CASE REPORT

Rhinology, 48, 251-, 2010

Cardiac arrest induced by submucosal injection of epinephrine in a patient with variant angina*

Ji Young Lee, Sung Jin Hong, Jin Young Chon, So Young Kwon

Department of Anesthesiology and Pain Medicine, Catholic University of Korea College of Medicine, Seoul, Korea

SUMMARY

This case report describes a 35-year old male who experienced ventricular tachycardia induced by intramucosal injection of epinephrine. Under general anaesthesia with desflurane inhalation, 1.5% lidocaine containing 1:100,000 epinephrine was injected into the nasal mucosa for septoplasty. ST segment elevation and QRS widening occurred after 10 minutes and progressed to pulseless ventricular tachycardia. A sinus rhythm was restored after cardiopulmonary resuscitation with electrical cardioversion. The cardiac enzymes were significantly elevated after the event. Exercise-stress testing and coronary angiography were normal. However, an injection of acetylcholine into the coronary artery provoked vasospasm in the left anterior descending and circumflex arteries. This case illustrates an unusual response to low dose epinephrine with cardiac arrest induced in a patient with undiagnosed variant angina.

Key words: epinephrine, general anesthesia, nasal surgery, variant angina

INTRODUCTION

Variant angina is usually suggested by a history of angina at rest, which is associated with typical ST-segment elevation on ECG. If the history is not clear, recognition during routine preoperative evaluation might not be possible. There are a few case reports of coronary vasospasm associated with anaesthesia or surgery in patients with normal coronary arteries. In these patients, coronary vasospasm could occur anytime during the perioperative period, especially during the induction of general anaesthesia [(1)], epidural anaesthesia [(2)], or autonomic nerve stimulation [(3)]. Only one prior case of coronary vasospasm caused by nasal packing with epinephrine (1:100,000) has been reported in a patient with unrecognized variant angina [(4)]. It occurred during the induction period of inhalation anaesthesia; the patient remained hemodynamically stable during the episode of ventricular tachycardia. Here, we report a case of cardiac arrest induced by intramucosal injection of epinephrine-containing lidocaine solution, for nasal surgery, during general anaesthesia in a patient with variant angina.

CASE REPORT

A 35-year old man, height 167 cm and weight 70 kg, had a tonsillectomy and nasal septoplasty planned. The patient was in good health with no systemic illness, history of hypertension or ischemic heart disease. Preoperative laboratory findings, the chest x-ray and ECG were within normal limits.

In the operating room, the patient's arterial pressure was 100/65 mmHg and the heart rate was 80 beats/min. Anaesthesia was induced with remifentanil 400 μg/hr, propofol 160 mg and rocuronium 50 mg. Tracheal intubation was performed and anaesthesia was maintained with air (50%)-oxygen (50%)-desflurane (4 vol%) and a continuous infusion of remifentanil (400 μg/hr). The patient's hemodynamic status was stable during the tonsillectomy. Fifty minutes after anaesthesia induction, 7.5 ml of 1.5% lidocaine containing 1:100,000 epinephrine was injected into the nasal mucosa for septoplasty. The patient's arterial blood pressure and heart rate increased to 135/85 mmHg and 92 beats/min, but quickly stabilized.

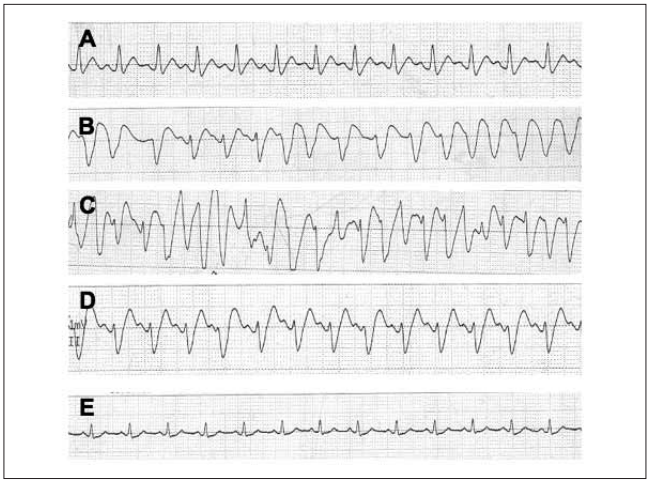

Figure 1. Changes of ECG. A) 10 minutes after epinephrine injection, B) Ventricular tachycardia, C) External cardiac massage, D) After cardioversion (marked QRS widening ST depression, E) Restoration of normal sinus rhythm.

