

ANAESTHETIC MANAGEMENT OF SURGICAL REVASCULARIZATION IN A PATIENT WITH CARDIOGENIC SHOCK AND MULTI-ORGAN DYSFUNCTION FOLLOWING ACUTE MYOCARDIAL INFARCTION

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Abstract: Cardiogenic shock continues to be the leading cause of death in patients of acute myocardial infarction (AMI) despite improvement in coronary care. Conventional medical therapy is associated with an 80-90% mortality rate. The presence of multi organ failure makes the prognosis worse. The dictum "time is muscle" following acute myocardial infarction is more appropriate in the presence of cardiogenic shock. Urgent medical stabilization and intra-aortic balloon counter pulsation help to support and optimize the heart before a definitive intervention. Coronary revascularization if performed early has led to a reduction in the hospital mortality rate to less than 50% as shown by various studies. We report such a case of successful surgical revascularization after initial stabilization by pharmacological and mechanical means in a patient of cardiogenic shock and multi-organ dysfunction following AMI.

Keywords: Cardiogenic shock (CS), percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), early revascularization (ERV).

CS is clinically defined as a state of end-organ dysfunction caused by hypoperfusion secondary to low cardiac output with associated hypotension. A decreased cardiac index ($< 2.2 \text{ L/min/m}^2$) and elevated left ventricular filling pressures (pulmonary capillary wedge pressure $>15 \text{ mmHg}$) defines the hemodynamic criteria of CS¹. CS occurs in 7% of patients with ST elevated MI and 3% with non ST elevated MI². It may also follow unstable angina. It has been shown that aggressive management in the form of early revascularization (percutaneous coronary intervention or coronary artery bypass surgery) results in a 13.2% absolute and 67% relative improvement in six years survival compared to initial medical stabilization³. Supportive measures such as intravenous vasopressor or IABP can complement the benefits of definitive revascularization.

Case Report:

A 64 years old male was referred to our set up in cardiogenic shock and type I respiratory failure on ventilator, inotropic support (dopamine infusion $10 \mu\text{g/kg/min}$) and maximum IABP augmentation (1:1 frequency) with a blood pressure of 101/67 mmHg, heart rate 120 beats per minute and pulse oximetry (SpO_2) reading of 90% at an inspired oxygen fraction (FiO_2) of 1. Chest auscultation revealed bilateral basal crepitations. He had a history of acute onset chest pain and breathlessness two days back. His past medical history was suggestive of type II diabetes mellitus for 10 years and hypertension for 15 years. He was on gliclazide, metformin, metoprolol and atorvastatin. On physical examination, patient was found to be sedated, paralyzed with cold extremities and feeble peripheral pulses. Pupils were of normal size and reacting sluggishly to light. No organomegaly was detected. After admission to the intensive care unit and connecting to pressure regulated volume control mode of ventilation, invasive monitors including central venous and pulmonary artery floatation catheter were inserted which revealed central venous pressure of $12 \text{ cmH}_2\text{O}$, pulmonary artery pressure 46/22 mmHg and pulmonary capillary wedge pressure 25 mmHg. Cardiac output measured by thermo dilution technique was found to be 2.1 l/min . Baseline laboratory work up showed deranged kidney function test (blood urea nitrogen 51 mg/dl , serum creatinine 2.1 mg/dl), coagulation profile [prothrombin time 21.3 sec, International normalized ratio of 1.80, activated partial thromboplastin time (APTT) 37/27 seconds], liver function test [total serum bilirubin 2.67 mg/dl , serum glutamic oxaloacetic transaminase (SGOT) 6364 units/lit , serum glutamic pyruvic transaminase (SGPT) 2267 units/lit , alkaline phosphatase 137 units/lit , lactate dehydrogenase 5830 units/lit] and cardiac enzymes (CPK MB 71.26 ng/ml). Coronary angiography report available with the patient documented triple vessel disease with involvement of the left main coronary artery. Two-dimensional echocardiography showed concentric left ventricular hypertrophy, diastolic dysfunction, moderately severe systolic dysfunction and an ejection fraction of 35%. There was no evidence of mitral

