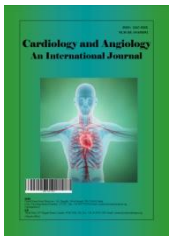

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Etiology and Age Distribution of Syncope in Delta Area Patients

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Syncope is a Total Loss of Consciousness (TLOC) due to cerebral hypo-perfusion, characterized by a rapid onset, short duration, and spontaneous complete recovery. It shares many clinical features with other disorders; it therefore presents in many differential diagnoses. This group of disorders is labelled TLOC. The aim of the present study was to determine the etiologies and the age distribution of patients with syncope in Delta area.
Methods: This study included thirty patients who had at least one episode of syncope. All patients in this study were subjected to history taking, clinical examination, laboratory investigations, standard 12-lead Electrocardiogram (ECG), Resting Transthoracic Echocardiography (TTE). Some patients were subjected to Prolonged ECG monitoring (Holter, Event, ILR), Brain computed tomography (CT) and Magnetic resonance imaging (MRI), Tilt table test and Electrophysiology study (EP study).
Results: Most common ECG rhythm was normal sinus rhythm, complete heart block, left ventricular hypertrophy, Slow Atrial fibrillation (AF), Left bundle branch block (LBBB), ventricular tachycardia. The most common diagnosis of syncope was vasovagal syncope, idiopathic syncope then cardiac syncope. Incidence of idiopathic syncope in populations under 40 years was significantly higher than in populations over 40 years, meanwhile regarding cardiac-related etiology for syncope in patients under age 40 was significantly lower than those patients over age 40.
Conclusion: For age distribution, the incidence of idiopathic syncope in populations under 40 years was significantly higher than in populations over 40 years, meanwhile cardiac-related etiology for syncope in patients under age 40 was significantly lower than those patients over age 40.

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Keywords: Etiology; age distribution; syncope; vasovagal syncope.

1. INTRODUCTION

"Syncope is a common clinical problem which is defined as a transient loss of consciousness (T-LOC) due to transient global cerebral hypo-perfusion characterized by rapid onset, short duration, and spontaneous complete recovery, which is not compatible with other states of altered consciousness (seizure, coma, migraine, metabolic disorders including, hypoxic ventilation, hypoglycaemia, epilepsy, transient ischemic attack, cataplexy, drop attack, somatization disorders)" [1].

"The etiological classification remains the basis for both risk stratification and subsequent clinical management. Syncope is a presenting symptom, not a diagnosis. While most causes are benign and self-limiting not requiring extensive in-hospital evaluation, others are potentially severe" [2]. "Causes of syncope differ between cardiac, neurological, orthostatic hypotension, vasovagal. Other causes as endocrinological causes that can be presented as orthostatic hypotension to cause autonomic dysfunction or hypovolemia. As examples, chronic adrenal insufficiency and hypopituitarism can be cited. Diabetes insipidus and salt-losing nephropathies since they result in volume depletion, and pheochromocytoma and carcinoid syndrome, due to vasoactive substances, can also be causes of syncope. Hyperventilation and psychiatric disorders, due to cerebral hypoperfusion" [3]. "Among the causes of syncope, the mediated neural reflex, known as neurocardiogenic or vasovagal syncope, is the most frequent, accounting for one third of the causes and reaching 66% of cases in emergency units. The diagnosis of syncope can be made by clinical method associated with the electrocardiogram (ECG) in up 50% of patients" [4].

"The optimal evaluation of patients with syncope follows a risk- adapted diagnostic algorithm exclude life-threatening conditions and identify those with high risk for further deterioration, such as structural heart diseases requiring further diagnostic evaluation. Low risk patients can be discharged without further work-up" [5]. "Prognosis is determined by the underlying etiology specifically the presence and severity of cardiac disease. And it is imperative to identify its cause and risk stratification for positive impact in reducing morbidity and mortality" [6]. The aim of the study was to determine the etiologies and the age distribution of patients with syncope in Delta area.

2. PATIENTS AND METHODS

The study was carried out at the department of cardiology Tanta University Hospitals. Thirty patients were included in this study. This study was done over a period of six months from September 2019 until February 2020. The inclusion criteria were patients who had at least one episode of syncope. The exclusion criteria included patients with loss of consciousness from reasons other than hypoperfusion of the brain (seizure, concussion and trauma, intoxication, hypoglycaemia, psychogenic pseudo-syncope, seizure, head injury). Patients who were unable to give either written or verbal informed consent.

