J Anesth (2010) 24:761–764
DOI 10.1007/s00540-010-0991-5

CLINICAL REPORT

Efficacy of amiodarone on refractory ventricular fibrillation resistant to lidocaine and cardioversion during weaning from cardiopulmonary bypass in aortic valve replacement for severe aortic stenosis with left ventricular hypertrophy

Yoshihisa Morita · Ju Mizuno · Tatsuya Yoshimura · Shigeho Morita

Received: 17 March 2010 / Accepted: 26 June 2010 / Published online: 28 July 2010

Abstract Intravenous injection of amiodarone, a class III anti-arrhythmic is widely used for persistent refractory arrhythmias. We present a case report suggesting the efficacy of amiodarone in refractory ventricular fibrillation (Vf) during weaning from cardiopulmonary bypass (CPB). A 66-year-old woman with hypertension had a medical examination as a result of an episode of palpitations and syncope. Echocardiography and an invasive hemodynamic study revealed severe aortic stenosis (AS) with left ventricular (LV) hypertrophy because of calcified degeneration in a congenital bicuspid aortic valve (AV). Aortic valve replacement (AVR) was scheduled under general anesthesia and CPB. Intraoperative diagnosis was AS with calcified AV, LV hypertrophy, and aneurysm of ascending aorta (Ao). AVR with a biological valve, artificial vessel replacement of ascending Ao, and excision of the outflow myocardial septum were performed under CPB with intermittent antegrade blood cardioplegia at a body temperature (BT) of 24°C. The patient suffered from Vf at a BT of 35.3°C. Vf was not responsive to lidocaine 100 mg and 10 direct current (DC) shocks. After continuous intravenous infusion of amiodarone 225 mg/h for 10 min and a single intravenous injection of amiodarone 150 mg followed by a single DC shock, she returned to normal sinus rhythm. Sinus rhythm was maintained by continuous intravenous infusion of amiodarone 60 mg/h. Total CPB time was 5 h 43 min. Aortic cross-clamping time was 3 h 50 min. Administration of amiodarone is effective for refractory Vf resistant to lidocaine and cardioversion during weaning from CPB in cardiac surgery for heart diseases with LV hypertrophy.

Keywords Class III antiarrhythmic · Persistent ventricular fibrillation · Rewarming · Lidocaine · Cardioversion

Introduction

Ventricular fibrillation (Vf) is a common event during weaning from cardiopulmonary bypass (CPB) in cardiac surgery. Sustained Vf during rewarming is undesirable because of increased myocardial oxygen consumption. We sometimes experience Vf resistant to lidocaine and direct current (DC) shocks. Intravenous injection of amiodarone, a class III anti-arrhythmic used for persistent refractory arrhythmias [1], although it had already been used in European countries and the US, was approved for clinical use in Japan in 2007. We report a case which demonstrated the efficacy of amiodarone on refractory Vf resistant to lidocaine and cardioversion during weaning from CPB in aortic valve replacement (AVR) for severe aortic stenosis (AS) with left ventricular (LV) hypertrophy.

Case report

The case was a 66-year-old woman (weight 44 kg: height, 154 cm), who had an episode of palpitations and syncope for about 10 s while at rest. She was taking an angiotensin receptor blocker and a calcium channel blocker for hypertension. She had undergone myomectomy for uterine myoma under general anesthesia when 31 years old.

Y. Morita · J. Mizuno (✉) · T. Yoshimura · S. Morita
Department of Anesthesiology, Teikyo University School of Medicine, 2-11-1 Kaga, Itabashi-ku, Tokyo 173-8605, Japan
e-mail: mizuno_ju6@yahoo.co.jp