regurgitation or aortic stenosis. Emergency CABG was planned with informed high-risk consent. Patient was shifted to the operation theater on intermittent positive pressure ventilation (IPPV), IABP and dopamine infusion at 15 μ /kg/min along with heparin and insulin infusions. Just before shifting, his blood pressure was 80/46 mmHg, pulmonary artery pressure 48/27 mmHg and SpO₂ of 89% at a FiO₂ 1. In the operation theater, blood pressure dropped to 70/40 mmHg. Dopamine was increased to 20 μ /kg/min. Dobutamine and nor-adrenaline infusions were started at 10 μ /kg/min and 0.05 μ /kg/min respectively. Anaesthesia was maintained on small incremental doses of fentanyl, oxygen, nitrous oxide, isoflurane and vecuronium bromide. After heparinization, cardiopulmonary bypass (CPB) was initiated and bypass grafting of left anterior descending, diagonal and obtuse marginal arteries done on a beating heart using saphenous venous graft as conduit. The patient came off CPB in normal sinus rhythm with dopamine 10 μ /kg/min, dobutamine 5 μ /kg/min and nor-adrenaline 0.05 μ /kg/min support. Immediately after CPB, his blood pressure was 90/48 mmHg, with pulmonary artery pressure of 41/24 mmHg and 10 cmH₂O central venous pressure. He was shifted to the intensive care unit (ICU) in IPPV, IABP and dopamine, dobutamine support of 5 μ /kg/min each with a positive intraoperative fluid balance of 125 ml (haemofiltration removed 1400 ml of fluid). In the ICU, his blood pressure was 105/64 mmHg with pulmonary artery pressure 36/22 mmHg, pulmonary capillary wedge pressure 20 mmHg, SpO₂ 90% and heart rate 120 beats per min. Cardiac output was 2.5 l/min. He was electively ventilated by pressure regulated volume control mode of ventilation. From the first postoperative day (POD), the patient started showing signs of clinical improvement. Arterial blood gas values (table 1) and laboratory parameters (table 2) improved significantly. Cardiac output gradually improved to 3.5 l/min on the 2nd POD and dopamine was tapered off. IABP was removed on the 3rd POD along with dobutamine. Patient was extubated on the 4th POD and discharged from the ICU on the 6th day.

Discussion:

The classical paradigm of CS is progressive myocardial ischemia and dysfunction following an episode of AMI. This is followed by a vicious cycle of reduced cardiac output (CO), hypotension, further coronary insufficiency, and further reduction in contractility and CO. Various other causative mechanisms have also been implicated in its pathogenesis. Patients with CS may develop a systemic inflammatory response syndrome similar to that seen in patients with sepsis with complement activation and release of inflammatory cytokines (interleukin 6, interleukin 1, tumor necrosis factor α). Expression of inducible nitric oxide (NO) synthase and inappropriate vasodilation by high level of NO may play an important role in the evolution and outcome of CS⁴.

Triple vessel disease is found to be present in 60% cases and left main coronary artery disease in 20% cases of CS⁵. In the presence of multivessel CAD, the ability of the unaffected myocardium to be recruited to preserve left ventricular function is impaired⁶. The loss of normal compensatory mechanism of hyper contractility in unaffected myocardium results in the inability to maintain cardiac output, which leads to progressive ischemia in remote territories and further deterioration in function. Inadequate oxygen delivery to tissues may be assessed by alterations in mixed venous oxygen saturation, lactate level and base deficit. Clinically end organ perfusion can be assessed by urine output, serum creatinine, mental status and liver function. Except examination of mental status, all other parameters were affected in our patient

Various options are available for restoring coronary perfusion, such as, systemic fibrinolytic therapy; percutaneous coronary intervention in the form of selective fibrinolysis, angioplasty and stenting; and surgical revascularization. We continued IABP for preoperative stabilization of the patient. IABP increases the aortic diastolic pressure and thereby improves coronary perfusion and also decreases systemic afterload without increasing myocardial oxygen demand. In the 'SHould we emergently revascularize Occluded Coronaries for cardiogenic shock (SHOCK) trial' registry, initial stabilization of patients in CS using IABP was associated with a 20% absolute risk reduction in mortality^{7,8}. The American College of Cardiology/ American Heart Association guidelines for ST elevation MI has given IABP therapy a class I recommendation for CS not quickly reversed with pharmacological therapy as a stabilizing measure for angiography and ERV. Thrombolytic therapy is not beneficial in patients with CS probably due to reduced lysis of thrombi as a result of low perfusion pressures⁹. IABP can improve the result of thrombolytic therapy by increasing the perfusion pressure. In our patient thrombolytic therapy was ruled out because of the late presentation to our set up.

