Neurology Asia 2022; 27(2) : 309 – 315

Transient loss of consciousness in children: Syncope or epileptic seizure?

Abdullah Taşdelen, Arzu Ekici

University of Health Sciences, Bursa Yüksek İhtisas Training and Research Hospital, Department of Pediatrics, Bursa, Turkey

Abstract

Background & Objective: Transient loss of consciousness (TLOC) is often benign from syncope but may be due to epileptic seizure. In this study, we aimed to evaluate the clinical differences between TLOC from epileptic seizures and syncope. *Methods:* The medical records of 300 children with TLOC were analyzed retrospectively. Age, sex, clinical symptoms, trigger factors, laboratory findings, ECG, ECHO, EEG and neuroimaging findings, as well as the causes of TLOC were reviewed. Results: Out of the 300 children admitted with TLOC, 288 (96%) were diagnosed as syncope. Of these patients, 231 (77%) were diagnosed as vasovagal syncope, 19 (6.3%) as orthostatic hypotension, and 38 (12.7%) as unclassifiable syncope. Twelve (4.2%) patients were diagnosed with epilepsy. Patients with epilepsy showed significantly higher rates of collapse and eye deviation during TLOC, as well as occurring in sitting and supine position (p=0.012, p=0.039, p=0.03 respectively).
Conclusion: For a differential diagnosis, TLOC accompanied by collapse, eye deviation and occurrence in sitting or supine position are suggestive of epilepsy.

Keywords: epileptic seizure, orthostatic hypotension, syncope, transient loss of consciousness, vasovagal syncope

INTRODUCTION

A common complaint of admission to pediatric emergency and neurology outpatient clinics is transient loss of consciousness (TLOC), which is characterized by amnesia for the duration of unconsciousness, impaired motor control, and lack of responsiveness.[1] TLOC is often benign; however, it may also be from epileptic seizure or cardiac dysfunction.

Syncope is a brief loss of consciousness caused by global cerebral hypoperfusion, accompanied by a loss of postural tone and complete spontaneous recovery.[2,3] Children's syncope was found to be mostly caused by a vasovagal event, at a rate of 61 to 80%.[4] The International League Against Epilepsy (ILAE) defines epileptic seizure as a brief occurrence of signs and/or symptoms caused by abnormally excessive or synchronized neuronal activity in the brain.[5] The most perplexing signs in the differential diagnosis of syncope and seizures are myoclonic jerks and tonic spasms. Asystole can occur in epileptic seizures, albeit it is uncommon.[6] Conversely, muscle contractions due to diminished cerebral perfusion can also be seen in prolonged syncope.[7] According to recent studies, approximately 20% of children who present with syncope are diagnosed with epilepsy.[8] Twenty-five percent of patients who were treated for epilepsy, were misdiagnosed.[9]

The distinction between syncope and epileptic seizures is critical, although not always easy. In this study, we aimed to evaluate the clinical differences between epileptic seizures and syncope associated with TLOC.

METHODS

This study included 300 patients aged 6-18, who presented with the complaint of TLOC to the pediatric neurology clinic of Health Sciences University, Bursa Yuksek Ihtisas Training and Research Hospital between 2016 and 2019. Patients with chronic disease younger than 6 years old were excluded from the study. Age, gender, pre-syncope and accompanying symptoms, pre-syncope positions, syncope-triggering variables, systemic and neurological results, and sitting and

Address correspondence to: Arzu Ekici, MD. University of Health Sciences, Bursa Yüksek İhtisas Training and Research Hospital, Department of Pediatrics, Bursa, Mimar Sinan Avenue, Yıldırım/Bursa/Turkey. Tel: +90224 295500/3106, email: drarzuekici@gmail.com

Date of Submission: 17 May 2021; Date of Acceptance: 12 April 2022

https://doi.org/10.54029/2022nfr

standing blood pressure measures were among the assessment parameters. The results of a complete blood count (CBC), biochemical testing (serum glucose, electrolytes, liver and kidney function tests), and serum vitamin B12 and folic acid, ECG, echocardiogram (ECHO), EEG, and neuroimaging were documented. The diagnosis of anemia was based on the WHO hemoglobin reference ranges, vitamin B12 and folate levels of less than 200 pg/ml and 3 ng/ml were defined as deficient.

