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Case Report

Deep Vein Thrombosis

A Case Report on Challenging Deep Vein Thrombosis

Dr.Logeswari B.M[1] and Dr.Vatsala Shahi[2]

[1] *Assistant Professor, Department of Obstetrics and gynecology, Sree Balaji Medical College and Hospital, BIHER, Chromepet, Chennai-6000444*

[2] *Junior Resident, Department of OBG, Sree Balaji Medical College and Hospital, BIHER Chromepet, Chennai-600044*

Abstract: In pregnancy, venous thromboembolism (VTE) remains a leading cause of direct maternal mortality. It is approximately five times more common in pregnant women, with an incidence of 0.5-2 in 1000. The highest risk of deep venous thrombosis (DVT) leading to pulmonary embolism (PE) is during the postnatal period, especially after a cesarean section. A clinician who does not treat pregnant women regularly may not routinely identify pregnancy-specific risk factors for VTE. In patients with DVT, treatment with anticoagulation is associated with a high risk of bleeding during the immediate postpartum state, and patients do well with monitored anticoagulation and proper supportive treatment, as described in this case report. We are reporting a case of a 20-year-old primigravida who presented with labor pains at 39 (+6) weeks of gestation at Sree Balaji Medical College and Hospital, Chrompet, Chennai. The patient had no comorbidities and did not report any past or family history of VTE. An emergency cesarean section was performed due to fetal distress, and on the first postoperative day, she developed swelling pain and tenderness in the left lower limb. Color Doppler ultrasound showed left anterior tibial vein thrombosis, following which anticoagulant therapy was started with low molecular weight heparin. The patient was closely monitored for signs of bleeding or PE and was eventually discharged on oral anticoagulation. In conclusion, all women should be assessed for the risk factors of DVT during the antenatal period, and early ambulation in the postoperative period is crucial to prevent such deadly complications.

Keywords: Deep vein thrombosis, Pregnancy, Prothrombin time/INR, LMWH

***Corresponding Author**

Dr.Logeswari B.M , Assistant Professor, Department of Obstetrics and gynecology, Sree Balaji Medical College and Hospital, BIHER, Chromepet, Chennai-6000444

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1. INTRODUCTION

One of the leading global causes of morbidity and mortality that can be prevented is deep vein thrombosis (DVT). The annual death toll from venous thromboembolism, which includes DVT and pulmonary embolism, is estimated to be 1 per 1,000 persons.[1] A careful balance between components that promote and inhibit coagulation is essential for normal blood physiology. Virchow's triad summarizes the plethora of DVT risk variables into three fundamental components that encourage thrombus formation: venous stagnation, vascular damage, and clot formation. Diagnostic evaluation for DVT involves a detailed history, thorough clinical examination, laboratory analysis including D-dimer levels and markers for hypercoagulability, and ultrasound Doppler examination of lower limb veins.[2] The management of DVT requires anticoagulation treatment. DVT and its complications are the major causes of maternal mortality.[2] All three elements of Virchow's triad—venous stasis, endothelial injury, and hypercoagulability- are present in pregnancy, which is prothrombotic. Pregnancy and puerperium are well-established risk factors for venous thromboembolism, as women are up to five times more likely to develop DVT during pregnancy than when not pregnant.[3] Pulmonary embolism (PE) is the most dreaded complication of DVT and the leading cause of maternal death. The prevalence of Venous thromboembolism in pregnancy varies between 0.5 - 2 per 1000 pregnancies. Synthesis and circulating levels of clotting factors are physiologically increased during pregnancy to minimize bleeding after placental separation. Venous stasis during pregnancy is caused by enhanced lower limb venous system expansibility. Compression of the inferior vena cava by the gravid uterus also contributes to venous stasis. Injury to pelvic blood vessels may also occur during delivery, which can accelerate thrombus formation. Pulmonary embolism caused by migration of lower limb venous thrombus into the pulmonary circulation is a consequence of DVT associated with high fatality. [2,4] The most common symptoms of DVT are pain and swelling of the lower limbs. These are confounding features for an obstetrician, as pain and swelling over lower limbs are common during the third trimester due to obstruction of lymphatic flow caused by a gravid uterus and also in pre-eclampsia.[4] Risk factors for DVT include obesity, advanced age, smoking, blood type other than O, inherited and acquired thrombophilia, prolonged bed rest or immobility, malignancy, congestive cardiac failure, and high blood pressure. Pre-eclampsia, eclampsia, and delivery by LSCS are all pregnancy-related potential causes and risk factors.[5] Factor V Leiden and factor II mutations are the most frequent inherited causes of DVT. DVT at a young age, family history of thrombosis, arterial thrombosis, history of estrogen medication, and thrombosis during pregnancy entails investigations for genetically inherited coagulation disorders. Among such patients, factor V Leiden and prothrombin G20210A mutations, protein C-S, antithrombin 3, and homocysteine levels have often been studied; however, one should be cautious about the timing of the tests and the interfering factors as ongoing anticoagulation for treatment may give false results.[2] Anticoagulation therapy is the first line of treatment for patients with lower limb DVT. In contrast, those with life-threatening PE may need thrombolytics, too.[2] Evaluating and preventing risk factors, early diagnosis, and management is vital for preventing maternal fatalities among pregnant women. The present study describes a case of a young primigravida diagnosed with DVT in the left leg after an emergency LSCS.

