



Original article

Favorable outcomes of patients with vasospastic angina associated with cardiac arrest



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ABSTRACT

Background: The long-term survival of vasospastic angina (VSA) patients is generally good, so long as they remain on calcium channel blockers (CCBs) and avoid smoking. However, the pathogenesis, appropriate treatments, and prognosis of VSA associated with cardiac arrest remain unclear. This study aimed to elucidate the clinical features and long-term outcomes of patients with VSA associated with cardiac arrest.

Methods and results: Eighteen consecutive patients with VSA associated with cardiac arrest [13 patients resuscitated after out-of-hospital cardiac arrest (OHCA) and 5 resuscitated after in-hospital cardiac arrest] were retrospectively analyzed. Sixteen of the eighteen patients were smokers. None had other cardiac diseases possibly causing cardiac arrest. Although 1 patient resuscitated after OHCA later died of cerebral hypoxia, the remaining 17 were discharged without complications. One patient died of cancer 50 months after resuscitation. The other 16 patients are still alive and none have shown ventricular arrhythmias, syncope, or cardiac arrest during a mean follow-up of 67 months. All are treated with long-acting CCBs/nitrates and successfully quit smoking. Six patients received implantable cardioverter defibrillators (ICD). However, none demonstrated any ventricular arrhythmias and appropriate ICD actuation was achieved.

Conclusion: Appropriate medical treatment can achieve favorable long-term outcomes even for patients with VSA associated with cardiac arrest.

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Introduction

Vasospastic angina (VSA) is one of the causes of chest pain without coronary artery stenotic lesions. The long-term survival of VSA patients is generally good, so long as they remain on calcium channel blockers (CCBs) and avoid smoking [1]. However, recent clinical studies have demonstrated that VSA might play an important role in the pathogenesis of unexpected sudden cardiac arrest (SCA) [2–11]. Takagi et al. reported that VSA patients who survived out-of-hospital cardiac arrest (OHCA) were a high-risk population [2]. Therefore, decision-making on appropriate therapy including implantable cardioverter-defibrillators (ICD) for VSA patients resuscitated from SCA is an important topic in current clinical investigations. This study aimed to elucidate the clinical

features and long-term outcomes of patients with VSA associated with SCA in order to determine appropriate treatments for this specific patient population.

Methods

This was a retrospective, observational study in a single emergency medical care center in Japan. The present study was approved by our institutional ethics committee. Informed consent from individual patients was not required under Japanese law as the present study was purely observational. Our hospital is a tertiary care referral center with intensive care units, coronary care units, and a coronary intervention facility available 24 h a day, 7 days a week. In total, 18 VSA patients resuscitated after SCA between 1992 and 2012 were identified in our hospital records. Thirteen patients were resuscitated after OHCA. The other five patients were resuscitated after in-hospital cardiac arrest; two patients (#14, 17) had been admitted to our hospital to receive detailed evaluation of unexplained syncope, two (#15, 18) had been admitted to undergo non-cardiac operations, and one (#16) had been admitted for

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diabetes mellitus control. All patients had angiographically normal coronary arteries. All patients fulfilled the following criteria as described in previous studies [4–9]: (1) documented ventricular fibrillation (VF) or sustained rapid ventricular tachycardia (VT) or pulseless electrical activity; (2) the absence of significant narrowing due to coronary atherosclerosis or any structural cardiac abnormalities possibly causing cardiac arrest; (3) no previous history of angina pectoris or acute myocardial infarction; (4) absence of identifiable or reversible causes of lethal ventricular arrhythmias. A definite diagnosis of VSA was made in all patients according to the Guidelines for Diagnosis and Treatment of Patients with Vasospastic Angina of the Japanese Circulation Society [12]. VSA was considered to be the cause of SCA if one of the following features was noted: (1) spontaneous transient ST segment elevation around the time of resuscitation; (2) spontaneous coronary artery spasm during the emergent coronary angiogram obtained immediately after resuscitation; (3) a positive provocation test for coronary artery spasm and negative results of an electrophysiological study (EPS) to exclude idiopathic VF. The acetylcholine (ACh) provocation test was performed according to the method included in the Guidelines for Diagnosis and Treatment of Patients with Vasospastic Angina of the Japanese Circulation Society [12]. The protocol for EPS has already been described [3,8,13,14]. Briefly, the EPS protocol included burst pacing of up to 240 bpm and programmed stimulation with up to 3 extra stimuli (minimum 200 ms) with basic cycle lengths of 600 and 400 ms from 2 sites in the right ventricle with and without isoproterenol infusion. EPS was identified as negative when sustained ventricular tachyarrhythmias were not induced. Clinical characteristics, past medical history, situation at the time of SCA, documented initial rhythm at the time of SCA, diagnostic basis of SCA due to VSA, therapeutic contents post-resuscitation, and follow-up data were evaluated in each patient.

