Evaluation of the patients with syncope during the first month after coronary artery bypass graft

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ABSTRACT

Background: Syncope is a well-known risk factor for adverse cardiovascular event in patients with coronary artery disease, especially those with previous myocardial infarction (MI) or left ventricular dysfunction. The aim of this study was to assess electrophysiologic findings and results of head-up tilt test (HUTT) in patients with syncope and without orthostatic changes in blood pressure during the first month after coronary artery bypass graft (CABG). **Materials and Methods:** A total of 20 patients with syncope during the first month after CABG were prospectively enrolled in this study from June 2002 to April 2006. Electrophysiologic study (EPS) was performed in all of them. HUTT was performed in all of the patients regardless of the result of EPS. **Results:** The mean age of patients was 60.3±11 years. Twelve patients were males. EPS was negative in 18 patients. HUTT was positive in 10 patients. Six patients had old MI. Ischemic insult occurred in one patient after CABG. Left bundle branch was present in two patients. There was a significant relationship between the duration of bed rest after CABG and positive HUTT (*P* value = 0.021). All of the patients except one did not experience syncope during the follow-up period. **Conclusion:** In patients with syncope during the first month post CABG, in whom an arrhythmic cause is suspected, the other cause of syncope like orthostatic intolerance should be considered. Being bedridden for an extended period of time post CABG can be a predisposing factor.

Key words: Coronary artery bypass graft, electrophysiologic study, head-up tilt test, syncope

INTRODUCTION

Syncope represents a great challenge to the electrophysiologists. Syncope is an important risk factor for adverse cardiac event in patients with ischemic heart disease, especially those with myocardial infarction (MI) and/or left ventricular (LV) dysfunction and ventricular conduction defects.[1-4] The initial evaluation of a patient with syncope consists of history, physical examination including orthostatic blood pressure measurement and electrocardiography (ECG). Initial evaluation may lead to diagnosis based on symptoms, signs, and ECG findings (e.g., situational syncope, orthostatic syncope, arrhythmia-related syncope, i.e., complete heart block, rapid supraventricular tachycardia, or ventricular tachycardia). There are many studies about syncope in patients with various structural heart diseases. Syncope may occur after coronary artery bypass graft (CABG) and often requires hospital admission to rule out the acute and life-threatening events. First episode of syncope or recurrence of syncope may occur after CABG due to hemodynamic collapse. This may be related to changes in blood pressure or autonomic failure. MI and ischemic event may be the other cause of syncope. There is not any study about the results of electrophysiologic

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DOI: 10.4103/0975-3583.85267

studies and clinical significance of syncope in this group of patients. The present study assessed the electrophysiologic study (EPS) and head-up tilt test (HUTT) results in patients with syncope during the first month after CABG.

MATERIALS AND METHODS

Study population

Among eight thousand patients with CABG, 50 patients were admitted in our hospital from June 2002 to April 2006, who had syncope within the first month after CABG. All of them underwent standard workup including history, physical examination, 12-lead ECG, 24-hour Holter monitoring, echocardiography, carotid sinus massage, and neurological evaluation (history and neurological examination). Exclusion was made on those with orthostatic changes in hemodynamic (n = 30). EPS was done in the patients without orthostatic changes in blood pressure. Orthostatic hypotension was defined by a fall in pressure response of >20 mm Hg systolic and/or >10 mm Hg diastolic in response to moving from supine to standing position after 3 minutes. They were followed for two years after initial workup.

Electrophysiologic study

The EPS included measurement of corrected sinus node recovery time, HV interval in baseline and during incremental pacing, inducibility of ventricular arrhythmia by means of programmed ventricular stimulation utilizing two drive cycle lengths (600 ms, 400 ms) up to three extrastimuli. Procainamide was infused in all patients. EPS was considered diagnostic in the presence of (1) an abnormal sinus node recovery time; (2) baseline HV interval ≥100 ms, second- or third degree His-Purkinje block demonstrated by incremental atrial pacing or elicited by intravenous procainamide (10 mg/kg over 10 minutes); and (3) induction of sustained monomorphic ventricular tachycardia (SMMVT) or induction of rapid supraventricular tachycardia that reproduced symptoms.

