Systolic Anterior Motion of the Mitral Valve Triggered by the Intra-Aortic Balloon Pump

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THE INTRA-AORTIC balloon pump (IABP) is the simplest and most widely used mechanical assist device in the setting of cardiogenic shock.[1] The use of the IABP is currently a class IIa indication in the American College of Cardiology/American Heart Association guidelines for the management of ST-elevation myocardial infarction who do not respond to pharmacotherapy.[2] There are patients, however, in whom its use may be more detrimental than beneficial. The authors present a case where the IABP did not give the desired effect.

CASE REPORT

A 65-year-old male presented to the hospital with acute coronary syndrome, affecting the postero-inferior territory, and atrial fibrillation. The patient was treated pharmacologically and cardioverted into normal sinus rhythm. The following day, the patient underwent cardiac catheterization that revealed 70% occlusion of the left main artery, 70% occlusion of the right coronary artery, 50% occlusion of the circumflex artery, 70% occlusion of the second obtuse marginal artery, and 85% occlusion of the posterior descending artery. Left ventricular (LV) function was normal. In the context of significant left main disease and 3-vessel obstruction, an IABP, 40 mL, 7.5-fr, (Datascope Corp., Maquet Getinge Group, Fairfield, NJ) was inserted prophylactically into the right femoral artery.

The patient underwent surgery on day 4 following admission. The initial intraoperative transesophageal echocardiogram (TEE) revealed good diastolic and systolic function with an ejection fraction of 65%. There was concentric LV hypertrophy and normal cardiac valves. There was no evidence of systolic anterior motion (SAM) of the mitral valve. The TEE confirmed adequate positioning of the IABP. Cardiopulmonary bypass (CPB) was initiated and the patient was cooled to 34°C. Once the aorta was clamped, the IABP was switched to internal mode at 40 beats per minute to offer pulsatile flow during CPB in an attempt to optimize microvasculature perfusion. Four coronary artery bypass grafts were done with an endarterectomy of the left anterior descending artery.

The aortic clamp was removed after 99 minutes. A ventricular pacemaker was installed. During the separation from CPB, right ventricular dysfunction was treated with 5 mg of milrinone nebulized via the endotracheal tube. The patient was weaned from CPB after 128 minutes with the aid of the IABP. Despite adequate timing of IABP inflation, the tracing showed a diastolic augmentation lower than the assisted systolic pressure. Proper position of the IABP was reconfirmed by TEE. The patient's mean arterial pressure was adequate, with the aid of appropriate vasopressor therapy. The central venous pressure was 14, and the patient's heart rate (HR) was 65 beats/min. However, the patient showed increased hemodynamic instability with falling systemic pressures and rising pulmonary artery pressures (PAPs). After these events, the IABP was paused for a surgical intervention on the aorta. At this point, the systemic blood pressure increased from 70/40 to 100/55 mmHg and PAPs decreased from 63/38 to 40/25 mmHg. The hemodynamics seemed to worsen with IABP augmentation. Cardiac function was reassessed by TEE, showing acceleration of blood flow in the left ventricular outflow tract (LVOT) with the IABP functioning. Further evaluation revealed a posteriorly directed eccentric mitral regurgitation jet, which had increased from mild-to-moderate in severity. This was due to systolic anterior motion (SAM) of the mitral valve. The acceleration of flow in the LVOT and SAM completely resolved with cessation of the IABP. Following these observations, intravenous fluids were administered to the patient, the IABP was set at a rate of 1 to 3 and the inflation was adjusted to 50%. The IABP was removed upon admission to the ICU.

DISCUSSION

Kantrowitz was the first to report the clinical advantage of the IABP, with improved systemic arterial pressure and urine output.[3] The IABP functions by counterpulsation in which the balloon inflates in diastole and deflates prior to systole. The inflation of the IABP causes a displacement of blood within the aorta by augmentation of the intrinsic Windkessel effect. This leads to an increase in coronary blood flow and a potential improvement of systemic perfusion. The deflation of the IABP decreases LV afterload via reductions in end-diastolic and systolic aortic pressures. However, the magnitude of effect induced by the IABP can be hindered by inappropriate balloon position and volume, poor IABP timing, reduced LV stroke volume, low mean arterial pressure, rapid heart rate, and poor aortic compliance (Table 1).