Results

Demographic and clinical data of the studied patients are outlined in Table 1. Fifteen patients (83%) were men and 16 (88%) were active smokers. Three patients (#9, 14, 17) were prescribed relatively low doses of CCBs (diltiazem 60 mg/day in 1 patient and amlodipine 5 mg/day in 2 patients) for treatment of hypertension

prior to SCA. Two patients (#14, 17) had a history of unexplained syncope. Chest pain as a prodromal symptom of SCA was documented in 7 patients (38%). Fourteen patients (77%) had suffered SCA while at rest. Two patients (#15, 18) had suffered SCA during a non-cardiac operation. VF as the initial rhythm at SCA was documented in most patients (16 patients, 88%).

Diagnostic bases of VSA associated with SCA in the present study population are listed in Table 2. Angiographically normal coronary arteries were confirmed by standard coronary angiography in all patients. No one showed Brugada type electrocardiograms. Spontaneous ST elevation around the time of SCA was documented in 9 patients (50%). Furthermore, ST elevation transitioning to VF was documented in 8 of these 9 patients. Spontaneous coronary artery spasm was documented in 3 other patients (16%) during emergent coronary angiography (CAG) immediately after resuscitation. These 3 patients demonstrated severe coronary spasm involving the entire right coronary artery. Intracoronary injection of isosorbide dinitrate (ISDN) relieved the spasm in all 3 patients. The remaining 6 patients (33%) were diagnosed based on positive results of the ACh provocation test. Idiopathic VF was excluded by EPS in 3 of these 6 patients. The other 3 patients refused EPS.

Treatments and clinical courses in all patients are summarized in Table 3. All patients were followed at our hospital. Although patient #13 died of cerebral hypoxia 7 days after resuscitation, the remaining 17 patients were discharged without complications. All discharged patients achieved cessation of smoking. Daily multiple doses of long-acting (24 h) CCBs were prescribed for most patients. Only 1 patient (#12) was prescribed a daily single dose of amlodipine because of intolerance to multiple doses of other CCBs. ISDN was prescribed for 14 patients and nicorandil for 8. Although 2 patients (#5,9) complained of chest pain during follow-up because of discontinuation of the medication, none showed evidence of coronary spasm recurrence while continuously medicated. Patients #5 and 9 resumed taking medications. Six patients underwent ICD implantation at their request. However, none showed ventricular arrhythmias. Therefore, the ICD was not activated. One patient died of cancer 50 months after resuscitation. The other 16 patients are still alive and none has developed ventricular arrhythmias, syncope, or cardiac arrest during a mean follow-up of 67 months.

Table 1
Demographic and clinical data at resuscitation.

Patient number	Age (yrs) and sex	Coronary risk factors	Prescribed CCBs prior to SCA	History of syncope	Prodromal symptom	Situation at SCA	Rhythm at SCA	LVEF (%)
Survivors from out-of-hospital cardiac arrest								
1	44 (M)	Smoking	0	0	Chest pain	At rest (morning)	VF	60
2	58 (F)	HT	0	0	Syncope	At rest (morning)	VF	80
3	55 (M)	Smoking	0	0	Syncope	At rest (day time)	VF	65
4	41 (M)	Smoking	0	0	Syncope	At rest (morning)	VF	65
5	68 (M)	Smoking/HT	0	0	Syncope	At rest (evening)	PEA	75
6	70 (F)	Smoking/HT/DL	0	0	Chest pain	At rest (morning)	PEA	87
7	44 (M)	Smoking	0	0	Chest pain	At rest (morning)	VF	66
8	46 (F)	0	0	0	Chest pain	At rest (morning)	VF	60
9	70 (M)	Smoking/HT	Diltiazem/60 mg ^a	0	Chest pain	At rest (morning)	VF	52
10	49 (M)	Smoking/HT	0	0	Syncope	At rest (day time)	VF	75
11	58 (M)	Smoking/HT	0	0	Nausea	At rest (day time)	VF	60
12	48 (M)	Smoking/DL	0	0	Syncope	During exercise	VF	67
13	59 (M)	Smoking	0	0	Syncope	During effort	VF	75
Survivors from in-hospital cardiac arrest								
14	65 (M)	Smoking/HT	Amlodipine/5 mg ^a	+	Chest pain	At rest (morning)	VF	64
15	65 (M)	Smoking	0	0	0	During operation	VF	64
16	64 (M)	Smoking/DM	0	0	Chest pain	At rest (morning)	VF	65
17	62 (M)	Smoking/HT	Amlodipine/5 mg ^a	+	Syncope	At rest (morning)	VF	76
18	77 (M)	Smoking	0	0	0	During operation	VF	70

CCBs, calcium-channel blockers; DL, dyslipidemia; DM, diabetes mellitus; F, female; HT, hypertension; LVEF, left ventricular ejection fraction; M, male; PEA, pulseless electrical activity; SCA, sudden cardiac arrest; VF, ventricular fibrillation.

^a CCBs were prescribed for hypertension in those patients.

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