Head-up tilt test

HUTT was performed in all of the patients. The patients received no oral intake for more than 6 hours before HUTT. An intravenous line was placed for fluid administration. At least 20-minute resting period after IV line placement, the patient was elevated to 70˚ or until syncope occurred. Sublingual nitroglycerin (400 μg) was used for drug provocation if passive phase had been negative. Duration of drug provocation phase was 15 minutes. The test was considered positive in case of bradycardia, hypotension (systolic blood pressure <70 mm Hg), and syncope.

Statistical analysis

All data were entered into the SPSS database (SPSS 13, Inc, Chicago, IL, USA). The continuous variables were expressed as mean ± SD. Parametric (*t*-test) test was used for comparison. Categorical variables were compared by chi-square test. Logistic regression analysis was used for multivariable analysis. $P<0.05$ was considered statistically significant.

RESULTS

Demographics and clinical presentation

Clinical characteristics of patients are shown in [Table 1]. The mean age of patients was 60.3 ± 11 years. Ten patients with normal EPS and two with abnormal EPS were males. Six patients had old MI. Syncope occurred in 50 patients during the first month after CABG. One patient had syncope before CABG. Thirty patients had orthostatic hypotension after undergoing initial workup. Ischemic insult occurred in one patient after CABG.

Results of the head-up tilt test

HUTT was positive in 10 (50%) patients [Tables 2 and 3]. Four patients had positive HUTT during passive phase. The vasodepressive type was noted in four patients, the cardioinhibitory type in three patients, and the mixed type in three patients. Salt and water ingestion, isotonic exercise, elastic housing, and tilt training were recommended in patients with a positive HUTT response. There was

Table 1: Baseline characteristics of patients

	Abnormal EPS	Normal EPS
Age (year)	60±0.1	60.7± 6
Sex (male, n)	2	10
MI (n)	2	4
LVEF (%)	37.5±3.5	47±6
Bed rest (n)*	-	14
Syncope before CABG (n)	1	
Drugs(n)	-	-
Beta-blockerö	2/2	18/18
Aspirin	2/2	18/18
ACE inhibitor	2/2	18/18
Statin	2/2	18/18
Antiarrhythmic drugs	-	-

MI: Myocardial Infarction, LVEF: Left ventricular ejection fraction, CABG: Coronary artery bypass graft, ACE inhibitor: Angiotensin-converting enzyme inhibitor more than 3 days of bed rest after discharge from hospital

Table 2: Results of paraclinical investigations

Abnormal EPS (n)	2
Positive HUTT (n)	10
Echocardiography (LVEF, mean ±SD, %)	46±6.5
ECG (LBBB, n)	2
His-Purkinje disease (n)	3

EPS: Electrophysiologic Study, HUTT: Head-up tilt test, LVEF: Left ventricular ejection fraction, LBBB: Left bundle branch block

Table 3: Type of response in patients with positive HUTT

Results of HUTT	HUTT phase (n, passive/ active)	Number of patients	Resting heart rate <60 bpm
Cardio-inhibitory	3/-	3	3
Mixed type	2/1	3	-
Vasodepressor	2/2	4	-

a significant relationship between the duration of bed rest after CABG and a positive HUTT response (*P* value = 0.021).

Electrophysiologic characteristics

All of the patients had sinus rhythm at baseline and two patients had left bundle branch block. One episode of non-sustained VT was seen in a patient with relatively good LV function during 24 hours Holter monitoring. EPS was done in all patients with syncope after exclusion of orthostatic hypotension. Result of EPS was negative in all of them except two. All of the patients with negative EPS were free of syncope during the follow-up period. Two patients had inducible SMMVT during EPS. Both of them had LV ejection fraction >35%. They underwent ICD implantation according to the ACC/AHA/ESC 2006 guidelines regarding the risk for cardiac death. One of them had anteroseptal MI and syncope before CABG and one episode of syncope two weeks after CABG. He had appropriate ICD discharge during the follow-up period. The second patient had MI after CABG and one episode of syncope ten days after CABG. He was free of the syncope and ICD discharge despite the inducible SMMVT during EPS.