2. CASE PRESENTATION

We report a 20-year-old primiparous woman admitted to Sree Balaji Medical College and Hospital, Chrompet, Chennai, complaining of a 39 (+6) weeks pregnancy with labor pains. She had no comorbidities and no past or family history of thromboembolism. The patient did not have any swelling in the lower limbs before delivery. The patient's physical examination findings and laboratory values were within acceptable limits on evaluation. However, there was cephalic disproportion with 'non-reassuring' fetal status on the non-stress test (NST), due to which an emergency cesarean section was performed. The cesarean was performed without any complications. On the first day of surgery, the patient developed swelling, redness, and pain in the left lower leg, as shown in Figure 1. Color Doppler ultrasound of the left leg showed a long segment thrombus that involved the left anterior tibial vein causing near-total occlusion, as shown in Figure 2. Table 1 summarizes the findings of the lower limb Color Doppler. Ascending venogram of the left leg was also obtained to confirm the diagnosis. Figure 3 shows a venogram with a filling defect in the anterior tibial vein. Her laboratory investigations showed: hemoglobin-10.8 g%, platelet count-1,50,000, prothrombin time-international normalized ratio (PT-INR)- 15.2-1.37, partial thromboplastin time- 29 seconds, fibrinogen- 866 mg/dl (200-400), ATIII-81 (80-120) and D-dimer- 1300 mcg/L (0-550).The patient was negative for anticardiolipin antibodies (IgG and IgM) and anti-nuclear antibody assay. Her ECG, chest x-ray (post-cesarean), and echocardiogram were normal, and she gave no history of chest pain, breathing difficulty, or palpitations throughout her illness. Despite no symptoms or signs of pulmonary embolism patient was shifted to the intensive care unit and continuously monitored for worsening of vital parameters. Anticoagulation therapy was started with low molecular weight heparin-enoxaparin 1mg/Kg given subcutaneously twice a day, followed by oral vitamin K antagonist-warfarin. The dose of warfarin was adjusted to maintain PT-INR between 2.0 to 3.0. details of prothrombin time measured for monitoring anticoagulation therapy are detailed in Table 2. To ensure comprehensive evaluation and management, a multidisciplinary team was involved in patient management, including an obstetrician, physician, cardiologist, and rheumatologist. The patient responded well to treatment and was discharged on oral warfarin, 2mg, taken once a day, and was advised to monitor PT-INR levels every two weeks. The patient was doing well on follow-up after four weeks of surgery and was advised to continue anticoagulation therapy.

