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Postpartum Ovarian Vein Thrombosis Following an Intrauterine Death Diagnosed at 31+2 Weeks

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Abstract

Ovarian vein thrombosis (OVT) is an uncommon cause of postpartum abdominal pain that may be difficult to recognise when systemic features are minimal. We report the case of a 26-year-old primiparous woman who presented with severe central abdominal pain 10 days after delivery of a stillborn infant at 31+5 weeks' gestation, nine days following discharge. She was haemodynamically stable with a mild tachycardia and minimally elevated inflammatory markers. Pelvic ultrasound did not identify a clear cause for her symptoms. Subsequent contrast-enhanced computed tomography (CT) of the abdomen and pelvis demonstrated a short-segment filling defect in keeping with left OVT. Therapeutic enoxaparin was commenced and subsequently changed to apixaban following haematology advice, with a planned three-month course of anticoagulation. Her pain resolved during the two-day admission, with no inpatient adverse events. Longer-term outcomes were unavailable. This case highlights the importance of considering OVT in patients with severe unexplained postpartum abdominal pain, including after stillbirth and during reported adherence to thromboprophylaxis, even when fever and substantial inflammatory abnormalities are absent.

Categories: Obstetrics/Gynecology, Hematology
Keywords: intrauterine fetal death, ovarian vein thrombosis, postpartum, puerperium, venous thromboembolism

Introduction

Postpartum ovarian vein thrombosis (OVT) is an uncommon but clinically important cause of abdominal pain in the puerperium. Its presentation can be non-specific, and diagnosis may be challenging when fever or substantial inflammatory abnormalities are absent. Its presentation can overlap with endometritis, retained products of conception, pelvic collection, appendicitis and urinary tract pathology. Potential complications include thrombus extension into the renal vein or inferior vena cava and pulmonary embolism [1].

The ovarian veins are a set of paired vessels that drain deoxygenated blood from the ovary, fallopian tubes and adjacent pelvic structures. The left ovarian vein drains into the left renal vein, whereas the right ovarian vein drains directly into the inferior vena cava [2]. OVT more commonly affects the right ovarian vein, although left-sided disease also occurs [1]. OVT is a rare condition typically seen in the postpartum period, pregnancy, abdominal or pelvic surgeries and gynaecological malignancies [3]. During pregnancy and the puerperium, raised oestrogen contributes to hypercoagulability and a reduction in circulating factors that inhibit clot formation [4].

Here we present the case of a patient who developed left-sided OVT in the postpartum period following intrauterine fetal death (IUFD) diagnosed at 31+2 weeks' gestation, followed by delivery at 31+5 weeks' gestation. Initial pelvic ultrasound was inconclusive, with contrast-enhanced computed tomography (CT) establishing the diagnosis. This case highlights the importance of considering OVT in patients with unexplained postpartum abdominal pain, even in the absence of fever or substantial inflammatory abnormalities and whilst receiving thromboprophylaxis.

Case Presentation

Initial presentation and antenatal findings

A 26-year-old primigravida attended maternity triage at 31+2 weeks' gestation following referral by her community midwife for a one-week history of reduced fetal movements, intermittent lower abdominal pain, and headaches. She had been previously well, with no medical, surgical, obstetric, or gynaecological history, and no family history of thrombophilia.

Her body mass index was 18.41 kg/m², and her blood group was B RhD positive. Her only regular medication was ferrous sulphate, commenced during pregnancy for anaemia, with a haemoglobin concentration of 96 g/L. The available growth scan report at 28+6 weeks documented fetal cardiac activity, a head circumference of 273 mm, abdominal circumference of 245 mm, a femur length of 55 mm and an estimated fetal weight of 1,307 g. Umbilical artery diastolic flow was present, with recorded pulsatility index values of 0.75 and 0.79.

Fetal heart activity could not be auscultated by the community midwife or on subsequent assessment in maternity triage. Ultrasound examination confirmed a single cephalic fetus with absent cardiac activity. Cardiovascular, respiratory and abdominal examinations were all normal. The fetal head circumference measured 233.6 mm, corresponding to 25+3 weeks' gestation. This represents an unresolved discrepancy in the available records and cannot be interpreted as a true reduction in fetal head size. On assessment, the patient was clinically and haemodynamically stable, with a temperature of 36.3°C, heart rate of 85 beats/minute, blood pressure of 107/59 mmHg, respiratory rate of 18 breaths/minute, and oxygen saturations of 98% on room air.

Laboratory investigations revealed the following: haemoglobin was 115 g/L, white cell count 4.86 × 10^9/L, and platelet count 137 × 10^9/L. Renal function, C-reactive protein (CRP), thyroid function, and liver function tests were within normal limits. Anticardiolipin antibodies and anti-beta2 glycoprotein I antibodies tested were reported as negative, with samples obtained before anticoagulation therapy was commenced.

