECG, while the other two patients were diagnosed with ajmaline test.

In a total of 42 (5.3%) patients (34 patients with syncope during exercise, four patients with syncope accompanied by palpitations/chest pain and four patients with syncope in supine position), we could not elucidate the etiology and classified them as idiopathic syncope (**Table 2**).

Table 2: Supportive findings at the time of initial diagnosis of cardiac syncope

N=115	N (%)		
Accompanied by palpitations and/or chest pain	40 (34.8)		
Syncop during exercise	39 (33.9)		
Family history of sudden death	8 (7)		
History of arrhythmia-CHD or cardiac surgery in the patient	Arrhytmia	CHD	Cardiac surgery
	14 (12.2)	12 (10.4)	4 (3)
Detection of arrhythmia during EEG in neurology clinic	10 (8.7)		
Detection of arrhythmia on standard 12 lead ECG	30 (26.1)		
Detection of arrhythmia on 24-hour rhythm holter monitoring	80 (69.6) (during EEG in neurology + 12 lead ECG + rhythm holter) 40 (34.8) (only rhythm holter)		
Detection of arrhythmia during cardiac exercise stress test	6 (5.2)		
Detection of structural heart disease by echocardiography	29 (25.2)		

In the arrhythmia group of patients with cardiac syncope, 2nd degree AV block (32 (27.8%) and long QT (15 (13%)) were the most common; in the structural heart disease group, valvular aortic stenosis was detected in 6 (5.2%) and HCMP in 6 (5.2%) patients (**Table 3**).

Table 3: Distribution of children diagnosed with cardiac syncope

	N (%)	Age (year)	F (%)	Frequency of Syncope
Cardiac Arrhytmia				
Long QT	15 (13)	12 (8-17)	7 (26.7)	1.4±0.3
Brugada	5 (4.3)	8 (8-13)	0 (0)	1.2 (1-2)
2nd degree AV block	32 (27.8)	11 (6-13)	15 (48)	1.5±0.3
3rd degree AV block	3 (2.6)	12 (5-10)	3 (100)	1.3 (1-2)
Sinus node dysfunction (pause lasting longer than 3 seconds)	5 (4.3)	10 (7-12)	3 (60)	1.3 (1-3)
Wolff Parkinson White syndrome	5 (4.3)	12 (12-17)	3 (60)	1.3 (1-2)
Paroxysmal supraventricular tachycardia	9 (7.8)	9 (5-14)	3 (33.3)	1.3 (1-3)
Sustained ventricular tachycardia	5 (4.3)	9 (7-15)	3 (60)	1.3 (1-2)
Catecholaminergic polymorphic ventricular tachycardia	2 (1.7)	12 (9-13)	1 (50)	1.5 (1-2)
Sinus bradycardia (heart rate less than 35/min)	5 (4.3)	15 (12-15)	4 (80)	1.3 (1-3)
Total	**86 (74.8)**	**11 (5-17)**	**42 (48.8)**	**1.4±0.3**
Structural Cardiac Disease				
Valvular aortic stenosis	6 (5.2)	7 (5-12)	3 (50)	1.3 (1-3)
Pulmonary stenosis	2 (1.7)	6 (7-14)	1 (50)	1.5 (1-2)
Hypertrophic cardiomyopathy	6 (5.2)	12 (8-16)	2 (33)	1.2 (1-2)
Dilated cardiomyopathy	5 (4.3)	11 (6-14)	2 (40)	1.2 (1-2)
Anomalous origin of coronary artery	2 (1.7)	10 (7-9)	4 (40)	1
Pulmonary hypertension	3 (2.6)	11 (8-14)	1 (33)	1.3 (1-2)
Acute myocarditis	5 (4.3)	10 (6-16)	2 (40)	1.4 (1-2)
Total	**29 (25.2)**	**10 (5-16)**	**14 (48.3)**	**1.3±0.3**
Total	**115 (100)**	**11 (5-17)**	**56 (48.7)**	**1.4 (1-3)**