All patients in this study will be subjected to the following: clinical history was performed, and each episode of syncope was well characterized about presence of prodrome, precipitating factors, conditions in which it occurred, position of the patient, and other associated signs such as nausea, pallor, diaphoresis, muscle twitching, confusion, physical injury, palpitations, Dyspnea, chest pain, and cyanosis. The personal medical history and family history were also taken to identify the cause of syncope. Clinical examination: All participants were subjected to complete physical examination including assessment of the general condition and vital signs as blood pressure and heart rate, it was comprehensive, including measurement of blood pressure in the supine position and within 3 min in the orthostatic position, the blood pressure measurements in both arms.

Syncope analysis for prodrome, precipitating factors, conditions in which it occurred, position of the patient, and other associated signs. Local examination of the heart: Heart sounds, additional sounds as S3 or S4 and cardiac murmurs.

Investigational studies included Standard 12-lead ECG: for clinical features suggestive of arrhythmias (palpitation, family history of sudden cardiac death, and syncope during exercise). Abnormal axis, ischemic changes, Brugada syndrome and conduction block (first, second- and third-degrees heart block, abnormal QT interval, bi-fascicular block, and Left bundle branch block (LBBB)) were also detected.

Resting Transthoracic Echocardiography (TTE): It is the most useful imaging study for evaluating the severity of underlying cardiac disease and for the risk stratification of patients who had unexplained syncopal events especially those with a prior known positive cardiac history or an abnormal ECG. Echocardiography in patients with a normal physical exam and normal ECG is not very helpful.

Echocardiographic images were obtained in the parasternal long-axis and short-axis and apical two-chamber and four-chamber views using standard transducer positions. Vivid 9, General Electric Healthcare (GE Vingmed, Norway) equipped with a harmonic M5S variable-frequency (1.7-4 MHz) phased-array transducer was used. left ventricle (LV) dimensions and wall thickness, EF, and left atrial diameter and volume were measured in accordance with the recommendations of the American Society of Echocardiography.

It was done mainly to diagnose structural heart disease causing arrhythmias, valvular heart disease as mitral valve prolapses and aortic stenosis, severe LV dysfunction, pericardial effusion, pulmonary hypertension and hypertrophic cardiomyopathy.

The following investigations were done for some patient: Prolonged ECG monitoring (Holter, Event, ILR): 48 hours ECG for those with paroxysmal arrhythmias.

Electroencephalogram (EEG): records included scalp surface routine EEG and EEG after sleep deprivation. EEGs lasted 20 to 30 minutes and included hyperventilation and photic stimulation. The EEGs were reported and reviewed by a board-certified neurologist and neurophysiologist. It was classified into (1) normal, (2) epileptiform discharges and (3) slow waves and separated according to the gender.

Brain computed tomography (CT) and Magnetic resonance imaging (MRI): to detect abnormal structures in brain (masses or AV malformations), brain hypoperfusion or stroke.

Tilt table test: Patients underwent tilt-table testing in a quiet room after they had fasted for 4 to 8 hours. They were comfortably restrained on an electric tilt table. Instrumentation consisted of a peripheral intravenous cannula and automatic and manual blood pressure cuff. The test ended after frank syncope during an infusion of 5 µg/min isoproterenol or to a total of 10 minutes in the head-up position with either presyncope or no symptoms. Heart rate, blood pressure, and symptoms were recorded each minute. The test was considered to be positive if it ended in syncope or in presyncope and a drop-in rate–systolic pressure.

Ambulatory blood pressure monitoring was conducted using (Fukuda Denshi, Ltd., Tokyo, Japan). Blood pressure (BP) and heart rate (HR) were measured automatically every 30 minutes for 24 hours (48 measurements). BP was measured by the Korotkoff microphone method or oscillometeric method. The average systolic blood pressure (SBP), diastolic blood pressure (DBP), and HR values and their standard deviations (SD) were calculated. All parameters were separated into daytime (7 am to 10 pm) and night-time (10 pm to 7 am).

