

Ventricular Failure After Pericardial Decompression- Successful Outcome By Early ECMO Implant

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ABSTRACT

We report a case of a 68-year-old woman referred to our hospital after being diagnosed of acute coronary syndrome with ST elevation but no evidence of coronary stenosis amenable of percutaneous or surgical approach. She was transferred to our institution due to pericardial effusion with incipient haemodynamic compromise and under suspicion of contained cardiac rupture. Pericardial collection was shown in a routine transthoracic echocardiogram study meanwhile the patient was recovering from coronary event in a different hospital with no Cardiac Surgery facilities. After sternotomy and removal of pericardial fluid, biventricular failure developed with no answer to inotrope support. Venous-arterial extracorporeal membrane oxygenation (VA-ECMO) through femoral vasculature was initiated in few minutes. The outcome of this patient was succesful and biventricular function was completely recovered.

Keywords

Biventricular dysfunction, Pericardial decompression syndrome, Extracorporeal membrane oxygenation.

Case Presentation

Pericardial decompression syndrome (PDS) is a rare clinical condition with a pathophysiological basis that is still unclear and results in a potentially fatal outcome for patients who suffer from it. The clinical picture consists of left, right or biventricular acute failure occurring in some patients after percutaneous pericardiocentesis or surgical pericardial window. A common indication to perform these procedures is pericardial effusion that causes hemodynamic instability (cardiac tamponade) or evolves towards that condition. In the current scientific literature there are about 35 cases of this syndrome published from 1983 to 2013 [1,2], although the term pericardial decompression syndrome was not coined until 2010 [3]. According to the current literature, the incidence could be around 4% after subxiphoid pericardiocentesis and 11% after surgical window, although the degree of ventricular dysfunction in each case is very variable. Mortality varies between 29 and 80% [1,2], although these data could be sensibly different due to little scientific evidence available.

We present the case of a patient who suffered a clinical setting consistent with PDS in a situation of pulse-free electrical activity that required implantation of VA-ECMO in the intraoperative period for pericardiotomy indicated by incipient cardiac tamponade. The patient was a 68-year-old woman with no allergies, no toxic habits, total thyroidectomy in 2004 with secondary hypothyroidism and hypoparathyroidism, hypocalcemia with hypercalciuria. Home treatment included statins, calcium, vitamin-D, hydrochlorothiazide, PPI and Levothyroxine. On September 29th, the patient presented an antero-lateral cardiac infarction Killip I (ultrasensitive Troponin I peak 2539 ng / L).

Coronariography study revealed a dissected diagonal branch (vessel <1mm) as a possible cause of the ischemia, but due to the small diameter this vessel was not percutaneously treated. She was admitted to the Critical Care Unit for medical surveillance and treatment, receiving double antiplatelet therapy. After 24-48 hours, the patient developed chest pain of pleuropericarditic characteristics accompanied by an echocardiogram imaging showing light to moderate pericardial effusion, with fibrin, and incipient data of hemodynamic compromise. Contrast-enhanced Chest CT was performed suggesting acute transmural ischemic lesion in the apex and free

wall of the left ventricle, medium-sized pericardial effusion that could correspond to complex fluid / haemopericardium, as well as small bilateral pleural effusion. Under clinical suspicion of cardiac rupture with hemopericardium she was transferred to our center to assess surgical treatment.

Upon admission to the Coronary Care Unit, the patient was hemodynamically stable, with tendency to slight hypotension and tachycardia, and the clinical symptoms of chest pain with pleuropericarditis characteristics persisted. Bedside echocardiogram revealed slight-moderate global pericardial effusion, with fibrin and partial collapse of right cavities, without significant transmitral flow variation during the respiratory cycle at this time. VCI was dilated, without inspiratory collapse. Severe LVH and preserved LVEF, with latero-apical hypokinesia were described. Progressive loss of hematocrit (40% to 33%) was observed. Expectant attitude was decided, but during the next hours impairing of haemodynamic situation despite fluid administration and echo signs of cardiac tamponade with flow variation through mitral valve, indicated urgent surgical intervention.

