SISLA MEDICAL PAEDIATRICS & GYNAECOLOGY JOURNAL

Volume 1 Issue 1 Case Report

Postpartum Right Ovarian Vein Thrombosis Following a Fifth Vaginal Delivery: A Case Report

Dr. Elmar Bayraktarov*

**Radiology, Diakonie Klinikum Kirchen, Germany.*

***Corresponding author:** *Dr. Elmar Bayraktarov, Radiology, Diakonie Klinikum Kirchen, Germany.*

Received Date: September 09, 2026; **Published Date:** November 06, 2026

© All Rights Reserved by Elmar Bayraktarov

Abstract

Background

Ovarian Vein Thrombosis (OVT) is a rare but potentially serious postpartum complication. In a recent retrospective cohort of 34,140 postpartum women, OVT occurred in 0.05% following vaginal delivery and 0.10% following cesarean delivery [1]. It is more frequently reported in association with puerperal infection and hypercoagulability [2,3]. Although the right ovarian vein is predominantly affected, the mechanisms underlying this laterality are multifactorial [4,5]. The possible contribution of multiparity to postpartum OVT remains insufficiently defined. The aim of this case report is to describe right postpartum OVT following a fifth spontaneous vaginal delivery and to discuss the potential contribution of high parity in the context of established anatomical, hemodynamic, inflammatory, and thrombotic factors.

Case Presentation

We report the case of a 27-year-old woman (G6P5) who developed right ovarian vein thrombosis following her fifth spontaneous vaginal delivery. The delivery was complicated by increased postpartum vaginal bleeding requiring uterine curettage and temporary intrauterine Celox® tamponade. On postpartum day 2, the patient developed fever up to 39°C with elevated inflammatory markers. Blood cultures were negative. Despite broad-spectrum antibiotic therapy, the inflammatory response persisted. Contrast-enhanced magnetic resonance imaging demonstrated thrombosis of the right ovarian vein with venous wall enhancement and surrounding inflammatory changes extending toward the inferior vena cava. Therapeutic anticoagulation with enoxaparin and prolonged intravenous antibiotic therapy resulted in clinical improvement. The patient was discharged on postpartum day 22 in good general condition. Thrombophilia investigations did not identify an inherited or acquired thrombophilic disorder.

Conclusion

Postpartum OVT should be considered in women presenting with persistent fever and inflammatory findings despite antibiotic therapy, including women following spontaneous vaginal delivery [2,6]. The marked right-sided predominance of OVT may be explained by a combination of anatomical and hemodynamic factors [4,5]. Although the present case raises the possibility that high parity may contribute to the development of postpartum OVT, current evidence is insufficient to establish multiparity as an independent risk factor. Further observational studies are warranted.

Keywords: *Ovarian vein thrombosis; Postpartum complications; Multiparity; Magnetic resonance imaging; Pelvic thrombosis; Puerperal infection*

Introduction

Ovarian vein thrombosis (OVT) is an uncommon form of venous thromboembolism that is most frequently encountered during the postpartum period. In a recent retrospective cohort of 34,140 postpartum women, OVT occurred in 0.05% following vaginal delivery (10/19,912) and 0.10% following cesarean delivery (14/14,228) [1]. OVT is clinically important because thrombus extension into the inferior vena cava or renal veins and pulmonary

embolism may occur [2].

The pathophysiology of postpartum OVT can be understood in the context of Virchow's triad, involving venous stasis, endothelial injury, and hypercoagulability. Pregnancy and the puerperium are physiologically hypercoagulable states, while uterine enlargement and pelvic venous compression contribute to venous stasis. Endothelial injury may occur during delivery or in association with local pelvic inflammation or infection [3,6].

The right ovarian vein is involved in the majority of postpartum OVT cases [4,5]. Several anatomical and hemodynamic mechanisms have been proposed, including the longer course of the right ovarian vein, its drainage directly into the inferior vena cava, physiological dextrorotation of the gravid uterus with compression of the right ovarian vein and inferior vena cava, and differences in postpartum venous flow [4,5]. These factors may collectively promote venous stasis and thrombus formation.