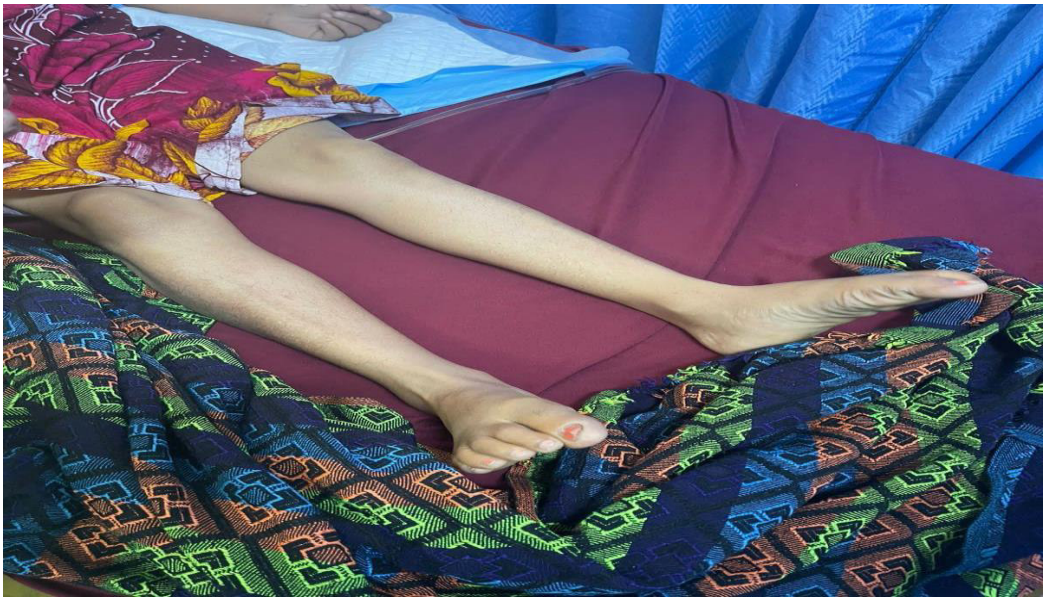

Fig 1: Picture showing swelling of the left lower limb.

On the first postoperative day, the patient complained of unilateral pain and swelling over the left lower limb, as seen in the above image.

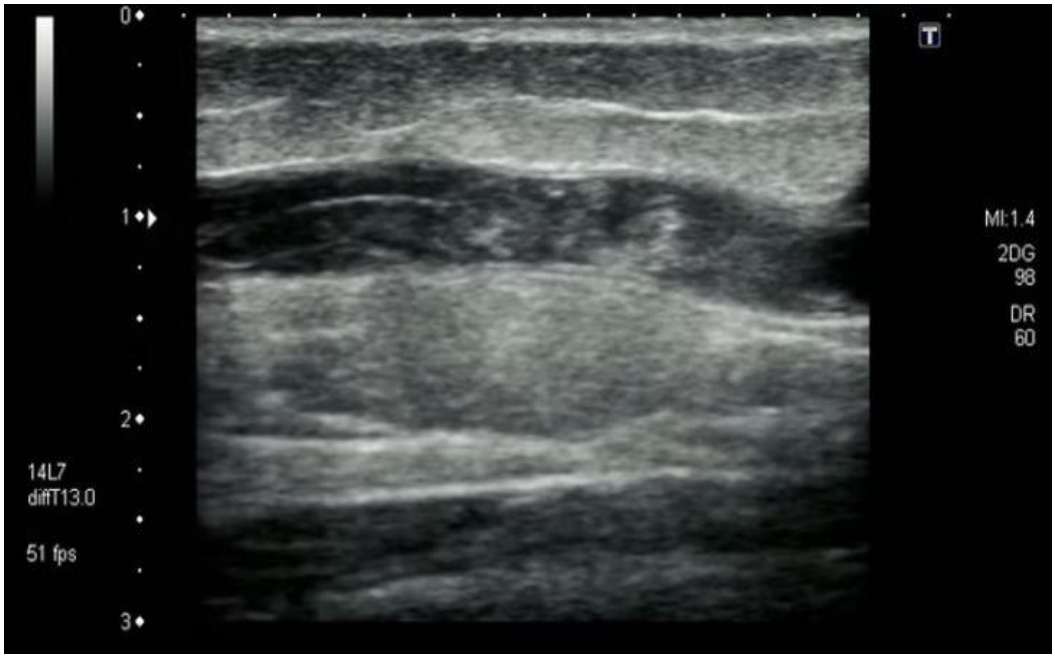

Fig 2: Ultrasound of left anterior tibial vein.