Electrophysiology study (EP study): Using local anesthesia, two or more multipolar electrode catheters were percutaneously introduced via the peripheral veins and positioned within the high right atrium, AV junction (His bundle) and right ventricle for local intracardiac ECG recordings or electrical stimulation. At least three surface ECG leads, intracardiac ECG and timelines were simultaneously displayed on a multichannel oscilloscope. All tracings were continuously recorded on a tape and subsequently reproduced on paper at a speed of 100-150 mm/sec. Cardiac stimulation was performed using a digital stimulator capable of delivering impulses with adjustable voltage and duration. The following pacing protocol was followed: Incremental atrial pacing up to cycle lengths sufficient to produce atrioventricular (AV) nodal Wenckebach phenomenon or initiation of arrhythmia. Incremental atrial pacing starting at rates just faster than sinus for a period of 30 seconds to evaluate the sinus node function. Atrial premature stimulation at one or more basic cycle length. Incremental ventricular pacing up to cycle length of 300 ms. Ventricular premature stimulation with single beats (V2) during one or more ventricular paced drives. Bursts of rapid ventricular pacing were delivered [4-6 beats] at progressively shorter cycle lengths until ventricular muscle was refractory to every other stimulus.

Laboratory investigations: Blood samples at hospital admission were drawn in the emergency room from the antecubital vein by careful venepuncture using a 21-gauge needle attached to a sterile syringe without stasis. Complete blood count (CBC), liver function test, renal

function test, electrolytes panel and thyroid profile were measured. When the diagnosis was not determined after these evaluations and targeted tests, the results of diagnostic procedures (cardiology and neurology) were re-evaluated. Diagnosis of typical neurocardiogenic syncope was based on clinical history [syncope occurring during prolonged standing, instrumentation, stress, or fear with typical prodromal symptoms (diaphoresis, warmth, and nausea)] and the result of tilt-table test.

2.1 Statistical Analysis

Statistical analysis was done by SPSS v26 (IBM Inc., Chicago, IL, USA). Quantitative variables were presented as mean and standard deviation (SD). Qualitative variables were presented as frequency and percentage (%). A two tailed P value < 0.05 was considered statistically significant.

3. RESULTS

Demographic data, Risk factors and history in studied population shown in Table 1.

The most frequent symptoms of patients with syncope were dizziness, followed by palpitation, shortness of breath, chest pain, easy fatigability. While the most frequent signs of patients with syncope was basal crepitations followed by lower limb (LL) edema, meanwhile none of the patients had congested neck veins Table 2.

The most common ECG rhythm in patients presented with syncope was normal sinus rhythm, complete heart block, left ventricular hypertrophy, Slow atrial fibrillation (AF), LBBB, ventricular tachycardia. Table 3.

Echocardiographic data and laboratory investigations in studied population shown in Table 4.

The most common diagnosis of syncope was vasovagal syncope, which was diagnosed by history, idiopathic syncope which diagnosed after exclusion other causes of syncope by history, ECG, Holter ECG, echo, CT brain then cardiac syncope that includes CHB, VT, and slow A. Fib. which diagnosed by ECG, lastly orthostatic hypotension and neurological causes which diagnosed by measuring blood pressure during setting down and during standing and by CT brain. Table 5.

Regarding age distribution and incidence of Syncope End-Diagnosis in studied population, the incidence of idiopathic syncope in populations under 40 years was significantly higher than in populations over 40 years, meanwhile regarding cardiac-related etiology for syncope in patients under age 40 was significantly lower than those patients over age 40. However, no statistically significant difference was noted between age groups for vasovagal, orthostatic hypotension, neurological causes, pulmonary embolism Table 6.

Table 1. Demographic data, risk factors and history in studied population

Data		Mean ±SD	
Age (36 – 72 Ys)		52.84 ±8.4	
Age Distribution	≤40 Ys	N (/ 30)	Ratio (100%)
		17	57%
	>40 Ys	13	43%
Gender	Male	19	63%
	Female	11	37%
Hypertension	Yes	17	57%
	No	13	43%
Smoking	Yes	12	40%
	No	18	60%
Diabetes Mellitus (DM)	Yes	12	40%
	No	18	60%
Valvular Heart Disease	Yes	1	3%
	No	29	97%
ischemic heart disease	Yes	5	16%
	No	25	84%
Cardiomyopathy	Yes	1	3%
	No	29	97%
Chronic Kidney Disease	Yes	2	6%
	No	28	94%

