Syncope as the Sole Manifestation of Acute Right Ventricular Myocardial Infarction in A Non-Diabetic Patient

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Abstract

Syncope is a symptom defined as a transient, self-limited loss of consciousness, usually leading to a fall. The causes of syncope are numerous, but life-threatening conditions should be identified as the first priority in order to avoid a catastrophic outcome. Here we report a non-diabetic patient who presented syncope as the sole manifestation of a silent acute right ventricular myocardial infarction (RVMI). The cause of syncope in this patient was assumed to be hypoperfusion of sinus node artery arising from the right coronary artery, hence causing transient sinus node dysfunction. Syncope could be the sole manifestation of RVMI complicated with sinus node dysfunction. Lead II in ECG monitoring itself could only provide limited information and a panel of 12-lead ECG is required in all patients presenting with syncope, even in the absence of typical symptoms of acute coronary syndrome or hemodynamic instability. Continuous ECG monitoring and prolonged ED observation in a syncopal patient without immediately identifiable causes is warrant to avoid such potentially life-threatening condition.(J Intern Med Taiwan 2008; 19: 523-526)

Key Words ： **Syncope, Acute myocardial infarction, Sick sinus syndrome, Bradyarrythmia, Emergency department**

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Introduction

Syncope is a symptom defined as a transient, self-limited loss of consciousness, usually leading to a fall. The causes of syncope are numerous, but life-threatening conditions should be identified as the first priority in order to avoid a catastrophic outcome. Here we report a non-diabetic patient who presented with syncope as the sole manifestation of a painless acute right ventricular myocardial infarction (RVMI).

Case report

A 49 year-old healthy man presented to the emergency department (ED) because of syncope. The bystanders reported that he had a brief convulsion, upward gaze and urinary incontinence but regained consciousness briskly within 2 minutes. No prodromal symptoms, chest pain or crushing tightness, palpitation, tongue biting and postictal confusion was noticed. On arrival, he was oriented and stated having the first episode of near-syncope without any accompanying symptoms 6 hours earlier. He denied having any systemic illness or recreational drug use. He smoked cigarette 1 pack per day since the age of 18 years. He had no history of epilepsy and head trauma. His vital signs included blood pressure of 148/66 mmHg, respiratory rate 16 per minute, and pulse rate 80 beat per minute. The conjunctiva was not pale and the lung was clear to auscultation. The remaining physical and neurologic examinations were without abnormalities. While waiting for the neurologist consultation, he was put on the electrocardiographic (ECG) monitoring. He developed another episode of syncope and the simultaneous ECG monitoring showed 6 seconds of sinus pause followed by junctional escape rhythm with a rate of 30 bpm. His blood pressure dropped to 60/34 mmHg then. He responded promptly to repeated intravenous atropine 0.5 mg twice and fluid resuscitation. A transcutaneous pacemaker was prepared and no inotropic agent was required. A 12-lead ECG showed deep Q wave and ST

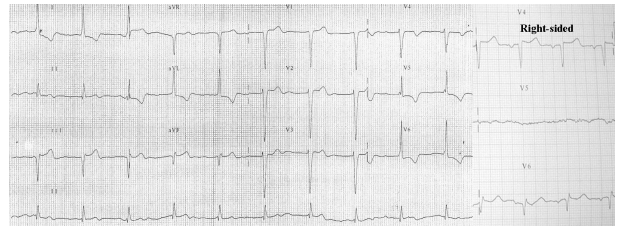

Fig.1.ECG obtained in ER

elevation in lead II, III and aVF as well ST elevation in V4R in right-sided ECG (Fig. 1), indicated RVMI. Elevated serum creatine kinase (514 U/L), creatine kinase MB (64 U/L) and troponin-I (7.14 ng/mL) further confirmed the diagnosis. He was treated with aspirin, unfractionated heparin and clopidogrel accordingly. A trans-thoracic echocardiogram demonstrated hypokinesia of the right ventricular and apical wall. No ventricular septal defect, rupture of papillary muscle or flail mitral valve was noted. The coronary angiogram demonstrated total occlusion of the proximal third of right coronary artery and percutaneous angioplasty to the culprit lesion was performed subsequently. The cause of syncope in this patient was assumed to be hypoperfusion of sinus node artery arising from the right coronary artery, causing transient sinus node dysfunction. On follow-up at 6 weeks, he was well and required no permanent pacemaker implantation.

