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# Emergent Surgical Pulmonary Embolectomy in a Pregnant Woman:

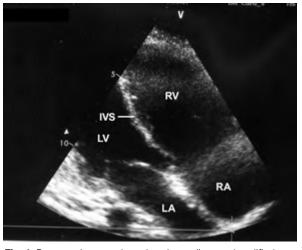
Case Report and Literature Review

Acute pulmonary embolism is a leading cause of death during pregnancy and delivery in the United States. We describe the case of a 25-year-old woman who presented in cardiogenic shock in week 38 of her first pregnancy. After the emergent cesarean delivery of a healthy male neonate, the mother underwent immediate surgical pulmonary embolectomy. We confirmed the diagnosis of pulmonary embolism intraoperatively by means of transesophageal echocardiography and removed large clots from the patient's pulmonary arteries. Mother and child were doing well, 27 months later. In addition to presenting our patient's case, we discuss the other relevant reports and the options for treating massive pulmonary embolism during pregnancy. **(Tex Heart Inst J 2014;41(2):188-94)** 

he prevalence of pulmonary embolism (PE) in pregnant women is 5 times greater than that in nonpregnant women.<sup>1,2</sup> Pooled data from 1991 through 1999 reveal that PE accounted for 20% of maternal deaths in the United States<sup>3,4</sup>—more than for hemorrhage (17%) or pregnancy-induced hypertension (16%).<sup>4</sup> The optimal management of acute PE during pregnancy, especially in the presence of unstable cardiopulmonary status, is challenging and poses severe risks for the mother and fetus.<sup>1</sup> Options include anticoagulation with heparin, thrombolytic therapy, transcatheter embolectomy, and surgical pulmonary embolectomy (SPE).<sup>5,6</sup> Earlier, SPE was considered to be the "last resort" because of the accompanying high mortality rates.<sup>7</sup> However, reports since 2005 suggest that a more aggressive strategy, involving a multidisciplinary approach, prompt diagnosis, and operative intervention, leads to better outcomes in patients with acute PE.<sup>8-15</sup> Surgical pulmonary embolectomy has undergone a resurgence after having been largely discarded except in cases of cardiogenic shock or cardiopulmonary resuscitation.<sup>16</sup> We report what to our knowledge is the 13th case of SPE in a pregnant woman.

# **Case Report**

In December 2011, a 25-year-old woman at week 38 in her first pregnancy was hospitalized because of knee joint luxation after a fall at home. She had undergone open knee-joint repositioning and stabilization with a splint. Postoperatively, she developed sudden dyspnea, tachycardia, intermittent hypotensive episodes, nausea, and vomiting. This raised a suspicion of PE and inferior vena cava compression syndrome, and she was referred to our hospital. Upon admission, her clinical cardiopulmonary status was stable. Her personal and family medical histories were not contributory. Abnormal laboratory findings were high levels of D-dimer, creatine kinase and its MB isoenzyme, cardiac troponin I, and B-type natriuretic peptide (BNP), which suggested myocardial injury and dysfunction. A transthoracic echocardiogram (TTE) showed a markedly dilated, hypocontractile right ventricle (RV) (Fig. 1), pulmonary hypertension, mild-to-moderate tricuspid valve insufficiency, a small left ventricular cavity, and paradoxical interventricular septal movement (Fig. 2). The TTE also showed distinct hypokinesia of the mid-free wall of the RV but normal motion of the apex. Thromboembolic clots were not directly observed. An electrocardiogram revealed evident signs of RV strain. Doppler ultrasonographic images of the lower extremities neither confirmed nor excluded deep vein thrombosis. Cardiac tocography yielded normal results, and the fetus was vital. Chest computed tomographic (CT)



**Fig. 1** Preoperative transthoracic echocardiogram (modified 3-chamber view) shows a markedly dilated, hypocontractile right ventricle and billowing of the interventricular septum into the left ventricle.

IVS = interventricular septum; LA = left atrium, LV = left ventricle; RA = right atrium; RV = right ventricle



**Fig. 2** Preoperative transthoracic echocardiogram (left parasternal long-axis view) shows deviation of the interventricular septum toward the left ventricle and its small cavity.

