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Sustained Ventricular Tachycardia During Dobutamine Stress Echocardiography

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ABSTRACT

Dobutamine stress echocardiography (DSE) has been frequently performed safely and renders important diagnostic information of underlying myocardial ischemia in patients with coronary artery disease. During DSE, death or other major events, such as myocardial infarction and sustained ventricular tachycardia (VT), have been extremely rare. We herein present three patients who developed three different types of sustained VT during DSE. These clinical observations indicate that various mechanisms may be involved in the development of this malignant arrhythmia during DES.

INTRODUCTION

Dobutamine stress echocardiography (DSE) has been proven to be an accurate and safe diagnostic procedure for the detection of myocardial ischemia. No death has ever been reported during a DSE study, whereas other major events, such as myocardial infarction and sustained ventricular tachycardia (VT) have been extremely rare [1,2]. Clinical predictors of occurrence of sustained VT during DSE have not been clearly defined. We present three patients who developed three different types of sustained VT during DSE. This clinical observation indicates that various mechanisms may be responsible for the development of this malignant arrhythmia during DES.

CASES

Patient 1

The first case is a 48-year-old man, smoker, with hypertension, hyperlipidemia, and family history of coronary artery disease, who presented to our outpatient clinic complaining of an atypical chest discomfort developed during the preceding week. The patient had a stress echocardiography study with continuous dobutamine infusion in five stages of incremental dosages (10-50 mcg/kg/min) for the detection of myocardial ischemia. Baseline electrocardiogram (ECG) was negative for ischemia. At the 30 mcg/kg/min stage of dobutamine infusion, the patient had a significant arterial blood pressure increase (220/120 mmHg) and reported severe retrosternal chest pain. Twelve-lead ECG showed ST-segment elevation in leads II, III, avF, V5 and V6, and ST-segment depression in leads I, avL and V2 (Figure 1, A), whereas both the inferior and the posteroseptal myocardial segments became akinetic. Nitrates and atenolol were administered intravenously. Patient's angina as well as the ST-segment changes resolved after 5 minutes and this was followed by a self-terminated episode of accelerated idioventricular rhythm (Figure 1, B). At that point, the patient was

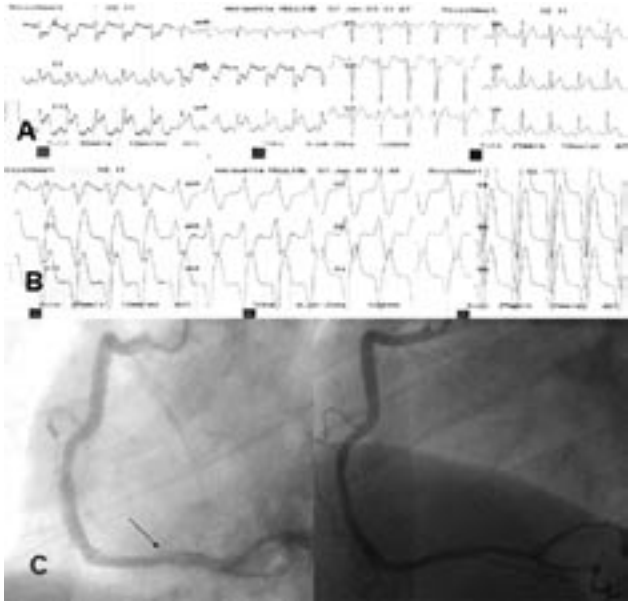

FIGURE 1. A: ST segment changes at the onset of angina during stress echocardiographic study. B: Idioventricular rhythm when angina began to relieve. C: Minor dissection of the distal part of the right coronary artery (arrow, left panel). Two days later complete restoration of the dissected right coronary artery wall (C, right panel).

free of any symptoms whereas the ECG was entirely normal. A coronary angiogram, performed the same day, revealed a localized, non-obstructive dissection at the distal part of the right coronary artery (Figure 1, C left panel). Since there was TIMI III flow in the artery, a conservative therapeutic strategy with the administration of enoxaparin and a IIb/IIIa platelet receptor antagonist was followed. Two days later, a repeat coronary angiogram showed complete restoration of the vessel lumen (Figure 1, C right panel).

Patient 2

The second case is a 54-year-old lady, smoker, with hypertension, and hyperlipidemia (LDL=250 mg/dl), totally asymptomatic who had a treadmill stress test as a part of her annual check up. At the third stage of the Bruce prorocol (8th min), she developed a 2-mm ST-segment depression in leads II, III, and aVF without any symptoms. Her physician suggested that she should have another stress test with an imaging modality and she was referred to us for a DES study. The study was done according to our routine protocol as mentioned above. At the 30 mcg/kg/min stage of dobutamine infusion, the patient started complaining of a constricting retrosternal chest pain, whereas a marked ST-segment elevation was displayed on the rhythm monitor of the echocardiographic equipment and the inferior wall became akinetic. Dobutamine infusion was discontinued immediately and we administered atenolol and nitrates. However, the patients had no clinical or ECG improvement and we decided to perform a coronary angiogram. While she was waiting to be transported to the catheterization laboratory she developed an episode of polymorphic VT with subsequent ventricular fibrillation (Figure 2, A). The patient was successfully cardioverted to sinus rhythm with a DC shock (300 joules). A coronary angiogram, performed the same day, revealed a non significant stenosis of the left circumflex artery (Figure 2, B) which was also confirmed by fractional flow reserve measurement (0.90).

