Coronary spasm during cardiac electrophysiological study following isoproterenol infusion

NARENDRA KUMAR1,*, ISMAIL AKSOY1,*, KEVIN PHAN2, JINDRA VAINER1, CARL TIMMERMANS1

1 Department of Cardiology, Maastricht University Medical Centre and Cardiovascular Research Institute Maastricht (CARIM), The Netherlands
2 Westmead Clinical School, Sydney Medical School, University of Sydney, Australia

*Corresponding author: Narendra Kumar, MD; Department of Cardiology, Maastricht University Medical Centre and Cardiovascular Research Institute Maastricht, PO Box 6202AZ, Maastricht, The Netherlands; Phone: +31-43-3877095; Fax: +31-43-3875104; E-mail: drnarendra007kr@gmail.com

+These authors have contributed equally to the case report.

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Abstract: Sudden cardiac death (SCD) remains the leading cause of death in industrialized world. The majority of SCD is caused by ventricular fibrillation associated with structural and/or ischemic heart disease. Ventricular fibrillation represents the final common pathway for SCD and, thus, is an attractive target for ablation. According to class I recommendation level of evidence A, an implantable cardioverter defibrillator (ICD) should be implanted for such patients [1]. Other than programmed electrical extrastimulus technique, isoproterenol infusion is commonly used in invasive cardiac electrophysiology labs for arrhythmia induction. We hereby report a rare case of transient coronary spasm during isoproterenol infusion for ventricular tachycardia induction testing.

Keywords: isoproterenol, electrophysiology study, complication, coronary spasm, sudden cardiac death

Introduction

Sudden cardiac death (SCD) remains the leading cause of death in industrialized world. The majority of SCD is caused by ventricular fibrillation associated with structural and or ischemic heart disease. Ventricular fibrillation represents the final common pathway for SCD and, thus, is an attractive target for ablation. According to class I recommendation level of evidence A, an implantable cardioverter defibrillator (ICD) should be implanted for such patients [1]. Other than programmed electrical extrastimulus technique, isoproterenol infusion is commonly used in invasive cardiac electrophysiology labs for arrhythmia induction. We hereby report a rare case of transient coronary spasm during isoproterenol infusion for ventricular tachycardia induction testing.

Case Report

A 47-year-old euglycemic normotensive Caucasian man was admitted to our hospital because of a successful resuscitation of an out of hospital cardiac arrest due to ventricular fibrillation. He was an occasional smoker without any significant past and or family history. The ambulance electrocardiogram (ECG) showed minimal ST elevation of the inferior leads, and a screening echocardiogram later at emergency department revealed normal left ventricular ejection fraction. The coronary angiography, which was done immediately, did not show any significant lesions. An invasive cardiac electrophysiology study was carried out under conscious sedation using Midazolam. Ventricular tachycardia could not be induced after using programmed extrastimulus
technique (up to 3 extrastimuli), ventricular burst pacing (Fig. 1), and isoproterenol infusion up to 2-microgram/minute infusion, leading to a heartbeat increase from baseline 60 later up to 90 beats per minute.

Ten minutes after stopping isoproterenol, the patient complained of chest pain (Canadian Cardiovascular Society grading of angina pectoris class IV) non-radiating type, progressive, and without any sweating. The 12 lead surface ECG showed ST segment elevation in the inferior leads. The ST-T changes were gradual and up to 12 millimeters above baseline (Fig. 2). Other vitals including blood pressure were normal. Figure 2 shows the baseline ECG changing to maximum changes and return to normal.

The patient was treated with sublingual nitroglycerine, and on-call interventional cardiologist was immediately consulted. The pain and the ECG changes decreased after 10 min. He became pain free before the emergency coronary angiography was done which did not show any significant coronary lesion (Fig. 3). The laboratory blood analysis (6 h after the event) revealed an increased levels of aspartate aminotransferase (AST) 68 mmol/L, creatine kinase (CK) 123 mmol/L, and high-sensitive troponin I 20 mmol/L compared to baseline values. In conclusion, the ECG change was due to temporary coronary spasm of the right coronary artery triggered by isoproterenol infusion. As sometimes, there may be late onset of drug action in spite of short half life and effect may persist even up to 30 min.

Patient was treated with standard anti-ischemic medications including isosorbide-5-mononitrate and metoprolol and discharged after ICD implantation.

Discussion

Coronary artery spasm plays an important role in development of angina. Parasympathetic nervous system might play a role in the pathogenesis of coronary spasm and/or angina [2, 3]. Development of coronary artery spasm is believed to result from an imbalance in the autonomic discharge [4, 5], although strong sympathetic stimulation under increased basal parasympathetic activity has been suggested as a possible mechanism. The role of both of them remains controversial in the coronary artery spasm induction [2, 6]. Isoproterenol is commonly used for increasing the heart rate and to enhance sinoatrial and atrioventricular conduction in a cardiac electrophysiology laboratory [7–10]. Isoproterenol can cause coronary vasospastic angina and Eisenberg et al.
Fig. 2. Showing base line surface and intracardiac ECG (at His bundle and right ventricular apex) evolving from early ST segment elevation to maximum ST segment elevation and returning back to normal ST segments during different stages.
reported 2 cases of polymorphic ventricular tachycardia due to coronary spasm, where the spasm was caused by ergonovine infusion [11]. Ming-Jui Hung reported in 2004 a total of 8 patients with coronary artery spasm with isoproterenol [3]. In our case, we report a patient with ventricular fibrillation due to coronary spasm, where the spasm was reinduced by isoproterenol infusion during invasive cardiac electrophysiological testing.

**Conclusions/Implications to Clinical Practice**

During invasive cardiac electrophysiology study, ventricular burst pacing and isoproterenol infusion are used to induce ventricular tachycardia. We need to be extra cautious considering isoproterenol’s tendency to cause coronary spasm.

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