Given her clinical stability, she was counselled regarding the diagnosis and management options. Following discussion, she elected to return home and re-attend the labour ward the following day for induction of labour.

Induction and delivery

On day two following presentation, the patient re-attended the labour ward with lower back pain. Speculum examination showed no evidence of labour. She received a single dose of oral mifepristone 200 milligrams (mg) and was discharged home.

On day three, the patient re-attended for induction of labour and received vaginal misoprostol 50micrograms (mcg), followed by two further doses of vaginal misoprostol 50 mcg at four-hour intervals. In view of the abdominal pain and intermittent uterine tightening, patient-controlled analgesia was commenced at her request. Delivery occurred approximately 12 hours after the first misoprostol dose.

On day four, she developed a temperature of 38.0°C. A septic screen was performed, with an initial lactate of 1.6 mmol/L, and intravenous cefuroxime 1.5 grams (g) three times daily and metronidazole 500 mg three times daily were commenced in accordance with local guidelines. One hour later, she delivered a stillborn infant weighing 1,190 g. Postpartum oxytocin was administered, and the placenta was delivered by controlled cord traction. Estimated blood loss was 150 millilitres (mL). Detailed gross placental and cord findings, placental histology, and stillbirth investigation results were unavailable at the time of writing. The cause of fetal death could not be established from the available records.

A postnatal venous thromboembolism risk assessment was completed, and prophylactic subcutaneous enoxaparin 40 mg once daily was commenced. Identified risk factors included systemic illness, preterm birth, and stillbirth. She also received immediate-release nifedipine 10 mg orally on two occasions, once before and once after delivery, for elevated blood pressure. Pre-eclampsia screens on both occasions were negative, with normal urinary protein:creatinine ratios, normal placental growth factor, normal liver function tests, and normal platelets.

On postpartum day 1, septic screen results demonstrated negative blood cultures, with a white cell count of 7.10 × 10^9/L and CRP of 17 mg/L. She remained clinically stable, with a temperature of 36.4°C, a heart rate of 98, saturating 98% in room air, and a blood pressure of 114/69 mmHg (mean arterial pressure (MAP) of 84). The patient was discharged with 10 days of subcutaneous enoxaparin 40 mg once daily, oral lactulose 15 mL twice daily, dihydrocodeine 30 mg four times daily, and a seven-day course of oral cefalexin and metronidazole as per local policy.

Postpartum readmission and diagnosis

On postpartum day 10, nine days following discharge, the patient re-presented via the emergency department and was admitted to the labour ward with an eight-hour history of severe periumbilical abdominal pain, rated nine out of 10 in intensity. The pain radiated to her back and flanks and was partially relieved by lying on her right side. The patient reported no clear exacerbating factors. There were no associated symptoms, including fever, night sweats, nausea, vomiting, altered bowel habit, or change in vaginal discharge. Lochia had been minimal since delivery. Her weight was 59.2 kg. She reported complete adherence to prophylactic enoxaparin 40 mg once daily and was still receiving prophylaxis when symptoms developed.

On examination, the patient was haemodynamically stable with mild tachycardia, and her observations were temperature of 37.1°C, heart rate of 102 beats/minute, blood pressure of 107/58 mmHg (MAP 74), respiratory rate of 17 breaths/minute, and oxygen saturation of 100% in room air. Her abdomen was soft and non-distended, with voluntary guarding on palpation of the periumbilical region but no signs of peritonism. The uterus was well contracted. There was no clinically detectable ascites, palpable mass, or organomegaly, and examination of the hernial orifices was unremarkable. Cardiovascular and respiratory examinations were normal. Initial differential diagnoses included intra-abdominal bleeding, intra-abdominal collection,

and OVTs.

Laboratory investigations showed a haemoglobin of 118 g/L, white cell count of 8.9 × 10^9/L, platelet count of 248 × 10^9/L, amylase of 51 U/L, and CRP of 8 mg/L. Additional laboratory results were as follows: alanine aminotransferase 22 U/L, alkaline phosphatase 75 U/L, bilirubin 7 micromol/L, albumin 30 g/L, sodium 133 mmol/L, potassium 4.5 mmol/L, creatinine 48 micromol/L, and urea 2.0 mmol/L. Urinalysis was negative for blood, protein, leucocytes and nitrites.

As the patient did not exhibit any biochemical or clinical features consistent with an infection, antibiotics were not given on readmission. Pelvic ultrasound performed six hours after admission did not identify a clear cause for her symptoms. There was no evidence of retained products of conception or a pelvic collection. The vessels visualised in the right adnexa appeared patent. The uterus was noted to be laterally rotated, which was reported as a soft marker for torsion; however, ovarian torsion was considered less likely in the absence of an associated ovarian cyst and with preserved stromal blood flow.