Syncope is defined as a sudden loss of consciousness and postural tone that resolves spontaneously.[2-3] Cardiac syncope (CS), vasovagal syncope (VVS), orthostatic syncope (OS), and unclassifiable syncope were the forms of syncope differentiated. VVS patients were those who experienced a syncope attack while in upright posture, lost consciousness for less than a minute, had pre-syncope variables (e.g., hunger, tension,), dizziness, and blackout during syncope. Orthostatic hypotension (OH) patients had a decrease in SBP of at least 20 mm Hg and a decrease in DBP of at least 10 mm Hg as compared to standing blood pressure when sitting. Obstructive heart diseases (aortic stenosis and hypertrophic cardiomyopathy.) and arrhythmias (e.g., long QT syndrome, SVT) were accepted as causes of cardiac syncope. Patients who could not be classified by other types of syncope were classified in the group of unclassifiable syncope. In patients who had epileptiform abnormalities in the first EEG, a follow up EEG was performed. Epileptic seizures were diagnosed in patients who had epileptiform discharge in their EEG and continued to experience episodes of TLOC with epileptic seizure characteristics. In the follow-up, individuals with demyelinating plaques on cranial MRI and lesions that met temporal spread on MRI according to 2010 Mc Donald MS criteria were diagnosed with multiple sclerosis (MS).[10]

Statistical analysis

Statistical analyses were performed with the Statistical Package for the Social Sciences (SPSS) version 22.0. Qualitative data were expressed as frequencies and percentages. However, quantitative data were expressed as arithmetic mean, standard deviation, minimum and maximum values. The normality distribution of the data was confirmed with the Kolmogorov–Smirnov test. Pearson's chi-square test or Fisher's exact test was used to compare qualitative data. The Student's t-test and Mann-Whitney U test were used, respectively, for the comparison of quantitative data and for normally distributed and non-normally distributed data consisting of two independent groups. Pearson's correlation coefficient was used to evaluate the relationship between continuous variables. All statistical analyses were performed at a 95% confidence interval, at a significance level of $p < 0.05$.

RESULTS

A total of 300 patients who presented with TLOC were identified and included in this study. Of these, 116 (38.7%) were male and 184 (61.3%) were female (male/female ratio 0.63). At the time of presentation, the mean age was 13.20 ± 3.05 years (6-18 years). One hundred and forty (46.7%) patients presented to the pediatric neurology outpatient clinic after their first TLOC attack, and 160 (53.3%) visited after two or more attacks. The average number of previous episodes of syncope at the time of presentation was 1.99 ± 1.22 (1-5). The duration of unconsciousness was <1 minute in 227 (92.3%) patients, between 1 and 5 minutes in 17 (5.7%) patients, and >5 minutes in 6 (2%) patients. During TLOC, 268 (89.3%) of the patients were standing, 24 (8.0%) of the patients were sitting, and 8 (2.7%) of the patients were supine. Hunger was the most common triggering cause in 94 (31.3%) of patients, followed by stress in 24 (8%). There were 145 patients (48.3%) who had no TLOC trigger. Prior to TLOC, 256 patients (85.3%) had dizziness, 228 (75.7%) experienced blackout, 16 (5.3%) experienced perspiration, 9 (3%) experienced palpitations, and 1 (0.3%) experienced flashing light. TLOC symptoms included collapse in 25 (8.3%), muscle contractions in 24 (8%), pallor in 19 (6.3%), cyanosis in 10 (3.3%), urine incontinence in 9 (3%), eye deviation in 8 (2.7%), and locking of jaw in 3 (1%) of the patients. Table 1 show position during TLOC, triggering events, and pre- and post-TLOC findings.

All of the patients' serum electrolyte levels, liver, and kidney function tests were normal. Fifty-six patients (18.7%) were anemic, with 5 (8.9%) having megaloblastic anemia and 51 (91.1%) having iron deficiency anemia (IDA). Blood vitamin B12 levels were measured in 289 (96.3%) of the patients, with low serum vitamin B12 levels found in 25 patients (8.7%). The mean serum vitamin B12 level was 342.29± 37.98 pg/ml (116-1023pg/ml). Serum folic acid levels were measured in 234 (78%) of the patients, with folic acid deficiency found in 6 (2.6%) patients. The mean serum folic acid level was 7.62±3.36 ng/ml (1.7-24ng/ml).