Established risk factors for OVT include pregnancy and the postpartum state, cesarean delivery, pelvic infection, pelvic or gynecological surgery, malignancy, inflammatory disease, and inherited or acquired thrombophilia [2,3,6]. Importantly, parity of three or more is recognized as a maternal VTE risk factor in the Royal College of Obstetricians and Gynaecologists (RCOG) risk-assessment tool and is assigned one risk point [10]. However, this risk score addresses pregnancy-associated VTE overall and does not establish multiparity as an independent risk factor for OVT. In two French prospective cohorts, 13 women with symptomatic postpartum OVT were identified, and 2 (15.4%) had antenatal parity of three or more [11]. In a three-case series of postpartum OVT within the spectrum of septic pelvic thrombophlebitis, two women had recorded parity values of 6+3 and 8+1, whereas the third was nulliparous (0+0) [12]. Conversely, in a multicenter cohort of 73,087 women from a cesarean delivery registry examining postpartum septic pelvic thrombophlebitis, 89 women developed the condition, and nulliparity was more common among cases than controls (51.1% vs. 23.3%) [13]. Taken together, these small and heterogeneous datasets do not establish high parity as an independent risk factor for OVT. Repeated pregnancies may theoretically expose the pelvic venous system to repeated periods of venous distension, compression, hormonal changes, and hypercoagulability, but evidence establishing multiparity as an independent risk factor remains limited.

We present a case of right-sided postpartum OVT following a fifth spontaneous vaginal delivery in a 27-year-old G6P5 woman. The case highlights the diagnostic importance of OVT in postpartum fever and discusses the possible contribution of high parity, as well as the anatomical and hemodynamic mechanisms underlying the predominance of right-sided disease.

Case Presentation

A 27-year-old woman (gravida 6, para 5) was admitted at 40+3 weeks of gestation because of the onset of labor. A healthy term infant was delivered vaginally.

For uterine atony prophylaxis, 10 IU of oxytocin was administered. Shortly after delivery, the patient developed increased vaginal bleeding. Uterine curettage was therefore performed, followed by placement of an intrauterine Celox® tamponade. The tamponade was removed uneventfully the following day.

On postpartum day 2, the patient developed a fever of up to 39°C. At that time, she did not report other significant clinical symptoms. Initial blood cultures

were negative. Laboratory investigations demonstrated elevated inflammatory markers, including a C-reactive protein level of 11.3 mg/L and a white blood cell count of 14.1 $\times 10^9$/L. The hemoglobin concentration was 9.8 g/dL.

Empirical antibiotic therapy with cefuroxime and metronidazole was initiated. Because of persistent fever and inflammatory findings, antibiotic therapy was subsequently escalated to piperacillin-tazobactam.

Despite antibiotic treatment, the patient continued to have fever and elevated inflammatory markers. Contrast-enhanced MRI of the pelvis performed on postpartum day 4 demonstrated thrombosis of the right ovarian vein, characterized by dilatation of the ovarian vein with intraluminal filling defect and surrounding inflammatory changes. The thrombus extended cranially toward the inferior vena cava without evidence of inferior vena cava thrombosis.

Therapeutic anticoagulation with enoxaparin, initially administered at a dose of 0.8 mL subcutaneously twice daily and subsequently adapted according to body weight, was initiated.

Despite ongoing low-grade fever during the initial treatment period, the patient's inflammatory markers progressively improved, and she remained hemodynamically stable. A repeat MRI performed on postpartum day 12 demonstrated persistent thrombosis of the right ovarian vein with more pronounced surrounding inflammatory changes.

Abdominal Doppler ultrasonography did not demonstrate thrombosis of the inferior vena cava or hepatic veins.

The patient was evaluated jointly by the internal medicine and gynecology teams. Therapeutic anticoagulation for at least 3 months was recommended. Intravenous antibiotic treatment was continued for a total duration of 21 days.

