Use of Intra-Aortic Balloon Pump (IABP) in Patient with Ventricular Septal Rupture

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Abstract

Intra-Aortic Balloon Pump (IABP) can be used among patients with Acute Coronary Syndrome (ACS) who experience Ventricular Septal Rupture (VSR) as a form of mechanical support for hemodynamics that has worsened due to cardiogenic shock. As for the cases reported, a 61-year-old male patient with ACS STEMI Infero-Anterior Extensive Killip 4 and VSR experienced chest pain accompanied by shortness of breath. Auscultatory examination found a heart murmur on 3/6 without thrills. The patient had received drugs to improve hemodynamics and was reported to have used an IABP machine. IABP uses semi-auto mode, 1:1 frequency with maximum augmentation. The patient's condition worsened, and he lost consciousness. The patient then had an endotracheal tube connected to a ventilator.

Keywords: IABP, Intra-aortic-counterpulsation, ventricular septal rupture.

Introduction

The IABP is a long, thin balloon that controls blood flow through the aorta. This device seeks to balance the supply and demand of blood and oxygen that the heart needs (Limbert & Amiri, 2019). Counterpulsation Intra-aortic balloon pump (IABP), announced over fifty years ago, has restored hemodynamic parameters and myocardial oxygen transportation in patients with myocardial ischemia and cardiogenic shock (González & Chaney, 2020).

A sequence of experiments by Adrian Kantrowitz and Arthur Kantrowitz demonstrated the principle of diastolic augmentation. They theorized coronary perfusion with systolic blood pressure (SBP) during diastole would improve coronary blood flow, and, in 1953, they confirmed in animals that perfusion of the coronary arteries with blood diverted from the femoral arteries postponed the arrival of the peak systolic blood pressure. Systolic pressure waves to myocardial diastole and improved coronary blood flow (González & Chaney, 2020).

The National Center for Health Statistics (NCHS) estimated that IABP was used in 42,000 patients in the US in 2002 (Krishna & Zacharowski, 2009). The ACC/AHA guidelines on STEMI suggest that the usage of IABP in cardiogenic shock is counted in the class IB recommendations (Antman et al., 2004). The benefits of using IABP with indicated patient conditions have been suggested in the guidelines so that the use of IABP at Dr. Hasan Sadikin in ACS patients with very high VSR.

This article discusses the use case of IABP in STEMI patients with ruptured ventricular septal (VSR). VSR is an infrequent complication of acute myocardial infarction (AMI). Before the era of reperfusion therapy, the occurrence of VSR ranged from 1-2% and diminished to 0.17-0.31% after reperfusion therapy (Siegel et al., 2002). The mortality rate from this complication is still very high. Through a data study, the Myocardial Infarction Data Acquired System (MIDAS) conveyed that patients with AMI with VSR complications had an augmented risk of death in the hospital, seven times greater than those without VSR. During 18 years of observation, the mortality rate in hospitals due to this complication was prone to be stationary at 50% (Moreyra et al., 2010).

Case

Mr. Y, 61 years old, 160 cm tall, came with NRS 4 chest pain accompanied by shortness of breath, cold sweat, with STEMI Inferior Coronary Artery Disease (CAD), Anterior Extensive Killip 4 with Ventricular Septal Rupture. ECG picture shows sinus tachycardia rhythm, QRS axis normoaxis, rate 115 x/minute, regular, P wave width 0.04 seconds, height 0.1 mV, PR interval 0.16 seconds, pathological Q waves in inferior leads II, III, aVF, QRS duration 0.04

seconds, QTC 0.475 seconds, ST segment: ST elevation in leads V2-V6, leads II, III, and aVF, inverted T waves in leads V2-V6, R/S V1 < 1.5. V1/V2 + R V5/V6 < 35 mm. RaVL + SV3 < 28mm, with the conclusion: sinus tachycardia, STEMI Anterior, acute inferior, and Left Ventricular Hypertrophy. The echo results showed reduced LV systolic function / LVEF 50-55% eye balling with akinetic apical, inferoseptal normokinetic segments. LV diastolic dysfunction, mild TR, normal RV systolic function, LV apical akinetic, VSR Left to Right Shunt 10-11 mm, (CO; 2.3) (CI; 5.3) (SV; 16) (SVI; 11) (SVR; 1983).

