Evaluation of children with syncope: a clinical audit at Assiut University Children Hospital

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Background
Syncope is one of the most prevalent paroxysmal disorders in children and adolescents. It is a transient, self-limited loss of conscious and postural tone that is followed by a full recovery spontaneously and without any neurological consequences.

Aim
The objective of this internal audit is to assess how much the staff at Assiut University Children Hospital (AUCH) are sticking to the agreed upon unit's protocol regarding how to deal with children or adolescent with syncopal attacks.

Patients and methods
This clinical audit was conducted on 150 pediatric patients with syncope who were admitted to Assiut University Children Hospital, Egypt during the period from the start of January 2021 up to the end of June 2022. Data was systematically collected regarding medical history, general, and systematic examination including chest, cardiac, abdominal, and neurological examination. Duration of syncopal attack (minutes), prodrome history, related symptoms (as chest pain or palpitations), and triggers (as hyperventilation, pain, stress, or effort) were all collected.

Results
The mean age of the studied participants was 11.96 ± 2.47 years (range 1.5–15 years), with slight female sex predominance. The neurocardiogenic causes (vasovagal, VVS) were the main cause for syncopal attacks among our studied cases, followed by neurologic causes in 17.3%. Collectively, we could say that our institution is successfully following the recommended protocol of our institution for the management of syncopal attacks in children and adolescents.

Conclusion
The recommended protocol of our institution for the management of syncopal attacks in children and adolescents was successfully followed by the staff members at Assiut University Children Hospital.

Keywords:
Assiut University Children Hospital, clinical audit, syncope

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Introduction

Syncope is a transient loss of consciousness accompanied by an inability to maintain a postural tone, which is followed by a rapid spontaneous recovery [1]. Most syncope victims are girls, and between 30 and 50% of youngsters experience it at least once before reaching adolescence. The syncope rate among 15 to 17-year-old adolescents is 9%, and it rises until the end of adolescence [2].

The prodrome is the most essential component of the history; warm or clammy feelings, nausea, dizziness, or visual changes (such as seeing spots, greying out, or tunneling) are all highly suggestive of vasovagal syndrome. Other symptoms include irritability, confusion, auditory changes, or dyspnea, and the lack of a prodrome raises the possibility that a cardiac cause may be the cause. Only 40% of children with cardiac conditions experienced prodromal symptoms, compared with 85% of children with vasovagal syncope. Pediatric cardiac causes of syncope have been linked to palpitations and chest discomfort [3].

The main goal of the current internal clinical audit study is to assess how much the staff in the Assiut University Children Hospital (AUCH) are sticking to the agreed-upon unit's protocol regarding how to deal with children or adolescents with syncopal attacks.

Patients and methods

One hundred and fifteen children and adolescents diagnosed with syncope were enrolled in this clinical audit. The study was conducted at AUCH in the period from the start of January 2021 up to the end of June

2022. The study adhered to the guidelines of Assiut University's Ethical Committee (IRB No: 17101444). All enrolled participants or their care givers were provided an informed written consent.

Eligible participants

All cases (of both sexes) who were diagnosed with syncope and admitted to AUCH during the study period were enrolled in this study. Patients or their care givers who refused to participate in this study were excluded.

All studied participants were subjected to the following checklist:

(1) Name.
(2) Age.
(3) Residency.
(4) Child born full term or preterm.
(5) Parental consanguinity.
(6) Family history.

Check list*	Yes	No	Comment
History			
Nausea			
Headache			
Greyout			
Tunneling			
Seeing spots			
Auditory changes			
Dyspnea			
Chest pain			
Body swelling			
Fatigue, Effort intolerance			
Duration of the attack			
Palpitation			
Cyanosis			
Emotional stress (fear-pain)			
Situational syncope: (sneeze, cough, laugh, swallowing, head turning, micturition defecation,)			
Mechanic carotid triggers			
Exercise			
Sudden change in position			
Foreign body inhalation			
Hyperventilation			
History of structural heart disease			
History of neurological disease: (cerebrovascular, increase intracranial pressure, migraine)			
Hypovolemia: (vomiting, diarrhea, bleeding)			
Hypoglycemia: (drugs as sulfonyl urea, insulin, sepsis, renal failure, liver disease)			
Psychological disease: (depression, panic attacks, anxiety, somatization)			
Family history of syncope			
Examination			
Heart Rate and rhythm			
Respiratory Rate			
Blood Pressure			
In supine position			
In sitting position			
Temperature			
Oxygen Saturation			
GCS			
Random blood glucose			
Chest			
Cardiac			
Neurological			
Abdominal			
Investigation			
CBC			
Kidney function tests			
Electrolytes			
ECG			
ECHO			
EEG			
CT			
Drug history			
Antihypertensive			
Diuretics			
Barbiturates			
Tricyclic antidepressant			
Alcohol			
Antiarrhythmics			
Macrolides			
Antihistamines			
Antipsychotics			
MAO inhibitors			
Levodopa			
Prazosin			
Benzodiazepines			

