RESEARCH PAPER

Clinico-Etiological Profile of Pediatric Syncope: A Single Center Experience

Sweta Mohanty,[1] C P Ravi Kumar[2] and Sowmyashree Mayur Kaku[3]

From Department of [1]Pediatric Cardiology, [2]Neurology, and [3]Clinical Neurosciences and Child and Adolescent Psychiatry, Aster CMI Hospital, Bengaluru, Karnataka, India.

Correspondence to: Dr Sweta Mohanty, Valmark Apas, Bangalore 560 076, Karnataka, India. doctorsweta@yahoo.com
Received: July 23, 2020; Initial review: August 31, 2020; Accepted: November 17, 2020.

Objective: To describe the clinical profile of children with syncope. **Methods**: Hospital records were reviewed for clinical and laboratory details of childrenpresenting withreal or apparent syncope. Five diagnostic categories were identified: neurocardiogenic syncope (NCS), psychogenic pseudosyncope (PPS), cardiac, neurological and indeterminate. **Results**: 30 children (aged 4 to 17 years)were included. The commonest cause of syncope was NCS (63.3%), followed by PPS (13.3%), cardiac (10%), neurological (10%) and indeterminate (3.3%). Exercise, loud noise or emotional triggers and family history were associated with cardiac etiology, and electrocardiogram (ECG) was diagnostic in the majority. Children with PPS and cardiacsyncope had frequent episodes when compared with other groups. Indiscriminate antiepileptic use was found in 5 children, including two cardiac cases.**Conclusion**: Frequent recurrences of syncope may suggest PPS or cardiac cause. Cardiac etiology may be readily identified on history and ECG alone.

Keywords: *Neurocardiogenic syncope, Psychogenic pseudosyncope, Head-up tilt table test, Management.*

Syncope is defined as a transient loss of consciousness due to transient global cerebral hypo-perfusion, and is characterized by rapid onset, short duration and spontaneous complete recovery [1]. Syncope is commonest in adolescent age group with a peak in incidence between 15 to 19 years [2].

Various classification systems broadly categorize syncope as neurocardiogenic (NCS), cardiac, neurologic, or psychogenic pseudosyncope (PPS) [2],the commonest cause being benign NCS [2,3]. Although cardiac causes of syncope are rare, they can be potentially life-threatening [4]. Even a benign syncopal event can generate extreme anxiety. As a result, syncope evaluation often leads to a battery of expensive low-yield tests [4].

We performed this study to document common etiologies and identify clinical features that may assist in differentiating between various diagnostic categories.

METHODS

We reviewed records of patients aged 1 to 18 years presenting to our center with syncope, over a 14-month period from January, 2019 to February, 2020. Outpatient visits as well as inpatient hospitalizations of the first presentations were included. For the purpose of this study, syncope was defined as a sudden and transient (<2 hours) loss of consciousness and postural tone, with spontaneous recovery [5,6]. Children with dizziness without loss of consciousness were excluded. Those with clinical presentation of seizure, focal neurological deficit or established causes of pathological syncope like cardiac disorder or trauma were excluded. The electronic medical records were retrospectively reviewed for demographic and clinical data and results of electrocardiography (ECG) or any additional testing.

Patients were classified into five diagnostic categories [1,2] viz., neurocardiogenic syncope (NCS), cardiac syncope, psychogenic pseudosyncope (PPS), neurological disorder and indeterminate cause. NCS was diagnosed by typical history, such as precipitating factors or prodromal symptoms, with supportive evidence on orthostatic vital testing or Head-up tilt table (HUTT) test in some cases. A confirmatory diagnosis of cardiac syncope was made based on ECG abnormalities and supplemental tests such as exercise stress test or 24-hour ECG monitoring (Holter). All cases of neurological disorder had abnormal electroencephalogram (EEG) with or without abnormal neuroimaging findings. Syncope was classified as indeterminate in absence of a clearly definable cause for an objective clinical manifestation. A diagnosis of PPS was made after the exclusion of other causes, and evaluation by a child psychiatrist.

Our pediatric syncope team follows a standardized clinical assessment and management plan to evaluate syncope patients. A standard 12-lead ECG is done in all

patients. Orthostatic vital sign testing in the clinic is considered positive when there is a drop in systolic blood pressure of greater than 20 mm Hg [7] or a rise in heart rate of more than 40 beats per minute on standing for 3 minutes [4]. Standard views for echocardiograms and standardized protocols for HUTT[8] and exercise stress testing[9] are followed. Holter monitoring consists of digital recording over 24 hours, analyzed using Digitrack (GE) software system with manual reviewing of all data. Psychological evaluation is done by the child psychiatrist, as indicated.