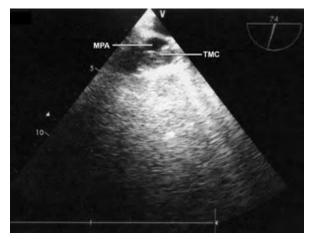
AML = anterior mitral leaflet; Ao = aorta; IVS = interventricular septum; LV = left ventricle; PML = posterior mitral leaflet; RV = right ventricle

and ventilation/perfusion (V/Q) scans of the lungs were intentionally not performed, because of the hazards of ionizing radiation.

The diagnosis of submassive PE was presumed. Heparin was administered as a 5,000-U bolus and then by continuous intravenous infusion. The patient was transferred to the intensive care unit for monitoring. Ten hours after admission, her clinical condition worsened to circulatory collapse with severe respiratory insufficiency, hypoxemia, and cyanosis. Tracheal intubation and mechanical ventilation were promptly initiated. Further circulatory support with catecholamines was necessary. We decided to deliver the infant emergently through a cesarean section and to subsequently perform SPE with the mother under cardiopulmonary bypass (CPB). An intraoperative transesophageal echocardiogram (TEE) confirmed the paradoxical interventricular septal movement, the severely dilated and poorly contractile RV, and tricuspid annular dilation with mild-to-moderate valvular insufficiency. The obstetrics team performed the cesarean delivery of a healthy male neonate. Immediately thereafter, TEE showed a sudden and subtotal occlusion of the mother's main pulmonary artery (PA) by thromboembolic material (Fig. 3). Median sternotomy with aortic and bicaval cannulation was performed, and CPB with mild hypothermia and crystalloid cardioplegic arrest was started. We made bilateral horizontal incisions in the branch PAs. The left PA was occluded with thrombus, and approximately three quarters of the lumen of the right PA was occluded, up to the segmental arteries. We used forceps and suction to remove all the clots (Fig. 4). We removed the clots in the main PA (which we did not open) by means of suction and with use of angulated forceps through the incisions in the branch PAs. The patient was uneventfully weaned from CPB, and her postoperative course was uncomplicated. As of March 2014, she and her child were doing well, and no recurrent PE was detected.

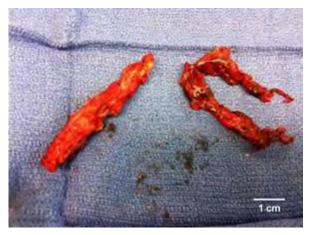
# Discussion

The prevalence of PE during pregnancy—0.3 to 3 per 1,000 pregnant women—is 5 times greater than that in nonpregnant women.<sup>2,6,17</sup> During pregnancy, the 3 elements of Virchow for deep vein thrombosis and



**Fig. 3** Intraoperative transesophageal echocardiogram immediately after cesarean delivery shows subtotal thrombotic occlusion of the mother's main pulmonary artery.

MPA = main pulmonary artery; TMC = thrombotic clots



*Fig. 4 Photograph shows thrombotic clots removed from the main and branch pulmonary arteries.* 

thromboembolic sequelae can occur: hypercoagulability, venous stasis, and vascular damage.<sup>5,6,18,19</sup>

To avoid negative consequences for the pregnant woman and fetus, an early and correct diagnosis of PE is crucial.<sup>1,19</sup> Diagnostic tests include chest radiography, V/Q imaging, spiral computed tomography, and pulmonary angiography.<sup>1,17</sup> The guidelines of the European Society of Cardiology for the diagnosis and management of acute PE state that these tests can be completed safely, because they fall below the minimum radiation dose of 50 mSv (50,000  $\mu$ Gy) that could harm the fetus.<sup>17</sup>

Both TTE and TEE are valuable in the diagnosis of PE.<sup>5,8-10,12,13,20,21</sup> Echocardiograms can show direct views of central thromboemboli and provide indirect evidence of PA obstruction and RV pressure overload, such as RV dysfunction, tricuspid regurgitation, leftward bowing of the interatrial septum, and systolic flattening of the interventricular septum.<sup>17,20,21</sup> Echocardiograms can reveal moderate or severe RV free-wall hypokinesia with preserved apical contractility (the McConnell sign) as indirect evidence of acute PE.17,20,21 Although D-dimer levels naturally rise during pregnancy, substantial increases have been observed in pregnant women with ongoing thrombosis.1,17 High levels of BNP and N-terminal pro-BNP biomarkers have been associated with RV dysfunction in patients with acute PE and are significant predictors of all-cause in-hospital or short-term death.22

