Low cardiac output syndrome after cardiac surgery with extracirculatory at cardiovascular center, E hospital

Hoang Van Trung¹, Pham Thi Thu Trang¹, Dam Hai Son¹, Nguyen Tran Thuy¹,²*

ABSTRACT

Background: Low cardiac output syndrome is one of the most severe complications after cardiac surgery, associated with 38% mortality, increased rates of respiratory complications, myocardial infarction, stroke, and heart failure. If the signs are detected early and diagnosed in time, the treatment will give better results.

Objectives: Description of clinical, subclinical characteristics and evaluation of early results of treatment of patients with low cardiac output syndrome after cardiac surgery at Cardiovascular Center, E hospital.

Methods: Retrospective report of 3 cases of low cardiac output syndrome with confirmed diagnosis, treatment and comparison with the literature.

Results: From January 2022 to May 2023, 3 patients with low cardiac output syndrome were confirmed at the cardiovascular center, E hospital. The mortality rate is 66.7%, mean age was 57.3 ±9.3, mean preoperative EF 62 ±13.7, mean postoperative cardiac index 1.5 ±0.2 l/minute/m², mean extracorporeal circulation time 172 ±63.4 minutes, mean aortic cross-clamp time 107 ±28.4 minutes.

Conclusion: Low cardiac output syndrome after surgery is a serious complication with high mortality risk. Doctors need to know the early signs for timely diagnosis and proper treatment to help patients recover best.

Keywords: Low cardiac output syndrome, open heart surgery

INTRODUCTION

Low cardiac output syndrome (LCOS) is one of the most serious complications after cardiac surgery, associated with 38% mortality rate [1],[2] an increased rate of respiratory complications, myocardial infarction, heart attack, stroke, kidney failure, and re-surgery [3]. The most common definition of LCOS includes a decrease in cardiac index (CI) < 2 l/min/m², systolic blood pressure < 90 mmHg, associated with signs of tissue hypoperfusion (cold, moist, irritated skin, confusion, oliguria, increased lactate) and no hypovolemia. The use of vasopressors or mechanical circulatory support is always required to improve the hemodynamics of the patient [4]. LCOS reduces blood flow, reduces oxygen to the liver, kidneys, lungs and even the coronary arteries that feed the heart muscle, thus impairing the function of these organs. Failure of these organs will negatively affect the cardiovascular and systemic functions, gradually the patient will have multi-organ failure and death.

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Table 1. Risk factors and prognosis of LCOS

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<th>Preoperative factors</th>
<th>Factors in surgery</th>
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<tr>
<td>Age &gt; 65</td>
<td>Long CPB time</td>
<td>Hemoglobin</td>
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<tr>
<td>EF &lt; 50%</td>
<td>Incomplete revascularization</td>
<td>Lymphocyte count &lt; 2000 cells/mcl</td>
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<td>CABG with CPB</td>
<td>Emergency surgery</td>
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*(EF: left ventricular ejection fraction, CABG: coronary artery bypass graft, CPB: Cardiopulmonary bypass)*

The treatment of this syndrome is still difficult and the mortality rate is high. Therefore, it is necessary to know the risk factors, causes and early signs of LCOS for timely diagnosis and treatment. We would like to present a series of cases diagnosed with LCOS at Cardiovascular Center, E Hospital.

**RESEARCH OBJECTIVE**: Describe the clinical and paraclinical characteristics and evaluate the early results of treatment of patients with LCOS after cardiac surgery at the Cardiovascular Center, E Hospital.

**STUDY SUBJECTS AND METHODS**: Retrospective report of 3 cases of LCOS diagnosed, treated and compared with the literature.

**RESULTS**: From January 2022 to May 2023, there were 3 patients with LCOS were diagnosed at Cardiovascular Center, E Hospital. The mortality rate was 66.7%, mean age was 57.3 ±9.3, mean preoperative EF 62 ±13.7, mean postoperative cardiac index 1.5 ±0.2 l/min/m², mean extracorporeal circulation time 172 ±63.4 min, mean aortic cross-clamp time 107 ±28.4 minutes.

**REPORTING CASES**

**Case report 1:**

Patient Phung Thi L, female, 51 years old, hospitalized on September 16, 2022, history of rheumatism 43 years ago, no previous history of cardiovascular disease was detected. 4 months before admission, the patient appeared tired, had difficulty breathing on exertion, was diagnosed with mitral stenosis, treated at the provincial hospital for 2 times without improvement, transferred to cardiovascular center. Examination: Patient awake, dyspnea NYHA III, heart rate complete arrhythmia, rate 90 beats/min, blood pressure 100/70 mmHg, diastolic murmur 4/6, clear alveolar murmur, soft abdomen, liver, spleen not palpable, no edema, urine 1300ml/24h.