In view of the inconclusive ultrasound findings and ongoing clinical concern, contrast-enhanced CT of the abdomen and pelvis was performed. This demonstrated a small, short-segment filling defect within the mid left ovarian vein, measuring approximately 2 cm, consistent with ovarian vein thrombosis (Figure *1*). The left ovary was not clearly delineated but appeared mildly enlarged and oedematous. The uterus was enlarged, with prominent parauterine and myometrial vessels, appearances felt to be in keeping with the postpartum state. A small right ovarian cyst was also noted posterior to the uterus. The uterus was normally positioned within the pelvis, with no evidence of deviation.



FIGURE 1: Coronal contrast-enhanced CT of the abdomen and pelvis. The red arrow indicates the reported short-segment filling defect within the left ovarian vein, consistent with left ovarian vein thrombosis.

Following the CT findings, therapeutic-dose low-molecular-weight heparin (LMWH) was commenced. Due to logistical factors, the patient was initially treated with subcutaneous enoxaparin 60 mg twice daily, representing therapeutic-dose LMWH based on the patient's weight. After discussion and guidance from the haematology team the next day, anticoagulation was changed to oral apixaban, with a loading dose of 10 mg twice daily for six days, followed by 5 mg twice daily for a planned total duration of three months. The choice

of anticoagulation represents an individualised approach in line with extrapolation from National Institute for Health and Care Excellence (NICE) guidance for treatment of deep vein thrombosis and pulmonary embolism, acknowledging the limited evidence supporting direct oral anticoagulants specifically in postpartum OVT [5]. Apixaban was chosen to facilitate outpatient anticoagulation and avoid prolonged subcutaneous injections. Her pain resolved with oral morphine during the two-day admission. No bleeding or other adverse events were documented during admission. She was discharged on postpartum day 12 with dihydrocodeine 30 mg four times daily for analgesia, alongside anticoagulation counselling and safety-netting advice. The clinical course is summarised in Table *1*.

Time	Event
31+2 weeks' gestation	Presented at 31+2 weeks' gestation with reduced fetal movements; ultrasound confirmed intrauterine fetal death.
Following day	Re-attended with lower back pain; received oral mifepristone 200 mg and was discharged home.
Prior to delivery	Induction commenced with vaginal misoprostol; patient-controlled analgesia started for pain.
Postpartum day 0	Developed fever; septic screen performed and intravenous antibiotics commenced. Delivered a stillborn infant. Prophylactic enoxaparin started following postnatal VTE risk assessment.
Postpartum day 1	Clinically stable and discharged with prophylactic enoxaparin, oral antibiotics, analgesia, and laxatives.
Postpartum day 10	Re-presented with severe periumbilical abdominal pain radiating to the back and flanks. Initial blood tests were largely unremarkable. Pelvic ultrasound did not identify retained products, collection, or clear adnexal pathology. CT of the abdomen and pelvis demonstrated left ovarian vein thrombosis; therapeutic-dose enoxaparin was commenced.
Postpartum day 11	Following haematology discussion, anticoagulation was changed to oral apixaban for a planned three-month course.

TABLE 1: Timeline of events and treatments; Delivery is designated postpartum day 0.

CT: computed tomography; VTE: venous thromboembolism.

Discussion

Postpartum OVT is a rare but important cause of acute abdominal pain in the puerperium. Its clinical significance lies in its non-specific presentation and potential complications, including thrombus extension into the renal vein or inferior vena cava, sepsis, and pulmonary embolism if diagnosis and treatment are delayed [1]. One single-centre study reports that OVT occurs in 0.05% of pregnancies resulting in live birth [6]. The presentation of OVT is often non-specific and may overlap with more common postpartum complications, including endometritis, retained products of conception, pelvic collection, ovarian torsion, appendicitis, or urinary tract pathology. Although OVT most commonly affects the right ovarian vein [7], this case involved the left ovarian vein, which is less frequently reported.

This case highlights an atypical presentation due to OVT occurring after IUFD and stillbirth. Furthermore, the patient had left-sided OVT, whereas right-sided disease is more typical. In addition, she presented with severe pain but minimal systemic features. Her observations and inflammatory markers were not markedly abnormal, highlighting that OVT may occur in the absence of classical infective or inflammatory features. Prior to discharge, our patient's venous thromboembolism risk was assessed as per local trust guidelines. The patient scored 3 points due to her being treated for systemic illness in addition to stillbirth and preterm birth. As a result, she was discharged with 10 days of prophylactic enoxaparin following local trust policy guided by the Royal College of Obstetricians and Gynaecologists (RCOG) [8]. The patient developed OVT despite reporting complete adherence to prophylactic anticoagulation.

