Recurrent Ventricular Fibrillation After Aortic Valve Replacement

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Citation

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Abstract

We report a patient who developed several episodes of sustained ventricular fibrillation after chest closure following stentless aortic valve replacement. Myocardial ischemia was suspected. Transesophageal echocardiography (TEE) was invaluable in confirming the diagnosis in the setting of acute ventricular fibrillation and new left bundle branch block. Ischemia may be due to coronary artery spasm, air or particulate embolus, and reperfusion injury. Iatrogenic injury to coronary arteries is a known complication of aortic valve surgery, and was the likely source of the ischemia and resultant arrhythmia. Treatment of recurrent ventricular fibrillation after aortic valve replacement was with coronary artery bypass grafting. Once the coronary artery flow was restored, there were no further arrhythmias, and outcome was good.

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INTRODUCTION

Because of the high risk of sudden death and diminished life expectancy, it is recommended that symptomatic patients with severe aortic stenosis (AS) undergo aortic valve replacement (AVR).[1] The risk of death or serious complications from AVR for AS is between 1-5%, depending on age of the patient, degree of left ventricular dysfunction or dilatation, and co morbidities.[2] We report a patient who developed several episodes of sustained ventricular fibrillation (VF) after chest closure, just prior to transfer from the operating room. The etiology and management of sustained VF after AVR is discussed.

CASE REPORT

A 58 year old woman with known AS and worsening dyspnea on exertion was scheduled to undergo stentless AVR. Her preoperative transthoracic echocardiography demonstrated severe AS with a valve area of 0.8 cm^2, peak gradient 53 mmHg, mean gradient 34 mmHg, a mildly dilated aortic root, mild left ventricular (LV) hypertrophy with no wall motion abnormalities and a LV ejection fraction of 60%. %. Preoperative coronary artery catheterization showed no coronary artery disease. Her past medical history was significant for hypertension and hypothyroidism; the patient's medications included amiloride, levothyroxine, metoprolol, sertraline and simvastatin.

Induction, line placement, and initiation of cardiopulmonary bypass were all uneventful. Following the valve replacement portion of the procedure, the surgeon noted a dense plaque surrounding the left coronary ostia and that it had partially separated from the intima; the intimal flap was tacked down to the sinus of Valsalva with sutures, and tied externally, allowing easy passage several times with a 3mm coronary probe. After the patient was rewarmed and the heart was defibrillated, CPB was discontinued and heparin was reversed. Vasopressin, 2u/hour was infused for low systemic vascular resistance. Transesophageal echocardiogram (TEE) showed normal LV and right ventricular function and a properly functioning bioprosthetic aortic valve without valvular or perivalvular leak. There was no evidence of dynamic LV outflow tract obstruction. There were no regional wall motion abnormalities. Blood gas showed pH 7.36, PCO2 41 mmHg, PO2 91 mmHg, bicarbonate 20.8 mmol/L, base excess -2.1 mmol/L. Electrolytes were within normal limits, and hemoglobin was 9.6 g/dL. After satisfactory hemostasis was achieved, the chest was closed and preparation was made for transfer to the intensive care unit (ICU). Vital signs were stable, with a mean arterial pressure between 70-80 mmHg. Cardiac index was 2.5 $l/min/m^2$.

While preparing to transfer to the ICU bed, VF occurred suddenly with loss of the arterial and pulmonary artery pressure waveforms. All anesthetics were discontinued. Ventilation was with 100% oxygen. External cardiac massage was started, and the patient was defibrillated within 2 minutes using external electrode patches. Blood pressure was restored. There was a new left bundle branch block. Lidocaine, 100 mg and magnesium, 2 Gm were given. The TEE probe was reinserted. New LV systolic wall motion abnormalities were now evident. There was hypokinesis of the basal and mid septal and anteroseptal walls; the posterior and lateral walls were hyperdynamic. There was no evidence of aortic dissection or prosthetic valve dysfunction. Amiodarone 150 mg, IV was given. VF occurred several times over the next 15 minutes and was successfully defibrillated. Epinephrine and amiodarone infusions were initiated.

Concern was raised of compromised left coronary blood flow. The patient was heparinized and CPB was initiated to allow bypass grafting of the left anterior descending and circumflex coronary arteries. TEE exam performed following coronary artery bypass grafting (CABG) demonstrated preserved LV and RV wall motion, no evidence of further stunning, and competent bioprosthetic aortic valve. The patient was transferred to the ICU on epinephrine, 0.025 mcg/kg/min, vasopressin, 2 u/hour, and amiodarone, 1 mg/min. She was weaned from vasopressors, and her trachea was extubated 8 hours postoperatively. Right lower lobe pulmonary infiltrate was noted on postoperative day number 3, and was treated with antibiotics, diuresis and pulmonary toilet. Otherwise, recovery was uneventful and no neurologic deficit was noted. The patient was discharged home on postoperative day 6.