The patient remained afebrile for several consecutive days, with progressive clinical and laboratory improvement. She was discharged on postpartum day 22 in good general condition.

Thrombophilia and Additional Risk-Factor Assessment

Because postpartum OVT may be associated with inherited or acquired thrombophilia, additional thrombophilia evaluation was performed [2,3].

The patient's personal history was negative for previous venous thromboembolism, and there was no known family history of thrombosis.

The thrombophilia investigations performed included Factor V Leiden mutation, prothrombin G20210A mutation, protein C activity, protein S activity, antithrombin activity, lupus anticoagulant, anticardiolipin antibodies, anti-β2-glycoprotein I antibodies, and homocysteine.

The thrombophilia work-up was negative, and no inherited or acquired thrombophilic disorder was identified in the patient.

Although thrombophilia has been reported in a subset of patients with postpartum OVT, the available evidence is heterogeneous [2,7]. In the present case, therefore, no specific thrombophilic predisposition was identified.

Imaging Findings

Contrast-enhanced MRI was the principal imaging modalities used for diagnosis and follow-up.

On MRI, the thrombosed right ovarian vein appeared as an enlarged tubular retroperitoneal structure containing an intraluminal filling defect, associated with venous wall enhancement and surrounding inflammatory fat stranding Figures 1&2. The thrombus extended cranially toward the inferior vena

cava.

Follow-up MRI on postpartum day 12 demonstrated persistent thrombus with more pronounced inflammatory changes.

The inferior vena cava and hepatic veins were evaluated by Doppler ultrasonography and showed no evidence of thrombosis.

MRI has been reported to have excellent diagnostic performance for OVT, with CT also providing high diagnostic utility, particularly in the acute setting [2]. Ultrasound may be used as an initial examination, although visualization of the ovarian veins can be limited by bowel gas and their deep retroperitoneal location [3,6].

Discussion

Postpartum OVT is a rare but clinically important condition that should be considered in the differential diagnosis of postpartum fever, particularly when the response to antibiotic therapy is incomplete [2,6]. The clinical presentation may be nonspecific and can overlap with postpartum endometritis, urinary tract infection, pyelonephritis, appendicitis, adnexal pathology, and other causes of pelvic inflammation.

In the present case, the diagnosis was particularly challenging because the initial clinical manifestation was fever with elevated inflammatory markers rather than prominent abdominal pain. The persistence of fever despite broad-spectrum antibiotics prompted further imaging, leading to the diagnosis of right OVT. This clinical course is consistent with previous reports emphasizing the importance of considering OVT in women with persistent postpartum fever despite antibiotic treatment [2,6].

Right-Sided Predominance of OVT

The marked predominance of right-sided OVT is a well-described feature of the disease [4,5]. Several anatomical and hemodynamic mechanisms may contribute to this phenomenon.

First, the right ovarian vein generally has a longer course than the left ovarian vein and drains directly into the inferior vena cava, whereas the left ovarian vein usually drains into the left renal vein. The right ovarian vein therefore follows a different anatomical and hemodynamic pathway [4,5].

Second, the gravid uterus undergoes physiological dextrorotation during pregnancy. The enlarged uterus may compress the right ovarian vein and inferior vena cava at the level of the pelvic brim, thereby reducing venous flow and promoting stasis [4,5].

Third, changes in venous flow during the postpartum period may contribute to right-sided susceptibility. Differences in flow direction between the right and left ovarian veins have been described, with antegrade flow reported in the right ovarian vein and retrograde flow in the left ovarian vein in the postpartum period [4,5].

Finally, the presence and competence of venous valves may influence local venous stasis. Dilatation of the ovarian veins during pregnancy may impair valvular competence and further facilitate thrombus formation [4,5].

Taken together, these anatomical and hemodynamic factors provide a plausible explanation for the predominance of right-sided OVT observed in our patient.

