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Intra-Aortic Balloon Counterpulsation in Low Cardiac Output Noncardiogenic Shock - Case Report and Review

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Abstract

Background: Intra-aortic balloon pump is one of the most frequently used circulatory assist device in cardiogenic shock. However, plenty of patients with low cardiac output shock from non-cardiogenic origin require additional supportive therapy apart from conventional treatment measures.

Case Presentation: We describe two clinical cases of patients with circulatory insufficiency presenting in low cardiac output state. The first one was admitted in our department with urosepsis and presented with low cardiac output and hemodynamic compromise. The second case was with hemorrhagic shock after puncture site complication. Both of the patients stayed refractive to conventional treatment measures and were stabilized by virtue of intra-aortic balloon counter pulsation.

Conclusion: We describe two cases that illustrate the role of IABP support in patients with circulatory insufficiency from non-cardiogenic origin and discuss the possible mechanisms of overall hemodynamics stabilization.

Keywords: Counterpulsation; Septic shock; Hemorrhagic shock

Introduction

The concept of counter pulsation in the management of left-ventricular failure was first described by Kontrowitz and Kontrowitz more than 60 years ago [1]. A few years later, in 1962, Moulopoulus et al. [2] introduced the counter pulsation via device inserted into the aorta. Since then the Intra-Aortic Balloon Pump (IABP) has become a valuable minimally invasive method of augmenting left-ventricular function. Although the use of IABP failed to improve short- and long-term survival after Myocardial Infarction (MI) complicated by Cardiogenic Shock (CS) [3], it is still one of the most frequently used circulatory assist device in CS. In addition, the application of IABP is currently recommended for treatment of mechanical complications after MI [4].

Cardiogenic shock consists of only 15% to 17% of the circulatory insufficiency in the intensive care unit [5]. Distributive and more precisely septic shock is the most common type of shock (62%), followed by cardiogenic and hypovolemic (16%). Plenty of patients with low Cardiac Output (CO) shock from non-cardiac origin require additional supportive therapy apart from conventional treatment measures. Mechanical assistance with IABP may be useful in those clinical situations.

Hereby we describe two cases from our clinical practice that represent common examples of patients with low output shock in the intensive care unit.

Case Presentation

Patient 1

A 72-year-old female patient with a history of nephrolithiasis presented to our emergency department with complaints of excessive fatigue and febrility. She looked in mild distress. Her vital signs on presentation were: Pulse 88 bpm, BP 100/60 mmHg, respiration rate 12/min, SatO2 97% on room air and body temperature of 38.1°C. Auscultation of her heart and lungs was normal. The laboratory results showed leukocytosis with white blood cells count of 15 G/l and increased C-reactive protein 42 mg/l (reference range <5 mg/l). Creatinine was also elevated and eGFR was 39 ml/min/1.73 m^2. Electrocardiogram showed sinus rhythm with left axis deviation, HR of 90 bts/min, no ST-segments abnormalities. Echocardiogram revealed EF 53% with normal kinetics and no significant valvular lesions. Probes from blood and urine were taken for microbiology test. While waiting for the results we started empiric broad spectrum antibiotic treatment with 1 g ceftriaxone IV and saline infusion.

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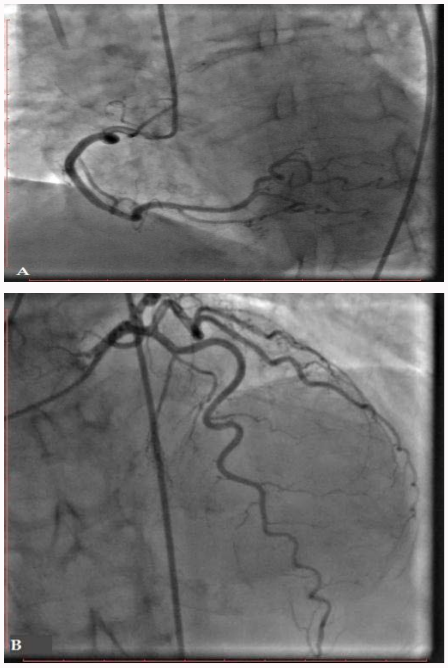

Figure 1: Coronary angiography revealing coronary vessels with no significant stenosis: A) Left anterior oblique view of the right coronary artery; B) Right anterior oblique view of the Left Anterior Descending (LAD) and Left Circumflex (LCx) artery.