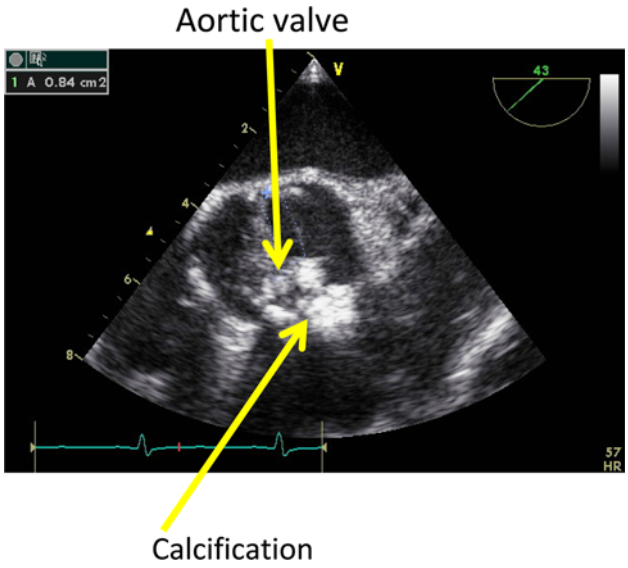

Fig. 1 Transesophageal echocardiography during the operation. Calcified degeneration in bicuspid aortic valve is shown

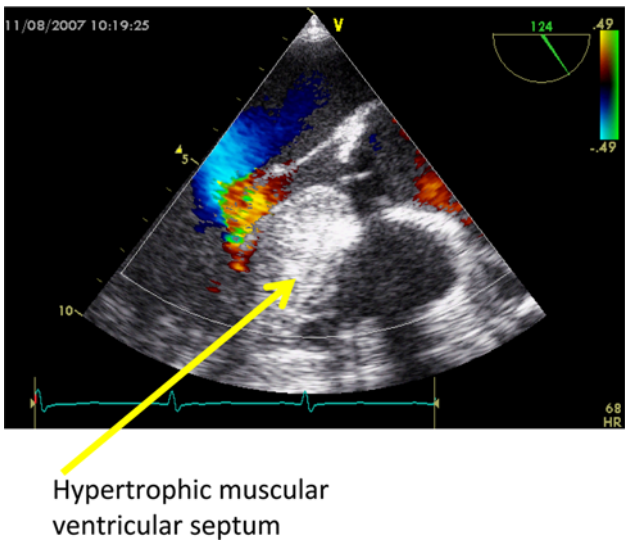

Fig. 2 Transesophageal echocardiography during the operation. Hypertrophic muscular ventricular septum is shown

Transthoracic and transesophageal echocardiography, and an invasive hemodynamic study revealed severe AS with LV hypertrophy because of calcified degeneration in a congenital bicuspid aortic valve (AV). Pressure gradient LV-aorta (Ao) was 76 mmHg at maximum and 45 mmHg at mean. AV area was 1.1 cm^2. The thickness of the interventricular septal wall and the LV posterior wall was 1.8 cm. LV dimension was 3.8 cm at diastole and 2.4 cm at systole. Ascending Ao was dilated to a maximum width of 4.7 cm. Ejection fraction was 63% and fractional shortening was 37%. Coronary angiography showed no significant coronary disease. Therefore, AVR was scheduled under general anesthesia and CPB.

General anesthesia was induced and maintained using oxygen, fentanyl, diazepam, vecuronium, and sevoflurane. Intraoperative diagnosis was AS with calcified AV (Fig. 1), LV hypertrophy (Fig. 2), and aneurysm of ascending Ao. AVR with a biological valve, artificial vessel replacement of ascending Ao, and excision of the outflow myocardial septum were performed under CPB with intermittent antegrade blood cardioplegia at a body temperature (BT) of 24°C.

The patient suffered from Vf at a BT of 35.3°C. Vf was not responsive to two consecutive intravenous injections of lidocaine 50 mg, five consecutive 20-J DC shocks, four consecutive 30-J DC shocks, and a single 50-J DC shock at a BT of 35.2°C. CPB restarted 4 min after discontinuation of the first CPB. Continuous intravenous infusions of dopamine and norepinephrine were administered. Arterial blood gas analysis and serum electrolytes were within normal limits. After continuous intravenous infusion of amiodarone 225 mg/h for 10 min and a single intravenous injection of amiodarone 150 mg followed by a single 30-J DC shock, sinus rhythm was restored. Sinus rhythm was maintained by continuous intravenous infusion of amiodarone 60 mg/h.