Multiparity as a Potential Contributing Factor

The possible relationship between multiparity and postpartum OVT is less well established. Most recognized risk factors for OVT relate to pregnancy-

associated hypercoagulability, cesarean delivery, pelvic infection, pelvic surgery, malignancy, inflammatory disease, and thrombophilia [2,3,6]. Although parity of three or more is included as a risk factor in the RCOG maternal VTE risk-assessment tool [10], this applies to pregnancy-associated VTE overall and does not demonstrate an independent association with OVT. Recent observational data remain limited: 2 of 13 women with symptomatic postpartum OVT in two French prospective cohorts had antenatal parity of three or more [11], while a small postpartum OVT case series included two multiparous women and one nulliparous woman [12]. In contrast, a multicenter cohort of postpartum septic pelvic thrombophlebitis found nulliparity to be more common among affected women [13]. These heterogeneous findings do not support a simple or established relationship between higher parity and postpartum OVT.

Repeated pregnancies may theoretically contribute to pelvic venous stasis through repeated uterine enlargement and compression of the pelvic venous system. Recurrent pregnancy-associated hormonal and coagulation changes may also contribute to a prothrombotic environment. In addition, repeated pregnancies may result in recurrent distension of the pelvic venous circulation.

However, these proposed mechanisms remain theoretical, and current evidence is insufficient to establish multiparity as an independent risk factor for OVT. The present case should therefore be considered hypothesis-generating rather than evidence of a causal association.

Inflammatory and Thrombotic Mechanisms

The patient developed high-grade fever and elevated inflammatory markers on postpartum day 2, followed by radiological evidence of ovarian vein thrombosis with venous wall enhancement and surrounding inflammatory changes.

The inflammatory component may reflect puerperal pelvic inflammation associated with ovarian vein thrombophlebitis. However, the negative blood cultures do not establish a microbiologically confirmed septic thrombophlebitis. The inflammatory changes may therefore represent either a septic/inflammatory component or inflammation secondary to the thrombotic process [3,6].

The postpartum state itself provides a strong prothrombotic background. Pregnancy and the puerperium are characterized by increased coagulation activity, reduced fibrinolytic activity, and venous stasis [2,3]. In the present patient, additional potential contributors included postpartum bleeding, uterine curettage, temporary intrauterine tamponade, and the local inflammatory response.

Because thrombophilia can be associated with OVT, documentation of thrombophilia testing is clinically relevant. In the current literature, thrombophilia has been identified in a subset of patients who underwent testing, although the available evidence is heterogeneous and based predominantly on observational data [2,7].

Diagnostic Imaging

with a Imaging plays a central role in the diagnosis of OVT because clinical findings are nonspecific [2,3]. Ultrasound with Doppler may be used as an initial examination, although visualization of the ovarian veins can be limited by bowel gas and their deep retroperitoneal location [3,6].

Contrast-enhanced CT can demonstrate an enlarged ovarian vein containing a central thrombus, peripheral venous wall enhancement, and surrounding inflammatory changes. CT is particularly useful in the acute setting because it can

simultaneously evaluate alternative causes of postpartum abdominal or pelvic symptoms and assess thrombus extension toward the inferior vena cava [2,3,6].

MRI provides excellent soft-tissue contrast and may be particularly useful when CT findings are equivocal or when radiation exposure is a concern. A recent scoping review reported the best diagnostic performance for MRI, followed by CT [2]. In the present case, both CT and MRI demonstrated the thrombotic and inflammatory changes of the right ovarian vein, while follow-up MRI was used to assess persistence and progression of the inflammatory component.

Management

Management of postpartum OVT generally includes anticoagulation, while antibiotic therapy is frequently used when an inflammatory or infectious component is suspected [2,6].

In the present case, therapeutic anticoagulation with enoxaparin was initiated after the diagnosis was established. Broad-spectrum intravenous antibiotics were continued because of the patient's persistent fever and inflammatory findings. The patient demonstrated progressive clinical and laboratory improvement and was discharged after 22 postpartum days.

The optimal duration of anticoagulation for OVT remains uncertain because of the rarity of the condition and the lack of high-quality randomized evidence. Published literature commonly describes anticoagulation for approximately 3–6 months in postpartum OVT [2,8]. In our patient, at least 3 months of therapeutic anticoagulation was recommended by the treating internal medicine and gynecology teams.

