Deep Vein Thrombosis and Pulmonary Embolism in the 8th Week of Pregnancy

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ABSTRACT

A 29-year-old woman in her 8th week of pregnancy was referred to our hospital for swelling in the lower extremities, rapid onset of dyspnea (1 hr) and pre-syncpe. Severe right ventricular dysfunction and moderate pulmonary hypertension were detected using 2-dimensional Doppler echocardiography. In addition, left calf vein and proximal thromboses were detected by venous compression ultrasound imaging. After successful thrombolytic treatment, the patient quickly recovered and was discharged from hospital on subcutaneous low-molecular-weight heparin. She delivered a normal, healthy infant at full-term (40 weeks). (Korean Circulation J 2007;37:130-133)

KEY WORDS : Pulmonary embolism ; Thrombolytic therapy ; Pregnancy.

Introduction

Pulmonary embolism(PE) among pregnant and postpartum women remains a major cause of maternal morbidity and mortality. Deep vein thrombosis(DVT) usually starts in the calf veins, and may extend to the proximal veins and cause a pulmonary embolism.1)2) Available data show that the risk period for deep vein thrombosis and pulmonary embolism is greatest during the third trimester and postpartum.3)4) However, herein is report a rare case of a patient who suffered from deep vein thrombosis and pulmonary embolism during the first trimester.

Case

A 29-year-old woman in her 8th week of pregnancy was referred to our hospital due to progressive swelling in the lower extremities over a 2-week period, acute onset of dyspnea(1 hr) and pre-syncpe. She had a 5-year history of hypothyroidism and daily Synthyroid (0.1 mg) administration. She had experienced a spontaneous abortion in the 12th week of pregnancy 1 year earlier. Otherwise, she had no significant past medical history and was taking no medication upon admission to the hospital. On examination, both her lower legs exhibited swelling and stasis, particularly the left lower leg. She weighed 87.6 kg and was 167 cm tall. Her blood pressure, heart rate and respiratory rate were 110/80 mmHg, 130 per minute and regular and 24 per minute, respectively. Arterial blood gas analysis on room air gave a pH of 7.43, PaCO2 30.9 mmHg, PaO2 56.5 mmHg, bicarbonate 20.0 mmol/L, oxygen saturation 90.6% and P(A-a)O2 30.5 mmHg. The prothrombin time, partial thromboplastin time and protein C were all within normal levels. Fibrin/fibrinogen degradation product and D-dimer values were 54.6 μg/mL and 6.7 mg/L, respectively, and the B-type natriuretic peptide(BNP) was 354.5 pg/mL. Serum titers of the anticardiolipin antibody(IgG, IgM) were within normal levels. Antinuclear and antineutrophil cytoplasmic autoantibodies(ANCA) were both negative. No factor V Leiden or prothrombin G20210A mutations were found. Deficiencies were observed in both the antithrombin III(18.0 mg/dl) and protein S activity(33%). ECG demonstrated sinus tachycardia and right axis deviation. A chest roentgenogram revealed mild hilar enlargement. A 2D echocardiogram revealed a normal left ventricular systolic function, dimension with a dilated right heart, an impaired right ventricular systolic function and an estimated pulmonary artery pressure of 55 mmHg(Fig. 1A). Venous ultrasonography demonstrated a left calf vein thrombosis, proximal vein thrombosis and incompressible posterior tibial veins(Fig. 2A-C). However, a ventilation perfusion...
(V/Q) lung scan and CT pulmonary angiography were not performed due to a labor-management dispute at our hospital, and because of concerns about the effects of radiation on the fetal development and the risk of developing breast cancer. The patient received thrombolytic therapy (tissue-type plasminogen activator: 50 mg for 1 hr, 50 mg for 2 hrs) followed by intravenous heparinization, and was closely monitored in the intensive care unit. She improved dramatically following the thrombolytic therapy, with a marked reduction in the dyspnea and significant improvement in her exercise capacity. Two weeks following the thrombolytic therapy, a 2D echocardiographic assessment revealed significant improvement in the right ventricular function, with reduction in the right ventricular dimension and an estimated pulmonary artery pressure of 37 mmHg (Fig. 1B). Her D-dimer level decreased to 0.4 mg/L. An obstetric assessment confirmed no evidence of fetal distress, with the baby continuing to progress. The patient responded very well over time and was continued on low molecular weight heparin (Fraxiparine®: nadroparin, 86 U/kg, SC, 2 times/day). Three weeks later, she made an uneventful recovery and was discharged with antepartum prophylaxis treatment (Fraxiparine® 3800 U, SC, 2 times/day). Finally, she delivered a normal, healthy infant at full-term (40 weeks).

**Discussion**

The overall incidence of deep vein thrombosis or pulmonary embolism during pregnancy has been reported as in 1 in 1000 to 1 in 2000 pregnancies. However, the relatively low incidence of venous thromboembolism (VTE) in Asians has been estimated, and may be related to a lower prevalence of genetic factors predisposing to VTE, such as factor V Leiden in Asians (0.5%) compared to Caucasians (5%). Several factors may be important in the development of VTE during pregnancy, such as hypercoagulability, venous stasis and vascular damage. Hypercoagulability results from increased levels of coa-
Thrombophlebitis associated with pregnancy occurs in about 40% of pregnancies and is characterized by inflammation of the veins, often accompanied by pain and swelling. This condition is associated with a significant risk of fetal injury and may contribute to postpartum venous thrombosis. Venous stasis begins in the first trimester and reaches a nadir at 36 weeks of pregnancy, due to the gravid uterus and compression of pelvic vessels by the right iliac artery where they cross. The risk of recurrent VTE is the same for patients with proximal DVT or PE, but the risk of a fatal PE is 2- to 3-fold higher after an episode of PE than of DVT. Therefore, most experts recommend that pregnant women at risk of VTE receive more aggressive prophylaxis than that traditionally recommended. In conclusion, because of the gravity of maternal morbidity and mortality, the need for prolonged heparin therapy during pregnancy and prophylaxis during subsequent pregnancies, physicians should not be reluctant to perform definitive radiologic studies whenever VTE is suspected in a pregnant woman. Further studies will be required to make a confident recommendation in pregnancy associated VTE.

REFERENCES