

## Iatrogenic Thoracoscopic Right Ventricular Laceration Resulting in Cardiovascular Collapse Treated With Extracorporeal Membrane Oxygenation

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**I**ATROGENIC CARDIAC LESION is an uncommon yet dangerous complication of thoracoscopic surgery. Its management is complicated by several factors including rapid blood loss with ensuing instant hemodynamic instability, difficulty of repair because of lateral positioning and limited access to the heart, heart motion, and blood in the pleural cavity obstructing the surgical visual field. The use of extracorporeal membranous oxygenation (ECMO) provides a valuable tool to manage iatrogenic laceration of the right ventricle (RV) during video-assisted thoracoscopic surgery (VATS) that results in cardiopulmonary arrest. ECMO allows aggressive and prompt volume expansion while diminishing blood flow to the RV, helping ease the repair of a right ventricular laceration and postoperative life support in the intensive care unit.

### CASE REPORT

A 54-year-old woman was injured in a motor vehicle accident with left pulmonary contusion and severe pneumohemothorax. The patient had no comorbid conditions or surgical history. A large-bore chest drainage tube was placed, and the patient subsequently recovered. The patient was discharged home after the chest tube removal 2 days later. A chest x-ray after chest tube removal showed no pneumohemothorax.

One month later, the patient complained of respiratory difficulty. A chest x-ray showed large left pleural effusion (Fig 1). VATS was scheduled to relieve her symptoms and find the source of pleural effusion. Preoperative evaluation revealed no other abnormalities, a normal electrocardiogram and laboratory profile. Hemoglobin was 11.1 g/dL, and hematocrit was 33.7%, which are similar to those before chest tube placement.

The patient was premedicated orally with 10 mg of diazepam 1 hour before surgery. Venous access was achieved with an 18-G catheter in the right forearm. An arterial catheter was inserted in the left radial artery. General anesthesia was induced with a bolus of 100 µg of fentanyl and 150 mg of propofol. Relaxation was achieved with rocuronium. A right endobronchial double-lumen tube was placed without difficulty, and the patient was put in the right lateral decubitus position. Anesthesia was maintained with sevoflurane, fentanyl, and rocuronium.

VATS instruments entered the left pleural cavity through the left 3rd, 6th, and 7th intercostal spaces. Upon entering the thoracic cavity, a large quantity of yellowish opaque fluid and multiple pleural dense adhesions were found, especially over the anterior aspect of the heart. Debridement of dense adhesions with sharp dissection was performed and an unintentional laceration was made into the anterior aspect of the RV, which resulted in severe bleeding into the thoracic cavity. The patient quickly lost an estimated 5 L of blood in several minutes, and heart rhythm showed pulseless electrical activity. The arterial pressure was fluctuating between 0 to 25 mmHg from hypovolemia. End-tidal carbon dioxide dropped from 30 to 6 mmHg (Table 1). Anesthesia personnel were assembled expeditiously for resuscitation. A fluid warming pressure infusion device (Level 1 Technologies, Inc, Rockland, MA) was used through the newly inserted left internal jugular vein Cordis introducer catheter (Edwards Lifesciences, Irvine, CA) for quick pressure infusion. Epinephrine (1-mg intravenous boluses) and vasopressin (one 40-U intravenous bolus) were administered, followed by epinephrine (25 µg/kg/min) and dopamine (8 µg/kg/min) infusions.

Open thoracotomy along the 7th intercostal space was executed immediately to control hemorrhage, initiate internal cardiac massage, and repair the laceration. Foley catheter hemostasis was attempted but



**Fig 1.** Chest radiography before the video-assisted thoracic decortication depicts left pulmonary fibrosis and massive pleural effusion.

failed because of the difficult location. Because of inadequate exposure, hemodynamic instability, and concern over extending the laceration of the right ventricular free wall in the bloody field, manual hemostasis by the thoracic surgeon was performed. This maneuver, together with aggressive volume resuscitation by the anesthesia team using pressure fluid infusion via the Cordis catheter, enabled venous return and hemodynamic parameters to improve but restricted the thoracic surgeons with other procedures. This prompted the decision of ECMO insertion by a second cardiac team to facilitate maintaining blood pressure, oxygenation, and right ventricular decompression for repair.

During volume resuscitation, internal cardiac compressions, and manual hemostasis, ECMO circuitry was established quickly by cardiac surgeons through the left femoral vein and artery with a straightened left leg. Unfractionated heparin, 300 U/kg, was administered in the priming solution to maintain an activated coagulation time >400 seconds. Venoarterial ECMO (CB2505 Medtronic, Minneapolis, MN)

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**Table 1. Vital Signs and Hemodynamic Support Before and After ECMO**

Variable	Before ECMO	During ECMO						After ECMO 72 h
		10 min	20 min	60 min	120 min	24 h	48 h	
MAP mmHg	0-25	40	35	45	42	64	65	70
HR (beats/min)	PEA	150	135	142	136	104	126	96
CVP (cmH <sub>2</sub> O)	—	—	—	24	18	14	10	8
ETCO <sub>2</sub> (mmHg)	6	13	15	18	33	29	32	31
SpO <sub>2</sub> (%)	—	85	92	96	98	98	97	99
Hb (g/dL)	4.1	5.2	8.6	9.1	9.7	10	9.7	11.2
ECMO flow (L/m)	—	3	3	2.3	2.3	1.8	1	—
Epinephrine (μg/kg/min)	1-mg bolus	25	25	18	8	6	—	—
Dopamine (μg/kg/min)	8	8	8	6	6	3	—	—
Norepinephrine (μg/kg/min)	—	12	12	12	4	—	—	—
Vasopressin (U)	40 bolus	—	—	—	—	—	—	—