In the case presented, the diastolic augmentation was lower than the patient's systolic pressure. The reason for this could not be explained by any causes described above, but instead resulted from a dynamic left ventricular outflow tract obstruction (LVOTO) caused by SAM of the anterior leaflet of the mitral valve. During systole, as blood passes through the

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Table 1. Factors Affecting Intra-Aortic Balloon Pump Diastolic Augmentation

Causes	Effect
IABP position	If the IABP is too low, the augmentation will be reduced.
IABP timing	Proper synchronization with the ECG or arterial pressure is required. If there is late inflation, the augmentation will be suboptimal.
Balloon volume	The volume of the IABP determines the volume of blood displaced during IABP inflation. If the balloon is too small in relation to the aorta, the augmentation will be reduced. The diameter of the balloon should not exceed 85% to 90% of the diameter of the descending thoracic aorta.[5]
LV stroke volume	The optimal stroke volume for maximal augmentation should be similar to the volume of the IABP.[5]
Arterial blood pressure	When the systolic arterial pressure is below 60 mmHg to 70 mmHg, the IABP is known to be ineffective, caused by an increased aortic compliance at low blood pressure.[4]
Heart rate	At heart rates above 120 beats per minute, there is significant decline in hemodynamic changes caused by the IABP. The inflation interval of the IABP is too short at high heart rates to produce optimal hemodynamic changes.
Aortic compliance	The greater the arterial compliance (reduced systemic vascular resistance), the lower the arterial changes caused by the IABP.[5]

Abbreviations: ECG, electrocardiogram; IABP, intra-aortic balloon pump; LV, left ventricle.

LVOT, the anterior leaflet of the protruding mitral valve is displaced by drag or "sail effect" towards the septum into the LVOT, thus obstructing it. As SAM progresses, the angle between the anterior leaflet and the blood flow increases, thus increasing the exposed surface of the anterior leaflet to the blood flow. As the surface area of the leaflet increases, more area is exposed to the drag forces, amplifying the effect of SAM.[6] The accelerated flow through a narrowed LVOT can push the mitral

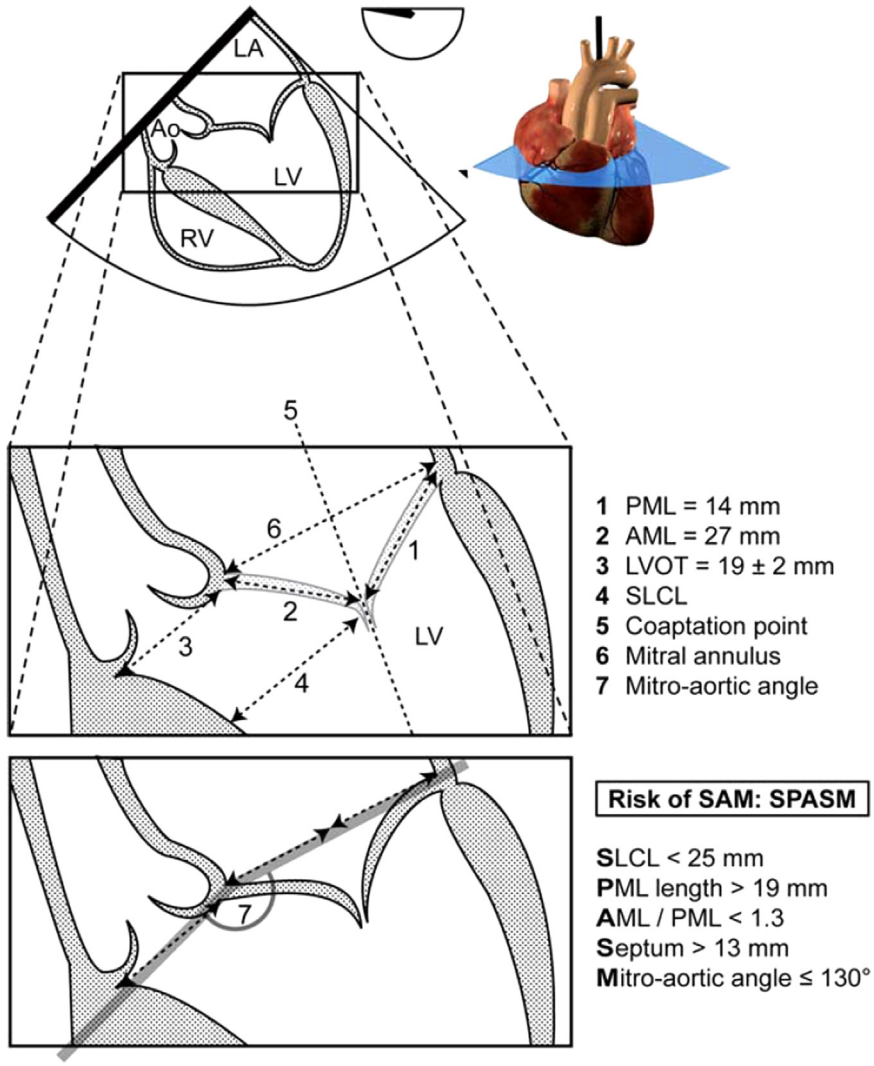

Fig 1. Risk factors for systolic anterior motion of the mitral valve.