Echocardiography: severe mitral regurgitation, moderate stenosis, leaflet thickening, calcification, margin adhesions, Dd (left ventricular end-diastolic diameter) 55 mm, Ds (left ventricular end-systolic diameter) 31 mm, EF 74%, LA (left atrial diameter) 53 mm, PAP (pulmonary artery pressure) 28 mmHg. The aortic valve and the tricuspid valve are normal.
Electrocardiogram: Atrial fibrillation, frequency 81 beats/min

At 4 pm on October 21, 2022, the patient underwent endoscopic mechanical mitral valve replacement, left atrial appendage suture, and atrial fibrillation with high-frequency waves according to Cox-Maze IV diagram. Extracorporeal circulation time is 243 minutes, mean aortic cross-clamp time is 139 minutes, intraoperative infusion of 700ml red blood cells. The patient was transferred to resuscitation at 22:45 on the same day in a state of sedation and mechanical ventilation, pulse 83 beats/min, sinus rhythm, blood pressure 129/80 mmHg (maintenance of noradrenaline 0.1 mcg/kg/min), extubated. administered at 10:00 am on October 22, 2022. At 20:00 on the same day, the patient appeared to have difficulty breathing with great exertion, cold skin, rapid pulse 133 times/minute, blood pressure 60/30 mmHg, CVP 11mmHg, urine 20ml/h. Arterial blood gas: pH 7.25, lactate 8. Tests show kidney failure, acute liver failure, urea 16 mmol/l, creatinine 168mcemol /l , GOT 625 U/l, GPT 284 U/l , echocardiography: poor contractility, CI 1.5l/min/ m², the patient was intubated, mechanically ventilated, maintained adrenaline 0.8 mcg/kg/min, noradrenalin 0.2 mcg/kg/min , dobutamine 10 mcg/kg/min, indicated intra aortic balloon counterpulsation. During resuscitation there are multiple ventricular tachycardias, the patient receives electric shock and medication for arrhythmias. After that, the patient's course improved, organ function gradually recovered, vasopressors gradually decreased, intra aortic balloon withdrawal after 5 days, endotracheal intubation after 14 days. Stopping all vasopressors after 18 days , total recovery time 18 days. The patient was discharged after 34 days of treatment with consciousness, NYHA I dyspnea, pink mucous membranes, no fever, urinary 1500ml/24h, no edema, mechanical artificial mitral valve echocardiography in place, operational, Dd 42 mm, Ds: 29 mm, EF 59%, no regional dyskinesia, sinus rhythm electrocardiogram frequency 97 times per minute.

Case report 2:

Patient Nguyen Thi Y, female, 68 years old, admitted to the hospital on August 18, 2022, history: mitral stenosis discovered 10 years ago, dilated valve once 8 years ago, regular drug treatment. This time the patient feels tired, has difficulty breathing with exertion, is admitted to the hospital in a conscious state, shortness of breath NYHA III, 75times/minute, blood pressure 110/70 mmHg, complete cardiac arrhythmia, diastolic 3/6, clear alveolar murmur, mild edema of the lower extremities, urinary 1000ml/24h

Electrocardiogram: Atrial fibrillation, rate 87 beats/ min

Echocardiography: Mitral stenosis, moderate regurgitation, leaflet thickening, calcification, retraction, margin adhesions, valvular orifice area 1.05 cm², PAP 40 mmHg, Dd 43 mm, Ds 25mm . EF 65%, LA 58 mm. Mild aortic valve and tricuspid valve mild regurgitation