Table 1: Position during transient loss of con-sciousness, triggering factors, pre- and during transient loss of consciousness findings

Variables	n=300	%
Position during TLOC		
Standing	268	89.3
Sitting	24	8.0
Supine	8	2.7
Triggering factors		
None	145	48.3
Menstrual cycle	7	2.3
Fasting	94	31.3
Blood donation	5	1.7
Stress	24	8.0
Hot environment	23	7.7
Circumcision	2	0.7
Pre-TLOC findings		
Dizziness	256	85.3
Grey out	228	75.7
Perspiration	16	5.3
Palpitation	9	3
Photopsia	1	0.3
Findings during TLOC		
Collapse	25	8.3
Muscle contractions	24	8
Pallor	19	6.3
Cyanosis	10	3.3
Urinary Incontinence	9	3
Eye deviation	8	2.7
Locking of teeth	3	1

TLOC: transient loss of consciousness

Nineteen patients (6.3%) had a reduction of at least 20 mm Hg in SBP and of at least 10 mm Hg in DBP while standing up compared to the sitting position with demonstrable OH. All of the patients had ECGs, and 298 (99.3%) exhibited a normal ECG. Two patients (0.07%) had sinus tachycardia. In 127 (42.3%) of the patients, an ECHO was performed, and abnormal results were found in 38 (30%). The most common findings of the patients were secundum ASD (11%), mild mitral insufficiency (8.7%), and mitral valve prolapse (5.6%). (Table 2). ECG and ECHO findings were thought to be not linked to cardiac syncope in any of the patients. Abnormal results were found in 60 (22.4%) of the 254 (84.7%) cranial MRIs. Arachnoid cyst (6.7%) and pineal gland cyst (5.9%) were the most common findings (Table 2). Dizziness and syncope were reported by four (1.6%) patients with demyelinating plaques on the cranial MRI. In later follow-ups, three of these patients (75%) were diagnosed with multiple sclerosis (MS) due to new lesions on the MRI that satisfied the time component of the 2010 Mc Donald MS criteria. VVS was diagnosed in these three patients. Cranial MRI findings were thought to be not associated with syncope except for these three patients.

An EEG was performed in 289 (96.3%) of the patients. The EEG was evaluated as abnormal in 17 (5.9%) patients. All these 17 patients had a follow up EEG. On follow up, the EEG was normal in 5 of the patients, and epileptiform discharges were detected in 12. Epilepsy was diagnosed in 12 (4.2%) patients who had recurring attacks and had persistent epileptic discharges on their EEG. Of the 300 children admitted with TLOC, 288 (96%) were diagnosed with syncope, and 12 (4%) with epileptic seizures. Two-thirds of the patients (77%) had VVS, 19 (6.3%) had OH, and 38 (12.7%) had unclassifiable syncope (Figure 1). However, none of the patients had cardiac syncope.

Table 2: Results of ECHO cardiography and cranial MRI findings

Variables	n	%
ECHO cardiography findings (n=127)		
Normal	89	70.1
Secundum ASD	14	11
Minimal mitral insufficiency	11	8.7
Mitral valve prolapse	7	5.6
Patent foramen ovale	6	4.7
Primum ASD	1	0.8
Minimal aortic insufficiency	1	0.8
Cranial MRI findings* (n=254)		
Normal	198	78
Arachnoid Cyst	18	6.9
Pineal Gland Cyst	16	6.1
Non-Specific Gliotic Foci	9	3.5
Demyelizing Plate	4	1.6
Cavum Septum Pellucidum	4	1.6
Choroid Plexus Cyst	2	0.8
Pituitary Microadenoma	1	0.4
Venous Angioma	1	0.4
Suprapineal Lipoma	1	0.4

*There was more than one finding in some (2) of the patients.

In terms of gender and age, there was no statistically significant difference between patients diagnosed with epilepsy (p> 0.05). Table 3 shows the symptoms before and during TLOC, the body position during TLOC, and the duration of unconsciousness in these patients.