RESULTS

A total of 30 patients, aged 4 to 17 years, presented with syncope. Seven of these patients required hospitalization; the remaining were evaluated and managed on outpatient basis. Of the 30 patients, 19 (63.3%) were diagnosed to have NCS, 4 (13.3%) had PPS, 3 (10%) had a cardiac cause, 3 (10%) had a neurological cause and 1 (3.3%) was of indeterminate etiology. Two patients with NCS had convulsive syncope, where tonic clonic movements were observed following the loss of consciousness (EEG was normal in both).

Sixteen (53.3%) children had a history of recurrent episodes of syncope. All cases of PPS presented with multiple episodes in a week, with complete disappearance during hospital observation. Postural changes (15, 84%) or accompanying acute febrile illnesses (6, 31%) were the predominant precipitating factors for NCS (**Table I**). Exercise was precipitating event for a child with NCS, but syncope in this case was post-exertional (occurring a minute after cessation of exercise). Only two (10.5%) patients of NCS had a positive orthostatic exam.

Web Table I shows the investigations performed with their diagnostic yield. ECG was performed in all children and revealed a cardiac diagnosis in three children (long QT syndrome, 2; sinus node dysfunction, 1). One child with sinus node dysfunction had significantbradycardia onholtermonitoring andrequired electrophysiology referral. EEG confirmed a diagnosis of idiopathic epilepsy in three patients, all of whom were started on antiepileptic medications. Lastly, a child with syncope following epistaxis,with similar parental history, was classified as indeterminate, after baseline investigations, including holter monitoring, revealed no abnormality.

All children with NCS were reassured, advised to increase fluid and salt intake and advised behavioral modifications on experiencing prodromal symptoms. The two siblings with long QT syndrome were started on beta-blockers and showed no recurrence on follow up at 3 months. For children with PPS, underlying stressors were identified, and two cases required psychotropic medications.Unindicated antiepileptic medications were being administered in five patients, with recurrent episodes (including children with cardiac syncope), and were discontinued.

Table I Demographic Features and Precipitating Events in Different Etiological Categories of Pediatric Syncope (*N*=30)

	Cardiac (n=3)	*NCS (n=19)*	*PPS (n=4)*	*Neurologic (n=3)*
Age (y)[a]	13.7 (1.1)	10.3 (3.5)	12.5 (1.7)	10 (2.6)
Females	1	9	1	2
Recurrent syncope	2	8	4	2
Family history	2	0	0	0
Precipitating event				
Exercise	2	1	0	0
Loud noise/emotion	1	0	0	0
Postural factors	1	15	3	2
Fever/acute illness	0	6	0	0
Accompanying symptoms				
Nausea/sweating	0	3	0	1
Palpitation/chest pain	1	0	1	0
Headache	1	2	3	2
Visual change	0	2	2	0
Injury during fall	0	2	1	0
Urination	0	0	0	1
Vomiting on awakening	1	3	0	1

NCS: Neurocardiogenic syncope; PPS: Psychogenic pseudosyncope; Values in numbers except [a]mean (SD).

DISCUSSION

In this retrospective study, the commonest diagnosis in children presenting with syncopewasneurocardiogenic, followed by psychogenic pseudosyncope. Specific features on history that suggested a cardiac etiology, as reported previously [1,10], included syncope associated with exercise, loud noise or fright, and syncope preceded by chest pain or palpitations in the absence of prodromal symptoms.

The frequency of episodes tends to be significantly higher in cardiac causes as compared to non-cardiac [11], with the exception of PPS. In a patient with unusual loss of consciousness occurring multiple times per day, unrelated to posture, with varying presentations, or with events lasting longer than 3 minutes, conversion disorder should be considered [7].

ECG is an essential component of evaluation of all children who present with syncope [5] and clinched the

WHAT THIS STUDY ADDS?

- Detailed history of precipitating factors and accompanying symptoms and a baseline electrocardiogram helps identify the cause of syncope in most cases.

diagnosis for the cardiac cases in this cohort. Echocardiogramwas found to be non-contributory, as also reported earlier [12]. HUTT is useful to differentiate NCS from PPS by the reproduction of symptoms during tilt testing in the absence of haemodynamic abnormalities in the latter [13]. However, as HUTT is time-consuming and not without risk [7], we selectively advised the test only when it was expected to bring about a change in management. EEG may be helpful in differentiating convulsive syncope, wherein extremity jerking usually occurs after loss of consciousness, from myoclonic jerks. It is common for syncope to be misdiagnosed and erroneously treated as an epileptic condition [14].

This study has the limitation of being a retrospective study, with a small sample size. However, our findings underscore that a detailed history is of paramount importance in making the diagnosis in syncope, and cases of syncope need an ECG to rule out potentially life-threatening cardiac causes.