13h30 on 8 September 2022, the patient underwent endoscopic surgery to replace the biological mitral valve and suture the left atrium. Extracorporeal circulation time is 152 minutes, mean aortic cross-clamp time is 97 minutes, intraoperative infusion of 700ml red blood cells. The patient was transferred to the intensive care
unit at 6:30 pm on the same day, in a state of sedation, mechanical ventilation, pulse 130 times/min, blood pressure 118/80 mmHg (dobutamine 7.5 mcg/kg/min). At 8:00 am on 09/09, the patient appeared cold skin, 3000ml/24h, pulse 150 beats/min, blood pressure 70/40mmHg, CVP 10 mmHg, 3 vasopressors adrenaline 0.4 mcg/kg/min, noradrenaline 0.3mcg/kg/min, dobutamine 15mcg/kg/min, arterial blood gas pH 7.56, lactate 6.6, acute liver failure, renal failure: urea 18 mmol/l, creatinine 171 mcmol/l, GOT 722 U/l, GPT 445 U/l, NTproBNP > 70000, procalcitonin: 79. Echocardiography: CI 1.7 l/min/m². The patient was maintained with 3 vasopressors, intra aortic balloon counterpulsation, and received intensive resuscitation treatment. After 2 weeks, the patient died in multiple organs failure.

**Case report 3:**

Patient Ta Thi T, female, 53 years old, with a history of mitral stenosis for many years, without treatment, this episode presented with shortness of breath, fatigue, and was admitted to the hospital in a state of shortness of breath NYHA IV, pulse 100 beats/min, blood pressure 125/82 mmHg, complete heart arrhythmia, 3/6 diastolic murmur, clear alveolar murmur, lower extremity edema, urine 1300ml/24h

Electrocardiogram: Atrial fibrillation, frequency 122 beats/min

Echocardiogram: mitral stenosis, severe regurgitation, leaflet thickening, calcification, retraction, margin adhesion, orifice area 0.92 cm², PAP 55 mmHg, Dd 56mm, Ds 42mm. EF 47%, LA 129 mm. Mild aortic valve regurgitation, moderate tricuspid valve regurgitation

At 2:20 p.m. on August 31, 2022, the patient underwent mechanical mitral valve replacement, left atrial appendage suture, and tricuspid valve repair. Extracorporeal circulation time is 121 min, mean aortic cross-clamp time is 97 minutes. During surgery, there is no need for infusion erythrocyte mass. The patient was transferred to the intensive care unit at 9:30 p.m. on the same day, in a state of sedation, mechanical ventilation, pulse 120 beats/min, blood pressure 120/76 mmHg (dobutamine 15 mcg/kg/min, noradrenaline: 0.5 mcg/kg/min, arterial blood gas: pH 7.55, Lactate: 6.3. At 7:40 am on September 1, the patient appeared ventricular fibrillation, circulatory arrest -> emergency circulatory arrest, electric shock. After 30 minutes of emergency, the patient's pulse returned, blood pressure was 80/50 mmHg, CVP 12 mmHg, maintained 3 vasopressors, anuric, and acute renal failure was tested. urea 16mmol/l, creatinine 205 mcmol/l, NTproBNP > 70000, GOT 892 U/l, GPT 95 U/l, Echocardiogram: CI 1.3 l/min/m² -> maintain 3 vasomotor, ECMO support.

3/9: sedation on mechanical ventilation, ECMO, blood pressure 60/40 mmHg (3 vasopressors at maximum dose), worsening condition and death

**DISCUSSION**

**1. Risk factors**

- **Age:** The average age of the patients in the study was 57.3 years old, the oldest patient was 68 years old, the youngest patient was 51 years old. Many studies have confirmed that advanced age, female are independent prognostic factors of LCOS after cardiac surgery, the study of RAO et al [5] showed that age over 70 (OR=1.5), female (OR=1.5) OR=2.5). Manganti
M’s study. [2] found that advanced age (OR=1.02) and female gender (OR=2.8) were found to be risk factors for LCOS after cardiac surgery. Author Sá MP et al. [6] studied 605 patients undergoing coronary artery bypass graft surgery and showed that age ≥60 (OR=2.1) is also a prognostic factor for LCOS. In our study, there were 2 patients < 60 years old and 1 patient > 60 years old.

- **NYHA, EF grade:** NYHA grade III, IV and low left ventricular EF, which are representative factors for preoperative left ventricular dysfunction, have also been shown to be related to the prognosis of needing cardiac drugs, vasomotor, mortality [6], [7], [8]. Our 3 study patients all had NYHA class III, IV dyspnea, 1 patient had preoperative EF 47%. According to Boosma [12] EF < 50% preoperatively is an independent risk factor for predicting an increased rate of right ventricular failure and postoperative mortality.

- **Pulmonary artery pressure:** In a study of 2149 patients undergoing coronary artery bypass surgery, Reich found that pulmonary arterial hypertension was one of four non-independent predictors of the need for inotropic drugs. [9]. The PAP of the patients in the study were 28 mmHg, 40 mmHg, and 55 mmHg, respectively.