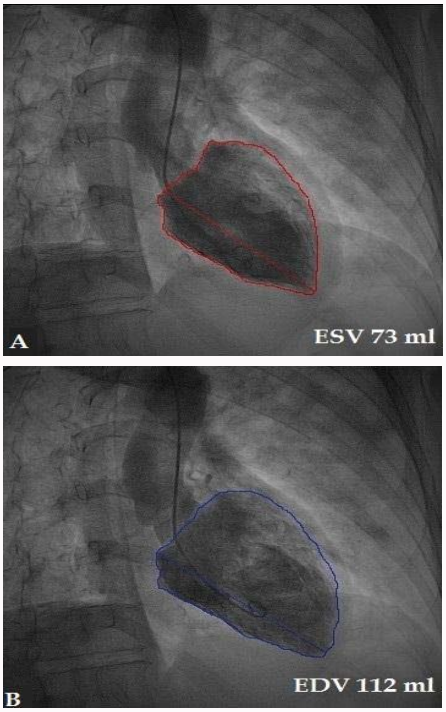

Figure 2: Left ventriculography A) End-Systolic Volume (ESV) 73 ml. B) End-Diastolic Volume (EDV) 112 ml.

In the next few hours there was a rapid deterioration in patient's condition. The blood pressure dropped below 90/60 mmHg, HR 110 bts/min. Patient became dyspneic, diaphoretic with cold and clammy skin. Repeat echocardiography revealed systolic dysfunction with EF 38% and diffusely hypokinetic left ventricle. We transported her to the catheterization lab for emergent examination. Coronary angiography revealed patent coronary arteries with no significant stenosis (Figure 1) and confirmed low EF-35% (Figure 2). Pulmonary angiography did not show pulmonary hypertension or embolism. We started IV infusion with dopamine, norepinephrine and fluid resuscitation. However, the patient's hemodynamics failed to improve and intra-aortic balloon was inserted through the femoral artery using a 7-French sheath. Counter pulsation was programmed in 1:1 mode.

Meanwhile control lab results showed increase in white blood cells 25 G/l and CRP 165 mg/l. On the next day results from blood culture and urine were ready–both positive for Pseudomonas aeruginosa sensitive to cephalosporins. The IABP support was continued for 48 h. During that period the inotrope infusion was stopped and the patient restored normal hemodynamics. After a week on IV antibiotics there was normal recovery.

Patient 2

A woman at 51 years was referred to our department for treatment of symptomatic lower extremity artery disease with already proven stenosis of anterior and posterior tibial arteries on both legs. Her history was significant for end stage renal disease undergoing hemodialysis, and chronic hepatitis C virus. She had previously performed coronary angiography with no significant stenosis on coronary arteries. On admission patient's vital signs included heart rate of 65 bpm, arterial blood pressure of 130/75 mmHg, respiratory rate of 13 breaths per min. Her airway, lung, and heart examinations were unremarkable. Weak peripheral arterial pulsations on popliteal, posterior tibial and dorsal pedal arteries were noted on both legs. Initial laboratory results were significant for anemia (hemoglobin level 93 g/l; red blood cells 3,0 T/l; hematocrit 0,29) and renal failure (Creatinine 1240 mcmol/l, eGFR 9.2 ml/min/1.73 m^2) with elevated potassium of 6.09 mmol. We performed angioplasty of posterior and anterior tibial arteries of the right leg via 6 French femoral sheaths. The procedure went uneventfully with no visible complications. However, 2 h later the woman complained of dyspnea and dizziness. She was diaphoretic, tachycardia, with BP 60/40 mmHg, HR 115 bts/min. Emergent control laboratory test showed significantly decreased hemoglobin level 32 g/l and red blood cells 1.00 G/l.

Urgently the patient was brought into the catheterization laboratory for repeat angiography. We visualized extravasation of contrast material near the puncture site on right femoral artery. A graft stent 6 mm/58 mm on right common femoral artery was implanted with control of the extravasation. Infusion of IV inotropes and iso group hemotransfusion was started. Due to the severe hemodynamic compromise we decided to insert 40 mL balloon IABP in 1:1 mode with arterial waveform synchronization. The patient was stabilized and dehospitalized on 10th day after the intervention.

