Emergency balloon aortic valvuloplasty in patients with critical aortic stenosis presenting with cardiogenic shock

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This report describes 2 cases of aortic valvuloplasty performed as emergency treatment in patients with critical aortic stenosis presenting with cardiogenic shock. This procedure can be life-saving, and allows the patients to undergo further evaluation for aortic valve replacement, or other definitive treatments such as the recently developed percutaneous heart valve implantation for patients with unacceptably high surgical risk.

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Introduction

Percutaneous aortic balloon valvuloplasty (PABV) was developed by Cribier et al.[1] in 1986 to provide a less invasive alternative to surgical aortic valve replacement. Although PABV often leads to immediate hemodynamic and clinical improvement, the short-term restenosis rate has been reported as a major limitation[2]. Moreover, PABV has not been shown to be effective in altering the natural history of aortic stenosis and was therefore abandoned in many centers[3]. The procedure is currently applied in few institutions and in restricted highly selected groups of patients[4].

We report 2 cases of critical aortic stenosis presenting with cardiogenic shock who were treated with emergency PABV, with the aim of discussing the possible role of this procedure in the modern era of interventional cardiology.

Description of cases

Case 1. A 73-year-old man with chronic atrial fibrillation, previous lateral myocardial infarction and impaired left ventricular function (ejection fraction 25% at echocardiography) was admitted to our department with a diagnosis of severe aortic stenosis and rest dyspnea. He was referred by his cardiologist to undergo general and cardiologic evaluation before aortic valve replacement. A peak gradient through the aortic orifice of 65 mmHg and a mean gradient of 42 mmHg were measured at echocardiography. The aortic valve area was estimated to be 0.6 cm^2. Coronary angiograms showed severe three-vessel disease, with very long and calcific stenosis of the proximal and mid-left anterior descending coronary artery, chronic total occlusion of the left circumflex artery and severe proximal and distal stenoses of the right coronary artery. Furthermore, both vessels of the left coronary artery showed a small reference diameter. Severe calcifications of the aortic root and of the ascending aorta were observed with fluoroscopy. Left and right heart catheterization confirmed severe aortic stenosis, with a mean transvalvular gradient of 55 mmHg and an aortic valve area of 0.4 cm^2, moderate mitral valve stenosis and moderate regurgitation, mild mixed pulmonary hypertension (pulmonary artery pressure 50/22 mmHg, mean 30 mmHg, mean pulmonary capillary wedge pressure 20 mmHg, pulmonary resistances 3.1 UR), severely reduced cardiac output (3.2 l/min) and cardiac index (1.8 l/min/m^2). Renal function was normal. Functional pulmonary testing revealed a moderate mixed restrictive-obstructive deficit. Echo color Doppler examination of the carotid arteries showed a mild bilateral disease without significant stenoses.

The case was discussed together with the cardiac surgeons, and the patient was not deemed suitable for aortic valve replacement, due to the unacceptably high

surgical risk (coronary anatomy not favorable for coronary bypass grafting, poor left ventricular function, concomitant mitral valve disease, severely calcified aorta, pulmonary function moderately reduced, peripheral vascular disease), and was therefore discharged home with medical treatment.

Two months later, he was readmitted to our department for acute inferior myocardial infarction complicated by cardiogenic shock. Systemic arterial pressure was 55/35 mmHg, and peripheral cyanosis was present together with evidence of low cerebral perfusion. A coronary angiogram revealed thrombotic occlusion of the distal right coronary artery (Fig. 1A). We therefore performed a primary angioplasty after the insertion of an intra-aortic balloon pump. Despite successful right coronary artery recanalization with balloon angioplasty and stenting (Fig. 1B), complete ST resolution at ECG, and intra-aortic balloon pump support, the patient's hemodynamics continued deteriorating severely (systemic blood pressure was persistently 30 mmHg and the patient required external cardiac massage). This prompted the decision to perform an emergency aortic valvuloplasty. The procedure was performed via the femoral retrograde approach. An extra stiff exchange wire was placed in the left ventricle. A 7F 20 × 45 mm Cristal Balloon™ (Balt, Montmorency, France) was introduced over this wire and positioned across the aortic valve, and then inflated with a stepwise dilation technique (Fig. 1C). After the third inflation, systemic pressure rose above 90 mmHg and the patient showed a clear improvement. No significant increase in differential aortic pressure suggestive of aortic regurgitation was observed. Echo-Doppler examination documented a maximum and mean aortic gradient of 46 and 30 mmHg respectively, with mild aortic regurgitation, and aortic valve area was estimated to be 0.7 cm^2. Left ventricular ejection fraction was 25%. After 7 days in the intensive coronary care unit the patient underwent angioplasty and stenting of the left anterior descending and right coronary arteries. The post-procedure hospital stay was uneventful and the patient was discharged home in good clinical conditions.