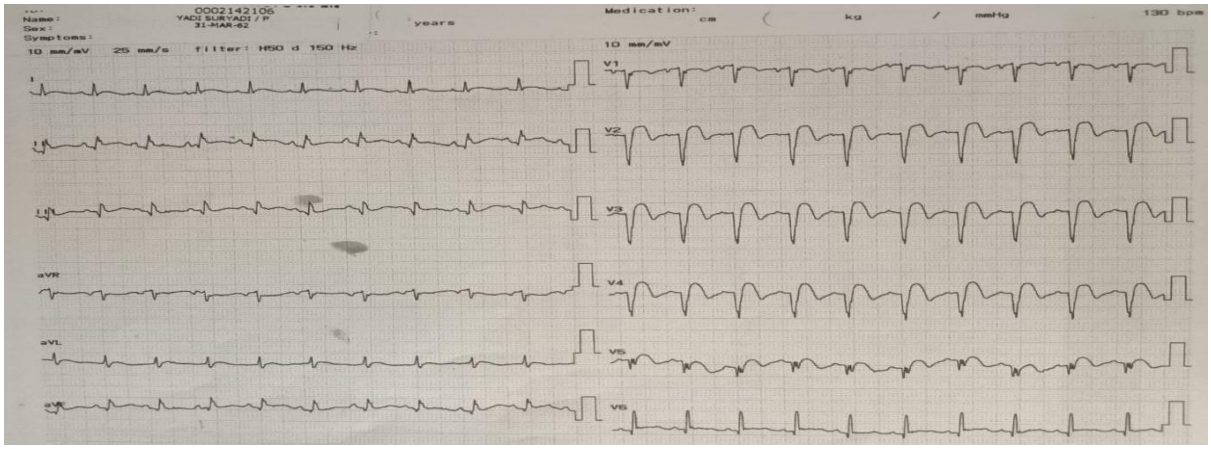

Figure 1. The results of the client's ECG examination, Mr. Y with STEMI Inferior, Wide Anterior

The patient has received therapy with Dobutamine 15 mcg/kg of body weight/minute, Norepinephrine 0.5 mcg/kgbb/minute, Aspilet 1x80 mg, Clopidogrel 1x75 mg, Atorvastatin 1x40 mg, Fondaparinux 1x2.5 mg, Lansoprazole 1x30 mg. The patient is being consulted by the cardiac surgery department for VSR Clossure surgery. The patient was treated in the Cardiac ICU and was immediately placed with the IABP in semi-auto mode, 1:1 frequency with maximum augmentation, and using a 30 cc IABP balloon. Because his condition had worsened, the patient was then intubated, installed ETT no 7.0 with a depth of 20 cm, with PC ventilator mode, IPL 12, PEEP 5, RR 12, FiO2 100%. Because his condition was worsening, the patient had a cardiac arrest 12 hours after the IABP and ventilator were installed. CPR was performed for ± 1 hour; the patient could not be saved and died.

Initial name	YS
Date of birth	January 21, 1942
Diagnosis	VSR
Location of IABP	right femoral artery
Tgl Pemasangan	May 20, 2023
Tgl Pencabutan	