Limitations

This report has the inherent limitations of a single-case observation. Although the patient's high parity raises the possibility that repeated pregnancies may contribute to the development of postpartum OVT, a causal relationship or an independent association between multiparity and OVT cannot be established from a single case.

Furthermore, the presence of inflammatory changes and fever raises the possibility of septic or inflammatory ovarian vein thrombophlebitis, but the negative blood cultures do not provide microbiological confirmation of infection.

Finally, the patient had postpartum bleeding requiring uterine curettage and temporary intrauterine tamponade, which represents an additional potential thrombotic or inflammatory contributor. Therefore, the development of OVT cannot be attributed to parity alone.

Conclusion

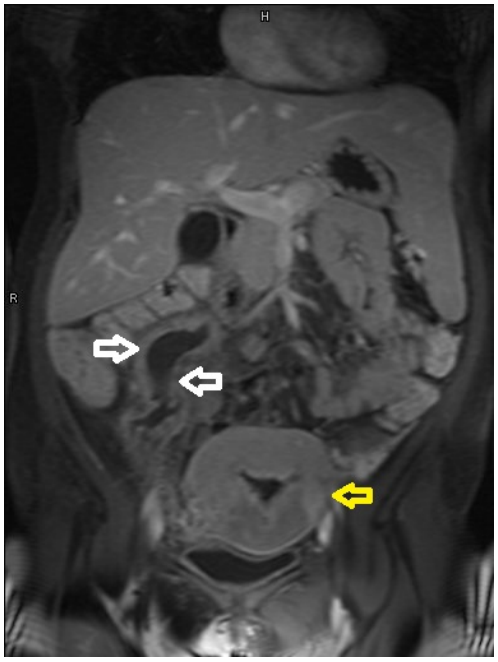
Congenital Postpartum ovarian vein thrombosis should be considered in women presenting with persistent fever and inflammatory findings after delivery, particularly when symptoms do not adequately respond to antibiotic therapy [2,6]. Although OVT is more commonly associated with cesarean delivery, it can also occur after spontaneous vaginal delivery.

The strong right-sided predominance of OVT is likely multifactorial and may be explained by anatomical and hemodynamic characteristics of the right ovarian vein, including uterine dextrorotation, compression of the right ovarian vein and inferior vena cava, the longer venous course, and differences in postpartum venous flow [4,5].

The present case raises the possibility that high parity may contribute to the development of postpartum OVT. However, current evidence is insufficient to establish multiparity as an independent risk factor. Larger observational studies are needed to clarify the potential relationship between parity and postpartum ovarian vein thrombosis.

Early cross-sectional imaging, particularly contrast-enhanced CT or MRI, is important for establishing the diagnosis and evaluating thrombus extension [2,3]. When an inflammatory or infectious component is suspected, combined anticoagulant and antibiotic therapy should be considered according to the individual clinical situation [2,6].

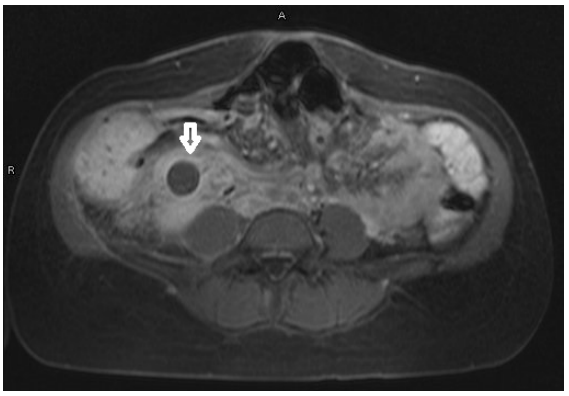
Figure 1:



Contrast-enhanced MRI of the abdomen:

Contrast-enhanced MRI demonstrating thrombosis of the right ovarian vein with an intraluminal filling defect, venous enlargement, peripheral wall enhancement, and surrounding inflammatory changes.