Five months later he underwent repeat angioplasty of the left anterior descending (edge stent restenosis) and right coronary arteries (in-stent restenosis) for recurrent angina pectoris. At that time left ventricular ejection fraction at echocardiography had risen to 40%, and the patient had been referred again for aortic valve replacement, and again refused because of his coronary anatomy and severe comorbidities. The patient died suddenly 8 months after hospital discharge. The relatives reported that during this period the patient had a satisfactory quality of life with moderate effort dyspnea.

Case 2. A 63-year-old woman was admitted to the emergency room of our hospital because of acute dyspnea, diaphoresis, and chest pain. Clinical history included type 2 insulin-dependent diabetes mellitus and previous (18 months earlier) surgery for pancreatic cancer. Physical examination revealed severe systemic hypotension (systolic blood pressure < 70 mmHg), a mild systolic ejective heart murmur, and diffuse pulmonary rales. Chest X-ray showed alveolar edema. The ECG examination revealed diffuse ST-segment depression with ST elevation in leads V_1 and aVR (Fig. 2A). After intravenous administration of 60 mg of furosemide, the patient was referred to the catheterization laboratory for suspected non-ST-elevation myocardial infarction complicated by shock and acute pulmonary edema. Despite intra-aortic balloon pumping, systemic arterial pressure remained around 50 mmHg. Coronary angiography showed normal coronary arteries (Fig. 2B). Severe calcification of the aortic valve was observed with fluoroscopy. During the procedure, tracheal intubation and mechanical ventilation was

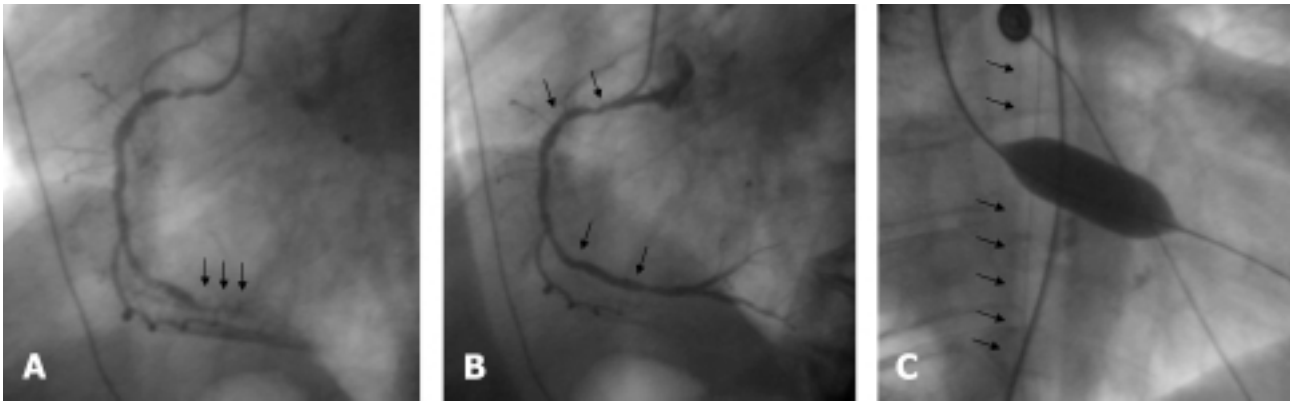

Figure 1. *Case 1.* A: *coronary angiogram showing right coronary artery severe proximal stenosis and distal thrombotic occlusion* (arrows). B: *the right coronary artery following successful recanalization with angioplasty and stenting. The* arrows *indicate the position of a 2.5 × 18 mm PENTA™ stent (Guidant Corporation, Santa Clara, CA, USA) in the distal right coronary artery, and the site of balloon dilation in the proximal segment. Despite the suboptimal result at this site, further stent implantation was not performed because of the progressive hemodynamic deterioration of the patient.* C: *balloon aortic valvuloplasty performed with a 7F 20 × 45 mm Cristal Balloon™ (Balt, Montmorency, France) via the retrograde femoral approach. The* arrows *delineate the profile of the intra-aortic balloon pump in the descending aorta (Fidelity™, Datascope Corporation, Fairfield, NJ, USA).*