*This checklist was driven from the recent recommended guideline (2017 ACC/AHA/HRS Guideline for the Evaluation and Management of Patients With Syncope) [4]. ACC, American College of Cardiology; AHA, American Heart Association; HRS, Heart Rhythm Society, GCS, Glasgow coma scale.

Statistical analysis

Data were gathered, edited, coded, and entered into the IBM SPSS (Statistical Package for Social Science, version 20). The qualitative data were presented as numbers (percentage) while quantitative data were presented as mean ± standard deviation (SD) or median and range when not normally distributed.

Results

The mean age of the studied cases was 11.96 ± 2.47 years (range: 1.5–15 years), 38.0% were males. Other demographic data was presented in Table 1. Causes of syncopal attacks among the studied participants were presented in Fig. 1.

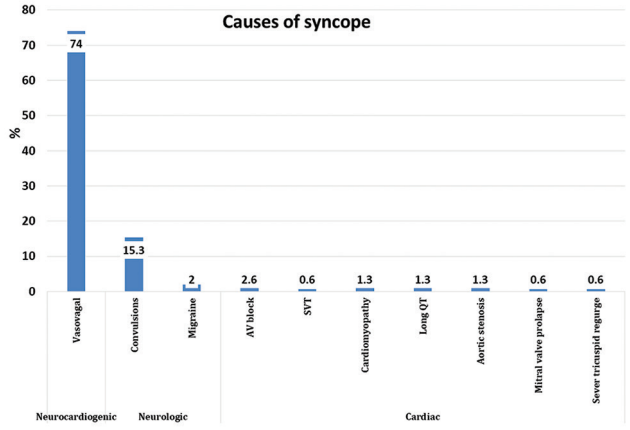
Table 2 shows that full history taking were asked for all studied cases. Positive findings were documented as follows; vomiting in 25.3% suffered from, headache in 16.6%, blurring of vision in 66.0%, dyspnea in 4.0%, chest pain in 9.3%, body selling was reported in only one 1 (0.7%), fatigue and/or effort intolerance

was reported in only 3 (2.0%) case, palpitations in 9.3%, cyanosis in 16.0%, and 6.0% have emotional stress. 10.0% suffered from syncopal attack during exercise, 2.7% suffered from syncopal attack during a sudden change in position, and 4.0% suffered from hyperventilation followed by syncopal attack. 8.0% had positive history of structural heart disease, 17.3% had history of neurological disease, and 3.3% had hypovolemia due to vomiting. 8.0% had a past history of cardiac disease, and 64.7% had history of prodroma.

The duration of syncopal attack was asked for all studied cases with a median duration of four minutes (range: 1–10 min). However, none of the studied cases were asked about the history of greyout, tunneling, seeing spots, auditory changes, or foreign body inhalation.

All studied participant received detailed general and systematic examination. Only the respiratory rate, temperature, and oxygen saturation were not checked for the studied participants in the current study, as shown in Table 3.

Figure 1



Causes of syncopal attacks among the studied participants.

Table 1 Demographic data of the studied participants

Demographic data	*n*=150
Age (y)	
Mean±SD	11.96±2.47
Median (range)	12 (1.5–15)
Sex, *n* (%)	
Male	57 (38.0)
Female	93 (62.0)
Residence, *n* (%)	
Urban	19 (12.7)
Rural	131 (87.3)
Family history of syncope	
Negative	150 (100.0)
Positive	0

Quantitative data are presented as mean±standard deviation and median (range), qualitative data are presented as number (percentage).

Detailed investigations among the studied participants were presented in Table 4; complete blood count (CBC) analysis was done for 22.6% cases, kidney function, and serum electrolytes in 6.0%.