Figure 2:



Contrast-enhanced MRI of the abdomen:

MRI demonstrating persistent thrombosis of the right ovarian vein with venous wall enhancement and surrounding inflammatory changes.

References

1. Chuanmei C, Jiang S, Liu D, Wei Z, Xu J, Shen Z (2025) Postpartum ovarian venous thrombophlebitis: clinical manifestations and the diagnostic value of non-contrast computed tomography semi-quantitative parameters. Clin Exp Obstet Gynecol 52(9): 41729.
2. Monnet M, Dufrost V, Wahl D, Morel O, Agopiantz M, Zuily S (2024) Epidemiology, natural history, diagnosis, and management of ovarian vein thrombosis: a scoping review. J Thromb Haemost 22(11): 2991-3003.
3. Bannow S BT, Leslie S (2017) Diagnosis and management of postpartum ovarian vein thrombosis. Hematology Am Soc Hematol Educ Program 8(1): 168-171.
4. Abrantes J, Teixeira E, Gomes F, Fernandes C (2019) Postpartum ovarian vein thrombosis and venous anatomical variation. BMJ Case Rep 12(6).
5. Jenayah AA, et al. Ovarian vein thrombosis. Pan Afr Med J 21:251.
6. Shi Q, Gandi DS, Hua Y, et al (2021) Postpartum septic pelvic

thrombophlebitis and ovarian vein thrombosis after caesarean section: a rare case report. BMC Pregnancy Childbirth 21: 561.

7. Salomon O, Apter S, Shaham D, et al. (1999) Risk factors associated with postpartum ovarian vein thrombosis. ThrombHaemost 82(3): 1015-1019.
8. Salomon O, Dulitzky M, Apter S (2010) New observations in postpartum ovarian vein thrombosis: experience of a single center. pascal-francis 21(1): 16-19.
9. Royal College of Obstetricians and Gynaecologists. Reducing the risk of thrombosis and embolism during pregnancy and the puerperium. Green-top Guideline No. 37a. 3rd ed. London: RCOG; 2015, amended 2023.
10. Laouenan E, de Moreuil C, Robin S, Morcel K, Anouilh F, Tromeur C, et al., (2025) E. Risk factors and prognosis of symptomatic postpartum ovarian vein thrombosis in 2 French prospective cohort studies. Res Pract Thromb Haemost 9(3): 102849.
11. Akbari EA, Majdalawi R, Harb DK, Hazari K, Abdelkareem W, Ammar A (2023) Postpartum septic pelvic thrombophlebitis in a tertiary maternity hospital in Dubai, UAE. Cureus 15(3): e36452.
12. Dotters-Katz SK, Smid MC, Grace MR, Thompson JL, Heine RP, Manuck T (2017) Risk factors for postpartum septic pelvic thrombophlebitis: a multicenter cohort. Am J Perinatol 34(11): 1148-1151. L, Mancini V, Sanguedolce F, Massenio P, Fucci L, et al. (2014) "Epidermoid cyst of the coronal sulcus mimicking penile cancer: a case report". J Med Case Rep 8: 179.
13. Guler Y, Tatar Z, Ucpinar B and Erbin A (2018) "An epidermal cyst of the penis after distal hypospadias surgery: a case report". J Med Case Rep 12: 378.
14. Kamal A, Arif M, Sinha S and Singh V (2018) "Epidermoid cyst of the penis: a rare cause of penile swelling". Urology Annals 10: 327-329.
15. Kumaraguru V, Prabhu R and Kannan NS (2016) "Penile epidermal cyst: a case report". J Clin Diagn Res 10: PD05-PD06.
16. Okumur A, Takagawa, K and Fuse H (2012) "Penile epidermoid cyst consisted of multiple foci: a case report". JCDSA 2: 214-215.
17. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H et al. (2013) "The CARE guidelines: consensus-based clinical case reporting guideline development". J Clin Epidemiol 67: 46-51.

Copyright: © 2026 Dr. Elmar Bayraktarov This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the