Discussion

The function of IABP should be accepted as a mean to facilitate left ventricular function without increasing oxygen demand. The balloon placed in the descending aorta is inflated and deflated synchronically with the cardiac cycle, triggered by the signal from electrocardiogram or the systemic arterial waveform. The increase in diastolic pressure during balloon inflation augments diastolic perfusion pressure gradient and coronary circulation [6]. What is more, end-diastolic deflation of the balloon reduces the resistance to left ventricular output, thus reducing after load and myocardial work. Due to reduced end-diastolic aortic pressure the aortic valve opens prematurely, which is leading to shortening of the isometric phase of left ventricular contraction [7]. This is eliciting additional reduction

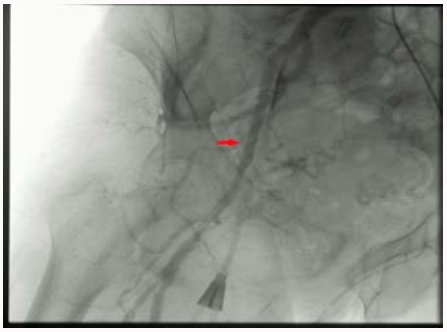

Figure 3: Peripheral angiography- arrow pointing extravasation of contrast material at the puncture site of right femoral artery.

in myocardial oxygen consumption and an overall effect of increase in the myocardial oxygen supply/demand ratio. The potential role of IABP counterpulsation in supporting hemodynamics in low CO shock from non-cardiac origin deserves consideration.

The impairment in cardiac function in sepsis results from a combination of factors. Many authors consider that severe hypotension in septic shock is responsible for reduced myocardial perfusion, leading to ischemic injury and myocardial depression [8,9]. However, others [10,11] argue in favor of a circulating myocardial depressant factor rather than hypoperfusion as the causative factor in septic shock-associated myocardial depression. Potential circulating inflammatory mediators that could lead to depression in cardiac contractility include platelet activating factor, histamine, TNF-alfa, prostaglandins and leukotrienes. What is more pathophysiologic production of NO may contribute to myocardial depression [12].

In studies of large animals with induced gram-negative septic shock, IABP improved survival times and hemodynamics [13,14]. Foster et al. report series of five patients with septic shock refractory to treatment with conventional therapy in which IABP was applied [15]. Two of the patients healed and were discharged from the hospital, but the other three stayed unresponsive to the IABP therapy and perished. Interestingly enough the three deceased patients presented in high cardiac output state – peripheral vasodilation with warm extremities. While the two survivors manifested with cold extremities as a sign of peripheral vasoconstriction and low cardiac output prior to intra-aortic balloon counterpulsation.

IABP may play important role in supporting not only left-ventricular function but overall hemodynamics also. It is known that in condition of hypovolemia and prolonged hypotension the splanchnic vasculature responds with intense arterial vasospasm [16]. It has been suggested that splanchnic visceral ischemia and the subsequent reperfusion injury play central role in the development of the multiorgan failure following shock due to release of inflammatory mediators such as arachidonic acid, cachectin, myocardial depressant factors and others. Landreneau et al. [17] studied the effect of IABP counterpulsation in animals with artificially induced hemorrhagic shock and the splanchnic blood flow response. In the IABP-augmentation group the animals maintained higher mean arterial pressure, cardiac output and peripheral vascular resistance during the hemorrhagic shock. The study shows that IABP counterpulsation may improve splanchnic hemodynamics following a period of ischemia. An interesting finding is that in the group treated with IABP there was an enhancement of renal perfusion [18]. This probably led to lower secretion of angiotensin II and vasopressin during shock, which is known mesenteric vasoconstrictors.

Each of these mechanisms may be important for improvement of myocardial function and hemodynamics. In condition of low cardiac output and circulatory insufficiency, IABP provides temporary assistance to the patient's circulation. It gives the clinician additional time for applying specific therapy and treating the underlying cause for the shock (Figure 3).

Conclusion

Both of the described cases show the development of low cardiac output noncardiogenic shock that was stabilized by virtue of IABP. Counterpulsation mechanical assistance may be of specific value in patients with hypodebit shock refractory to conventional treatment strategies.

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