Patient 3

Finally, our third case is an 80-year-old man with hypertension and a known history of an anterior myocardial infarction 20 years earlier resulting in left ventricular systolic dysfunction (ejection fraction=27%). He had been submitted to revascularization with bypass surgery twice in the past: 20 years earlier he had received venous grafts to the left anterior descending (LAD) and circumflex, and 7 years ago he had a redo operation with a left internal mammary artery grafted to the LAD coronary artery. Over the last year the patient had experienced many episodes of exertional angina, however his clinical status further deteriorated over the last month with episodes of angina even at rest. The patient was referred for a dobutamine stress echo. Up to the 20 mcg/kg/min stage of dobutamine infusion, systolic thickening was not observed in anterior, anteroseptal and apical myocardial segments which remained akinetic, whereas the rest of the ventricular wall demonstrated normal thickening without any evidence of

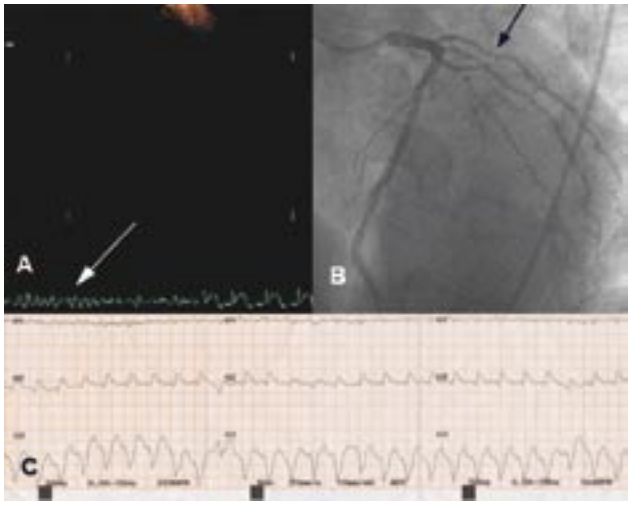

FIGURE 2. A: Polymorphic ventricular tachycardia which resulted in ventricular fibrillation (arrow). B: Coronary angiogram revealed a non significant stenosis of left circumflex artery (arrow). C: Asymptomatic sustained monomorphic ventricular tachycardia at the end of the 20 mcg/kg/min stage of dobutamine infusion.

ischemia. At the end of the 20 mcg/kg/min stage of dobutamine infusion, the patient developed an episode of sustained monomorphic VT without any symptoms or hemodynamic compromise (Figure 2, C). We initially administered 5 mg of atenolol without any success and tachycardia was finally terminated after infusing 700 mg of procainamide. The patient had a coronary angiogram the same day which revealed total occlusion of the venous grafts, significant stenoses at the distal part of the left internal mammary artery which was totally occluded at the site of anastomosis to the LAD, proximal total occlusion of the LAD and significant stenoses of a left dominant circumflex. Given the fact that the probability of functional recovery of the akinetic myocardial regions was low and that the patient was a high risk surgical candidate for a third operation, revascularization strategy was foregone and we proceeded to an electrophysiology study. Ventricular tachycardia was easily reproduced during the study and the patient finally received an implantable cardioverter defibrillator.

DISCUSSION

The clinical significance and the prognostic importance of DSE-induced VT have not been clearly defined. Ventricular tachycardia induced during DSE may be of different morphology and due to different mechanisms. In the cases we presented, idioventricular rhythm was caused by abrupt coronary blood flow restoration (first case), polymorphic VT was the result of ischemia due to the development of dynamic coronary artery stenosis (second case), and monomorphic VT developed on a reentrant circuit substrate of myocardial scar (third case). Previous studies [2-4] have shown that VT development during DSE in a population investigated for chest pain is not related to induction of ischemia. In patients with normal hearts, exercise-induced VT, which is usually not inducible during an electrophysiology study, can be seen and has been attributed to either catecholamine-sensitive enhanced automaticity, or catecholamine-sensitive delayed afterdepolarizations [5,6]. However, in patients with a previous myocardial infarction, dobutamine-induced ischemia facilitates induction of VT [7]. Nevertheless, we recently demonstrated that sustained monomorphic VT induced during a DSE study performed in an unselected population has very low predictive value for the identification of patients with coronary artery disease [8].

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