- **Extracorporeal circulation time and aortic cross-clamp time:** In our study, the mean extracorporeal circulation time was 172 minutes, the mean aortic cross-clamp time was 107 minutes. This is also equivalent to the studies in the literature that all agree on the duration of extracorporeal circulation time, aortic cross-clamp time is a predictor of post-operative circulatory dysfunction. According to Manganti, a long recovery time is a prognostic factor for LCOS (OR = 1.02 with p < 0.001) [1].

- **Transfusion of red blood cells in surgery:** According to author Le Xuan Duong et al. [10], infusion of ≥750 ml of red blood cells in surgery is also a risk factor for LCOS, erythrocyte mass contains biologically active products that stimulate systemic inflammatory responses, including nonspecific immune responses and local occlusion of capillaries leading to tissue hypoxia. In our study, 2 patients had to transfuse 700 ml of red blood cells.

- **Other factors:** Cardiac surgery with extracorporeal circulation triggers an inflammatory response in myocardial and systemic tissues, which is a consequence of intraoperative trauma, and organ reperfusion injury, which affects to cardiac rehabilitation after surgery. Many authors found that metabolic disorders are also a risk factor for cardiac rehabilitation after surgery, decreased pH, and metabolic acidosis of myocardium are prognostic factors for the use of inotropic drugs after extracorporeal circulation [11].

2. Diagnosis

Patients with all the criteria for the diagnosis of LCOS:

- Low CI (< 2l/min/m²)
- Low systolic blood pressure (<80mmHg), blood pressure measured by continuous monitoring of arterial blood pressure, including systolic blood pressure, diastolic blood pressure, mean blood pressure
- Signs of tissue hypoperfusion:
  - Cold skin: manifestation of peripheral vasoconstriction due to hypoperfusion
Arterial blood lactate is elevated (>6mmol/l). When peripheral perfusion is reduced, the tissues do not have enough oxygen to participate in metabolism, leading to anaerobic metabolism that produces a lot of lactic acid. High concentration of lactic acid converted to lattac salt.

- Patients without volume deficiency (CVP 10-12mmHg), who do not respond to infusion test

3. Treatment

- Ensure adequate ventilation and oxygenation. The patient is mechanically ventilated through an endotracheal tube, and the parameters on the ventilator are adjusted by arterial blood gas results

- Treatment of anemia or coronary artery spasm. In the early stages usually respond well to nitroglycerin

- Optimize preload:
  + Maintain a reasonable amount of fluid to avoid fluid loading but do not let the lack of fluid reduce blood volume.
  + Patients who do not respond to high-dose diuretics should receive continuous dialysis for water withdrawal, electrolyte balance, alkaline acidosis.

- Maintain proper heart rate (90-100 beats/min) and correct arrhythmias. Adjust to sinus rhythm to ensure synchronicity of atrioventricular and stroke volume. Moreover, when the heart is in sinus rhythm, mechanical support methods will be more effective

- If necessary, a temporary pacemaker can be used. If arrhythmia or tachycardia persists, electric shock may be used

- Improve myocardial contractility: With heart medication, vasopressors adrenalin, noradrenalin, dobutamin. If hemodynamic status does not improve, the patient is supported with intra aortic balloon counterpulsation, ECMO. In this study, all three patients were mechanically supported with intra aortic balloon counterpulsation and ECMO.

- Reducing afterload: For patients with left ventricular failure, it is necessary to reduce afterload to reduce the burden on the left ventricle. Use systemic vascular resistance to determine. If systemic vascular resistance is high, use vasodilators: Nitroprusside, nitroglycerin. If systemic vascular resistance is low, use vasopressors to maintain visceral perfusion pressure: Noradrenaline

- Intra aortic balloon counterpulsation: Immediately after being diagnosed with LCOS, the patient is placed in order to increase cardiac output, increase coronary perfusion, and reduce preload.

- Blood transfusion to Hemoglobin > 80g/dl, hematocrit > 26%. Plasma Transfer to Maintain Colloidal Pressure [2].

CONCLUSIONS

Low cardiac output syndrome is a serious and common complication following cardiac surgery with LVEF. Mortality rate is very high (3-45%), so it is necessary to identify risk factors before and during surgery to predict as well as plan emergency facilities. Doctors need to know the early symptoms as well as the diagnostic criteria in order to timely detect and treat the patient in the right way to help the patient recover the best.
REFERENCES


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