From Puerperal Fever to Ovarian Vein Thrombosis: An Historical Journey and Contemporary Challenges in Diagnosis and Management

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Abstract

Puerperal sepsis (PPS) is a severe postpartum infection that remains a significant maternal health concern. Recent evidence suggests a potential link between PPS and ovarian vein thrombosis (OVT), a rare but life-threatening complication occurring in 0.01 to 0.18% of pregnancies. Despite the historical significance of PPS and its well-documented consequences, its association with thrombosis remains underrecognized in obstetric practice. This narrative review explores the historical context, clinical presentation, diagnosis, and management of PPS and OVT while emphasizing the need for increased awareness and preventive strategies. Sepsis triggers a hypercoagulable state through inflammatory cytokine release, endothelial injury, and coagulation activation, contributing to thrombotic complications such as OVT. The right ovarian vein is more commonly affected due to anatomical factors, including uterine dextrorotation during pregnancy. OVT typically presents with abdominal pain and fever, requiring imaging modalities such as Doppler ultrasound and magnetic resonance imaging for diagnosis. Although anticoagulation therapy is widely used for deep vein thrombosis, its application in OVT remains inconsistent, despite comparable recurrence rates between the two conditions. The review also highlights the lack of consensus on thromboprophylaxis in septic postpartum patients. Although guidelines from major obstetric organizations are inconsistent, emerging evidence suggests that low-molecular-weight heparins may reduce thrombotic risk in PPS. In the absence of large-scale randomized trials, observational studies remain essential for guiding clinical decisions.

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- puerperal infection
- embolism and thrombosis
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Puerperal sepsis (PPS), a severe postpartum infection, is increasingly recognized as a potential risk factor for ovarian vein thrombosis (OVT), though existing evidence remains limited and inconclusive. Although rare-occurring in 0.01 to 0.18% of pregnancies,[1] OVT carries a significant risk of mortality. Historically, PPS was a devastating condition that afflicted women within days of childbirth, presenting with severe abdominal pain, fever, and profound weakness.[2,3] While numerous studies and guidelines have been published to facilitate early diagnosis and treatment of PPS, less attention has been given to thrombosis, a complication that can further exacerbate the disease.

Despite the life-threatening nature of postpartum infections and PPS, there remains a striking lack of robust epidemiological data and clinical awareness regarding their association with OVT, leaving this risk underappreciated in routine obstetric practice. Integrating PPS into risk assessments for OVT is crucial, as untreated infections can induce hypercoagulable states and vascular inflammation, significantly increasing thrombotic potential. Failure to recognize this connection may lead to delayed diagnosis and anticoagulant therapy, underscoring the need for heightened clinical vigilance to prevent avoidable maternal morbidity.

This narrative review aims to revisit the historical context of PPS as a relevant risk factor for OVT while summarizing the current understanding of its clinical presentation, diagnosis, and management. We will also discuss thromboprophylaxis strategies and explore available diagnostic and treatment options to enhance clinical decision-making.

Methods

We conducted a comprehensive search of the MEDLINE database up to January 2025, using the following keywords: "sepsis AND hemostatic system, sepsis AND puerperium, PPS AND history, PPS AND venous thromboembolism (VTE), and PPS AND thromboprophylaxis." Articles published in English were included without restrictions on publication type, year, or geographic location. Only peer-reviewed articles relevant to the review's objectives were considered. Screening was performed by reviewing titles and abstracts, followed by a critical appraisal of full-text articles based on clinical relevance, study methodology, and reported findings.

Brief History

The Hippocratic Corpus, a collection of 60 Greek medical texts, documented puerperal infections as part of various infectious syndromes.[4] However, the term puerperal fever first appeared in scientific literature in the early 18th century, as noted by Irvine Loudon.[5] A more detailed clinical description was provided in 1822 by Scottish physician William Campbell,[6] who observed that the disease typically began on the third day postpartum with rigors, headache, fever, chills, and thirst. Abdominal pain-initially mild but progressively worsening—was a hallmark symptom, often localized to the hypogastric and iliac regions. Other symptoms included painful urination with dark urine, respiratory

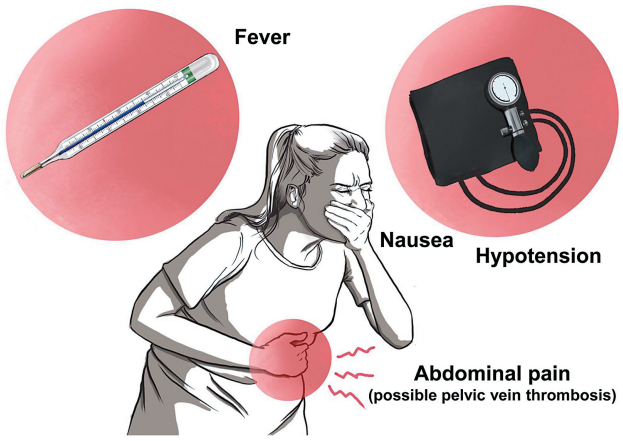

Fig. 1 Shows the most common clinical signs observed in women with puerperal sepsis.