All cases received electrocardiogram (ECG) examination and positive finding was documented in seven cases (4.7%). All cases received echocardiography (ECHO) examination and positive finding was documented in ten cases (6.7%). Electroencephalogram (EEG) examination was done for 23 (15.3%) cases, with positive finding in 20 (13.3%) cases. Computed tomography (CT) examination was done for 23 (15.3%) cases, with positive finding in 3 (2.0%) cases in the form of atrophic changes.

All studied cases were asked about used medications; only 18.7% of studied cases received medication. Table 5 summarize delayed list of used medications among the studied participants.

Discussion

Syncope is a common pediatric problem and is characterized as a transient loss of consciousness and postural tone followed by a spontaneous recovery [5]. The main goal of the current prospective clinical audit study is to assess how much the staff in the AUCH are sticking to the agreed upon unit's protocol regarding how to deal with children or adolescent with syncopal attacks.

In the history

Our institution is successfully following the recommended protocol for history taking, to investigate possible etiologies of these attacks, for appropriate management and better outcomes.

However none of the studied cases were asked about history of greyout, tunneling, seeing spots, auditory changes, or foreign body inhalation, all of these are component of prodrome which is the most important component of the history. Vasovagal syncope is strongly suggested by a warm or clammy feeling, nausea, lightheadedness, or vision abnormalities (such as seeing spots, greying out, or tunneling). Anger, disorientation, hearing abnormalities, dyspnea, or abdominal discomfort are additional symptoms. Absence of a prodrome should raise the suggestion of cardiac etiology [3,6]. This could be considered as a point of weakness in our institution, and must be taken into consideration as we could expect the underlying cause of syncope just by patient's history.

Table 2 Manifestations of syncope among the studied participants

History	*n* (%)
Nausea	
Not asked	0
Asked	150 (100.0)
Positive finding	38 (25.3)
Headache	
Not asked	0
Asked	150 (100.0)
Positive finding	25 (16.6)
Blurring of vision	
Not asked	0
Asked	150 (100.0)
Positive finding	99 (66.0)
Greyout	
Not asked	150 (100.0)
Asked	0
Tunneling	
Not asked	150 (100.0)
Asked	0
Seeing spots	
Not asked	150 (100.0)
Asked	0
Auditory changes	
Not asked	150 (100.0)
Asked	0
Dyspnea	
Not asked	0
Asked	150 (100.0)
Positive finding	6 (4.0)
Chest pain	
Not asked	0
Asked	150 (100.0)
Positive finding	14 (9.3)
Body swelling	
Not asked	0
Asked	150 (100.0)
Positive finding	1 (0.7)
Fatigue and/or effort intolerance	
Not asked	0
Asked	150 (100.0)
Positive finding	3 (2.0)
Duration of the attack (minutes)	
Not asked	0
Asked	150 (100.0)
Median (range)	4 (–10)
Palpitation	
Not asked	0
Asked	150 (100.0)
Positive finding	14 (9.3)
Cyanosis	
Not asked	0
Asked	150 (100.0)
Positive finding	24 (16.0)
Emotional stress (fear and/or pain)	
Not asked	0
Asked	150 (100.0)
Positive finding	9 (6.0)
Situational syncope	
Not asked	0
Asked	150 (100.0)
Positive finding	0
Mechanic carotid triggers	
Not asked	0
Asked	150 (100.0)
Positive finding	0
Exercise	
Not asked	0
Asked	150 (100.0)
Positive finding	15 (10.0)
Sudden change in position	
Not asked	0
Asked	150 (100.0)
Positive finding	4 (2.7)
Foreign body inhalation	
Not asked	150 (100.0)
Asked	0
Hyperventilation	
Not asked	0
Asked	150 (100.0)
Positive finding	6 (4.0)
History of structural heart disease	
Not asked	0
Asked	150 (100.0)
Positive finding	12 (8.0)
History of neurological disease	
Not asked	0
Asked	150 (100.0)
Positive finding	26 (17.3)
Hypo-volemia	
Not asked	0
Asked	150 (100.0)
Positive finding	5 (3.3)
Hypoglycemia	
Not asked	0
Asked	150 (100.0)
Positive finding	0
Psychological disease	
Not asked	0
Asked	150 (100.0)
Positive finding	0
Past history of cardiac disease	
Not asked	0
Asked	150 (100.0)
Positive finding	12 (8.0)
History of prodroma	
Not asked	0
Asked	150 (100.0)
Positive finding	97 (64.7)