*Treating Acute Massive Pulmonary Embolism.* Heparin anticoagulation, thrombolytic therapy, transcatheter embolectomy, catheter-directed thrombolytic therapy, and SPE are possible treatments for acute massive PE during pregnancy.<sup>5,6,9,10,18,19,23</sup> Heparin anticoagulation is the chief therapy.<sup>17,23</sup> Either unfractionated or low-molecular-weight heparin is safe in pregnant women, because neither substance crosses the placenta or is found in breast milk in significant amounts.<sup>17,23</sup> *Literature Review.* Our review of the English-language medical literature from 1970 through 2012 yielded 12 other case reports that described SPE for massive PE during pregnancy.<sup>6,18,24-33</sup> Table I summarizes the clinical characteristics, presenting symptoms and signs, diagnostic methods, clinical courses, indications for SPE, and fetal and maternal outcomes.

In all 13 patients, (age range, 21–39 yr), the clinical manifestation of PE was not dominated by one symptom or sign, but rather by combinations. The most frequent presentations were respiratory and cardiac: dyspnea in 9 patients, tachycardia in 5, cyanosis in 4, tachypnea in 4, hypoxemia in 2, acute respiratory distress in 1, and palpitations in 1. Heparin at therapeutic doses in 9 patients was insufficient to resolve their unstable hemodynamic conditions. In all 13 patients, SPE was indicated because of rapidly worsening hemodynamic status and the onset of cardiogenic shock. Cardiopulmonary resuscitation was necessary in 2 patients.<sup>30,31</sup> All patients underwent CPB: ours and one other<sup>6</sup> with cardioplegic arrest, and 2 others without it.<sup>32,33</sup> No data were available about cardioplegic arrest in the other 9 cases. The thrombi were removed through an opening in the main PA in all patients but ours. Two maternal deaths<sup>28,31</sup> and 3 fetal deaths<sup>24,26,31</sup> occurred, constituting a 15.4% maternal mortality rate and a 23% fetal mortality rate.

*Thrombolytic Therapy.* According to data from a metaanalysis of 11 randomized controlled trials,<sup>34</sup> thrombolytic therapy was no more beneficial than heparin in the initial treatment of unselected patients with acute PE. However, thrombolysis was advised in patients who were at highest risk of recurrent PE or death. Although single case reports have documented successful thrombolysis for massive PE in pregnant women,<sup>5,17,19,23</sup> no prospective randomized studies have been conducted to evaluate the efficacy and safety of thrombolytic agents during pregnancy.

Because of the risk of bleeding, thrombolytic agents are not for routine use and should be reserved for pregnant women who are hemodynamically unstable, particularly those with systemic hypotension.<sup>5,17,19,23</sup> With urokinase therapy, there is a risk of bleeding, teratogenicity, and premature abruption of the placenta.<sup>5</sup> In 2009, te Raa and colleagues<sup>35</sup> successfully treated massive PE with streptokinase after intravenous heparin therapy failed, in a 34-year-old woman who had a compromised hemodynamic status, hypoxemia, tachypnea, and tachycardia at 25 weeks of pregnancy. The streptokinase therapy enabled rapid improvement, and she delivered a healthy child at term. In their literature review, those authors found 13 patients who had undergone thrombolysis for PE during pregnancy. The outcomes included no maternal deaths, 4 nonfatal maternal major bleeding sequelae, 2 fetal deaths, and 5 preterm deliveries.35 Another success was achieved with recombinant

TABLE I. Cases of Massive Pulmonary Embolism in Pregnancy with Treatment by Surgical Pulmonary Embolectomy

