CASE REPORT

Intraoperative cardiac arrest during transcatheter aortic valve implantation: A case report

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occurring after balloon aortic valvuloplasty which was managed by urgent cardiopulmonary bypass and postoperative extracorporeal membrane oxygenation, the elderly patient had a good prognosis after TAVI.

ABSTRACT

Balloon aortic valvuloplasty and rapid ventricular pacing have been identified with inherent risks that can result in hemodynamic disturbance and severe complications during Transcatheter Aortic Valve Implantation (TAVI). Here, we report a case of cardiac arrest

Key Words: Transcatheter Aortic Valve Implantation (TAVI); Balloon Aortic Valvuloplasty (BAV); Cardiac arrest

INTRODUCTION

ranscatheter Aortic Valve Implantation (TAVI) has emerged as an effective intervention for patients with severe aortic stenosis (valve area <1 cm², mean gradient >40 mmHg) and is less-invasive alternative in high-risk patients [1]. Despite continuous simplification of the TAVI procedure, Balloon Aortic Valvuloplasty (BAV) and Rapid Ventricular Pacing (RVP), which are the crucial steps before valve deployment during TAVI, have been identified with inherent risks that can result in hemodynamic disturbance and severe complications [2-3]. Here, we report a case in which cardiac arrest occurred during TAVI.

CASE PRESENTATION

An 83-year-old male patient has suffered from chest stuffiness and polypnea after activity for more than 3 years, worsening with dizziness (New York Heart Association (NYHA) Class III) for over 10 days. He was admitted for aortic valve stenosis and was scheduled for TAVI under Monitored Anesthesia Care (MAC), combined with a history of poorly controlled hypertension. Transthoracic Echocardiography (TTE) showed severe aortic valve stenosis and calcification with an effective orifice area of 0.5 cm² and a mean gradient of 44 mmHg, dilated left atrium, moderate mitral regurgitation and moderatesevere pulmonary hypertension with preserved ejection fraction of 67% (Figure 1). MSCT presented calcification of the aortic arch wall and two coronary artery wall, chronic bronchitis and emphysema. Electrocardiography (ECG) indicated left ventricle hypertrophy and ST-T wave changes. The 6-Minute Walking Test (SMWT) was recorded as 197 meters. The STS risk score and logistic EuroSCORE score were 2.764 and 7.18, respectively. The patient was deemed frail with high surgical risk.

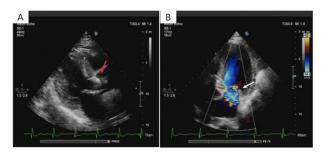


Figure 1) (A) Severe aortic severe aortic valve stenosis (red arrow) (B) Moderate mitral regurgitation (white arrow)

The patient was monitored by ECG, pulse oximetry, Bispectral Index (BIS) and external defibrillation electrodes, cardiac function was detected by TTE intermittently. Soon after nasopharyngeal airway and end-tidal carbon dioxide monitoring tube established, the patient was sedated by 1mg midazolam and 5 mg sufentanil intravenously. Radial artery puncture catheterization and right internal jugular vein catheterization were performed under local anesthesia. Vasoactive drugs combined with continuous infusion of propofol, remifentanil, dexmedetomidine and lidocaine were administrated to maintain the mean Arterial Blood Pressure (ABP) above 75 mmHg and proper depth of sedation. About 1.5 h after the operation, a balloon was expanded during RVP at 180 bpm, and then sinus rhythm recovered at a pacing rate of (50-60) bpm, ABP fluctuated around (50-60) mmHg/(30-35) mmHg and did not increase much after metaraminol and norepinephrine. Ventriculography showed severe aortic valve