Qualitative data are presented as number (percentage). Situational syncope include: cough, sneeze, laugh, head turning, swallowing, defecation, micturition. History of neurological disease include: cerebrovascular, increase intracranial pressure, migraine. Hypo-volemia induced: vomiting, diarrhea, bleeding. Hypoglycemia induced by: drugs as sulfonyl urea, insulin, sepsis, renal failure, liver disease. Psychological diseases include; depression, panic attacks, anxiety, somatization. ASD, atrial septal defect; MR, mitral rugrge; RA, right atrium; RV, right ventricle; TR, tricuspid regurge; VSD, ventricular septal defect.

Table 3 Examination results among the studied participants

Examination	*n* (%)
Heart Rate (beats/minute)	
Not checked	0
Checked	150 (100.0)
Respiratory Rate	
Not checked	150 (100.0)
Checked	0
Blood Pressure	
Not checked	0
Checked	150 (100.0)
Temperature	
Not checked	150 (100.0)
Checked	0
Oxygen Saturation	
Not checked	150 (100.0)
Checked	0
GCS	
Not checked	0
Checked	150 (100.0)
Median (range)	15 (15–15)
Random blood glucose (mg/dl)	
Not checked	0
Checked	150 (100.0)
Median (range)	97 (65–200)
General examination	
Not done	0)
Done	150 (100.0)
Positive finding	1 (0.7)
Generalized edema due to heart failure	1 (0.7)
Cyanosis	0
Clubbing	0
Dysmorphic features	0
Chest examination	
Not done	0
Done	150 (100.0)
Positive finding	1 (0.6)
Median sternotomy scar	1 (0.6)
Cardiac examination	
Not done	0
Done	150 (100.0)
Positive finding	
By Auscultation	5 (3.3)
Tachycardia	2 (1.3)
Pan systolic murmur	2 (1.3)
Ejection systolic murmur over the second right intercostal space	1 (0.7)
By palpitation	
Normal apex site	145 (96.7)
Abnormal apex site	5 (3.3)
Neurological examination	
Not done	0
Done	150 (100.0)
Positive finding	0
Abdominal examination	
Not done	0
Done	150 (100.0)
Positive finding	0

Quantitative data are presented as median (range), qualitative data are presented as number (percentage), GCS, Glasgow coma scale.

In the current study 97 (64.7%) children had a prodrome, in line with this finding Chen and colleagues found that among 154 children with syncope, 85% of children with VVS had a prodrome, whereas only 40% of those with cardiac problems showed prodromal symptoms [7].

Regarding the examinations

Our institution is successfully following the recommended protocol for general and systematic examination to evaluate causes of syncopal attacks, as all studied cases were received detailed clinical examination. Only the respiratory rate, temperature, and oxygen saturation were not checked for the studied participants in the current study. This is another point of weakness. Respiratory physiology has received very little attention in this context of syncopal attack. However, it is widely recognized that when a person hyperventilates, whether spontaneously or as a symptom of an anxiety disease, faintness or loss of consciousness may happen. Additionally, the vasoconstrictor impact of hyperventilation-induced hypocapnia over the cerebral vascular bed is well documented and may be utilized as a therapeutic strategy in the management of intracranial hypertension [8]. And already in the current study we observed that 6 (4.0%) cases suffered from hyperventilation which has been followed by syncopal attack.

It is unknown whether seasonal, cyclical, weekly, or temperature-dependent patterns are present in cardiac syncope and maybe other syncope aetiologies. Understanding these patterns may offer new insights on the pathophysiology of syncope. While reflex and orthostatic syncope increased with decreasing outside temperature, while cardiac syncope does not appear to have a seasonal or temperature-dependent pattern [9]. We, therefore, must assess the body temperature of the pediatric patients with syncope, thus me help us to differentiate the causes of syncope.