necessary, as well as a short period of external cardiac massage, because of extreme hypotension and persistent severe pulmonary edema. After stabilization of the systolic pressure around 60-70 mmHg with infusion of inotropes, we performed an echocardiographic examination. Echocardiography revealed mild hypertrophy of the left ventricle, severely impaired systolic function, moderate dilation of the left atrium, mild mitral valve regurgitation, normal volume and function of the right ventricle, and severe calcification of the aortic valve with a peak transvalvular gradient of 60 mmHg. We therefore decided to perform an emergency PABV. We measured a mean gradient of 51 mmHg, with a peak gradient of 75 mmHg (Fig. 2C). Cardiac output was measured with the Fick method as 3.8 l/min, and the aortic valve area was estimated to be 0.36 cm^2. Aortic valvuloplasty was performed as previously described. After repeat balloon inflations the mean transvalvular gradient was reduced to 18 mmHg, and aortic valve area increased to 0.77 cm^2 (Fig. 2D). The patient left the catheterization laboratory with a systemic arterial pressure of 120/70 mmHg, with no evidence of important aortic regurgitation. During the following days she slowly recovered and was transferred to the cardiology department to undergo further evaluation for aortic valve replacement. Eight months later she was still alive. A last oncologic evaluation before inclusion on the waiting list for aortic valve replacement was scheduled for 1 month later.

Discussion

We report 2 cases of critical degenerative aortic stenosis presenting with cardiogenic shock. In the first case, a complete clinical evaluation had been performed 2 months earlier, and the patient was not considered suitable for aortic valve replacement due to the high surgical risk. Two months later, he was referred to our catheterization laboratory to undergo primary angioplasty for acute inferior myocardial infarction. Despite the rapid recanalization of the culprit vessel, progressive hemodynamic deterioration developed, and this prompted emergency aortic valvuloplasty. Con-