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regurgitation after balloon dilation (Figure 2). Subsequently, ventricular fibrillation was confirmed by ECG in the absence of pulse wave. Defibrillation (biphasic, 200]) was immediately delivered followed by extracorporeal Cardiopulmonary Resuscitation (CPR), and in the meantime, a 26 mm Edwards SAPIEN 3 balloonexpandable aortic valve (Edward Lifesciences, Irvine, CA) was implanted and replacing the non-functional one, Ventriculography suggested severe artificial valve regurgitation and paravalvular leakage (Figure 3). After endotracheal intubation for mechanical ventilation, a Transesophageal Echocardiograph (TEE) probe was inserted. Drugs were administrated during the resuscitation period included intravenous boluses of adrenaline (3 mg in total), norepinephrine (0.4 mg in total), atropine (4 mg in total), methylprednisolone (80 mg) and omeprazole (40 mg), combined with continuous infusion of adrenaline, norepinephrine and sodium bicarbonate (5%, 200 ml). An ice pack was placed around the head for the cerebral protection. Four times of shocks failed to defibrillate the patient to a sinus rhythm. Cardiopulmonary Bypass (CPB) was rapidly instituted via a transfemoral approach after heparinization. The mean ABP rose to 80 mmHg under extracorporeal circulation support while the rhythm was sustained ventricular fibrillation. Surgeons again inserted a second 26 mm aortic valve via right 14 Fr femoral artery sheath, the implantation had good attachment with the former and functioned well, the aortic regurgitation was significantly reduced, which were identified by ventriculography (Figure 4). At that point, masses of pink frothy secretions were spilling out of the endotracheal tube accompanied by the peak airway pressure (Paw) increasing to 50 mmHg, and acute pulmonary edema was suspected. By reducing the extracorporeal blood flow, administrating diuretics and endotracheal suction, the peak airway pressure went down below 30 mmHg. 150 mg amiodarone and repeated external defibrillation were applied until ECG waveform converted to sinus rhythm. The patient received 3u of packed red blood cells, 600 ml of fresh frozen plasm, and 1000 ml of autologous blood. Electrolyte disturbance was corrected based on arterial blood gas analysis. By the end of the operation, CPB was changed to Extracorporeal Membrane Oxygenation (ECMO) with regard to cardiopulmonary insufficiency.

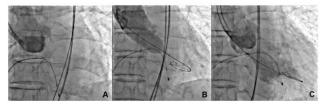


Figure 2) (A) Aortic root angiography (B) Pre-dilatation (C) Severe aortic valve regurgitation

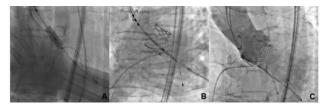


Figure 3) (A) Positioning (B) Deployment (C) Severe regurgitation and paravalvular leakage

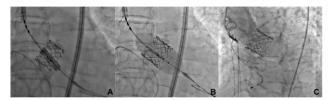


Figure 4) Valve-in-valve implantation (A) Positioning (B) Deployment (C) Minimal regurgitation and paravalvular leakage

The patient was then transferred to coronary care unit and underwent supportive and symptomatic therapies including anti-infection therapy and inotropic agents. ECMO treatment was terminated successfully on postoperative day 2, the tracheal tube was removed on postoperative day 9, and oral liquid diet was resumed 2 weeks after operation. He was admitted to the general cardiology ward 2 days later. Nearly 1 month later the patient made a good recovery and was discharged from our hospital without neurological abnormality.

DISCUSSION

Among the anesthesia methods of TAVI reported in China, general anesthesia accounts for approximately 55%, MAC 44% and local anesthesia 1% [4]. In this case, transfemoral approach was schemed by surgeons. MAC was selected reasonably as the patient had preserved ejection fraction and could tolerate supine position for a long time in the absence of dyspnea, he could also cooperate with surgeons' instructions, advantages of MAC were also taken into consideration including hemodynamic stability, increased cardiac preload and reduced residence time in operating room [5,6].