Longitudinal view of anterior tibial veins showing long segment thrombus causing near-total occlusion.

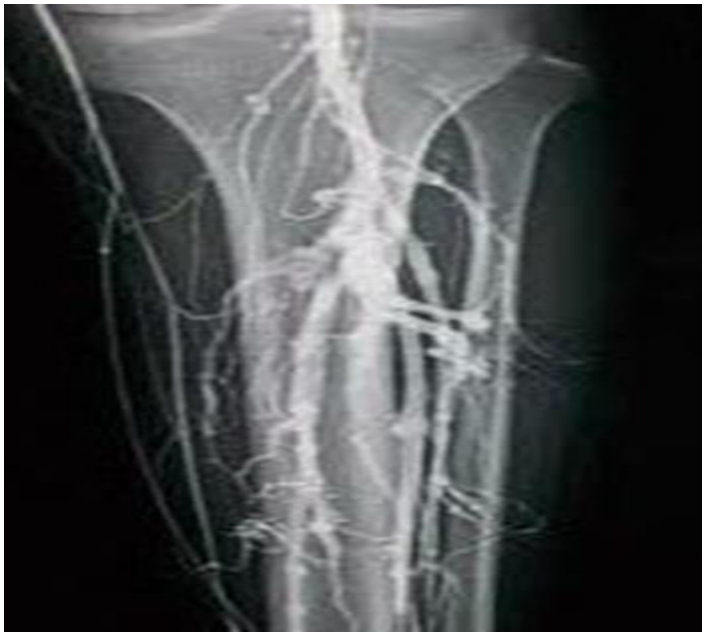

Fig 3: Venogram showing thrombus in anterior tibial vein.

Ascending venogram of the left leg with a filling defect in the anterior tibial vein.

Table 1: Findings on Color Doppler ultrasound of left lower limb.

S.no.	Vein	Thrombus
1	Inferior vena cava	None
2	Common Iliac	None
3	External Iliac	None
4	Femoral	None
5	Popliteal	None
6	Anterior Tibial	long segment thrombus causing near-total occlusion
7	Sural	None

The above table summarizes the distribution and extent of thrombus in the left lower limb venous system.

Table 2: Prothrombin Time values in the case discussed.

Postoperative day	Prothrombin Time	INR
1	15.2	1.37
2	14.8	1.33
5	106.5*	9.05*
5#	14.6	1.31
6	11.9	1.06
7	13	1.17
9	14.6	1.31
13	17	1.54
15	26.3	2.41

Prothrombin time was analyzed to monitor the anticoagulation therapy. *Unusually high prothrombin time and INR on day 5; however, upon #repeat testing after a few hours, the results were normal. INR: International normalized ratio

Table 3. Pathogenesis of the increased risk of VTE in pregnancy

Factors Predisposing To VTE In Pregnancy	Pathogenesis
Venous stasis	• The hormonally driven decline in venous tone and uterine enlargement blocks venous flow causing venous stasis. • By weeks 25 to 29, there is a 50% drop in the venous flow velocity in the legs. After giving birth, this lasts for roughly six weeks until returning to normal venous velocities. • The left lower extremity is the most typical location for DVT in pregnant and postpartum women (82%). A possible anatomical cause is the right common iliac artery compressing the left common iliac vein, amplified by the growing uterus.