As proposed by Rudolf Virchow in 1856, vascular thrombosis is promoted by hypercoagulability of blood, venous stasis and vessel wall injury [9]. All three may be present in pregnancy and the puerperium. Venous stasis is promoted by compression of pelvic veins by the gravid uterus, hormone-mediated venodilatation, and reduced venous flow in the puerperium. Furthermore, vessel wall injury can be a result of delivery of the placenta from the uterine wall, instrumental deliveries and caesarean sections. The resulting vascular trauma activates the extrinsic coagulation cascade [10]. Finally, pregnancy has been demonstrated to result in an increase in fibrinogen and clotting factors V, VII, IX, X and XII, acquired activated protein C resistance

and reduced protein S activity [11,12], contributing to a hypercoagulable state.

The diagnosis of OVT can be challenging because symptoms are non-specific and may mimic other postpartum abdominal or gynaecological pathology. In this case, the initial differential diagnoses included intra-abdominal bleeding, intra-abdominal collection, ovarian torsion, and OVT. The absence of fever, leucocytosis, or significantly raised CRP may have reduced clinical suspicion; however, the severity of the patient's pain and lack of an alternative diagnosis warranted further imaging.

Pelvic ultrasound is a useful first-line investigation in postpartum patients, particularly to assess for retained products of conception, pelvic collection, or adnexal pathology [13]. However, visualisation of the ovarian veins may be limited, and ultrasound may not reliably exclude OVT. In this case, contrast-enhanced CT was diagnostic, demonstrating a short-segment filling defect within the left ovarian vein. This supports the use of cross-sectional imaging when postpartum abdominal pain is severe, persistent, or unexplained by initial assessment.

Stillbirth is associated with an increased risk of postpartum venous thromboembolism, although evidence specifically linking it to OVT remains limited. Sultan et al. identified stillbirth as the strongest postpartum venous thromboembolism risk factor, reporting an incidence rate ratio of 6.2 [14]. This estimate, however, relates to postpartum venous thromboembolism overall and should not be interpreted as an OVT-specific risk. Li et al. reported a case of Group B Streptococcus infection-associated right ovarian vein thrombosis diagnosed during caesarean section in a patient with placental abruption and intrauterine fetal demise [15]. Lee et al. presented a case similar to the case we have described, where a woman developed postpartum OVT after an IUFD at 25+6 weeks' gestation due to hydrops fetalis [16]. Ultrasound and CT findings were initially suggestive of appendicular abscess, and OVT was diagnosed at laparotomy. She was subsequently treated with anticoagulation. In their case, Lee et al. had confirmed the most likely cause for the IUFD but could not comment on an association between hydrops fetalis and postpartum OVT. They had considered the need for prophylactic antepartum anticoagulation in future pregnancies, but this remains debated [16]. These observations support considering OVT in patients with unexplained abdominal pain after stillbirth, whilst recognising that the available evidence does not establish fetal death as an independent cause of OVT.

Pain resolved during the readmission, but longer-term symptom status, completion of the planned three-month anticoagulation course, and radiological resolution were unavailable at the time of writing. The absence of subsequent outcome data prevents conclusions about sustained recovery, recurrent thrombosis, or the longer-term effectiveness and safety of apixaban in this patient. The negative antiphospholipid antibody results did not exclude all inherited or acquired thrombophilias. Interpretation of thrombophilia investigations during pregnancy also requires caution. Other limitations include the unresolved fetal head circumference discrepancy and unavailable placental and stillbirth investigation results.

Conclusions

OVT is a rare yet important differential to consider in patients with acute abdominal pain in the puerperium. Diagnosis can be made using a combination of a patient's history and presentation, risk factors, and imaging. Absence of fever or substantial inflammatory abnormalities does not exclude the diagnosis; mild tachycardia was present in this case. Whilst ultrasound is a low-cost and useful tool to exclude common postpartum complications, it may not reliably identify OVT. Early CT or MRI should be considered when symptoms are disproportionate, persistent, or unexplained by initial clinical assessment. Symptomatic postpartum OVT is commonly managed with therapeutic anticoagulation, often for a three-month duration, although treatment should be individualised with haematology input due to limited OVT-specific guidance.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Nazar I. Mohammed, Leena Khan

Acquisition, analysis, or interpretation of data: Nazar I. Mohammed, Leena Khan

Drafting of the manuscript: Nazar I. Mohammed, Leena Khan

Critical review of the manuscript for important intellectual content: Nazar I. Mohammed, Leena Khan

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Nazar Mohammed and Leena Khan contributed equally to the work and should be considered co-first authors.

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