Cardioplegic Arrest as Pharmacological Defibrillation; A Novel Approach for Refractory Ventricular Fibrillation

To the Editor,

Although occurrence of transient ventricular tachycardia/ventricular fibrillation (VT/VF) is common (around 24%),[1] but refractory VT/VF is a rare event while coming off cardiopulmonary bypass (CPB). In most of the cases, VT/VF occurring after removal of aortic cross-clamp revert back to normal spontaneously or after electrical/pharmacological defibrillation. In very rare instances, VT/VF does not respond to any of these interventions. We hereby share our experience of managing refractory VF while coming off CPB not responding to multiple electrical defibrillation and bolus doses of amiodarone and lidocaine.

A 56–year-old male, with height of 158 cm, weight of 52 kg was taken for double valve replacement (DVR) with severe aortic stenosis (AS), mean gradient 64 mm Hg, left ventricular (LV) wall thickness of 15 mm, along with severe mitral stenosis (MS), moderate mitral regurgitation (MR) having normal systolic function. In the operating room, patient was monitored according to the American Society of Anesthesiologist standards. Patient was induced with 0.1 mg/kg midazolam and 10 μg/kg fentanyl; his trachea was intubated with 8.5 mm cuffed endotracheal tube after administration of vecuronium bromide 0.2 mg/kg and he was put on mechanical ventilation with a mixture of 50% oxygen in air. Transesophageal echocardiography (TEE) probe was inserted for perioperative echocardiography.

A standard median sternotomy was done, after full heparinization and optimal activated clotting time, cardiopulmonary bypass was instituted with ascending aortic and bicaval cannulation. Patient was cooled to 32°C and a flow of 2.0 L/m^2 of body surface area (BSA) per minute was maintained. Cardiac arrest was achieved with 20 ml/kg of antegrade cold blood cardioplegic solution containing 16 mmol/l of potassium, 16 mmol/l of Magnesium, and 1 mmol/l of Procaine (PLEGIOCARD®, Samarth Life Sciences, India). Subsequent doses of cardioplegia was repeated every 20 min with 10 ml/kg cardioplegic solution containing 8 mmol/l of potassium. Mitral valve was replaced through a left atrial approach with 31 mm mechanical valve (St. Jude Medical Inc. Minnesota, USA) and aortic valve was replaced with size 25 mm mechanical valve (St. Jude Medical Inc. Minnesota, USA). After the completion of the procedure, rewarming was started. As per our protocol of deairing; head end of the table was raised (reverse trendelenburg position) and chambers were passively allowed to fill with blood by transiently decreasing the venous drainage. Aortic root vent cannula was allowed to suck the blood with simultaneous manual manipulation of heart, sideways tilting of operation table along with forcible manual ventilation to push any air trapped in pulmonary veins. Once there were no echocardiographic evidence of air in the chambers, trendelenburg position was done, 150 mg of amiodarone, and 1 gm magnesium sulphate was administered as per institutional protocol, and aortic cross-clamp was removed after achieving the core body temperature of more than 35°C. Gradually the pump flow was decreased to half, the perioperative TEE showed good prosthetic valve function with no paravalvular leak. While coming off CPB, the patient developed VF, immediately full CPB flow was re-established and mean arterial pressure of 75 mm of Hg was maintained. Core body temperature of 36.5°C was achieved, four escalating dose of defibrillation starting from 5J to 30 J were attempted; amiodarone 150 mg and lidocaine 1 mg/kg were administered. Despite serum potassium of 4.7 mmol/l, serum magnesium of 2.1 mmol/l and normal blood gas analysis, and no echocardiographic evidence of air in the LV cavity, the heart continued to have VF. Due to refractory nature of the VF, finally it was decided to arrest the heart with cardioplegia. Immediately aortic cross-clamp was re-applied and antegrade

normothermic blood cardioplegia 10 ml/kg containing 8 mmol/l of potassium was given. After achieving the cardioplegic arrest, aortic cross-clamp was opened while maintaining the full pump flow of 2.4 L/m^2 of BSA/minute. After 5 min, heart started beating in junctional rhythm and gradually regained sinus rhythm.

Gradually the CPB was weaned off without any difficulty. The TEE revealed normal prosthetic and biventricular functions with no regional wall motion abnormalities. The heparin was neutralized with protamine and decannulation was done. The total aortic cross-clamp time was 72 min and the total bypass time was 98 min. After completion of the surgery, the patient was transferred to the intensive care unit and put on ventilator with stable hemodynamics and rhythm, and was extubated after 8 h. Rest of the postoperative course was uneventful and the patient was discharged on 6th postoperative day.

VF is characterized by unsynchronized myocardial contraction with higher myocardial energy expenditure, and if left untreated, it may lead to permanent myocardial damage. The mechanism of VT/VF following removal of the aortic clamp is multifactorial. Air emboli or release of particulate debris from calcified aortic valve may occlude the coronary arteries, poor myocardial protection, electrolytes and acid–base disturbances, and conduction abnormalities among others, play a role.[2] Further, iatrogenic injury to left cercumflex coronary artery during mitral valve surgery because of its proximity to the mitral valve annulus can also lead to regional left ventricular dysfunction or arrhythmias.

Despite animal experimental models showing beneficial anti-fibrillatory effect of potassium, there have been a very few clinical reports on the use of potassium to convert ventricular fibrillation (VF) after cardioplegic arrest.[2]

In conclusion, we suggest that the potassium cardioplegia-induced transient asystole may conserve myocardial energy, foster chemical defibrillation, and can be used as a resetting technique for persistent VT/VF after aortic declamping; thereby improving VF arrest outcome.[2-5]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Submitted: 09-Jan-2019
Revised: 13-Jan-2019
Accepted: 25-Aug-2019
Published: 19-Oct-2020

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Quick Response Code:

Website: www.annals.in

DOI: 10.4103/aca.ACA_6_19

How to cite this article: Chowdhry V. Cardioplegic arrest as pharmacological defibrillation; A novel approach for refractory ventricular fibrillation. Ann Card Anaesth 2020;23:541-2.