Observation date	May 20, 2023							
Time		18	19	20	21	22	23	24	01
Systolic Pressure		77	80	80	82	71	69	66	76
Diastolic Pressure		58	54	56	57	52	52	51	48
MAP		81	79	81	80	73	72	69	70
augmentation		118	120	121	123	115	107	109	99
Filling Pressure									
Central vein									
Pulmonary Artery									
Right Atrium									
Pulse frequency		133	135	138	136	135	134	140	140
IABP frequency		1:1	1:1	1:1	1:1	1:1	1:1	1:1	1:1
Trigger		ECG	ECG	ECG	ECG	ECG	ECG	ECG	ECG
IABP Inflation Point		-1.2	-1.2	-1.2	-1.2	-1.2	-1.2	-1.2	-1.2
IABP Deflation Point		0	0	0	0	0	0	0	0
IABP Augmentation		Max	Max	Max	Max	Max	Max	Max	Max
IABP Fill									
Lower extremity pulsation		+	+	+	+	+	+	+	+
Dorsalis Pedis Right/Left		+/+	+/+	+/+	+/+	+/+	+/+	+/+	+/+
Popliteal Right/Left		+/+	+/+	+/+	+/+	+/+	+/+	+/+	+/+
Femoralis Right/Left		+/+	+/+	+/+	+/+	+/+	+/+	+/+	+/+
Lower Extremity Warmth									
Right Extremity		H	H	H	H	H	H	H	H
Left Extremity		H	H	H	H	H	H	H	H
Skin color									
Right Extremity		M	M	M	M	M	M	M	M
Left Extremity		M	M	M	M	M	M	M	M
Information									
IABP frequency	1:01	1:02							
IABP Inflation	0 -1 -2 -3 1 2 3								
IABP Deflation	0 -1 -2 -3 1 2 3								
Trigger	ECG	Pressure	Pacer A	Pacer V	A-V Interna				
IABP Fill	Auto/Manual								
Warmth	Warm (W)	Cold (C)							
Skin color	Red (R)	Pale (P)	Cyanosis (C)						

Figure 2. IABP Observation Sheet Mr. YS

Discussion

VSR is a condition resulting from perforation of the interventricular septum so that it forms a shunt amid the two ventricles. The primary etiology of this disorder is myocardial infarction, which extends to transmural infarction. Anterior and inferior infarctions have the same frequency to cause VSR (Moreyra et al., 2010). In the Crenshaw study in patients with anterior (70%) versus inferior (29%) infarctions, occlusion most frequently originated in the left anterior descending artery (64%) and right coronary artery (28%) (Crenshaw et al., 2000).

The pathologic process of rupture alters over time. Coagulation of new necrotic sites begins throughout the first 24 hours. The development of rupture begins with neutrophil invasion into the infarcted tissue. Neutrophils will then undergo apoptosis and release lytic enzymes (Moreyra et al., 2010). This enzyme induces the disintegration of necrotic myocardial tissue. Early rupture happens in infarcts with significant intramural hematomas that cut into the tissues. If the patient remains alive, fibrotic tissue remodeling will occur after several weeks (Moreyra et al., 2010)

The rupture consequences in a left-to-right shunt are accompanied by right ventricular (RV) volume excess, enlarged pulmonary blood flow, and left atrial and left ventricular secondary volume excess. An incessant process will cause left ventricular systolic function to reduce. The body will receive negative feedback by amplified systemic vascular resistance through peripheral vasoconstriction (Moreyra et al., 2010). However, this mechanism causes the shunt flow to be more robust. If it lasts for a long time, the left ventricle can experience pump failure so that the systolic pressure will decrease (Moreyra et al., 2010)

The main indication for placing the IABP is for hemodynamic support and stabilization, while the most common place for placing the IABP is in the catheterization laboratory (Oktaviono, 2016). VSR is an infrequent complication of acute myocardial infarction, often occurs with cardiogenic shock, and is associated with high in-hospital mortality, despite prompt intervention. Although urgent surgery is recommended for patients who cannot be effectively stabilized, the ideal timing of intervention is controversial, and mechanical circulatory support (MCS) such as IABP allows hemodynamic stabilization and delays definitive intervention even in critically ill patients (Ronco et al., 2021). Moreover, deaths related to IABP are around 0.05% (Oktaviono, 2016).