As we mentioned above that the most frequent cause of syncope in children and young adults is neurocardiogenic syncope. In spite of that, NCS seldom results in death; it can have serious deleterious effects. This makes it crucial for patients who experience frequent NCS episodes to receive the proper diagnosis and treatment. Ayers and colleagues expected that regional tissue oxygen saturation would fall during an Neurocardiogenic syncope (NCS) episode but remain steady in patients without syncope [10]. Based on this finding we recommend adding monitoring of oxygen saturation via near-infrared spectroscopy (NIRS) monitoring during head-up tilt-table test (HUTT) as these

Table 4 Investigation results among the studied participants

Investigation	*n* (%)
Complete blood picture (CBC)	
Not done	116 (77.3)
Done	34 (22.6)
Kidney function tests	
Not done	141 (94.0)
Done	9 (6.0)
Electrolytes*	
Not done	141 (94.0)
Done	9 (6.0)
ECG	
Not done	0
Done	150 (100.0)
Positive finding	7 (4.7)
SVT	1 (0.7)
First degree AV block	1 (0.7)
Second degree heart block	1 (0.7)
Third degree AV block	2 (1.3)
Long QT	2 (1.3)
ECHO	
Not done	0
Done	150 (100.0)
Positive finding	10 (6.7)
Aortic stenosis	4 (2.7)
Cardiomyopathy	2 (1.3)
ASD	2 (1.3)
Mitral regurge with dilated all cardiac chambers	1 (0.7)
Severe tricuspid regurge with pulmonary hypertension	1 (0.7)
EEG	
Not done	127 (84.6)
Done	23 (15.3)
Positive finding	20 (13.3)
Generalized epileptic discharge	10 (6.6)
Multifocal epileptic discharge	7 (4.6)
Epileptic focus	3 (2.0)
CT	
Not done	127 (84.6)
Done	23 (15.3)
Positive finding	3 (2.0)
Atrophic changes	3 (2.0)

*Serum electrolytes include: Sodium, Potassium, and Calcium.
Qualitative data are presented as number (percentage).
CBC, complete blood picture; CT, computerized tomography; ECG, Electrocardiogram; ECHO, echocardiography; EEG, Electroencephalogram.

processes will yield a trustworthy outcome before any clinical symptoms or indicators appear. Additionally, it aids in identifying NCS from psychogenic syncope.

Regarding the investigations

In our protocol for management for syncopal attacks we mentioned the following investigations; CBC, kidney function tests, serum electrolytes, ECG, ECHO, EEG, and CT. However actually we observed that only ECG, and ECHO were performed for all studied cases.

Table 5 History of medications received by the studied participants

History of medications	*n* (%)
Not asked	0
Asked	150 (100.0)
Positive finding	28 (18.7)
Levetiracetam	13 (8.6)
Sodium valproate	9 (6.0)
Inderal	2 (1.3)
ACE inhibitors + diuretics + digoxin	1 (0.7)
Antihypertensive	5 (3.3)
Diuretics	4 (2.6)
Barbiturates	0
Tricyclic antidepressant	0
Alcohol	0
Antiarrhythmics	1 (0.7)
Macrolides	0
Antihistamines	0
Antipsychotics	15 (10.0)
MAO inhibitors	0
Levodopa	0
Prazosin	0
Benzodiazepines	0

Qualitative data are presented as number (percentage).

However, the precise significance of ECG and/or echocardiography in the evaluation of syncope remains unknown, particularly in patients with no cardiac history and no positive findings in the history, physical examination, or first evaluation [11]. It is often recommended to be included in the evaluation of syncope in individuals with a cardiac history or an abnormal clinical examination [4,12]. Therefore, routine ECG and echocardiogram done on individuals who have a normal physical examination and no history of heart disease is not particularly beneficial and just increases the patient's additional costs and length of stay [11].

Thus we need individualized standardized approach to teach pediatrician how to deal with children with syncopal attacks to be implemented in our institution, in order to eliminate variation in practice, maximize resource usage, and improve patient care. Although an identical management strategy based on particular red flag history and physical exam findings should be established for use by general pediatric physicians, and by pediatric cardiologists.

Regarding to the initial management of syncopal attacks

All studied patients were asked about past and currently used medication, also we could say that our institution is successfully following the recommended protocol, except for some medication that not used by our studied cases, this could be contributed to many reasons as patients age, medical history, the presence of associated manifestations, and also the underlying cause of syncope.

We recommend individualized pharmacological management of syncope based on the last update in international guidelines for management of syncope in children and adolescents.

Conclusion

The management of children with syncope at Assiut University Children Hospital was successfully following the Unit's agreed upon protocol. However further studies are needed to compare the Unit's agreed upon protocol to one of the international guidelines and to identify every defect in management at AUCH and prescribe how to correct these defects, in order to prioritize our decision making in management of those patients to improve their quality of life, and to minimize the case fatality rate among them.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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