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Case Report

Pregnancy with Post Inferior Vena Cava Balloon Angioplasty with Uterine Arterio-Venous Malformation

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A B S T R A C T

Venous thromboembolism (VTE) complicates one in 1000 pregnancies. The standard therapy in venous thromboembolism are anticoagulants, heparin, or warfarin. Uterine artery malformation is rare but can result in life-threatening bleeding which needs immediate intervention. A 33-year-old female, G2A1 at 36 weeks period of gestation, with h/o inferior vena cava (IVC) thrombus and uterine arterio-venous (AV) malformation, which was treated with uterine artery embolisation and Inferior vena cava (IVC) balloon angioplasty 3 years back, was successfully managed during her antenatal, intrapartum and postpartum period with anticoagulants at therapeutic doses. Both mother and baby were discharged in stable condition. The balance between prevention of thrombus formation and bleeding complication with the use of anticoagulants is critical in such patients. Successful management of both VTE and bleeding uterine artery malformation needs intensive monitoring and requires both anticoagulant and haemostatic therapy under close supervision of multidisciplinary team.

Keywords: Venous Thromboembolism (VTE), Arterio-venous Malformation, IVC Angioplasty, Pregnancy, Uterine Artery Embolisation

Introduction

Pregnancy is a hypercoagulable state because of physiological changes in coagulant and anticoagulant factors. Therefore, pregnant women are more predisposed to thromboembolic events that can sometimes lead to maternal mortality. The standard therapy in venous thromboembolism (VTE) consists of anticoagulants like heparin or warfarin, depending on the gestational age and foetal risks. Of late, invasive procedures like placement of Inferior Vena Cava (IVC) filters are being used in pregnancy with thrombus, whereas, IVC angioplasty is the procedure of choice in non-pregnant women. Uterine artery malformation is rare but can result in life-threatening bleeding which needs immediate intervention. The balance between prevention of thrombus formation and bleeding complication with the use of anticoagulants is critical in such patients. A multidisciplinary team involving an obstetrician, cardiologist, anaesthetist, intervention radiologist, and neonatologist is required for managing high-risk VTE in

pregnancy. Here we present a case of post-IVC angioplasty for spontaneous inferior vena cava thrombus with uterine artery embolisation for uterine arterio-venous (A-V) malformation with term pregnancy.

Case

A 33-year-old female, married for 14 years, G2A1 with 36 weeks period of gestation, had a h/o IVC thrombus and uterine arteriovenous malformation 3 years back in 2018, which was treated by uterine artery embolisation, admitted from antenatal OPD for anticoagulant switchover.

In 2018, she had a spontaneous second-trimester abortion followed by heavy bleeding per vaginum. Magnetic resonance imaging (MRI) pelvis revealed multiple tortuous serpiginous flow voids of size 3.1 x 2.3 cm in anterior myometrium in the region of fundus and body of uterus, which was suggestive of uterine AV malformation. She underwent uterine artery embolisation. The post-procedure uterine cavity was normal with no AV malformation. During the evaluation, she was found to have dilated abdominal veins. Ultrasonography (USG) whole abdomen showed hepatic vein outflow tract obstruction. Contrast-enhanced MRI with whole abdomen MRI venogram showed loss of signal void within infradiaphragmatic part of IVC measuring 20 mm in length with multiple collaterals, suggesting IVC thrombus. Her thrombophilia screening was normal. Upper gastrointestinal (GI) endoscopy was reported normal with no evidence of portal hypertension. Pre-operative prothrombin time - international normalized ratio (PT/INR) was 1.2. IVC balloon angioplasty was done under fluoroscopic and ultrasound guidance. Pre angioplasty run showed occlusion of infrahepatic and suprahepatic IVC. Post angioplasty run showed opening of occlusion and disappearance of collaterals. She was put on oral anticoagulant with warfarin 5 mg once daily with PT/INR at 1.9.

In the present pregnancy, she was on low molecular weight heparin (LMWH) therapeutic dose until 16 weeks followed by oral anticoagulant warfarin. Her first-trimester nuchal translucency (NT) scan was normal; no biochemical aneuploidy screening was done. Ultrasound at 15 weeks of gestation showed a viable foetus of gestational age 15 weeks with multiple uterine artery collaterals with low resistance flow. Anomaly scan done at 26 weeks was normal. Her blood pressure recordings and diabetic screening were normal. Her foetal growth scan showed appropriate growth for gestational age. She was detected to have subclinical hypothyroidism and started on Tab Eltroxin once daily. She was switched over to inj. enoxaparin 40 mg once daily at 36 weeks after consultation with a cardiologist as her platelet count was 0.9 L/mm.[3] During pre-bridging time, PT/INR was 13/1.3 and after starting enoxaparin, PT/INR was 12.3/1.6. She underwent elective caesarean section at 39 weeks gestation in view of breech presentation. Post-operatively, she received enoxaparin 40 mg twice daily subcutaneously after 12 hours of surgery, followed by bridging therapy with warfarin for 5 days and a complete switch over to warfarin 5 mg with PT/INR of 12/1.7. Baby and mother were discharged in stable condition, and mother was advised to continue tablet warfarin 5 mg once daily. On follow-up at 5 months after delivery, she was on tablet warfarin 5 mg once daily with regular PT/INR monitoring. She was having regular menstrual cycles with no complaint of heavy bleeding.