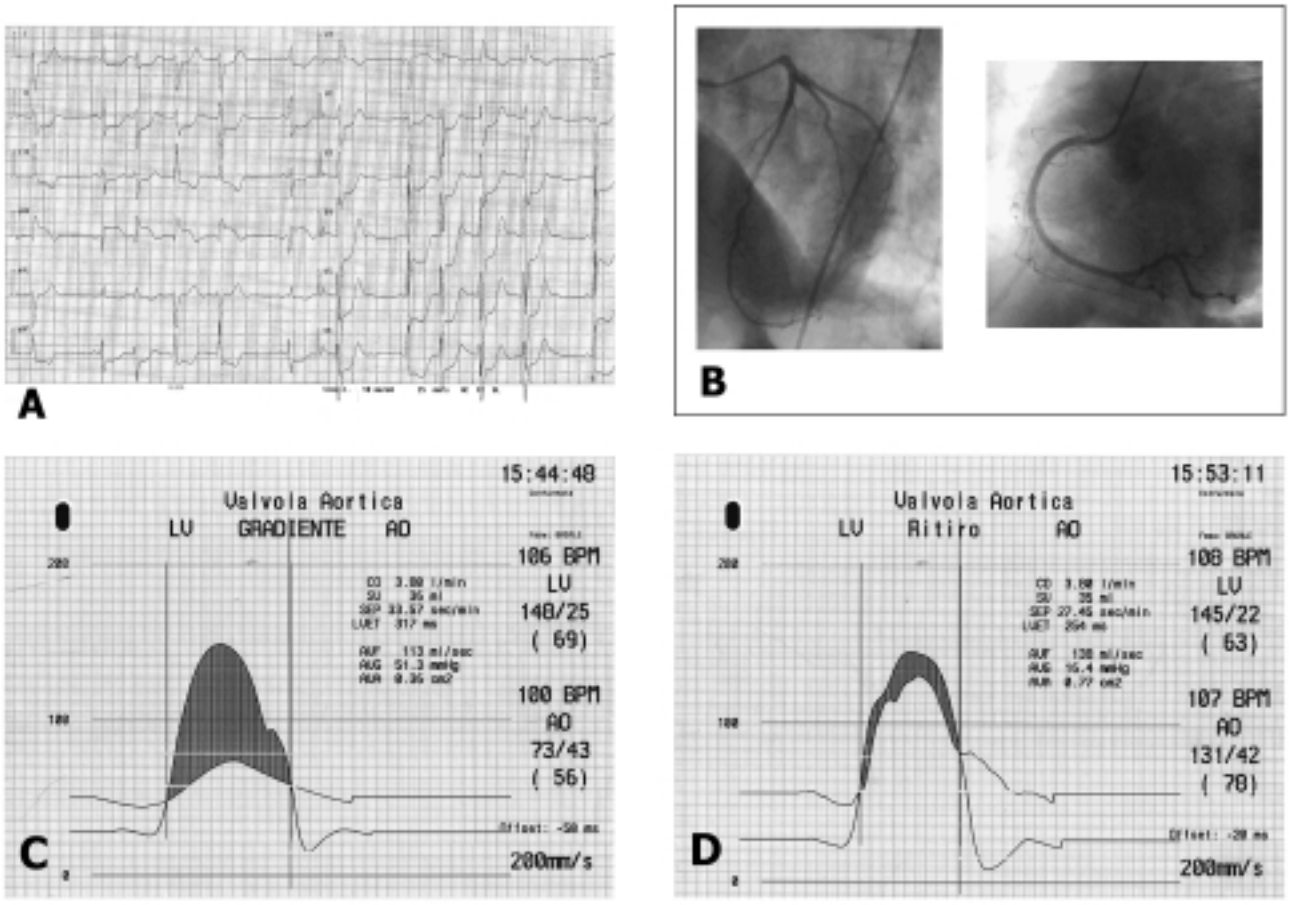

Figure 2. *Case 2.* A: *the ECG showed diffuse ST-segment depression with ST elevation in leads V_1 and aVR and the patient was referred to the catheterization laboratory for the suspect of non-ST-elevation myocardial infarction complicated by shock.* B: *coronary angiography showing normal left and right coronary arteries.* C: *pressure tracings in the ascending aorta and in the left ventricle at baseline, showing a mean gradient of 51 mmHg and a peak gradient of 75 mmHg. The aortic valve area was estimated to be 0.36 cm^2 by an automated program.* D: *pressure tracings in the ascending aorta and in the left ventricle following aortic valvuloplasty. The mean gradient was reduced to 18 mmHg and the aortic valve area increased to 0.77 cm^2, with a rise in systemic pressure above 100 mmHg.*

versely, in the second patient cardiogenic shock was the first manifestation of the disease.

PABV in calcific aortic valve stenosis is essentially a palliative procedure[3,5]. The main indications for PABV are prophylactic utilization in patients undergoing major non-cardiac surgery[6], bridge to aortic valve replacement in critically ill patients[7], compassionate use in inoperable patients[8]. The 2 cases described confirm previous reports that PABV can be life-saving in patients with cardiogenic shock[7]. Patient 1 was deemed inoperable for the coexistence of severe left ventricular dysfunction and severe comorbid conditions (chronic obstructive pulmonary disease). As expected, in this patient the valvuloplasty procedure combined with primary angioplasty was able to resolve the shock and to partially relieve the symptoms, although the patient died suddenly 13 months later. The recent development by Cribier et al.[9] of percutaneous implantable prosthetic aortic valves, could in the future offer an alternative to patients in similar clinical conditions and possibly improve the long-term prognosis. Ideally, all inoperable patients with critical aortic stenosis might be treated with non-surgical implantation of prosthetic aortic valve, and PABV might be used as a bridge to this procedure in selected critically ill patients. However, further data are necessary before this procedure can be introduced into clinical practice. Patient 2 was referred to our laboratory with suspected acute coronary syndrome, and PABV was performed following coronary angiography based on a clinical and echocardiographic diagnosis of severe aortic stenosis formulated in the interventional cardiology department. This case is paradigmatic of the necessity to consider aortic stenosis in the diagnostic process for patients presenting with cardiogenic shock. Although not very commonly, cardiogenic shock may in fact represent the first clinical manifestation of critical aortic stenosis. In these situations emergency PABV can be life-saving and permit recovery from shock and starting of the subsequent workout directed toward valve replacement, which remains the treatment of choice for this condition.

In conclusion, PABV may be life-saving in patients with degenerative calcific aortic stenosis presenting with cardiogenic shock, thus allowing the access to subsequent definitive treatment.

Compassionate use of this technique in patients permanently not suitable for cardiac surgery remains controversial, although recent developments in interventional cardiology such as percutaneous valve implantation may open up new possibilities for these patients.

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