DISCUSSION

In our study including patients admitted to the pediatric cardiology outpatient clinic with syncope, we found that 14.5% of the syncope had cardiac causes; 10.8% of which were arrhythmia and 3.7% of which were structural heart disease. Syncope is a very common health problem that can be seen at any age and is responsible for 3% of emergency room visits. 40% of people have experienced syncope at least once in their lifetime. 17% of children have had at least one syncope attack until the end of adolescence (1,3). The majority of syncope seen in childhood is benign, such as syncope due to VVS and OH, and those that may be life-threatening are those that occur due to a sudden decrease in cardiac output due to arrhythmia or structural heart disease. The AHA guideline reported the sudden death rate in unselected cases in the pediatric period as 0.00001 cases per patient year in cases with syncope (2).

Cui et al reported that in their study including 1,947 children and adolescents aged 1-18 years (mean 11.1 ± 3.1) diagnosed with syncope within 30 years (female rate 55.37%), the rate of syncope increased over the years, the rate of neurally-mediated syncope increased while the rate of idiopathic syncope gradually decreased. It was also reported that the rate of cardiac syncope was 8.91% between 1992 and 2001 and 5.50% between 2012 and 2021. Zavala et al reported noncardiac syncope rate as 77.7%, cardiac 4% and idiopathic 18.3% in their meta-analysis including 11 articles and 3700 children aged between 3 months and 21 years. Landwehr et al reported reflex syncope in 69.8%, presyncope in 13.7%, idiopathic syncope in 13.4% and cardiac syncope in 3.1% of 262 patients (female 61.5%) aged 12±3.9 years admitted to general pediatrics with syncope. Bozlu et al in their study of 1020 cases (59.2% female) between the ages of 1-18 years (12.8± 4.8) admitted to the pediatric emergency outpatient clinic with syncope (12.8± 4.8%) reported the etiologies as: reflex/neural mediated 55%, orthostatic 23.72%, epilepsy 4.90% and cardiac syncope 5.39%. In another study from our country, 65.7% VVS, 9% POTS, 7.5% epilepsy, 4.5% hysterical seizure, 4.5% OH, 3% exercise-related syncope, 3% cardiogenic syncope and 3% breath holding were reported. In our study, children presenting with cardiac syncope constituted 14.5% of the cases (Arrhythmia: 10.8% of total syncope cases and 74.8% of cardiac syncope cases - Structural heart disease: 3.7% of total syncope cases and 25.2% of cardiac syncope cases), while those presenting with noncardiac causes constituted 85.5% (VVS 60.6%, OH 18.6%, situational 1% and 4.8% idiopathic). The reason why we found the rate of cardiac syncope higher than the literature may be attributed to the fact that our case group consisted of patients referred to the pediatric cardiology outpatient clinic and had undergone a certain preliminary evaluation and screening by emergency and pediatric physicians prior to their presentation to us.

Studies have shown that syncope occurs more frequently in girls than boys during childhood. This is due to the fact that noncardiac causes such as VVS and OH, which

constitute the majority of syncope, are more common in girls. No gender difference has been reported among cardiac syncope cases. Prodromal symptoms such as pallor, sweating, nausea and hypotensive complaints are mostly seen in adolescent girls, but cardiac syncope including arrhythmia and structural heart diseases can occur at any age; therefore, noncardiac syncope is common in older girls, whereas cardiac syncope is found at younger ages and equally in both sexes (2-4,7-10). In our study, we found that the age and female ratio of patients presenting with cardiac syncope were lower than in the noncardiac group. Cases with cardiac syncope occur more unexpectedly (not during prolonged standing, sudden standing up and seeing blood; without prodromal symptoms and accompanied by exercise-palpitation and chest pain). In addition, a family history of sudden death-arrhythmia-cardiac disease and pathologic cardiovascular examination findings are also common in these patients. Therefore, patients usually present to the emergency physician or pediatrician at the first episode and are then referred to the pediatric cardiology department. Cases with noncardiac syncope occur in young adolescents with typical findings and do not cause much concern in the community because they are common; in addition, since these patients are well known by emergency physicians and pediatricians, they are usually not consulted by a pediatric cardiologist at the first episode. Therefore, in our study, the number of syncope episodes was lower in the cardiac syncope group compared to the noncardiac syncope group (1.4±0.3 and 2±0.4, $p<0.001$).

