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Original Article

Characteristics of syncope in patients with dilated cardiomyopathy

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ABSTRACT

Background: Syncope carries a poor prognosis among patients with dilated cardiomyopathy (DCM).

Objectives: To assess the prevalence, describe the underlying mechanisms and to identify risk factors for syncope in patients with DCM.

Methods: One thousand six hundred and ten medical files of 897 patients with a diagnosis of DCM were reviewed. Patients with syncope were identified and their clinical and paraclinical profiles were compared to an equal number of age- and sex-matched patients with DCM without syncope.

Results: Thirty patients (27 males) with an average age of 62.5 years were identified, corresponding to a prevalence of syncope of 3.3%. A cardiac origin of syncope was identified in 56% of patients ($n = 17$): ventricular arrhythmias in 33% ($n = 10$), and conduction disorders in 23% ($n = 7$). Other mechanisms of syncope were neurally mediated in 7% ($n = 2$) and orthostatic hypotension in 7% ($n = 2$). In 30% of cases ($n = 9$), the etiology was unidentified.

There were no significant differences regarding the etiology of DCM, ejection fraction (35.3% vs 35.3%, $p = 1.0$), NYHA class (mild or advanced, $p = 0.79$) and associated conditions (hypertension, $p = 0.36$; diabetes, $p = 0.75$; atrial fibrillation, $p = 0.43$; and dyslipidemia, $p = 0.33$) between the two groups. However, among patients with syncope, patients with a noncardiac cause were more likely to have hypertension (61.53% vs 23.52%, $p = 0.08$) and diabetes (46.15% vs 5.88%, $p = 0.03$).

Conclusion: In patients with DCM, syncope is a relatively rare finding. Cardiac causes (arrhythmias and conduction disorders) are responsible for the majority of cases. Risk factors for syncope in these patients remain to be determined.

1. Introduction

The occurrence of syncope in patients with congestive heart failure (HF) and left ventricular (LV) dysfunction is related to an increased risk of overall mortality and sudden cardiac death (SCD).[1,2] The one-year risk of SCD can be as high as 45% in subjects with advanced HF and syncope, compared to a significantly lower SCD risk of 12% in patients with advanced HF but without syncope[2]. Several studies identified syncope to

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be a negative prognostic factor for nonischemic dilated cardiomyopathy (DCM) patients.[3,4]

However, the occurrence of syncope in patients with DCM is not yet completely understood. The cause often remains undiagnosed after standardized evaluation and the relationship between syncope and death in HF patients remains vaguely characterized. The mechanisms of syncope in DCM patients are diverse, including cardiac diseases such as ventricular and supraventricular arrhythmias, bradycardia, conduction disorders, and valvular stenosis. Noncardiac causes are neurally mediated[5] and those attributed to orthostatic hypotension (OH) or neurological pathology[6,7].

Since syncope is associated with an increased mortality in patients with DCM, identifying risk factors for the occurrence of syncope in these patients is important. In the general population, risk factors for syncope include advanced age[8], the presence of an underlying heart disease, autonomic dysfunction, drugs (vasodilators, diuretics, alcohol), and volume depletion[9]. Whether patients with DCM have the same risk factors or whether there are other risk factors for syncope in these patients is less known. Therefore, the aim of this study was to assess the prevalence of syncope, to describe the underlying mechanisms and to identify risk factors for syncope in patients with DCM.

2. Methods

All data were collected retrospectively from the patients' medical records. The medical files of 897 patients with a diagnosis of DCM, admitted for syncope from January 2008 to December 2013 to the Cardiology Department of the Rehabilitation Hospital in Cluj-Napoca, Romania were reviewed. Patients with syncope were identified and their clinical and paraclinical profiles were compared to an equal number of age- and sex-matched patients with DCM without syncope. Patients from the control group were chosen in a chronological order according to the admission date.

The studied parameters included DCM etiology, left ventricular ejection fraction (LVEF), New York Heart Association (NYHA) class severity, and the presence of associated conditions: atrial fibrillation (AF), type 2 diabetes mellitus (DM II), hypertension, and dyslipidemia.

2.1. *Patient workup for syncope*

The protocol for the assessment of patients with syncope at our hospital includes

- detailed history taking and a complete physical examination.
- blood pressure measurement in both arms, both in a supine position and during standing, to identify patients with orthostatic hypotension.
- a neurological exam performed by a certified neurologist, to rule out neurological causes of transient loss of consciousness.
- blood sample testing, including a complete blood count, blood glucose level, blood urea nitrogen, serum creatinine, total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, aspartate amino transferase (ASAT), alanine amino transferase (ALAT), uric acid, Quick time, INR.
- a standard 12 lead ECG carefully assessed for the presence of brady- or tachy-arrhythmias, conduction disorders such as AV block or bundle branch block, the presence of ischemic changes, ventricular pre-excitation, QT prolongation or shortening, and Brugada-like changes.
- a trans-thoracic echocardiography using an Esaote MyLab 50 echocardiograph, to identify anatomical modifications such as atria or ventricular dilation, the presence of hypertrophy or cardiac masses, to quantify the LV ejection fraction (LVEF), to assess valve status, the presence of pericardial effusion, signs of cardiac tamponade, aortic dissection, and pulmonary embolism, to characterize the diastolic function, global and regional kinetics, and to measure the pulmonary artery pressure.
- a 24 h Holter ECG monitoring using a BTL CardioPoint H600 device, to assess the minimum, average and maximum heart rates, as well as the presence of arrhythmias and conduction disorders.
- a head-up tilt table test and carotid sinus massage, performed in patients in which history taking suggests elements in favor of a vaso-vagal syncope or carotid sinus hypersensitivity.
- an electrophysiological study (EPS), in patients in which an arrhythmia or a conduction disorder is suspected based on noninvasive tests.

Based on these clinical and paraclinical data, the clinician's judgment established the cause of syncope.

2.2. *Definitions*

Syncope was defined as an episode of transient loss of consciousness with incapacity to maintain postural tone, with sudden onset, short duration and complete, and spontaneous recovery.[9]

The definition of DCM was based on the existence of a progressive heart muscle disease with cavity enlargement and diminished performance of the left ventricle (LVEF $<$ 55%), in the absence of left ventricular hypertrophy, with or without enlargement of the right ventricle. The upper limit of the normal left ventricular diastolic diameter for males and females was defined as 59 mm and 53 mm, respectively [10]. The etiology of cardiomyopathy was differentiated into primary (idiopathic) and secondary causes. DCM was considered idiopathic after secondary causes (such as ischemia, severe valvular stenosis or regurgitation, a history of alcohol abuse, a history of myocarditis, general systemic diseases, muscular distrophies, neuromuscular disorders, use of anthracyclines, irradiation and peripartum cardiomyopathy) were excluded.

Atrial fibrillation was diagnosed using the 12 lead ECG or Holter ECG monitoring. Patients with all forms of atrial fibrillation: paroxysmal, persistent, and long-standing persistent were included.

Diabetes mellitus was defined as 2 values of fasting plasma glucose $>$126 mg/dl, or an abnormal oral glucose tolerance test, with a 2-h serum glucose after the ingestion of 75 g of glucose of $>$198 mg/dl.[11]