On statistical analysis comparing patients diagnosed with epilepsy and the others, dizziness before TLOC was less common (p = 0.019). During TLOC, patients with epilepsy showed higher rates of collapse (p = 0.012), and eye deviation (p=0.039). Muscle contractions were reported by six (50%) of the 12 patients diagnosed with epilepsy during TLOC (p<0.001). TCOC was more common to occur in epilepsy patients in the sitting and supine positions (p=0.03). The duration of loss of consciousness was between 1 and 5 minutes in 4 (33.3%) of the epilepsy patients (p = 0.006).

DISCUSSION

TLOC, which causes great concern in families, is one of the most common reasons for referral to

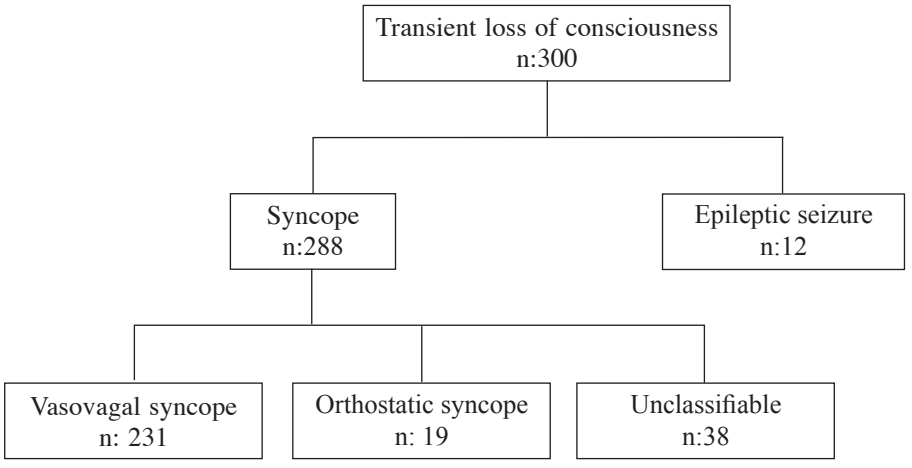

Figure 1: Causes of temporary loss of consciousness

Table 3: Findings of transient loss of consciousness prior and during the event, position and duration of unconsciousness with and without epilepsy

Variables	Epilepsy (n=289)*				p
	Yes (n=12)		No (n=277)		
	n	%	n	%	
Pre-TLOC findings					
Dizziness	7	58.3	240	86.6	0.019
Grey out	7	58.3	215	77.6	0.157
Perspiration	1	8.3	14	5.1	0.479
Palpitation	-	-	8	2.9	0.999
Photopsia	-	-	1	0.4	-
Findings during TLOC					
Collapse	4	33.3	20	7.2	0.012
Muscle contractions	6	50	18	6.5	<0.001
Pallor	-	-	19	6.9	0.999
Cyanosis	2	16.7	8	2.9	0.059
Urinary Incontinence	-	-	9	3.2	0.999
Eye deviation	2	16.7	6	2.2	0.039
Locking in teeth	-	-	3	1.1	0.999
Position during TLOC					
Standing	8	66.7	250	90.3	
Sitting	3	25.0	20	7.2	0.030
Supine	1	8.3	7	2.5	
Duration of unconsciousness					
<1 minute	8	66.7	259	93.5	0.006
1-5 minutes	4	33.3	12	4.3	
>5 minute	-	-	6	2.2	

*One hundred and eighty-nine patients with EEG were evaluated.
TLOC: transient loss of consciousness

pediatric neurology outpatient clinics. It is often benign, but may be a symptom of an epileptic seizure or rarely of various heart diseases. A detailed evaluation is required to work out the diagnosis in a patient who present with TLOC. Syncope can strike anyone at any age; however, it is more common among adolescent girls. Zhang *et al*.[11] reported an average age of 12 ± 3 years for syncope onset. In this study, the mean age of onset was 13.2 ± 3.05 years, with a male/female ratio of 0.63.