Abbreviations: MAP, mean arterial pressure; HR, heart rate; PEA, pulseless electrical activity; CVP, central venous pressure; ETCO<sub>2</sub>, end-tidal carbon dioxide; SO<sub>2</sub>, pulse oximeter saturation; Hb, hemoglobin.

was initiated at 3 L/min in about 10 minutes. The ECMO also provided for aggressive volume expansion of priming crystalloid solution and packed red cells that raised the mean arterial pressures from 0 to 20 mmHg to 60 to 70 mmHg. Blood collected from the left chest cavity via the ECMO cell saver was reused.

When the condition was stabilized with ECMO, the heart could be inspected thoroughly. The laceration was irregular and 4 cm in length, and the coronary arteries were intact. During a period of diminished venous return to the RV from ECMO, the repair was performed with a 3-cm Teflon pledget and a 4-0 polypropylene suture. The reduced bleeding also lessened the possibility of air embolism.

After pulmonary decortication, two 32F chest drainage tubes were placed in the pleural cavity. Twenty minutes after ECMO started, the patient's hemoglobin was brought up from 4.1 g/dL to 8.6 g/dL. As such, supine sternotomy cardiopulmonary bypass conversion was unnecessary. The patient was transferred to the surgical intensive care unit with ECMO and inotrope support in relatively stable condition. Central venous pressure was monitored from the Cordis catheter. The total blood loss was about 6,000 mL. A total of 800 mL of autologous salvaged blood, 3,000 mL of homologous packed red cells, and 2,000 mL of crystalloid fluid were administered. Table 1 summarizes relevant hemodynamic values, inotropes, and vasopressor use before, during, and after ECMO. It reflected that the vital signs were enhanced by ECMO.

The patient regained consciousness in 48 hours. Decannulation required only minor vessel repair without inotropic support. The intensive care unit and hospital course were uneventful; the patient was discharged on the 6th postoperative day (Fig 2). She had no neurologic sequelae when followed up on the 25th day (Fig 3).

## DISCUSSION

Rapid deployment of ECMO allowed timely stabilization of hemodynamics and oxygenation that established conditions for right ventricular repair. It also facilitated the surgical repair by diminishing right ventricular venous return. ECMO extended beyond the operative period and allowed for myocardial tissue recovery after operation.

Refinements in VATS technique and technology have expanded its applications. Complications associated with VATS are infrequent, but fatal complications may develop that require open thoracotomy.<sup>1</sup> In this case, cardiovascular collapse after a large iatrogenic right ventricular laceration resulted from VATS.

When used for cardiac support, ECMO circuitry can be coated completely with heparin bonding and used without systemic heparin for up to 48 hours. Thus, it is useful in the setting of coagulopathy and in trauma patients in whom instant setup is an important issue.<sup>2-4</sup> Because of experience and technology, the duration of ECMO therapy also has extended from days to weeks.<sup>5</sup>

Anastasiadis et al<sup>6</sup> reported a case of left ventricular rupture after myocardial infarction that was managed successfully with the use of ECMO for resuscitation, surgical repair, and postoperative hemodynamic support. Conversion to conventional cardiopulmonary bypass for surgical repair was avoided. In the present case, ECMO was used to repair iatrogenic right ventricular rupture and promoted postoperative myocardial recovery. A literature search did not find a similar report of ECMO for this indication.



**Fig 2. Three days after video-assisted thoracic decortication, chest radiography showed improvement in the expansion of the left lung, with 2 chest tubes in place.**



**Fig 3.** Twenty-five days after video-assisted thoracic decortication, chest radiography showed that the condition improved in comparison with previous films.

A number of factors, such as myocardial infarction,<sup>7</sup> infection,<sup>8,9</sup> and steroid use,<sup>10</sup> may make myocardial tissue susceptible

to rupture under mechanical stress. If unrecognized, hemorrhage from a laceration of the inflamed thin right ventricular wall can be massive. The authors speculated, however, that the present patient, who developed pleural fibrosis for only one month, did not have enough time for the development of afore-mentioned risks that weakened or thinned the RV wall. Therefore, the authors believe it was iatrogenic in origin.

This case reflects the need for constant vigilant monitoring of hemodynamics, blood loss, and the surgical field for unforeseen complications that often result from rare combinations of various risk factors. Rapid availability of anesthesia personnel and surgical teams and readiness for unexpected life-threatening complications should be included in routine daily anesthesia practice to decrease critical time and hasten recovery.

In summary, anesthesia and cardiothoracic surgical teams must keep in mind that prompt ECMO therapy can be useful in the event of a difficult right ventricular trauma associated with intractable cardiovascular collapse. It can be particularly useful in terms of facilitating right ventricular repair, quick volume expansion, stabilizing vital signs, and promoting postoperative myocardial recovery.

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