After monitoring and anesthetic induction, sternotomy and pericardiectomy were performed, with leakage of serous fluid under tension, with a yellowish appearance, ruling out cardiac rupture and suspecting inflammatory pericarditis after ischemic insult. Seconds after the pericardic decompression, patient presented hemodynamic deterioration, electrical activity without pulse and no response to inotropic drugs. Open thorax massage was administered and implantation of VA-ECMO through left femoral vessels was decided. Tissue perfusion was restored thanks to the device. Intraoperative transfusion of 5 packed red cell concentrates, 1 platelet pool, 4 units of fresh frozen plasma and 2 grams of Tranexamic Acid. After sternotomy closure, coronary angiography was performed with no evidence of stenosis or dissection of any coronary branch and ventriculography revealed global akinesia. Under diagnosis of pericardial decompression syndrome (in the absence of another explanation), she was admitted to the Postoperative Critical Care Unit.

During the first 72 hours of admission, the patient was under mechanical ventilation, with VA-ECMO at 4.2 bpm needing high FiO₂ in both ECMO and ventilator to maintain oxygenation, Dobutamine, epinephrine and levosimendan perfusions were administered. Portable X-Ray showed acute pulmonary edema and bilateral pleural effusion. Renal function was preserved and no neurologic deficits were found. Despite no anticoagulation, diffuse significant bleeding by cannulae entrance area and by sternotomy (patient was under double antiplatelet therapy, platelet mapping indicated almost complete inhibition of platelet activity), requiring polytransfusion including recombinant factor VII (transfusion therapy guided by thromboelastography and platelet mapping) and surgical review for hemostasis and tentative thorax closure by a pericardial patch.

On the other hand, patient had persistent hypocalcemia (hypoparathyroidism) which needed correction in order to avoid

impairment in contractility and hemostasis, requiring 6 grams of calcium chloride every 24 hours.

On day four coagulopathy was corrected and bleeding reduced, patient kept without anticoagulation and VA-ECMO around 3500 rpm to avoid coagulation of the system. Daily transesophageal echocardiogram was performed, which initially showed a severely depressed ejection fraction with apical akinesis, but in subsequent echocardiograms, gradual recovery of ventricular function was observed, although alterations in apical contractility persisted. On the 5th day of admission, inotropic support was withdrawn. The 7th day of admission ECMO-VA was removed (after about 180 hours of use). From respiratory point of view, the patient also presented gradual improvement with negative fluid balances, protective ventilation and steroids, requiring inhaled O₂ around 50%. On the 10th day of admission, the sternum was definitively closed. The patient was extubated on day 11th after 288 hours of VMI, oxygen therapy through high-flow nasal cannulas was initiated. From the 12th to the 22nd day of admission, respiratory and hemodynamics optimization were prosecuted: second levosimendan perfusion, respiratory physiotherapy, and negative fluid balance was continued. On day 20th of admission surgical revision due to subpectoral hematoma was required. On day 23th: discharge to conventional hospitalization floor.

During the first week of December, the patient was discharged from hospital.

Discussion

Pericardial decompression syndrome (PDS) is a term for a well-defined but rather underreported complication of pericardial drainage [3].

Normal response to pericardial decompression is haemodynamic improvement and relieving of symptoms but in some rare cases a paradoxical response appears after drainage [4]. This clinical syndrome was first described in 1983 by Vandike alluding to pulmonary edema after pericardiocentesis [5]. The term PDS was coined by Angouras and Dosios in 2010.

Pathophysiology of this syndrome has been explained through several routes [6]:

- The hemodynamic hypothesis [5]: A mismatch between preload and afterload develops with a rapid drainage of a large pericardial effusion, so that a sudden increase in venous return and consequently in right ventricle preload produces a left ventricle overload.
- Ischemic hypothesis [7]: Increased pericardial pressure causes a decrease in coronary blood flow.
- Sympathetic overdrive hypothesis [8]: Pericardial fluid is a stimulus for the sympathetic nervous system that, once relieved, may unmask an underlying ventricular dysfunction supported during sympathetic stimulus.

Whatever the origin of this syndrome, its rarity makes it is underestimated and, in some cases, an underlying pathology that

may affect myocardial function (malignant disease, chemotherapy induced cardiomyopathy) [3].

The originality of our case is the ultra-early implant of VA-ECMO therapy when we could check the absence of response to inotropic support. In the scientific literature, we could only find another article describing the use of ECMO for successful resuscitation of a patient with PDS [4].

ECMO therapy is not free from complications, and, as we described above, our patient needed surgical review due to severe bleeding and also a deferred chest closure was necessary. Despite these derivative effects, extracorporeal support was definitive to achieve hemodynamic and respiratory stability so that this patient fully recovered and was discharged from hospital.

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