Taking a detailed history is of great importance in the evaluation of children presenting with TLOC. Syncope episodes are often brief, lasting only a few minutes at most. The prolongation of the episode may make it difficult to distinguish it from other causes.[12] Dizziness, greying out, nausea, sweating, tinnitus, palpitations, and weakness are all prodromal symptoms of syncope. Fasting, fatigue, stress, being in an extremely hot and stuffy environment, prolonged standing, standing up suddenly, intravenous drug administration or blood donation, receiving or donating blood, painful interventions, circumcision, and menstrual periods are all common syncope-stimulating factors. Syncope can be accompanied by muscle contractions, cyanosis, urinary incontinence, weakness, and nausea.[13] Asymmetric generalized, tonic, or clonic movements might occur as a result of decreased cerebral perfusion during syncope. However differenting from other diagnosis may be difficult because severe VVS can be accompanied by epilepsy-like tonic-clonic contractions.[7] Dizziness and greying out were the most common prodromal signs of syncope in a study by Yılmaz *et al*.[14] At the time of their syncope episode, 69.3% of the patients were standing. Fasting, excitement, and fear are the most prevalent causes of syncope.[14] In this study, the majority of patients had a TLOC episode that lasted less than one minute (92.3%). Dizziness was the most common prodromal symptom (85.3%), followed by greying out (75.7%).

An ECG, CBC, and biochemical analysis should be performed in all patients.[15,16] Yılmaz *et al*.[17] reported that none of the patients in their study had hypoglycemia or an electrolyte abnormality, but 11.5% had anemia. In this study, 18.7% of the patients had anemia, 8.7% patients had vitamin B12 deficiency, and 2.6% patients had folic acid deficiency. According to some report, there is an association between vitamin B12 deficiency and syncope.[18] In Turkey, anemia and vitamin B12 deficiency are widespread.[19] In a study conducted in Turkey involving children aged 1-18 years, anemia was found at a rate of 18.8% and vitamin B12 deficiency at a rate of 16.9%.[20] As a result, it is not possible to link TLOC with anemia and vitamin B12 insufficiency.

In children with TLOC, cranial neuroimaging is not suggested as a routine assessment unless there is evidence of increased intracranial pressure or new focal impairments.[21] A cranial MRI was performed on 254 (84.7%) of the patients in this study, and normal results were noted in 77.6%. Arachnoid cysts (6.7%) and pineal gland cysts were the most common findings (5.9%). Syncope was not linked to images other than demyelinating plaques (n:4, 1.6%). Dizziness was experienced by three patients with demyelinating plaques. These three patients were diagnosed with MS during follow-up. It is possible that the association of syncope with MS is coincidental. However, autonomic dysfunction is common in MS patients. Furthermore, in MS, studies have found postural orthostatic tachycardia syndrome linked to autonomic dysfunction, as well as episodes of syncope linked to orthostatic hypotension or cervical cord involvement.[22]

Cardiac syncope in children should be distinguished from other causes, as although it is infrequent, it can be life-threatening. The frequency of cardiogenic syncope in children has been reported to be 3 to 5%.[11,23] Conditions like MVP, ASD, and PDA have been identified as underlying causes of cardiac syncope in some investigations. Despite the significant prevalence of syncope in patients with MVP, it is believed that MVP is not the direct cause of syncope.[24] In this study, we performed an ECHO in 42.3% of the patients, normal outcomes was seen in 70.1%. Secundum ASD (11%) and mild mitral insufficiency (11%) were the most common findings (8.7%). Two (0.07%) patients had sinus tachycardia, which was observed on the ECG. In none of the patient did an ECHO or an EKG confirm cardiac syncope. However, we did not perform further cardiac examinations such as tilt table test and Holter monitoring.

In our study, 288 cases were classified as syncope (96%) and twelve (4%) as epileptic seizures. Of the patients with syncope, 231 (77%) had VVS, 19 (6.3%) had OH, and 38 (12.7%) had unclassifiable syncope. The number of unidentified syncope may be high because the study was retrospective and psychogenic syncope was not examined. Epilepsy is one of the important causes of TLOC in children. Parents are anxious about the TLOC episode, especially because of the possibility of an epileptic seizures. Aura, tonic-clonic contractions, involuntary movements (mainly myoclonic jerks), fecal and urine incontinence, episodes of syncope in supine position, extended loss of consciousness, postictal confusion, and delayed recovery of consciousness should all be suggestive of epileptic seizures. Syncope rarely causes extended limb jerks or postictal disorientation. Syncope may however, cause brief myoclonic contractions from cerebral ischemia.[7] Syncope is frequently associated with symptoms such as prodromal diaphoresis, palpitations, or provocation by prolonged sitting or standing.[25]