Operation time was 8 h 20 min. Anesthetic time was 9 h 50 min. Total CPB time was 5 h 43 min. Aortic cross-clamping time was 3 h 50 min. Total circulatory arrest was 36 min. Retrograde cerebral perfusion was 34 min. Her postoperative course was uneventful, she was extubated on postoperative day (POD) 1, and discharged on POD 17.

Discussion

It is known that the causes of persistent refractory Vf during weaning from CPB in cardiac surgery include intraoperative surgical trauma, reperfusion injury, ischemic damage [2], inadequate deairing, mechanical irritation by an LV drainage catheter, inadequate myocardial protection, inadequate supply of cardioplegic solution, electrolyte and metabolic derangements, and uncorrected hypothermia. Hearts with significant LV hypertrophy are essentially susceptible to sustained Vf. LV hypertrophy may further reduce subendocardial perfusion, leading to compromised myocardial performance during rewarming from CPB. Myocardial protection provided by retrograde continuous blood cardioplegia would have been ideal for this patient.

A bolus injection of the sodium channel blocker lidocaine, a class Ib anti-arrhythmic, reduces the incidence of Vf in cardiac surgical patients during reperfusion following aortic cross-clamp release [3]. Therefore, lidocaine has traditionally been used as the initial anti-arrhythmic for Vf that is resistant to cardioversion while weaning from CPB.

However, use of lidocaine is associated with a reduced chance of successful resuscitation from in-hospital cardiac arrest [4]. Administration of lidocaine is not useful for improving outcomes in patients with out-of-hospital cardiac arrest and persistent Vf after the first defibrillation attempt [5].

Administration of amiodarone before ischemia reduces the incidence of reperfusion-induced Vf [6]. Amiodarone slows intraventricular conduction by blocking the sodium channel, slows the heart rate, impedes atrioventricular node conduction by blocking the β-adrenergic receptors and calcium channels, and prolongs atrial and ventricular repolarization by inhibiting the potassium channels [7, 8]. In the Amiodarone in the Out-of-Hospital Resuscitation of Refractory Sustained Ventricular Tachyarrhythmia (ARREST) study, which was a randomized, double-blind placebo-controlled study in patients who had cardiac arrest with Vf or pulseless ventricular tachycardia (VT) and who had not been resuscitated after receiving three or more precordial shocks, treatment with amiodarone resulted in a higher rate of patients surviving to hospital admission [9].

Animal experiments show that intravenous injection of amiodarone effectively improves the rate of defibrillation in acute myocardial infarction-related Vf that is refractory to lidocaine, epinephrine, and up to five attempts of DC cardioversion [10]. In a randomized, double-blind placebo-controlled study, compared with lidocaine, amiodarone led to substantially higher rates of patients with shock-resistant out-of-hospital Vf surviving to hospital admission [11]. Moreover, 24-h survival of patients with shock-resistant VT is higher with amiodarone than with lidocaine [12]. Amiodarone should be administered as an intravenous bolus injection after the third unsuccessful DC shock, and lidocaine only is used if amiodarone is not available or if its use is contraindicated [13]. One case report shows that amiodarone injection into the aortic root suppresses persistent Vf resistant to lidocaine and cardioversion during weaning from CPB in AVR for aortic valve regurgitation [14]. In our patient, intravenous injection of lidocaine 100 mg and 10 attempts at DC cardioversion did not revert Vf. However, amiodarone's electrophysiologic and pharmacologic actions on specialized and non-specialized atrial and ventricular tissue could be responsible for the effectiveness of DC shock and restoration of sinus rhythm.

Recent guidelines on cardiac arrest with refractory Vf recommend amiodarone as anti-arrhythmic of first choice [15, 16]. Moreover, intravenous infusion of amiodarone provides a useful tool in preventing acute relapse of life-threatening Vf and VT in Japanese patients [17].

In conclusion, administration of amiodarone is effective for refractory Vf resistant to lidocaine and cardioversion during weaning from CPB in AVR for AS with LV hypertrophy. Amiodarone is a reasonable option in patients with persistent Vf during weaning from CPB in cardiac surgery for heart diseases with LV hypertrophy.