Improved survival after CABG has been shown for patients with significant left main coronary artery disease and those with involvement of three or more coronary arteries¹⁰. The SHOCK trial had demonstrated the merits of emergency CABG in patients of MI complicated by CS^{7,8}. In the trial, one-year survival was found to be 46.7% for patients in ERV group compared to 33.6% in the initial medical stabilization group. In the trial, patients chosen for surgical revascularization were more likely to have left main disease and triple vessel disease than those treated with PCI. On the basis of the trial, the American College of Cardiology/ American Heart Association (ACC/AHA) has recommended ERV for shock as a class I indication, particularly for patients less than 75 years of age¹¹. PCI was not tried in our patient because of the diffuse coronary disease and haemodynamic instability.

Although patients with CS represent a minority of the total population of CABG, it accounts for almost 14% of deaths in CABG

patients¹². Anaesthetic management of patients in CS undergoing CABG is very challenging. Goals of anaesthetic management include maintenance of myocardial oxygen demand and supply ratio, monitoring for and prevention of further myocardial ischemia by maintaining normal (or usual for the particular patient) arterial blood pressure, low heart rate, low filling pressures, control of afterload and prevention of acidosis. Haemodynamically unstable patients can be supported by cardiopulmonary bypass (CPB) as soon as possible. While on CPB, the heart is emptied of blood and allowed to rest, which greatly diminishes myocardial oxygen demand while coronary perfusion pressure is maintained by CPB. We used vecuronium bromide as muscle relaxant to avoid further rise in heart rate. Maintenance of proper depth of anaesthesia is important for preventing awareness and undue sympathetic stimulation and subsequent rise in myocardial oxygen demand. Use of pulmonary artery catheter (PAC) is indispensable in the management of CS patients. It helps in measuring pulmonary artery pressure, left ventricular filling pressure (preload of the left ventricle) and cardiac output. PAC can be used as a guide for fluid and vasoactive drug administration in the perioperative period also as central venous pressure is not an accurate guide for measuring LV filling pressure¹³. Sustained atrial and ventricular arrhythmias or heart block should be reverted back to sinus rhythm by means of drugs or temporary pacing to maximize CO.

Table 1: Arterial Blood Gas values

Parameter	Preoperative	Immediate Postoperative	1 st POD	2 nd POD	3 rd POD	4 th POD	5 th POD
FiO₂	0.8	0.8	0.6	0.6	0.6	0.4	0.3
pH	7.39	7.27	7.45	7.49	7.42	7.46	7.47
PCO₂	39.1	51.7	44.7	40.7	45.9	41.0	34.6
PO₂	75.8	69.6	69.5	68.5	67.2	68.9	65.5
Glucose	137	174	155	160	140	136	155
Lactate	3.2	5.5	1.2	1.4	1.7	1.2	1.3
SO₂	93.2	85.4	93.8	93.8	92.8	93.5	92.2
HCO₃	23.8	23.0	30.4	31.1	29.1	28.8	25.0
Base Excess	0.3	-3.5	5.9	7.5	4.4	5.0	2.1

Table 2: Blood Biochemistry

Parameters	Pre operative Values	1st POD	2nd POD	3rd POD	5th POD
Haemoglobin (gm/dl)	10.2	10.9	11.1	10.1	11.4
Haematocrit (%)	30.4	32.1	32.5	30.7	33.8
Total Leucocyte Count	10200	12000	11700		
Blood Urea Nitrogen (mg/dl)	51	52	62	93	76
Serum Creatinine (mg/dl)	2.1	1.8	2.1	3.3	2.5
Prothrombin Time (sec)	21.3	19.5	16.7	16.2	
International Normalized Ratio (INR)	1.80	1.61	1.32	1.23	
APTT (sec)	37/27	33/27	32/27		
Total Serum Bilirubin (mg/dl)	2.67	2.76		2.79	
SGOT (units/lit)	6364	3264		762	
SGPT (units/lit)	2267	1477		744	
Alkaline Phosphatase (units/lit)	137	140		158	
Lactate Dehydrogenase (units/lit)	5830	3798		1576	
CPK MB (ng/ml)	71.26	52.6			

Conclusion:

CS is a rapidly progressive, often fatal complication of MI. Early establishment of aggressive reperfusion therapy, along with various pharmacological and mechanical means, is essential for better outcome in such patients. Rapidly re-establishing infarct related artery (IRA) blood flow is the cornerstone of management. IABP is recommended when shock is not quickly reversed with pharmacological therapy, as a stabilizing measure for patients who are candidates for further invasive care. Fibrinolytic therapy with IABP counterpulsation should be considered if revascularization therapy is not rapidly available. Survivors, however,

should be shifted to a set up with revascularization capability as soon as possible for further management.

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