In a report by Yılmaz *et al*.[17], EEG examination was performed in 65.1% of the patients who presented with syncope During the follow-up period, the authors reported epileptic activity in 15.9% of the patients and commenced therapy with a diagnosis of epilepsy in 14%. In another study, epileptiform abnormalities on the EEG were found in 9.7% of syncope patients.[26] In this study, EEG was performed in 96.3% of the patients; it was found to be abnormal in 17 (5.9%). The presence of epileptic discharge on the EEGs and recurrent episodes of TLOC accompanied with epileptic seizure findings led to the diagnosis of epilepsy.

In conclusion, it can be challenging to determine the etiology when a child presents with TLOC. For differential diagnosis between epilepsy and syncope, a thorough history-taking is required. To distinguish epileptic seizures from syncope, attacks in the sitting or supine position, symptoms of muscle contractions and eye deviation are helpful. EEG should also be performed to look for epileptiform abnormalities. On the other hand, laboratory studies and neuroimaging are often only useful in excluding significant underlying illness.

The study's limitations are first, it is a single-center retrospective study, and no follow-up studies such as Tilt Table Test, Holter ECG, and video EEG were performed. Psychogenic syncope was also not evaluated.

DISCLOSURE

Ethics approval: The study protocol was approved by the Institutional Ethics Committee with the decision number of 2011-KAEK-25 2019/06-19.