Discussion

Here we present a non-diabetic male patient who developed episodes of syncope as the sole manifestation of painless RVMI complicated with SSS. The causes of syncope are either cardiac (4-36%) or non-cardiac (17-52%) or remain unexplained (13-48%)[1,2]. In those of cardiac origin, arrhythmias are present in 86.5%, with ventricular tachycardia been the most frequent (36.8%). Less common are the sick sinus syndrome associated with bradycardia (15.7%), supraventriculr tachycardia (10.8%) and blocks of AV-pathway (8.6 %). Prinzmetal's angina with prolonged asystole had been also reported as the cause

of syncope[3]. History taking is the corner stone of the evaluation of a patient with transient loss of consciousness[4]. Clinical features suggestive of cardiogenic syncope include presence of severe structural heart disease; attacked during exertion, or supine; proceeded by palpitation or accompanied by chest pain; and family history of sudden cardiac death. The evaluations of a patient with syncope consist of careful history, physical examination, including orthostatic blood pressure measurement and a standard 12-lead ECG. Certain ECG presentations in patients with syncope will not only provide a reason for the loss of consciousness but also guide early therapy and disposition of these individuals. In addition, identification of certain ECG morphologic findings, including ST-segment and T-wave abnormalities of acute coronary syndrome; ventricular preexcitation as seen in the Wolff-Parkinson-White syndrome; right bundle branch block pattern with ST-segment elevation in the right precordial leads in Brugada syndrome; prolonged QT interval in the diverse long QT interval syndrome; and right ventricular hypertrophy in hypertrophic cardiomyopathy, are valuable in the acute care setting[5]. Predictors of arrhythmia or 1-year mortality in ED patients presenting with syncope include: age greater than 45 years, presence of an abnormal ED ECG and histories of ventricular arrhythmias or congestive heart failure[6].

Syncope could be the initial complaint in 5-12% of patients with acute myocardial infarction (MI) and is related to arrhythmia or pump failure. Vasovagal reactions, bradyarrythmia and atrio-ventricular blocks can be caused by ischemia. Patients suffering from acute MI are susceptible to neurally mediated syncopal or presyncopal attacks. Sympathetic withdrawal seems to be one of the most likely mechanism of syncope in patients with acute MI. The symptoms of acute MI may be affected by gender, age, smoking, infarction size, hypertension, diabetes and hypercholesterolemia. Women with diabetes represent a high-risk subgroup for painless onset followed by various other symptoms. The proportion of painless MI is greater in patients with diabetes mellitus, and it increases with age. Patients with painless MI are at risk for delays in seeking medical attention, less aggressive treatments and in-hospital mortality. Many patients with diabetes suffer from an autonomic dysfunction that impairs their quality of life and predisposes to life-threatening cardiovascular complications. Neuropathy and dysfunction of autonomic nerve fibers supply the myocardium might be responsible for the altered pain sensitivity. Other less common presentations, with or without pain, include sudden loss of consciousness, a confusional state, a sensation of profound weakness, the appearance of an arrhythmia, evidence of peripheral embolism, or merely an unexplained drop in arterial pressure. It is also worth noting that painless MI has a strong predictive value for all cardiac events and in particular for major adverse cardiac events in diabetic patients. RVMI developed in approximately 30% of patients with inferior MI and the triad of jugular vein distension, clear lung sounds, and hypotension are hallmarks. Patients with RVMI have higher risk of death and greater major complications. ST elevation in lead V4R in a right-sided ECG is needed for the diagnosis of RVMI. Asymptomatic RVMI had been occasionally reported. Utilization of ECG monitoring has resulted in a significant reduction of deaths from arrhythmias during acute MI. Sudden death outside the hospital, however, continue to be a major cause of premature mortality in patients with arteriosclerotic heart disease.

In summary, syncope could be the sole manifestation of a painless RVMI complicated with SSS. Though Lead II in ECG monitoring itself could provide only limited information, continuous monitoring is mandatory in patients with higher risks for cardiogenic syncope. A panel of 12-lead ECG is required in all patients presenting with syncope, even in the absence of typical symptoms of acute coronary syndrome or hemodynamic instability. When there are

clinical features that raise the suspicion of cardiac syncope, continuous ECG monitoring and prolonged ED observation in a syncopal patient without immediately identifiable causes is warrant to avoid such potentially life-threatening condition.

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昏厥爲一非糖尿病病患出現右心室心肌梗塞之唯一臨床表現

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摘 要

昏厥（syncope）係指自限性的，短暫的意識喪失，以致無法保持姿勢。造成昏厥的原因很多，但是有潛在生命危險的狀況應予早期診斷。在此我們報告一男性非糖尿病病患以昏厥爲其右心室心肌梗塞之唯一症狀，探討其造成昏厥的原因爲的右側冠狀動脈竇房結動脈分支缺血所造成的一過性竇功能失調。完整的12導程心電圖應爲昏厥病患之基本檢查。在高危險的昏厥病患猶應提供持續的心電圖監視及較長時間的留觀以避免耽誤治療。