DISCUSSION

Syncope in patients with coronary artery disease, especially those with MI or LV dysfunction, may have cardiac causes. First episode of syncope or recurrence of syncope may occur after CABG due to hemodynamic collapse. This may be related to changes in blood pressure or autonomic failure. MI and ischemic event may be the other cause of syncope. This study demonstrated that patients with syncope during the first month after CABG without recent ischemic insult usually have noncardiac syncope, especially those with prolonged bed rest after CABG. In our series, patients who developed syncope and sinus bradycardia after CABG were more prone to vasovagal syncope, especially cardioinhibitory type. On the other hand, in patients who had syncope before CABG or new transmural MI after CABG an arrhythmic cause was highly likely. So, EPS indicated in these patients and induction of SMMVT was considered endpoint for ICD implantation. Fourteen patients were in bed for a few days after hospital discharge due to false cultural believe. We suggested that deconditioning is a cause of syncope during first month after CABG in this patient group. Early ambulation of the patients after CABG in and out of hospital is necessary for normal autonomic system function and control of the hemodynamic. Patients who have been restricted to the bed rest following CABG routinely develop orthostatic hypotension or frank syncope during their initial attempt at ambulation. Orthostatic intolerance and reduced exercise capacity secondary to the bed rest is accompanied by reduced circulating blood volume, lower cardiac output, attenuated cardiac baroreflex responses, and limited baroreflex-mediated vasoconstrictive reserve.[5] Prolonged period of bed rest and physical inactivity removes baroreceptor unloading caused by regular upright standing and induces attenuation of cardiovascular baroreflex responses. The magnitude of reduced baroreflex sensitivity following bed rest is related to the degree of orthostatic hypotension. Reduction in vascular volume caused by bed rest or progressive hypovolemia does not affect carotid-cardiac baroreflex function. In contrast, intense exercise that increases arterial baroreceptor loading causes an acute increase in carotid baroreceptor sensitivity and has been associated with enhanced orthostatic stability following exposure to simulated microgravity. Endurance exercise training designed to enhance orthostatic stability was associated with increased blood volume and vasoconstrictive reserve, but no change in the carotid baroreflex response.[6] Calf vein compliance increases following bed rest after aortocoronary bypass surgery. Late postoperative ambulation (>7 days) increases the risk of orthostatic hypotension.[7] But alterations in venous compliance do not play a major role in reduction of exercise capacity.[8] Deconditioning changes affecting the cardiovascular system are decreased venous flow, decreased orthostatic tolerance, and decreased work capacity.[9] According to the result of this study, some patients after CABG may develop syncope despite the absence of orthostatic changes in heart rate and blood pressure. They

are those patients who were restricted to the bed rest more than 3 days after hospital discharge. Majority of syncope episodes are due to transient change of autonomic function and have a good prognosis.

Study limitation

The major limitation of this study was the small sample size. Thus, further evaluation of syncope in these patients requires a large multicenter study.

As in other studies of syncope, an abnormal EPS or HUTT was considered to be a surrogate of the true cause of syncope. The etiology of syncope is difficult to establish. Although the patients with orthostatic changes in blood pressure were excluded, the concomitant EP abnormalities could not be precluded. The clinical significance of syncope in this group of patients (with a negative EPS and a positive HUTT) should be clarified, in the future, by a large study with a long-term follow-up.

CONCLUSION

Syncope in patients without recent ischemic insult is a rare symptom during the first month after CABG. In selected patients with syncope during the first month post CABG, in whom an arrhythmic cause is suspected, the other cause of syncope like orthostatic intolerance should be considered. Being bedridden for an extended period of time post CABG can be a predisposing factor.

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How to cite this article: Alizadeh A, Kiavar M, Assadian-Rad M, Morady B, Alasti M, Sadeghi A, Emkanjoo Z. Evaluation of the patients with syncope during the first month after coronary artery bypass graft. J Cardiovasc Dis Res 2011;2:186-9.
Source of Support: Nil, **Conflict of Interest:** None declared.