Table 2.Symptoms and signs in studied population

Symptoms			
		N (/ 30)	**Ratio (100%)**
Palpitation	Yes	8	24%
	No	22	76%
Dizziness	Yes	10	34%
	No	20	66%
Easy Fatigability	Yes	1	3%
	No	29	97%
Shortness of breath	Yes	8	24%
	No	22	76%
Chest Pain	Yes	5	16%
	No	25	84%
Signs			
		N (/ 30)	**Ratio (100%)**
Congested Neck Vein	Yes	0	0
	No	30	100%
Fine basal Creps	Yes	3	9%
	No	27	91%
L.L edema	Yes	2	6%
	No	28	94%

Table 3. E.C.G Rhythm in studied population

Rhythm			
		N (/ 30)	**Ratio (100%)**
CHB	Yes	7	24%
	No	23	76%
NSR	Yes	17	58%
	No	13	42%
LBBB	Yes	1	3%
	No	29	97%
Slow AF	Yes	1	3%
	No	29	97%
LVH	Yes	2	6%
	No	28	94%
VT	Yes	1	3%
	No	29	97%

CHB= Congenital heart block, NSR= Normal sinus rhythm, LVH= Left ventricular hypertrophy, VT= Ventricular tachycardia

Table 4. Echocardiographic data and laboratory investigations in studied population

Data	Mean ±SD
LV EF (40 – 74%)	62.0 ±0.9
Urea	54.8 ±44.2
Serum Creatinine	1.49 ±1.2
K^+	4.4 ±0.6
TSH	2.7±1.3
Hb	14.3±1.3
WBCs	13±2.9
Plts	279.3±77.5

K^+= Potassium, TSH= thyroid stimulating hormone, Hb= Hemoglobin, WBCs= white blood cells, Plts= platelets

Table 5. End-diagnosis of syncope in studied population

Syncope End-Diagnosis			
		N (/ 30)	**Ratio (100%)**
Orthostatic		3	9%
Idiopathic		9	31%
Vasovagal		11	39%
Neurological		2	6%
	CHB	2	6%
Cardiac	**VT**	1	3%
	AF	2	6%
Pulmonary embolism		0	0%

Table 6. Age distribution and incidence of syncope end-diagnosis in studied population

		(AGE ≤ 40) N= 17 (%)	**(AGE > 40) N= 13 (%)**	**P-value**
Vasovagal		6 (35.2)	5(38.4)	0.985
Idiopathic		7 (41.2)	2 (15.4)	0.0001*
Orthostatic		2 (11.7)	1 (7.7)	0.074
Neurological		1 (5.8)	1 (7.7)	1.000
Cardiac:	**AF**	1 (5.8)	1 (7.7)	0.0001*
	VT	0	1 (7.7)	
	CHP	0	2 (15.4)	
Pulmonary Embolism		0	0	1.000

4. DISCUSSION

"Syncope is a common clinical problem accounting for 3% of all emergency room visits and 1% to 6% of all hospital admissions. It can be remarkably debilitating and associated with high health care costs; its true incidence is difficult to estimate due to variation in definition, differences in population prevalence and underreporting in the general population. Causes of syncope differ between cardiac, neurological, orthostatic hypotension, vasovagal. The evaluation and management of patients with syncope is one of the more challenging issues in clinical practice. Although many underlying causes of syncope are benign, others are associated with substantial morbidity or mortality, including cardiac arrhythmia, myocardial infarction, pulmonary embolism, and occult hemorrhage" [7].

Discordant to our results, Mahmudy et al. [8] which assessed the syncope risk factors among military training soldiers, 50 soldiers experienced syncope and the mean age was 22.94 ± 2.69 years.

In Awan et al. [1] a study conducted on 152 patients to identify a correlation between the various etiologies of syncope and age in a predominantly African American sample population there were 75 men and 77 female patients with 25 patients below the age of 40 and 127 patients above the age of 40.

In Ghani et al. [9], a study assessed 139 patients admitted to an observation unit with a primary diagnosis of syncope to assess the role of echocardiography in diagnostic evaluation of patients with syncope, there were 42% male and 52% female patients.