*Received for publication: March 26, 2009; accepted: April 22, 2009

DOI:10.4193/Rhin09.059

About 10 minutes after the local injection, ST-segment elevation was noted in lead II, QRS widening, and frequent ventricular premature contractions were noted on the electrocardiography. Intravenous lidocaine, 50 mg, was administered and a continuous intravenous infusion of isosorbid dinitrate was started. The ventricular arrhythmias subsided for several minutes; however, about 15 minutes after the local injection, the ST-segment elevation was aggravated and the cardiac rhythm progressed to a ventricular tachycardia (Figure 1). The arterial blood pressure could not be obtained. There was no pulse tone at the femoral arteries and no pulse wave on the pulse oximetry.

Cardiopulmonary resuscitation was started with chest compression, 0.5 mg of epinephrine intravenous injection, and electric defibrillation with biphasic 150 J. Lidocaine 80 mg was administered intravenously. A sinus rhythm was restored after the third trial of electrical shock with 1 g of intravenous magnesium sulfate. After restoration of a sinus rhythm, the arterial pressure was 120/90 mmHg and the heart rate was 125 beats/min. The ventricular tachycardia lasted for 8 minutes.

The surgery was cancelled and the patient awakened. A transthoracic echocardiography, performed immediately after the resuscitation, demonstrated no significant abnormalities except for a slight decrease in the anterior wall motion. At the end of anaesthesia, the patient responded to verbal stimulation and spontaneous respirations were restored. However, pinkish frothy sputum emerged from the endotracheal tube and the breath sounds were coarse with rales in both lung fields. Pulmonary oedema was suspected, therefore 5 cmH_2O of positive end expiratory pressure (PEEP) with 70% oxygen was provided. The patient recovered after 1 hour and the endotracheal tube was removed. The blood chemistry studies, drawn after the CPR, demonstrated marked increase of the cardiac enzymes (CPK: 1196 IU/l, CK-MB: 24.9 ng/ml, Troponin I: 5.67 ng/ml).

The patient was referred for cardiology evaluation. The 24-hour ambulatory ECG showed no abnormalities except intermittent T wave inversions in lead V5. The exercise stress test showed normal cardiac functional capacity without arrhythmias or ST segment changes. Coronary angiography showed no arterial narrowing in the resting state. However, more than 90% spasm of the left anterior descending and circumflex arteries was provoked by a 50 μg acetylcholine intra-coronary arterial injection (Figure 2). The diagnosis of variant angina was confirmed. The patient was discharged on a calcium channel blocker and nitrates.

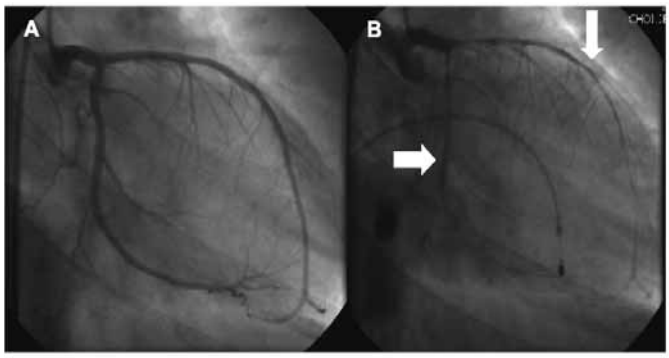

Figure 2. Coronary angiography showed normal coronary arteries. (A) Administration of 50 μg acetylcholine into the coronary artery induced severe stenosis in the left anterior descending and circumflex arteries (B).

DISCUSSION

Patients with Prinzmetal or variant angina tend to be younger than patients with stable or unstable angina secondary to coronary atherosclerosis, and usually do not exhibit classical coronary risk factors on presentation. Variant angina occurs at rest without any precipitating factors such as physical exertion or emotional stress, and can lead to myocardial infarction, life-threatening ventricular arrhythmias and sudden death. The sites of vasospasm may correspond to areas of focal atherosclerosis, however, coronary angiography is apparently normal in some patients; only the provocation test with acetylcholine or ergonovine can confirm the diagnosis [5]. In asymptomatic patients with a normal ECG, the recognition of variant angina during a preoperative evaluation might be impossible. In the present case, the patient had no significant cardiac risk factors and the cardiac functional capacity was normal.