Cardiac causes of syncope can be divided into two groups: arrhythmias and structural heart diseases. Unlike noncardiac causes of syncope, cardiac syncope can occur at any age, including infancy, and in both sexes at the same rate. Arrhythmias that may lead to syncope include long QT, severe AV blocks and tachyarrhythmias such as SVT-VT, while structural heart diseases include left ventricular outflow tract stenosis including hypertrophic cardiomyopathy, acute myocarditis and dilated cardiomyopathy (1.2,5,7, 10, 11). A family and personal history of arrhythmia/structural heart disease, association with exercise-chest pain and palpitations, and absence of prodromal symptoms have been associated with cardiac syncope (12). In order to diagnose these patients, 12-lead standard ECG, echocardiography, 24-hour rhythm holter, exercise ECG, tilt test, genetic and electrophysiologic examinations can be performed. In a study of 3122 children presenting with syncope, Uysal et al. reported a low rate of 2.4% for the diagnosis of arrhythmia-related cardiac syncope (13). In our study, we performed 12-lead ECG, echocardiography and rhythm holter examination in all patients suspected of cardiac syncope. We also performed exercise ECG test in exercise-related cases that we could not diagnose with these. The rate of rhythm holter examination to diagnose syncope due to cardiac arrhythmia was 9.6% in patients without previously detected arrhythmia. As a result of our study, being younger and presenting at the first syncope attack seemed to be risk factors for cardiac syncope. In addition, being female was not found to be a risk factor in contrast to noncardiac syncope. In the arrhythmia group, 2nd degree AV block and long QT were most common, while in the structural heart disease group, AS and HCMP were found. While genetic mutation was reported in 75% of long QT cases in the literature (14), it was found to be 53.3% in our study. The reason why we found a lower rate is that we could not screen all mutations that may cause long QT in our genetic laboratory. Cardiac syncope was diagnosed in 55.6% of patients with palpitations and/or chest pain during syncope, 53.4% of patients with syncope during exercise and 44.4% of patients with family history of sudden death. Among patients presenting with cardiac syncope, 26.1% had a previously known arrhythmia or structural heart disease that could cause syncope. Cardiac syncope was diagnosed in 57.1% of patients who underwent exercise ECG testing. Ten of the arrhythmia-related syncope cases (8.7% of cardiac syncope cases) were detected during EEG in patients presenting with atonic seizures in the neurology clinic.

Tilt test has been used for many years to diagnose reflex syncope,OH and psychogenic syncope. However, if hypotensive susceptibility occurs in syncope other than reflex syncope, false-positive results may be obtained regardless of etiology (including cardiac causes of syncope such as arrhythmias, aortic stenosis and hypertrophic cardiomyopathy) and in patients with true reflex syncope, positive results cannot always be obtained even with pharmacological provocation (false-negative rate is also high). In addition, the diagnosis of reflex syncope can be made to a great extent if a detailed anamnesis is taken and a complete physical examination including the cardiovascular system is performed. Today, the availability of cardiac evaluations such as ECG, echocardiography, rhythm holter and exercise ECG greatly reduces the need for tilt testing for diagnostic purposes. For these reasons, the indication for tilt testing is limited to patients with frequent recurrent syncope in whom the diagnosis of reflex syncope cannot be made after all investigations despite suspicion, and for whom tilt training and pharmacologic treatments will be planned (1,2).

Limitations

Our study had limitations such as its retrospective design, not accurately reflecting the general rate of cardiac syncope in the population because it consisted only of patients admitted to the pediatric cardiology outpatient clinic of a tertiary university hospital, and not performing tilt testing in patients.