Altered Blood Coagulability	• In a healthy pregnancy, a hypercoagulable condition starts to develop. The greatest significant risk factor for thrombosis during pregnancy is this. The levels of coagulation factors (II, VII, VIII, X) are all elevated, whereas fibrin production and fibrinolytic activity are lowered. • Protein S levels gradually decline, and activated protein C has developed resistance. All these alterations show how the body prepares physiologically for the coagulation challenge in delivery. • Increased levels of hemostatic activation indicators such as prothrombin fragment F1+2 and D-dimer show this hemostatic activation.
Vascular damage	• Venous hypertension or at the time of labor can both cause damage to the endothelium of the pelvic veins. However, DVT during pregnancy (6-11%) and the puerperium are caused by pelvic vein thrombosis, which is unusual other than the cause of pregnancy.

The above table elaborates on the pathogenesis of increased factors for developing deep venous thrombosis during pregnancy.

3. DISCUSSION

All women should undergo a documented assessment of risk factors for VTE before pregnancy, in early pregnancy, or immediate postpartum period. The pathogenesis of the increased risk of VTE in pregnancy is summarized in Table 3. Risk assessment should be repeated if the woman is admitted to the hospital for any reason or develops other intercurrent problems. The case presented in our study was closely monitored for vitals and specific complaints. The hypothesis was made after careful observation of symptoms and prognosis, which guided us to suspicion of DVT post-LSCS and management protocols through appropriate decision-making. The patient was intensively monitored, administered appropriate medication, and regularly followed up. Due to the risk for fetal and maternal problems and the absence of pertinent high-quality studies, the treatment and prevention of pregnancy-associated VTE are difficult.[3,5] Except for those with heparin-induced thrombocytopenia, or considerable renal impairment, low molecular weight heparin (LMWH) is the preferred medication for treating and preventing venous thromboembolism in pregnancy. In individuals with severe renal impairment, unfractionated heparin is preferable.[6,7] Most women do not need anticoagulation, although there is a higher risk of thrombosis during pregnancy and postpartum. Anticoagulation therapy is not devoid of adverse effects, and according to reports, heparin and low molecular weight heparin (LMWH) carry a 2% risk of maternal bleeding problems.[8] Thromboprophylaxis to prevent VTE during pregnancy is challenging ,because it requires long-term parenteral administration of LMWH or unfractionated heparin(UFH).[9] Both medications have risks of bleeding, osteoporosis, and heparin-induced thrombocytopenia (HIT); however, these side effects, particularly HIT, are extremely rare with LMWH. LMWH is the preferred agent for preventing and treating DVT during pregnancy due to its advantages over UFH; however, the extended half-life of LMWH compared to UFH is a drawback and can cause issues during birth.[10]. Choosing which patients need to take thromboprophylaxis has always been difficult. Finding the women with a higher risk of thrombosis and precisely estimating that risk is necessary for its sensible administration. Because postnatal DVT risk is higher than antenatal DVT risk and warfarin is appropriate during this period even if the mother is feeding the baby since warfarin is not excreted in breast milk, the threshold for suggesting postpartum prophylaxis is lower than for antepartum prophylaxis. Given that the risk of DVT is distributed rather evenly across all trimesters, it is recommended that antenatal prophylaxis be started as soon as possible in the first trimester in women with high risk.[8,11,12] According to the currently available information, recurrent DVT is reported in 2-10% of pregnant women with a history of VTE. However, the efficacy of thromboprophylaxis in expecting mothers with past DVT has not been studied in significant clinical trials.[13] Pregnant women at high risk of VTE should be counseled regarding anticoagulant thromboprophylaxis's benefits, risks, and complications. Detailed instructions for administering injectable LMWH or UFH at home and warning signs of abnormal bleeding and other possible adverse effects should be given. It is vital to stop anticoagulants at least 24 hours before an elective cesarian section or at the onset of natural labor.[14]

4. CONCLUSION

One of the high-risk populations for the development of VTE includes women during pregnancy and postpartum. Even though the measures needed to lower the risk and complications of this disease are extremely valuable, they are not without significant difficulties because the primary considerations in making treatment decisions are the potential risks to the fetus and maintaining the mother's general health during pregnancy.

5. AUTHORS CONTRIBUTION STATEMENT

Logeswari. B.M.: Concept, manuscript writing, and Vatsala Shahi: Data collection, manuscript draft.

6. CONFLICT OF INTEREST

Conflict of interest declared none.

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