Reference	Age of Mother (yr)	Weeks of Gestation	Clinical Presentation	Associated Morbidity	Diagnostic Method ECG, chest radiography, and clinical suspicion	
Marcinkevicius A, et al. <sup>24</sup> (1970)	30	24	Unconsciousness, deep cyanosis, tachycardia, tachypnea, and left-leg swelling	No		
Cohn LH and Shumway NE <sup>25</sup> 1973)	21	First trimester	Dyspnea, pleuritic chest pain, and No weakness		ECG, PA, right-sided heart catheterization, and chest radiography	
Duff P and Greene /P <sup>26</sup> (1985)	35	13	Diaphoresis, disorientation, tachypnea, Pancreatic and hypotension neoplasm		PA	
Richards SR, et al. <sup>27</sup> 1985)	25	35	Acute respiratory distress, substernal chest pain, and active labor hypoxemia	No	Clinical suspicion, ECG, BGA, and V/Q scan	
Girz BA and Heiselman DE² <sup>8</sup> 1988)	31	29	Dyspnea, hypoxia, hypotension, and fetal bradycardia	No	РА	
Blegvad S, et al.² <sup>9</sup> 1989)	26	28	Dyspnea, palpitations, syncope, chest pain, and cyanosis	No	Right-sided heart catheterization, PA, ECG, and clinical suspicion	
Splinter WM, et al. <sup>30</sup> (1989)	27	32	Syncope, tachypnea, cyanosis, tachycardia, and fetal bradycardia	No	V/Q scan	
Lau G³¹ (1994)	33	8	Left-calf pain and swelling, sudden collapse, and cardiac arrest	DIC	Clinical suspicion and TTE	
Woodward DK, et al. <sup>32</sup> (1998)	25	38	Sudden dyspnea, chest tightness, dizziness, cyanosis, tachycardia, and tachypnea	Thrombocyto- penia	Clinical suspicion, ECG, and PA	
<sup>E</sup> unakoshi Y, et al. <sup>18</sup> 2004)	32	38	Dyspnea and swelling of legs	Protein S deficiency and PFO	Chest CT and TEE	
āniguchi S, et al. <sup>33</sup> 2008)	35	18	Dyspnea, tachypnea, tachycardia, and hypoxemia	HIT, anemia, and myelodysplastic syndrome	Chest CT	
lajj-Chahine J, et I. <sup>6</sup> (2010)	39	25	Threatened premature labor, severe dyspnea, and ARDS	HIT and previous history of PE	TEE	
Current case	25	38	Dyspnea, intermittent hypotensive episodes, vomiting, nausea, and tachycardia	No	Clinical suspicion, laboratory findings, TTE, and TEE	

ARDS = acute respiratory distress syndrome; BGA = blood-gas analysis; CPR = cardiopulmonary resuscitation; CT = computed tomography; DIC = disseminated intravascular coagulopathy; ECG = electrocardiography; HIT = heparin-induced thrombocytopenia; IVC = inferior vena cava; NA = not applicable; NS = not specified; PA = pulmonary angiography; PE = pulmonary embolism; PFO = patent foramen ovale; Postop = postoperative; preop = preoperative; SPE = surgical pulmonary embolectomy; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography; V/Q = ventilation/perfusion **TABLE I** (continued). Cases of Massive Pulmonary Embolism in Pregnancy with Treatment by Surgical Pulmonary Embolectomy

Reference	Survival of Mother	Survival of Fetus	Type of Delivery	Initial Heparin Therapy	Indication for SPE	Cardioplegic Arrest	: IVC Filter	Postop Follow-Up
Marcinkevicius A, et al. <sup>24</sup> (1970)	Yes	No	Extraction of fetus	Yes	Shock	NS	No; caval ligation	6 mo
Cohn LH and Shumway NE <sup>25</sup> (1973)	Yes	Yes	Abdominal, at term	Yes	Shock	NS	No; partial caval interruption	12 mo
Duff P and Greene VP <sup>26</sup> (1985)	Yes	No	Spontaneous abortion	Yes	Shock	NS	No	NS
Richards SR, et al. <sup>27</sup> (1985)	Yes	Yes	Abdominal, at term	Yes	Shock	NS	No	NS
Girz BA and Heiselman DE <sup>28</sup> (1988)	No	Yes	Cesarean	No	Shock	NS	No	NA
Blegvad S, et al. <sup>29</sup> (1989)	Yes	Yes	Abdominal, at term	No	Shock	NS	No	4 mo
Splinter WM, et al. <sup>30</sup> (1989)	Yes	Yes	Cesarean	Yes	Shock and CPR	NS	No	NS
Lau G³ (1994)	No	No	Extraction of fetus	NS	CPR and shock	NS	No	NA
Woodward DK, et al. <sup>32</sup> (1998)	Yes	Yes	Cesarean	Yes	Shock	No	Yes (temporary); was removed on postop day 10	10 d
Funakoshi Y, et al. <sup>18</sup> (2004)	Yes	Yes	Cesarean	No	Shock	NS	Temporary; caval ligation	54 mo
Taniguchi S, et al. <sup>33</sup> (2008)	Yes	Yes	Cesarean	Yes	Shock	No	Yes (preop)	NS
Hajj-Chahine J, et al.º (2010)	Yes	Yes	Abdominal, at term	Yes	Shock	Yes	No	NS
Current case	Yes	Yes	Cesarean	Yes	Shock	Yes	No	27 mo