There are few cases of coronary artery spasm reported during surgery and anaesthesia in patients with normal coronary arteries [1-4]. The cause of perioperative coronary artery spasm is unknown. However, altered humoral factors and increased catecholamine responses associated with surgery and anaesthesia are suggested mechanisms. Increases in blood pressure, excessive alpha-adrenergic activity, and stimulation of the parasympathetic nervous system are thought to provoke coronary artery spasm. There are a few reports of coronary vasospasm induced by the therapeutic use of epinephrine [6], where an acute myocardial infarction occurred after administration of epinephrine for the treatment of anaphylactic shock.
Epinephrine is known to induce coronary vasospasm in susceptible patients; the mechanism for this is thought to be alpha-adrenergic receptor mediated. Especially in patients with variant angina, vasoconstrictor stimuli induce generalized vasoconstriction throughout the entire coronary tree, and administration of epinephrine may induce diffuse coronary artery spasm [5]. In the present case, we assume that the infiltrated epinephrine was slowly absorbed into the systemic circulation, which took over 10 minutes, and coronary vasospasm was triggered by the epinephrine.

The judicious use of epinephrine is recommended for the purpose of haemostasis or prolonging the effect of local anaesthet-

ics in patients with heart disease. Recent studies [7,8] have shown that local anaesthesia with epinephrine does not cause additional ischemic risk in patients with coronary artery disease; these studies were performed under local anaesthesia with spontaneous respiration. Our patient was under general anaesthesia with desflurane, and moderately hyperventilated, therefore the volatile anaesthetic agent and hyperventilated state may have contributed to the ventricular tachycardia. Volatile anaesthetics have been known to decrease the threshold of catecholamine-induced ventricular tachycardia [9]. In the present case, the pulseless ventricular tachycardia was sustained and we had to perform cardioversion three times to restore normal sinus rhythm. The epinephrine administered intravenously at the start of CPR may have aggravated the vasospasm.

After the resuscitation, although the ECG, echocardiography, and stress test were normal, the marked elevation of cardiac enzymes supported the diagnosis of an acute myocardial infarction. Pulmonary oedema following the cardiac arrest regressed spontaneously after restoration of normal cardiac function. Variant angina was confirmed by coronary vasospasm provoked by acetylcholine. The mainstay of therapy for variant angina is a calcium antagonist alone or in combination with nitrates; these drugs are known to be extremely effective for the prevention and treatment of coronary artery spasm [5].

In conclusion, we would like to emphasize the importance of cardiac monitoring during surgery and anaesthesia, even in patients with an apparently low risk of cardiac disease. Anaesthetists and surgeons should be aware of that epinephrine use during general anaesthesia might cause cardiac arrest in patients with variant angina.

REFERENCES

1. Sidi A, Dahleen L, Gaspardone A. Coronary vasospasm during anesthesia induction: awareness, recognition, possible mechanisms, anesthetic factors, and treatment. J Clin Anesth 2008; 20: 64-69.
2. Krantz EM, Viljoen JF, Gilbert MS. Pinzmetal's variant angina during extradural anaesthesia. Br J Anaesth 1980; 52: 945-949.
3. Choi SS, Lim YJ, Bahk JH, Do SH, Ham BM. Coronary artery spasm induced by carotid sinus stimulation during neck surgery Br J Anaesth 2003; 90: 391-394.
4. O-Uchi J, Komukai K, Tohyama J, et al. Coronary artery spasm discovered in thorough examination of perioperative VT in a 26-yearold Japanese male. Jpn Heart J 2003; 44: 1021-1026.
5. Cannon CP, Braunwald E. Unstable angina and non-ST elevation myocardial infarction. In: Zipes DP, Libby P, Bonow RO, Braunwald E, eds. Braunwald's Heart disease: a textbook of cardiovascular medicine. 7th ed. Philadelphia: Elsevier Saunders, 2005; 1264-1267.
6. Shaver KJ, Adams C, Weiss SJ. Acute myocardial infarction after administration of low-dose intravenous epinephrine for anaphylaxis. CJEM 2006; 8: 289-294.
7. Neves RS, Neves IL, Giorgi DM, et al. Effects of epinephrine in local dental anesthesia in patients with coronary artery disease. Arq Bras Cardiol. 2007; 88: 545-551.
8. Niwa H, Sugimura M, Satoh Y, Tanimoto A. Cardiovascular response to epinephrine-containing local anesthesia in patients with cardiovascular disease. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001; 92: 610-616.
9. Moore MA, Weiskopf RB, Eger EI 2nd, Wilson C, Lu G. Arrhythmogenic doses of epinephrine are similar during desflurane or isoflurane anesthesia in humans. Anesthesiology. 1993; 79: 943-947.

Sung Jin Hong, MD, PhD
Department of Anesthesiology and Pain Medicine
The Catholic University of Korea, College of Medicine
St. Mary's Hospital
62 Yoidodong Yongdungpogu
Seoul 150-713
Korea

Tel: +82-2-3779-1097 / 1322
Fax: +82-2-783-0368
E-mail: hongs@catholic.ac.kr