CONCLUSION

Cardiac syncope should be suspected if the child has a family history of sudden death at an early age, a history of arrhythmia/congenital heart disease/heart surgery, pathologic findings on cardiovascular examination, absence of prodromal symptoms, syncope accompanied by chest pain/palpitation or developing during exercise and seen in any position including supine position. Routine standard 12-lead ECG, echocardiography and rhythm holter examination should be performed in these patients; exercise ECG examination, head-up tilt test, genetic arrhythmia/cardiomyopathy investigations and electrophysiologic study should be performed in selected cases. In addition, simultaneous ECG monitoring during EEG examination in patients presenting with atonic seizures will be very useful in terms of clarifying the etiology.

ETHICAL DECLARATIONS

Ethics Committee Approval: This study was approved by Bursa City Hospital Scientific Researches Ethics Committee (Date: 29.05.2024, Decision No: 2024-9/4).

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

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

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REFERENCES

1. Brignole M, Moya A, de Lange FJ, et al; ESC Scientific Document Group. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J. 2018;39(21):1883-948.
2. Shen WK, Sheldon RS, Benditt DG, Cohen MI, Forman DE, Goldberger ZD. 2017 ACC/AHA/HRS Guideline for the Evaluation and Management of Patients With Syncope: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Circulation. 2017;136(5):e60-e122. Erratum in: Circulation. 2017 Oct 17;136(16):e271-e272.
3. Hu E, Liu X, Chen Q, Wang C. Investigation on the Incidence of Syncope in Children and Adolescents Aged 2-18 Years in Changsha. Front Pediatr. 2021;9:638394.
4. Cui Y, Liao Y, Zhang Q, Yan H, Liu P, Wang Y. Spectrum of underlying diseases in syncope and treatment of neurally-mediated syncope in children and adolescents over the past 30 years: A single center study. Front Cardiovasc Med. 2022;9:1017505.
5. Zavala R, Metais B, Tuckfield L, DelVecchio M, Aronoff S. Pediatric Syncope: A Systematic Review. Pediatr Emerg Care. 2020;36(9):442-5.
6. Landwehr K, Meyer S, Flotats-Bastardas M, Poryo M. Synkopenabklärung bei Kindern und Jugendlichen – Handeln wir gemäß der aktuellen Leitlinie? [Syncope in children and adolescents: are the current guidelines being followed?]. Wien Med Wochenschr. 2021;171(7-8):157-64.
7. Bozlu G, Durak F, Duman D, Karpuz D, Kömür M, Kılınç OH. Çocuk acil servise senkop nedeniyle getirilen 1020 olgunun geriye dönük değerlendirilmesi. Mersin Üniv Saglık Bilim Derg 2020;13(1):8-12.
8. Duras E, İrdem A, Körbeyli HK, Aygün F, Dursun H. Çocuk kardiyoloji polikliniğine senkop nedeniyle başvuran olguların etiyolojik olarak değerlendirilmesi. J Contemp Med 2018;8(4):345-9.
9. Ganzeboom KS, Colman N, Reitsma JB, Shen WK, Wieling W. Prevalence and triggers of syncope in medical students. Am J Cardiol. 2003;91:1006–8.
10. Stewart JM, van Dijk JG, Balaji S, Sutton R. A framework to simplify paediatric syncope diagnosis. Eur J Pediatr. 2023;182(11):4771-80.
11. Francisco Pascual J, Jordan Marchite P, Rodríguez Silva J, Rivas Gándara N. Arrhythmic syncope: From diagnosis to management. World J Cardiol. 2023;15(4):119-41.
12. Yeom JS, Woo HO. Pediatric syncope: pearls and pitfalls in history taking. Clin Exp Pediatr. 2023;66(3):88-97.
13. Uysal F, Bostan ÖM, Çetinkaya F, Deniz T, Çil E. Syncope in Children: Is Rhythm Holter Monitoring Necessary? J Curr Pediatr 2016;14:124-8.
14. Balestra E, Bobbo M, Cittar M, Chicco D, D'Agata Mottolese B, Barbi E. Congenital Long QT Syndrome in Children and Adolescents: A General Overview. Children (Basel). 2024 May 11;11(5):582.