ARDS = acute respiratory distress syndrome; BGA = blood-gas analysis; CPR = cardiopulmonary resuscitation; CT = computed tomography; DIC = disseminated intravascular coagulopathy; ECG = electrocardiography; HIT = heparin-induced thrombocytopenia; IVC = inferior vena cava; NA = not applicable; NS = not specified; PA = pulmonary angiography; PE = pulmonary embolism; PFO = patent foramen ovale; Postop = postoperative; preop = preoperative; SPE = surgical pulmonary embolectomy; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography; V/Q = ventilation/perfusion tissue plasminogen activator (rtPA) in a 26-year-old woman at 24 weeks.<sup>36</sup> The authors found 18 reports of thrombolysis in pregnant women with PE: 10 patients were given rtPA, 6 streptokinase, and 2 urokinase, with beneficial maternal and fetal effects in terms of mortality and complication rates and with acceptable bleeding risks. Major nonfatal bleeding was observed in 4 streptokinase patients. Two patients on rtPA therapy and 3 on streptokinase delivered preterm. The 2 fetal deaths, 1 each in the streptokinase and rtPA groups, were not attributed to fetal hemorrhage.<sup>36</sup>

*Catheter-Directed Thrombolytic Therapy.* Few reports are available about the successful use of catheter-directed thrombolysis, either alone or with thromboembolic mechanical fragmentation, in treating massive PE peripartum or postpartum.<sup>37-40</sup> Catheter-directed thrombolytic therapy affords potential advantages, such as more rapid clot lysis with consequent improvement in pulmonary blood flow, normalization of hemodynamic status, uterine perfusion, and lower risk of bleeding; however, no clear or strong evidence supports the superiority of local catheter-directed thrombolysis over systemic thrombolysis or heparin anticoagulation.<sup>40</sup>

*Transcatheter Thrombectomy.* Sato and colleagues<sup>5</sup> reported successful emergent transcatheter thrombectomy for massive PE in a hemodynamically unstable patient after emergent cesarean delivery at 30 weeks, followed by the intravenous administration of heparin and urokinase therapy for 2 weeks.<sup>5</sup> However, transcatheter thrombectomy can fragment the embolic material, which can then propagate into the peripheral pulmonary vasculature and increase the risk of pulmonary hypertension.<sup>13</sup>

*Insertion of an Inferior Vena Cava Filter.* The indication for inferior vena cava filter placement during pregnancy is the same as in nonpregnant patients.<sup>23</sup> In 1981, Scurr and colleagues<sup>41</sup> reported the first successful use of a filter in a pregnant woman. In 1986, Hux and associates<sup>42</sup> reported good maternal and fetal outcomes after Greenfield filter placement in 6 pregnant patients with thromboembolic disease. In 1997, Thomas and co-authors<sup>43</sup> described good outcomes in 8 pregnant patients after Greenfield filters were prophylactically inserted because of a high risk of PE. Instead of a permanent filter, a retrievable filter—removable within 10 days—might protect the patient in the high-risk period during and after the acute event.<sup>22</sup>

## Conclusion

Cooley and colleagues, who in 1961 first used temporary CPB when surgically treating massive PE in a 37-year-old woman after her pelvic surgery, said, "an aggressive attitude toward treatment of these cases should permit the saving of many patients who otherwise are doomed."<sup>44</sup> This aggressive attitude, in diagnosis and treatment, should be applied to every patient who pre-

sents with signs and symptoms of life-threatening massive PE.

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