In this case, we encountered the unexpected occurrence of hemodynamic collapse after balloon dilation. Theoretically, there are several common causes which can lead to a sharp drop in blood pressure during balloon valvuloplasty, including coronary occlusion or obstruction, valve dysfunction, aortic root rupture, pericardial tamponade, annulus ruptured and perivalvular leakage, among which acute aortic regurgitation, is one of the relatively frequent complications after pre-dilatation [2, 7]. circulatory deterioration occurs, echocardiography intraoperative angiography may help to identify specific causes. Gang Zhang et al. reported collapsed blood pressure after balloon dilatation, which was resulted from obstruction of the right coronary artery confirmed by the ventriculography [8]. Pasic et al. also reported aortic annular rupture diagnosed by TEE and angiography during TAVI [9]. According to the TTE and ventriculography, extensive diastolic reflux of blood from aorta back to the left ventricle resulted in volume and pressure overload and progressive LV dilatation, which gave rise to acute left heart failure and pulmonary edema. Furthermore, reduced coronary flow and inadequate myocardial perfusion in diastole. These pathological conditions may contribute subsequent ventricular fibrillation. In the presence of malignant arrhythmias, CPR is always the preferred treatment undoubtedly, rapid replacement of the valve is the key step expected to reverse circulatory collapse. Nevertheless, it did not work on this case as aortic regurgitation was not reduced by the deployed aortic valve. Moreover, given the patients' conditions of acute left heart failure with low cardiac output and refractory ventricular fibrillation, we initiated CPB and conducted following another aortic valve implantation.

Trenkwalder et al. reported that 1.8% of 1810 patients required emergency Extracorporeal Life Support (ECLS) during TAVI, life-threatening complications were the indications, which included ventricle rupture (30%), low cardiac output (15%), bleeding (12%), coronary artery impairment (9%), malignant arrhythmias (6%), severe aortic regurgitation (6%), aortic rupture or dissection (6%), and unclear cause for circulatory instability [10]. Patients with high risk are supposed to undergo TAVI in a hybrid operating room in the presence of a standby heart-lung machine or other advanced mechanical circulatory support (ECMO and catheter-based devices such as intra-aortic balloon pump) in case of emergency conversion [11,12].

Both prophylactic and emergent initiation of ECMO peri-TAVI were reported in previously published data. There were no standard or agreed indications for prophylactic use, the reasons reported in previous studies were usually severely depressed cardiac function, preprocedural high dose vasopressors and/ or inotropes and borderline hemodynamic (mean pulmonary artery pressure>40 mmHg, central venous pressure>20 mmHg) which were predictable [11, 13-15]. While it was found that prophylactic use of ECMO was associated with better outcomes when compared to emergent use [11, 14], indicating that prophylactic ECMO may be useful in predictable high-risk patients. In review of 249 patients undergoing TAVI under both MAC and general anesthesia in our hospital from April 2018 to February 2019, 19 patients (7.6%) suffered intraoperative cardiac arrest, 15 patients (78.9%) of which had mitral regurgitation, 13 (68.4%) had aortic stenosis with regurgitation, 10 (52.6%) had pulmonary artery hypertension. It remains unclear whether these factors are associated with peri-TAVI cardiac arrest, and more clinical trials with large sample need to be carried out. As for this patient, at the end of TAVI, left ventricular systolic dysfunction with reduced ejection fraction confirmed by TEE and impaired respiratory function suggested by the pink frothy sputum indicated rational utilization of ECMO.

CONCLUSION

In conclusion, patients are at high risk for cardiac decompensation and life-threatening complications during TAVI especially around the period of BAV and RVP, effective emergency scheme is critical to patients' outcomes when cardiac arrest is confirmed resulted from deadly aortic regurgitation after BAV. Immediate CPR without the interruption of rapid placement and deployment of prosthetic valve usually works. If not, mechanical circulatory support should be initiated without hesitation. Postoperative support of ECMO is recommended if a patient is hemodynamically unstable or has insufficient cardiopulmonary function.

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