Ethics clearance: Institutional ethics committee; Aster CMI Hospital; No IEC/033/2019-20, dated March 16, 2019.
Contributors: SM: drafting of manuscript, analysis of data, review of literature; RK: acquisition and interpretation of data, final approval of paper; SMK: interpretation of data, critical revision of paper.
Funding: None; *Competing interests*: None stated.

REFERENCES

1. Courtheix M, Jalal Z, Bordachar P, et al. Syncope unit in the paediatric population: A single-centre experience. Arch Cardiovasc Dis. 2016;109:199-206.
2. Friedman KG, Alexander ME. Chest pain and syncope in children: A practical approach to the diagnosis of cardiac disease. J Pediatr. 2013;163:896-901.
3. McLeod KA. Syncope in childhood.Arch Dis Child. 2003;88:350-53.
4. Paris Y, Toro-Salazar OH, Gauthier NS, et al. Regional implementation of a pediatric cardiology syncope algorithm using standardized clinical assessment and management plans (SCAMPS) methodology. J Am Heart Assoc. 2016;5:e002931.
5. Massin MM, Malekzadeh-Milani S, Benatar A. Cardiac syncope in pediatric patients. ClinCardiol. 2007;30:81-85.
6. Driscoll DJ, Jacobsen SJ, Porter CJ, Wollan PC. Syncope in children and adolescents. J Am CollCardiol. 1997;29:1039-45.
7. Anderson JB, Willis M, Lancaster H, Leonard K, Thomas C. The evaluation and management of pediatric syncope. Pediatr Neurol. 2016;55:6-13.
8. Grubb BP, Temesy-Armos P, Moore J, Wolfe D, Hahn H, Elliott L. The use of head-upright tilt table testing in the evaluation and management of syncope in children and adolescents. Pacing Clin Electrophysiol. 1992;15: 742-8.
9. Fletcher GF, Ades PA, Kligfield P, *et al*; American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee of the Council on Clinical Cardiology, Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular and Stroke Nursing, and Council on Epidemiology and Prevention. Exercise standards for testing and training: A Scientific Statement from the American Heart Association. Circulation. 2013;128(8):873-934.
10. Gupta A, Menoch M, Levasseur K, Gonzalez IE. Screening pediatric patients in new-onset syncope (SPINS) study. ClinPediatr (Phila). 2020;59:127-33.
11. Hegazy RA, Lofty WN, Ammar RI, Fattouh AM. Diagnostic dilemma of cardiac syncope in pediatric patients. Indian Pacing Electrophysiol J. 2008;8:22-31.
12. Ritter S, Tani LY, Etheridge SP, Williams RV, Craig JE, Minich LL. What is the yield of screening echocardiography in pediatric syncope? Pediatrics. 2000;105:E58.
13. Capitello TG, Placidi S, Di Mambro C, *et al*. Dysfunctional behaviors in children and adolescents with neurocardiogenic syncope. J Child AdolescBehav. 2014;2:4.
14. Viswanath D, Prabhuji MLV, Menon VV, Kailasam S, Kumar M. Syncope in children. J Indian Acad Oral Med Radiol. 2013;25;294-99.

Web Table I Diagnostic Tests Obtained in Evaluation of Syncopal Episodes (*N*=30)

Name of test	*Obtained in n (%)*	*Indication (n)*	*Abnormal result, n (%)*	*Assisted in management, n(%)*
ECG	30 (100)	All cases of syncope (30)	3 (10)	3 (10)
HUTT	5 (17)	Parental anxiety in suspected NCS (3) Suspected PPS (1) Suspected POTS (1)	2 (40)	4 (80)
Echo-cardiography	16 (53)	ECG abnormality (2) Past cardiac surgery (1) Injury during syncope (1) Required CPR (1) Parental anxiety (11)	0	0
Holter	6 (20)	Suspected LQTS (1) Sinus node dysfunction (1) Concerning family history (1) Frequent episodes in suspected PPS (2) Injury during syncope (1)	1 (17)	4 (66)
TMT	1 (3)	Suspected LQTS (1)	1 (100)	1 (100)
Genetic test	1 (3)	Suspected LQTS (1)	1 (100)	1 (100)
EEG	13 (43)	Suspected seizure (2) Unconfirmed diagnosis (11)	3 (23)	3 (23)
Neuro-imaging	7 (23)	Suspected seizure (1) At outside hospital (6)	1 (14)	1 (14)

HUTT: Head-up Tilt Table test; NCS: Neurocardiogenic syncope; PPS: Psychogenic pseudosyncope; POTS: Postural orthostatic tachycardia syndrome; Echo: Echocardiogram; CPR: Cardiopulmonary resuscitation; LQTS: Long QT syndrome; TMT: Treadmill exercise test.