In Gilfrich et al. [10] a study assessed the Syncope as a health risk for soldiers-influence of medical history and clinical findings on the sensitivity of head-up tilt table testing on 100 consecutive patients aiming to identify major and minor criteria that might suggest diagnostic benefit from head-up tilt testing included 55% female and45% male patients with mean age of population study 53 years.

Regarding to risk factors: Hypertension and diabetes are widespread risk factors and common co morbidities [11]. Regarding to Awan et al. [1] there were 29 (18.7%) hypertensive, 33 (21%) diabetic and 49 (31.6%) patients with heart disease.

Regarding to Ghani et al. [9] there were 63% hypertensive patients, 16% diabetic patients,

10% of patients with prior myocardial infarction, 11.5% of patients with chronic kidney disease, 10% with prior syncope and 8.6% of patients with Prior cerebrovascular accident.

Comparable to our results, MacCormick et al. [12] study which assessed the symptoms and signs associated with syncope in young people with primary cardiac arrhythmias in 35 consecutive patients Showed that dizziness was the most frequent symptom, being experienced by 47% of patients. 45%of patients had witnessed seizure-like activity and 40% of patients had urinary incontinence. Nineteen patients could describe the post-syncopal period, of whom 79% of patients reported symptoms, the most common 65% of patients being drowsiness or exhaustion.

Nishijima et al. [13] study assessed the ECG predictors of cardiac arrhythmias in older adults with syncope showed that 15.3% of patients with CHB, 27.3% of patients with symptomatic bradycardia and 22.9% of patients with VT.

Quinn and McDermott et al. [14] assessed the ECG findings in emergency department patients with syncope stated that 14% of patients with predicted cardiac outcomes had LBBB, 7% sinus ECG, 25% bradyarrhythmia 3% svt, 6% pvcs.

Discordant to our results, Rami et al. [15] a study assessed the characteristics of syncope in patients with dilated cardiomyopathy showed that the mean Left ventricular ejection fraction (LVEF) was 35.3%.

In Seol et al. [16] assessed patients presented with complete atrioventricular block presenting with syncope showed that TSH level was more than 100 mIU/L (normal range 0.27 - 4.2) and the Creatinine level was 3.3 mg/d.

Comparable to our results, Mitro et al. [17] a prospective study of the standardized diagnostic evaluation of syncope for 529 patients showed that 5 % of patients had CHB, 2.7% of patients had slow AF, 6% of patients had VT and 7.3% of patients had sick sinus syndrome.

Runser et al. [18] stated that cardiac syncope occurs in approximately 20% of syncope presentations. It is most often caused by an arrhythmia, and less often by a structural cardiac abnormality. In a prospective cohort study, patients with cardiac syncope had a twofold increase in mortality over 17 years.

Awan et al. [1] showed that 13 (8.3%) patients with cardiac syncope, 18 (11.6%) patients with orthostatic syncope, 43 (27.7) patients with vasovagal syncope, 60 (38.7%) patients with idiopathic syncope. They also showed that idiopathic syncope was higher above the age of 40, this is maybe due to small sample size and most of the patients experiencing syncope review neurology before cardiology clinic.

O'Brien and Kenny et al. [19] showed that One-third of cases of syncope in the older patient are caused by cardiac disorders. There is a higher morbidity and mortality associated with cardiac syncope. Cardiac syncope is characterised by little or no prodrome, occurrence when supine or during exercise and association with palpitations or chest pain. However, the older patient may not recall these symptoms.

Recommendations and limitations were further studies on larger sample size and on large geographical scale to emphasize our conclusion. Additional and specifically designed studies are needed to establish the optimal management of these patients. Further studies need to be undertaken to determine the etiologies and the age distribution of patients with syncope in Delta area. Finally, currently available risk stratification scores should not be used alone to perform risk stratification in the Emergency Department.

5. CONCLUSIONS

The most common cause of syncope is vasovagal (42%) followed by idiopathic causes (31%). For age distribution, the incidence of idiopathic syncope in populations under 40 years was significantly higher than in populations over 40 years, meanwhile cardiac-related etiology for syncope in patients under age 40 was significantly lower than those patients over age 40.

CONSENT AND ETHICAL APPROVAL

The study was done after approval from the Ethical Committee Tanta University. An informed written consent was obtained from the patient' guardians or relatives.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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