DISCUSSION

The operative mortality for AVR varies with the patient's age and co-morbidities. In the Society of Thoracic Surgeons National Database, which contains tens of thousands of patients, the operative mortality is approximately 4% for isolated AVR and 7% for AVR combined with CABG.[2] Excessive postoperative bleeding may require re-exploration of the mediastinum in 2-5% of patients; perioperative myocardial infarction occurs in 1-2% of patients, particularly if coronary artery disease is present.[2]

Figure 1

Table 1. Coronary Ostial Injury after AVR. Literature Review

Author, year	Type of Aortic Valve	Diagnostic	Onset of	Treatment	Outcome
patient number		Symptom	Symptoms		
Midell(1), 1976	Starr-Edwards	angina	5m	CABGx1	asymptomatic
Midell(2)	Starr-Edwards	angina	4m	CABGx1	massive intraoperative MI
Midell(3)	not reported	angina	1m	CABGx1	asymptomatic
Midell(4)	not reported	angina	5m	CABGx1	postop death- CHF + arrhythmia
Pande(1) 1995	Carbomedics	angina	6m	CABGx2	postop MI, survived
Hadjimiltiades(1) 2005	ATS	angina	3m	CABGx2	asymptomatic
Hadjimiltiades(2)	Bioprosthetic	angina	50d	PCI	not reported
Li(1) 2003	not reported	VF	3d	EP ablation	asymptomatic
Winkelmann(1) 1993	Duromedics	angina	4m	antianginal meds	asymptomatic
Winkelmann(2)	St Jude	angina	5m	CABGx2	asymptomatic
Winkelmann(3)	St Jude	angina	5m	CABGx2	rethoracotomy for bleeding; asymptomatic
Winkelmann(4)	St Jude	angina	3.5m	CABGx2	asymptomatic
Winkelmann(5)	St Jude	angina	6m	CABGx2	asymptomatic
Winkelmann(6)	St Jude	angina	1.5m	CABGx2	asymptomatic
Winkelmann(7)	St Jude	angina	3m	Atherectomy	hemolytic anemia
Funada(1) 2006	Bentall	angina	2m	PCI (refused CABG)	Restenosis of RCA, PCI
Funada(2)	Bioprosthetic	angina	3m	PCI	asymptomatic, no restenosis on angiogram
Funada(3)	Bioprosthetic	angina	4m	PCI	asymptomatic, no restenosis on angiogram
Murakami 2005	Medtronic freestyle	angina	18m	PCI	asymptomatic, no restenosis at 18m
Yates(1) 1974	Starr-Edwards	angina	6m	Vineberg procedure	occasional angina relieved by NTG
Yates(2)	Starr-Edwards	angina	4m	attempted PCI	dissection of LCA. Massive MI and death
Yates(3)	Starr-Edwards	arrythmia	5y	CABGx1	Apical MI. Recovered
Yates(4)	Starr-Edwards	angina	3.5m	CABGx1	Endocarditis. Died after redo AVR
Yates(5)	Starr-Edwards	angina	1.5m	CABGx1	Died intraoperatively
Yates(6)	Starr-Edwards	angina	5m	CABGx1	AFib, continued angina
Yates(7)	Starr-Edwards	angina	2m	CABGx1	AFib, asymptomatic
Yates(8)	Björk-Shiley	angina	3m	CABGx1	AFib, asymptomatic
Pennington(1) 1982	Björk-Shiley	angina	3m	none	asymptomatic
Pennington(2)	Björk-Shiley	angina	5m	CABGx1	asymptomatic
Pennington(3)	Björk-Shiley	angina	8m	CABGx1	asymptomatic
Pennington(4)	Björk-Shiley	angina	3m	CABGx2	Diffuse MI. Died
Pennington(5)	Björk-Shiley	angina	4m	CABGx3	Cardiogenic shock. Hemorrhage. Died postop
Pennington(6)	Björk-Shiley	angina	post-op	none	uneventful recovery
Pennington(7)	Angell-Shiley Porcine xenograft	angina	2.5y	refused CABG	Dyspnea. No angina
Chavannon(1) 2002	Ionescu-Shiley	angina	6m	CABGx3	Severe adhesions, injury of LIMA-LAD. Died of CHF 4m postop
Chavannon(2)	Carbomedics	angina	11m	CABGx1	asymptomatic
Chavannon(3)	Carbomedics	angina	11m	CABGx2	LIMA injury, perioperative MI
Chavannon(4)	Carbomedics	angina	6m	CABGx2	severe adhesions, injury of LIMA-LAD
Chavannon(5)	Carpentier Edwards Bioprosthesis	angina	9m	CABGx1	asymptomatic
Chavannon(6)	Carbomedics	angina	4m	CABGx2	progressive CHF
Chavannon(7)	Carbomedics	angina	4m	CABGx2	severe adhesions; VF. Redo CABG
Lesage (1) 1970	Starr-Edwards	angina	post-op	none	continued angina, no intervention
Rath(2) 1988	Starr-Edwards	angina	5m	CABG	data not reported
Force(1) 1980	Starr-Edwards	angina	7m	CABGx1	uneventful recovery
Sethi(1) 1979	Hancock bioprosthesis	angina	3m	CABGx2	uneventful recovery
Place(1) 2005	Bioprosthetic	angina	1y	PCI	asymptomatic, no restenosis
Hazan(1) 1974	Starr-Edwards	angina	4m	CABGx1	asymptomatic, no restenosis
Hazan(3)	Starr-Edwards	angina	6m	CABGx1	satisfactory at 1 year
Pillai (1) 2004	Bioprosthetic	angina	3m	CABGx2	satisfactory at 3m