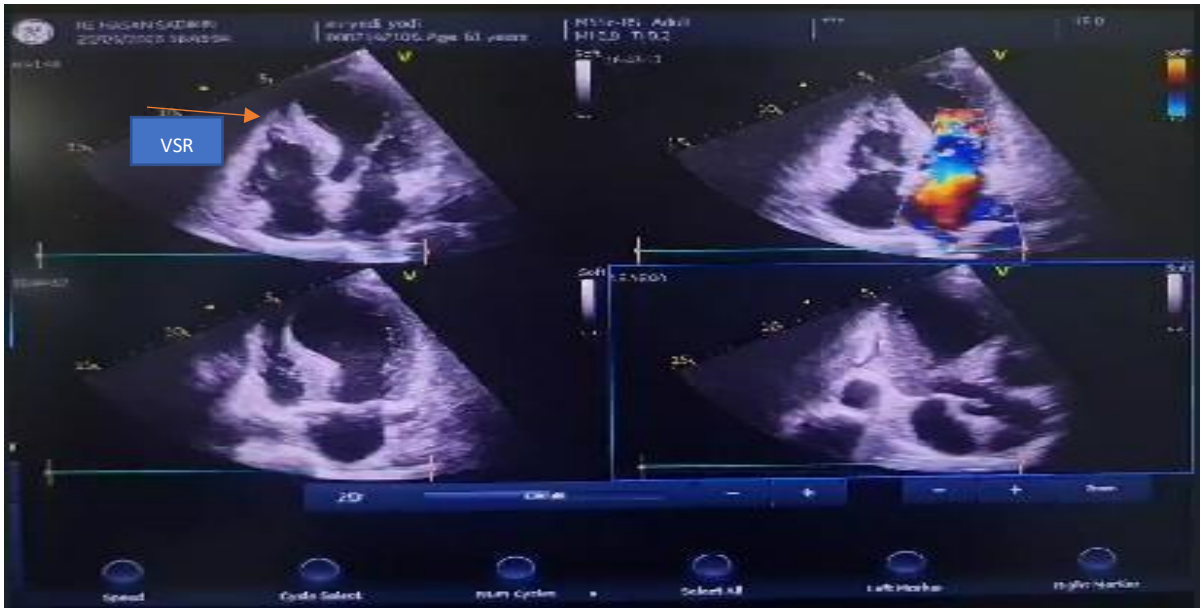

Figure 3. Patient's Echo Bedside

Triggering and Timing

Optimal inflation arises immediately after AV closure, and optimal deflation occurs shortly before AV opening. The most common trigger sources are ECGs (inflation triggered during T waves – P wave intervals, deflation triggered during R waves – T wave intervals); if the patient has atrial fibrillation, the IABP machine analyzes the QRS complex for time deflation with R waves, whether positively or negatively deflected (Webb et al., 2015). Nevertheless, with poor ECG image quality and or arrhythmias, ECG triggering becomes unreliable, requiring other trigger modes (Webb et al., 2015).

With pressure triggering, the aortic artery waveform triggers inflation and deflation by identifying the dicrotic notch and systolic upstroke. Suppose the aortic artery waveform is of poor quality (due to malfunction, small balloon, etc.). In that case, a peripheral arterial (radial, brachial, or axillary) blood pressure waveform can be used (González & Chaney, 2020). The best timing evaluation is by examining the arterial blood pressure waveform of the central lumen of the IABP (González & Chaney, 2020).The blood pressure waveform in the arterial pathway can be read with optimal timing. The augmented diastolic blood pressure is greater than the unassisted systolic blood pressure, the reduction of the assisted end-diastolic blood pressure and the assisted systolic blood pressure, and the assisted systolic blood pressure is less than unassisted systolic blood pressure. In this case, the IABP for Mr. Y had adjusted the trigger setting with the EKG and Timing inflation point -1.2 and depletion point 0.0.