Financial support: None

Conflict of interest: None

REFERENCES

1. Brignole M, Moya A, de Lange FJ, *et al*. 2018 ESC guidelines for the diagnosis and management of syncope. *Eur Heart J* 2018; 39:1883e948. doi: 10.1093/eurheartj/ehy037.
2. Sabri M, Mahmodian T, Sadri H. Usefulness of the head-up tilt test in distinguishing neurally mediated syncope and epilepsy in children aged 5–20 years old. *Pediatr Cardiol* 2006; 27(5):600-3. doi: 10.1007/s00246-006-1140-7.
3. Hadjikoutis S, O'Callaghan P, Smith PE. The investigation of syncope. *Seizure* 2004; 13(8):537-48. doi: 10.1016/j.seizure.2003.12.011.
4. Johnsrude C. Current approach to pediatric syncope. *Pediatr Cardiol* 2000; 21(6):522-31. doi: 10.1007/s002460010130.
5. Blume WT, Lüders HO, Mizrahi E, Tassinari C, van Emde Boas W, Engel J Jr. Glossary of descriptive terminology for ictal semiology: report of the ILAE task force on classification and terminology. *Epilepsia* 2001; 42(9):1212-8. doi: 10.1046/j.1528-1157.2001.22001.x.
6. Rugg-Runn FJ, Simister RJ, Squirrell M, Holdright DR, Duncan JS. Cardiac arrhythmias in focal epilepsy: a prospective longterm study. *Lancet* 2004;364:2212-9. doi: 10.1016/S0140-6736(04)17594-6.
7. Wolfe D, Grubb B, Kimmel S. Head-upright tilt test: a new method of evaluating syncope. *Am Fam Physician* 1993; 47(1):149-59. PMID: 8418578
8. Zaidi A, Clough P, Cooper P, Scheepers B, Fitzpatrick AP. Misdiagnosis of epilepsy: many seizure-like attacks have a cardiovascular cause. *J Am Coll Cardiol* 2000; 36(1):181-4. doi: 10.1016/s0735-1097(00)00700-2.
9. Brodie M, Shorvon S, Canger R, *et al*. Commission on European Affairs: appropriate standards of epilepsy care across Europe. *Epilepsia* 1997; 38(11):1245-50. doi: 10.1111/j.1528-1157.1997.tb01224.x.
10. Sadaka Y, Verhey LH, Shroff MM, *et al*. 2010 McDonald criteria for diagnosing pediatric multiple sclerosis. *Ann Neurol* 2012; 72(2):211-23. doi: 10.1002/ana.23575.
11. Zhang Q, Du J, Wang C, Du Z, Wang L, Tang C. The diagnostic protocol in children and adolescents with syncope: a multicentre prospective study. *Acta Pædiatr* 2009; 98(5):879-84. doi: 10.1111/j.1651-2227.2008.01195.x.
12. Hoefnagels W, Padberg G, Overweg J, Van der Velde E, Roos R. Transient loss of consciousness: the value of the history for distinguishing seizure from syncope. *J Neurol* 1991; 238(1):39-43. doi: 10.1007/BF00319709.
13. Brignole M. Task Force on Syncope, European Society of Cardiology, Guidelines on management of syncope-update 2004. *Europace* 2004; 6:467-537. doi:10.1016/j.eupc.2004.08.008.
14. Yılmaz ST, Binnetoğlu K, Babaoğlu K, Altun G. Predictors of vasovagal syncope recurrence in children and adolescents and value of head-up tilt table test. *Anadolu Kardiyol Derg* 2013; 13:688-94. doi: 10.5152/akd.2013.194.
15. Redd C, Thomas C, Willis M, Amos M, Anderson J. Cost of unnecessary testing in the evaluation of pediatric syncope. Pediatr Cardiol 2017; 38(6):1115-22. doi: 10.1007/s00246-017-1625-6.
16. Anderson JB, Willis M, Lancaster H, Leonard K, Thomas C. The evaluation and management of pediatric syncope. *Pediatr Neurol* 2016; 55:6-13. doi: 10.1016/j.pediatrneurol.2015.10.018.
17. Yılmaz Ü, Özdemir R, Katipoğlu N, Tuba D, Berksoy EA, Timur M. Çocuklarda Senkop Etiyolojisi Nörolojik ve Kardiyolojik İncelemelerin Tanıdaki Değeri. *Türkiye Çocuk Hast Derg* 2014; 8(2):64-70.
18. Ganjehei L, Massumi A, Razavi M, Wilson JM. Orthostatic hypotension as a manifestation of vitamin B12 deficiency. *Tex Heart Inst J* 2012; 39(5):722-3. PMID: 23109778
19. Koc A, Kocyigit A, Soran M, *et al*. High frequency of maternal vitamin B12 deficiency as an important cause of infantile vitamin B12 deficiency in Sanliurfa province of Turkey. *Eur J Nutr* 2006; 45:291-7. doi: 10.1007/s00394-006-0598-7.
20. Çolak A, Akşit MZ, Şimşek N, Bilgi P, Anıl M. Iron, folate and vitamin B12 status of children and adolescents: Single center study in the Aegean region. *İzmir Dr. Behçet Uz Çocuk Hast Derg* 2019; 9(3):183-190. doi:10.5222/buchd.2019.21704.
21. Martin K, Bates G, Whitehouse WP. Transient loss of consciousness and syncope in children and young people: what you need to know. *Arch Dis Child Educ Pract Ed* 2010; 95(3):66-72. doi: 10.1136/adc.2007.121103.
22. Kanjwal K, Karabin B, Kanjwal Y, Grubb BP. Autonomic dysfunction presenting as postural orthostatic tachycardia syndrome in patients with multiple sclerosis. *Int J Med Sci* 2010; 7(2):62-7. doi: 10.7150/ijms.7.62.
23. Steinberg LA, Knilans TK. Syncope in children: diagnostic tests have a high cost and low yield. *J Pediatr* 2005; 146(3):355-8. doi: 10.1016/j.jpeds.2004.10.039.
24. Ritter S, Tani LY, Etheridge SP, Williams RV, Craig JE, Minich LL. What is the yield of screening ECHO cardiography in pediatric syncope? *Pediatrics* 2000; 105(5):e58. doi: 10.1542/peds.105.5.e58.
25. Sheldon R, Rose S, Ritchie D, *et al*. Historical criteria that distinguish syncope from seizures. *J Am Coll Cardiol* 2002; 40:142-8. doi: 10.1016/s0735-1097(02)01940-x.
26. Nesrin C, Ummu A, Zeynep Selen K, Birce Dilge T, Cahide Y. Retrospective evaluation of patients admitted with complaints of syncope. *J Reprod Health Contracep* 2016; 1(3):15.