Discussion

VTE complicates one in 1000 pregnancies. Pregnancy is a high-risk condition for VTE because of a hypercoagulable state. Other risk factors that can co-exist are obesity, advanced maternal age, multiparity, multiple pregnancies, inherited and acquired thrombophilia, previous pregnancy-related VTE events, and other associated medical conditions. Delivery by caesarean section, prolonged hospital stays, restricted mobilisation after surgery, emergency delivery, postpartum haemorrhage, and sepsis are additional risk factors for VTE in postnatal patients.

VTE spectrum includes deep vein thrombosis (calf and pelvic veins), IVC thrombosis, and pulmonary embolism. Patients with high clinical suspicion of VTE should be advised for duplex compression ultrasound for both lower limbs, ventilation-perfusion (V/Q) scan, and computer tomography pulmonary angiogram (CTPA) for suspected pulmonary embolism.[1] IVC thrombus is a life-threatening condition, rarely diagnosed in pregnancy and postpartum period, and can lead to fatal pulmonary embolism. During pregnancy, these patients can be managed by placing temporary IVC filters along with therapeutic doses of anticoagulants.[2] IVC angioplasty is a promising option with lifelong anticoagulants.

American College of Obstetricians and Gynaecologists (ACOG) and Royal College of Obstetricians and Gynaecologists (RCOG) recommend various regimens for prophylactic and therapeutic doses of anticoagulants for patients with a high risk of venous thromboembolism.[3,4] Warfarin has potential anticoagulant action, but it is associated with high foetal complications. Foetal warfarin syndrome (bony defects in foetus) is the most common complication and is mainly due to deficiency of vitamin K dependent active osteocalcin protein. Warfarin dose of less than 5 mg can be advised during the first trimester of pregnancy without harmful effects on the foetus. Its therapeutic effect can be measured by PT/INR, keeping it at 1.5 to 2.5 times normal. Unfractionated heparin (UFH) and low molecular heparin, inhibitors of active Xa factor, have a less anticoagulant effect than warfarin but better foetal safety. Unfractionated heparin (UFH) doses can be monitored by APTT levels 1.5-2.5 times the control and factor Xa levels for LMWH at 0.8-

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1.2 U/ml.[3] Complications of long-term use of heparin are heparin-induced thrombocytopaenia (HIT), hypersensitivity, and osteoporosis. The use of LMWH has a fewer incidence of HIT. Osteoporosis is a well-recognised complication of long-term heparin use and supplementation of additional calcium supplements in 1 gm daily dose, and weight-bearing exercises are recommended. Fondaparinux, a synthetic pentasaccharide that selectively inhibits factor Xa, has better tolerance than LMWH in patients with heparin hypersensitivity.[5] RCOG guidelines in April 2015 state that non-vitamin K antagonist oral anticoagulants/ newer oral anticoagulants (NOACs) should be avoided in pregnant and lactating women.[6]

Uterine AV malformation is an uncommon condition, which can result in sudden and massive bleeding. Available management options for uterine AV malformation are expectant management with medications, invasive therapies like unilateral/ bilateral uterine artery ligation and conventional hysterectomy. Embolotherapy proved its effectiveness in controlling bleeding with the additional advantage of sparing the uterus for future pregnancies.[7]

In the case of haemodynamically unstable patients, urgent surgical interventions like uterine artery ligation and traditional hysterectomy are used conventionally.[8,9] Conservative management can be done in stable patients with various medications like estrogen-progestin, methyl ergometrine, danazol and prostaglandins (PGF2 alpha).[10] Chan et al. reported a case of recurrent uterine AV malformation, which was managed safely by repeated embolisation.[11]

Our patient had a history of both VTE and bleeding uterine AV malformation (haemorrhagic condition) which was successfully managed with a good outcome.

Conclusion

Management of both venous thromboembolism and bleeding uterine AV malformation needs intensive monitoring and requires both anticoagulants and haemostatic therapy under the care of a multidisciplinary team with precious inputs from physicians, cardiologists, and intensivists. We conclude that a team-based approach is key in the conservative management of such high-risk stable patients.

Conflict of Interest: None

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