Abbreviations: CABG: coronary artery bypass graft; CHF congestive heart failure; NTG nitroglycerin; PCI: percutaneous coronary intervention; LIMA left internal Mammary; VF ventricular fibrillation; LCA left coronary artery; MI myocardial infarction; m: months y: years;

Iatrogenic coronary artery ostial obstruction after AVR is a rare but life-threatening complication. Stenosis of the left main trunk and ostium of the right coronary artery after AVR was first described by Roberts and Morrow in 1967.[7] Most often its clinical onset occurs 1-12 months after the procedure, and is thought to be linked to trauma to the coronary endothelium related to cannulation of the ostia during direct administration of cardioplegia for myocardial protection.[8,9] Rarely, this complication may be due to a variable degree of direct obstruction of the coronary ostia by the prosthetic annular ring or stent secondary to poor valve-to-annular size matching, or unfavorable anatomy with a low coronary ostial origin.[10] It is also possible that a coronary ostial lesion was missed during angiography. An aortic sinus

injection should be performed to look for ostial stenosis if either no arterial pressure is recorded when the catheter is engaged, or there is no reflux of contrast into the aortic sinus.

Earlier presentation of myocardial ischemia and VF is due to macro-injury of the ostia, but micro-injuries, combined with a genetic predisposition to atherosclerosis can lead to intimal thickening and fibrous proliferation. [11,12] Turbulent flow around the prosthetic valve contributes to this pathophysiology. The incidence of coronary artery ostial stenosis following AVR has been estimated at 0.2-5%.[13] Coronary ostia obstruction after AVR is associated with a high mortality rate if left untreated. The usual treatment is CABG (Table 1), [14-20] but cases treated by percutaneous coronary intervention, balloon angioplasty, and catheter ablation have also been reported. [21-26] If significant stenosis occurs several months after surgery, the clinical picture includes severe angina, ventricular arrhythmias, or heart failure. [27-29]

VF can occur at any time during and after AVR. For this reason, the anesthesia and surgical team must remain vigilant and prepared to intervene, especially during periods of transfer from the operating room to the ICU. Monitoring may be an issue since transport monitoring systems do not have the same resolution or number of channels standard operating room monitors. The ability to rapidly perform diagnostic studies such as blood gases and TEE is obviously important. Aseptic conditions in the surgical suite must be maintained should the patient require another procedure such as CABG to treat the recurrent VF. At our institution, sterility is maintained until the patient has safely been transported to the ICU.

Other etiologies of recurrent VF must be sought and aggressively treated such as electrolyte abnormality, acidosis, hypoxia, and hypercarbia. Life threatening arrhythmias can also result should the pulmonary artery catheter be accidentally withdrawn into the right ventricle during transfer. (Table 2).

Figure 2

Table 2. Causes of Arrhythmias after AVR

Myocardial Ischemia
Electrolyte abnormality
Acidosis
Hypoxia
Hypercarbia
Displacement of the pulmonary artery catheter

Myocardial ischemia was suspected in our patient as there was no evidence of electrolyte abnormalities, difficulties with ventilation, or surgical bleeding. The pulmonary artery catheter position had not changed. TEE was invaluable in confirming new wall motion abnormalities in the setting of acute VF and new LBBB. Ischemia may be due to coronary artery spasm, air or particulate embolus, and reperfusion injury. For this case, iatrogenic injury to the coronary artery was felt to be the most likely source of coronary artery insufficiency, since the patient had normal coronaries at cardiac catheterization. Direct coronary injury (e.g., from plegetted sutures) is a known complication of aortic valve surgery, and was the likely source of the ischemia and resultant arrhythmia (Table 3).

Figure 3

Table 3. Causes of myocardial ischemia after AVR in the immediate postoperative period

Coronary artery spasm
Air embolus
Particulate embolus
Reperfusion injury
Direct coronary artery injury from suture or pledgets
Ostial obstruction from valve position or alignment
Proximal aortic dissection
Ostial rupture secondary to cannulation

Treatment of recurrent VF after AVR was directed at the cause - CABG. Once the coronary artery flow was restored, there were no further arrhythmias, and outcome was good.

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