distress, nausea, vomiting, tachycardia, and a characteristic white-coated tongue that darkened ominously before death[6] (►**Fig. 1**). Christine Hallett, in her comprehensive review,[3] reported that mortality rates during epidemics of PPS were as high as 70%, compared to approximately 30% in sporadic cases. Outbreaks were documented in Paris, London,[7] Dublin (1767), and Edinburgh (1773).[3] At the time, prevailing theories attributed the disease to inflammation and putrefaction.[8–10] Some believed that constipation during pregnancy led to the accumulation of putrid substances in the bloodstream, triggering sepsis. Consequently, treatments focused on inducing diarrhea and bloodletting, controversial practices that some clinicians argued could worsen the condition.[3] A significant breakthrough came with the observation that puerperal fever was often associated with erysipelas, paving the way for the germ theory in the late 19th century.[11] It was also recognized that the disease could be transmitted within clinical settings, particularly by physicians and midwives. The turning point occurred in 1847 when Hungarian obstetrician Dr. Ignaz Semmelweis noted that maternal mortality from PPS was three times higher in the clinic where medical students and physicians worked compared to the clinic staffed solely by midwives. He observed that doctors in the first clinic frequently performed autopsies, inadvertently exposing mothers to infectious material. In contrast, this issue did not arise in the second clinic, as midwives did not participate in autopsies. His hypothesis gained further support when his professor, Jacob Kolletschka, died from an infection following a finger puncture during an autopsy, exhibiting pathological findings identical to those of women who had died from PPS.[12] Semmelweis concluded that puerperal fever was caused by decaying organic matter—a revolutionary idea at the time, as bacteria had not yet been identified as the causative agents of infection. To fight the spread of infection, he introduced systematic handwashing with water, soap, and chlorine, reducing mortality rates by 3 to 10 times.[12] However, his findings were met with resistance from the medical community, and he was ultimately ostracized, dying in a psychiatric hospital at the age of 47. His work was only recognized

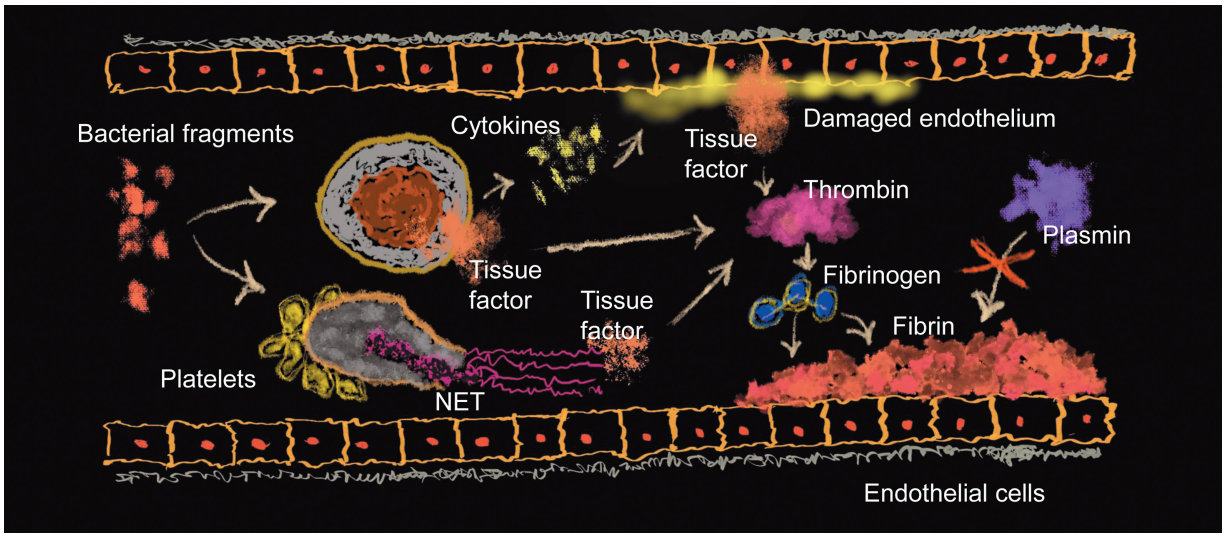

Fig. 2 Depicts the mechanism of thrombo-inflammation in sepsis. Bacterial fragments activate monocytes, leading to cytokine release and tissue factor exposure. Platelets contribute by forming complexes with neutrophil extracellular traps, which bind to tissue factor. Endothelial cell damage from cytokines further exposes tissue factor, initiating the coagulation cascade. This results in thrombin generation, converting fibrinogen into fibrin, while fibrinolysis is suppressed, promoting a pro-thrombotic state.