Physiological Effects

The goal of increasing coronary perfusion pressure induced by diastolic augmentation is achieved by balloon inflation, while balloon deflation reduces afterload and promotes LV ejection. Together, these effects lead to an increase in oxygen delivery with a decrease in myocardial oxygen consumption. This physiological effect has been demonstrated in many studies (Kettner et al., 2013). During cardiac cycles coupled with counterpulsation, balloon inflation at the start of diastole produces an increase in diastolic pressure in the coronary and systemic circulation. Whereas balloon inflation displaces blood volume, the effect on blood pressure is related to the formation of intra-aortic pressure waves rather than changes in bulk flow (De Silva et al., 2014). The effects identified in Mr.Y's patients were systolic pressure in the range of 60-70 mmHg and diastolic pressure in the range of 48-56 mmHg with MAP in the range of 69-82 mmHg.

Afterload reduction

When a boosted beat is compared to an unraised beat, balloon inflation in early diastole decreases systemic vascular resistance through increasing baroreceptor output in response to the transmission of 2 arterial pressure waves (LV ejection and diastolic augmentation with balloon inflation) (González & Chaney, 2020). Balloon deflation results in decreased afterload through a decreased impedance to left ventricular ejection, which results in reduced peak systemic blood pressure, decreased left ventricular systolic load, shorter left ventricular isometric contraction phase, and earlier opening of the aortic valve (González & Chaney, 2020). Meanwhile, Mr. Y, who has a VSR left to right shunt, will increase the workload on the right side of the heart.

Myocardial Oxygen Delivery

The effect of counterpulsation on myocardial oxygen delivery is best studied by examining the Diastolic Pressure Time Index (DPTI) and Tension Time Index (TTI). The DPTI is a surrogate marker for myocardial oxygen supply. It can be calculated from the area between the aortic and left ventricular pressure curves during diastole. Moreover, it depends on aortic diastolic pressure, left ventricular diastolic pressure, and the duration of the diastole. TTI is a surrogate marker for myocardial oxygen demand identified by the area under the left ventricular pressure curve during systole, and it is dependent on left ventricular pressure and afterload (Bonios et al., 2010). In Mr.Y's case, there has been a decrease in cardiac output, with echo results showing reduced LV systolic function / LVEF 50-55% eyeballing with akinetic apical, inferoseptal normokinetic segments. LV diastolic dysfunction, mild TR, normal RV systolic function, LV apical akinetic, VSR Left to Right Shunt 10-11 mm, (CO; 2.3) (CI; 5.3)

(SV; 16) (SVI; 11) (SVR; 1983). Medical applications or outcomes: Myocardial Ischemia, Myocardial Infarction, and Cardiogenic Shock The first documented clinical use of counterpulsation was in patients with cardiogenic shock secondary to acute myocardial infarction. Furthermore, the IABP offered a promising new technology to treat these critically ill patients, whose mortality ranges from 30% - 50% despite advances in therapy (Kantrowitz et al., 1968). Most clinical counterpulsation examinations have focused on patients with myocardial ischemia or infarction, 10% of whom have cardiogenic shock leading to end-organ dysfunction (Prondzinsky et al., 2003). Early observational studies show greater-than-expected survival when IABP is used in the setting of cardiogenic shock (González & Chaney, 2020).

Conclusion

Patients with an Intra Aortic Balloon Pump (IABP) in this VSR case, with a diameter of 10-11 mm, and a semi-auto mode setting, 1:1 frequency, maximum augmentation, and also given inotropic therapy support of 15 mcg/kg of weight/minute and norepinephrine 0.5 mcg/kg of weight/minute, can cause the heart's workload to get heavier. On the other hand, adjustments to hemodynamic stabilization require longer time, until VSR closure surgery is performed. IABP management competencies need to be possessed by nurses who monitor and evaluate patient conditions, as an effort to save patient lives. However, in this case the patient was able to survive 12 hours, until he finally experienced a cardiac arrest, CPR was immediately performed for ± 1 hour and he was declared dead.

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