References

1. Kowey PR, Levine JH, Herre JM, Pacifico A, Lindsay BD, Plumb VJ, Janosik DL, Kopelman HA, Scheinman MM. Randomized, double-blind comparison of intravenous amiodarone and bretylium in the treatment of patients with recurrent, hemodynamically destabilizing ventricular tachycardia or fibrillation. The Intravenous Amiodarone Multicenter Investigators Group. Circulation. 1995;92:3255–63.
2. Robicsek F. Biochemical termination of sustained fibrillation occurring after artificially induced ischemic arrest. J Thorac Cardiovasc Surg. 1984;87:143–5.
3. Landow L, Wilson J, Heard SO, Townsend P, VanderSalm TJ, Okike ON, Pezzella TA, Pasque M. Free and total lidocaine levels in cardiac surgical patients. Cardiothorac Anesth. 1990;4:340–7.
4. van Walraven C, Stiell IG, Wells GA, Hébert PC, Vandemheen K. Do advanced cardiac life support drugs increase resuscitation rates from in-hospital cardiac arrest? The OTAC Study Group. Ann Emerg Med. 1998;32:544–53.
5. Weaver WD, Fahrenbruch CE, Johnson DD, Hallstrom AP, Cobb LA, Copass MK. Effect of epinephrine and lidocaine therapy on outcome after cardiac arrest due to ventricular fibrillation. Circulation. 1990;82:2027–34.
6. Riva E, Hearse DJ. Anti-arrhythmic effects of amiodarone and desethylamiodarone on malignant ventricular arrhythmias arising as a consequence of ischaemia and reperfusion in the anaesthetised rat. Cardiovasc Res. 1989;23:331–9.
7. Kodama I, Kamiya K, Toyama J. Cellular electropharmacology of amiodarone. Cardiovasc Res. 1997;35:13–29.
8. Kowey PR, Marinchak RA, Rials SJ, Filart RA. Intravenous amiodarone. J Am Coll Cardiol. 1997;29:1190–8.
9. Kudenchuk PJ, Cobb LA, Copass MK, Cummins RO, Doherty AM, Fahrenbruch CE, Hallstrom AP, Murray WA, Olsufka M, Walsh T. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. N Engl J Med. 1999;341:871–8.
10. Anastasiou-Nana MI, Nanas JN, Nanas SN, Rapti A, Poyadjis A, Stathaki S, Moulopoulos SD. Effects of amiodarone on refractory ventricular fibrillation in acute myocardial infarction: experimental study. J Am Coll Cardiol. 1994;23:253–8.
11. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. N Engl J Med. 2002;346:884–90.
12. Somberg JC, Bailin SJ, Haffajee CI, Paladino WP, Kerin NZ, Bridges D, Timar S, Molnar J. Amio-Aqueous Investigators. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. Am J Cardiol. 2002;90:853–9.
13. Leeuwenburgh BPJ, Versteegh MIM, Maas JJ, Dunning J. Should amiodarone or lidocaine be given to patients who arrest after cardiac surgery and fail to cardiovert from ventricular fibrillation? Interact Cardiovasc Thorac Surg. 2008;7:1148–51.
14. Tempe DK, Gandhi A, Mehta V, Banerjee A, Datt V, Ramamurthy P, Goyal G. Administration of amiodarone into the aortic root for persistent ventricular fibrillation after aortic valve replacement. J Cardiothorac Vasc Anesth. 2007;21:414–6.
15. Nolan JP, Deakin CD, Soar J, Böttiger BW, Smith G, European Resuscitation Council. European Resuscitation Council

guidelines for resuscitation 2005. Section 4. Adult advanced life support. Resuscitation. 2005;67(Suppl 1):S39–86.
16. ECC Committee, Subcommittees and Task Forces of the American Heart Association. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2005;112(24 Suppl):IV1-203.
17. Katoh T, Ogawa S, Yamaguchi I, Kasanuki H, Hayakawa H. Efficacy and safety of intravenous amiodarone infusion in Japanese patients with hemodynamically compromised ventricular tachycardia or ventricular fibrillation. J Arrhythmia. 2007;23:131–9.