15 years later, even in the absence of microbiological evidence. His tragic story remains a powerful example of the Latin adage "Nemo propheta in patria" (no one is a prophet in his own land), as recorded in the four Gospels.

Postpartum Sepsis and Ovarian Vein Thrombosis: A Hidden Risk in Modern Obstetrics

Sepsis and the Hemostatic System

Sepsis arises from a dysregulated interaction between the immune and hemostatic systems in response to bacterial invasion. These systems share ancestral origins, both evolving to protect the host.[13] During sepsis, the hemostatic system becomes fully activated, with platelets and coagulation factors mobilized in response to cytokines released from monocytes upon exposure to endotoxins or viruses. These cytokines damage endothelial cells, triggering the expression of tissue factor and further activating coagulation.[14] Monocytes and neutrophils also contribute to this process, with neutrophils forming extracellular traps that entrap bacteria but simultaneously exacerbate coagulation.[15] While these mechanisms are protective, an excessive response can be detrimental. Elevated pro-inflammatory cytokines create a thrombo-inflammatory milieu, leading to fibrin deposition, impaired fibrinolysis, and disseminated intravascular coagulation (DIC), which promotes a prothrombotic state[16–19] (►**Fig. 2**).

PPS and Venous Thromboembolism

Sepsis is a prothrombotic condition that can trigger both DIC and VTE.[18] Kaplan et al reported a VTE incidence of 37.2% in patients with severe sepsis and septic shock, even when thromboprophylaxis was administered. In the postpartum period, rare but potentially life-threatening complications include ovarian and pelvic vein thrombosis.[20] In PPS, rare but potentially life-threatening complications include ovarian and pelvic vein thrombosis.[21,22] OVT should be suspected in postpartum patients with abdominal pain and fever.[23,24] Septic pelvic thrombophlebitis has an estimated incidence of 1 in 3,000 deliveries, occurring more frequently after cesarean section (1 in 800) than vaginal delivery (1 in 9,000). Imaging is essential for diagnosis, with magnetic resonance imaging (MRI) offering greater accuracy in detecting venous occlusion compared to ultrasound.[25] Although OVT is treated with anticoagulation less frequently than other forms of DVT (54% vs. 98%, $p < 0.001$),[26] recurrence rates were similar (2.3 vs. 1.8 per 100 patient-years; $p = 0.49$) in a retrospective multicenter cohort.[27]

Anatomic Characteristics of OVT

OVT primarily involves gonadal veins, which are anatomically and physiologically distinct. The ovarian veins arise from the ovarian venous plexus in the pelvis, which is interconnected with the uterine venous plexus within the broad ligament of the uterus. These veins ascend into the abdominal cavity, positioned anterior to the psoas muscle and intersecting the ureters around the third to fourth lumbar vertebra.[1]

The right ovarian vein directly drains into the inferior vena cava at an oblique angle, whereas the left ovarian vein drains into the left renal vein at a more acute angle. This anatomical difference has implications for the propensity of OVT, especially during pregnancy, where right ovarian vein compression occurs due to the dextrorotation of the uterus, increasing the risk for thrombus formation in this vessel.[28] Hormonal factors also contribute to the incidence of OVT, particularly in women using oral contraceptives or during pregnancy and postpartum, as hormonal changes can influence coagulation pathways and venous tone, leading to stasis and potential thrombosis in the ovarian veins.[1]

The symptoms and manifestations of OVT are often related to the underlying venous anatomy and physiological

Table 1 Summary of treatment outcomes from a systematic review and meta-analysis comparing ovarian vein thrombosis in all women versus pregnancy/puerperium[33]

Outcome	Rate and 95% CI in all OVT	Rate and 95% CI in pregnancy/puerperium OVT
Mortality	2.43% (0.54–5.41)[a]	0.85% (0.0–2.23)
Major bleeding	1.27% (0.48–2.38)	0.80% (0.0–2.17)
Recurrent VTE	3.49% (1.12–6.95)[b]	3.81% (0.42–9.63)
Vessel recanalization	89.4% (74.6–98.6)[c]	99.3% (97.0–100)

Abbreviations: CI, confidence interval; OVT, ovarian vein thrombosis; VTE, venous thromboembolism.
[a]Substantial heterogeneity among studies ($I2 = 53.8\%$, $p = 0.011$).
[b]Considerable heterogeneity among studies ($I2 = 63.5\%$, $p = 0.001$).
[c]Considerable heterogeneity among studies ($I2 = 80.6\%$; $p < 0.001$).

changes. In the context of pregnancy, alterations in blood flow dynamics, venous compression, and hormonal influences culminate in an increased risk of thrombus formation. The prevalence of right-sided OVT can also be attributed to anatomical considerations, including the length of the right ovarian vein and its drainage patterns, which may predispose it to conditions such as valve incompetence and thrombus propagation.[1] Understanding the anatomy and physiology of the ovarian veins is crucial for recognizing the factors that contribute to OVT and guiding appropriate prophylactic and therapeutic measures in susceptible patients.

Clinical Characteristics and Diagnostic Approaches

Pelvic pain can result from various conditions, including vasculitis, which causes inflammation of blood vessels and may lead to ischemia or tissue damage. OVT is a rare condition with nonspecific symptoms, requiring a high index of clinical suspicion for diagnosis. It is often associated with the postpartum period or pelvic surgeries, such as cesarean section, and typically presents with acute or subacute pelvic pain, fever, and tenderness.[29]

Ultrasound is the preferred initial imaging modality for diagnosing OVT. It is widely available, cost-effective, noninvasive, and does not expose the patient to ionizing radiation or require intravenous contrast. However, OVT shares clinical features with several other causes of lower abdominal pain—such as appendicitis, diverticulitis, ovarian torsion, and pelvic inflammatory disease—making accurate diagnosis challenging. Ultrasound can help identify a dilated, echogenic ovarian vein with associated thrombus, aiding in diagnosis. Still, its sensitivity is limited to about 56%, and diagnostic accuracy may be affected by operator dependency and technical factors such as overlying bowel gas.[30]

In more complex or inconclusive cases, advanced imaging techniques—such as computed tomography—offer higher diagnostic accuracy. MRI, including magnetic resonance angiography, is particularly valuable in patients for whom radiation exposure is a concern, such as pregnant individuals.[31]

The prognosis of OVT depends largely on timely diagnosis and the prompt initiation of appropriate treatment, underscoring the importance of early imaging and clinical vigilance. Most patients respond well to anticoagulation, which is associated with relatively low rates of recurrent VTE and major bleeding during treatment. However, recurrence remains a concern, particularly in high-risk groups.[32]

The incidence of OVT is highest in the postpartum period, with estimates ranging from 0.05 to 0.18% of deliveries. A recent systematic review and meta-analysis of 17 observational studies showed that nine studies enrolled primarily patients with pregnancy- or puerperium-related OVT, with heparin monotherapy used in 45.7% of cases. Among treated patients, mortality was rarely VTE-related, with only 11 deaths not attributed to VTE and one of unknown cause.[33] In a subgroup analysis of postpartum OVT, the rates of major bleeding and recurrent VTE were 0.80% (95% CI: 0.0–2.17) and 3.81% (95% CI: 0.42–9.63), respectively, (►**Table 1**) and 1.12% (95% CI: 0.32–2.34) and 1.78% (95% CI: 0.62–3.46), respectively, when analyzing only full-text studies.

Studies focusing on pregnancy-related OVT reported significantly higher rates of ovarian vein recanalization and a trend toward lower rates of adverse outcomes compared with studies of cancer-associated OVT. Specifically, the weighted mean recanalization rates were 99.3% (95% CI: 97.0–100; 87/87 patients across four studies) in the pregnancy-related group versus 91.3% (95% CI: 73.2–97.6; 21/23 patients in one study) in cancer-related cases ($p = 0.037$).[33]

While current evidence supports anticoagulation as an effective treatment, the authors emphasized the need for further high-quality studies to confirm these findings and refine optimal management strategies for OVT.

PPS and Thromboprophylaxis

It has been suggested that thromboprophylaxis should be considered in cases of PPS or postpartum infections, as these conditions are associated with an increased risk of OVT and other thromboembolic complications. In this respect, Giouleka et al recently compared guidelines from the Royal College of Obstetricians and Gynaecologists (RCOG), the Society for Maternal-Fetal Medicine (SMFM), the Society of Obstetric Medicine of Australia and New Zealand (SOMANZ), the World Health Organization (WHO), and the Society of Obstetricians and Gynecologists of Canada.[34] Only SOMANZ and SMFM recommend thromboprophylaxis in septic

Table 2 Guideline and recommendations on thromboprophylaxis in puerperal sepsis

Organization	Thromboprophylaxis in PPS	Type of heparin recommended	Grading of recommendations
RCOG	Not discussed	NA	–
SMFM[36]	Recommended	UFH/LMWH LMWH is preferred over UFH for a better safety profile. UFH may be preferred in specific clinical circumstances (e.g., allergy to LMWH, imminent delivery, etc.).	1B
SOMANZ[37]	Recommended	UFH/LMWH	Moderate quality evidence

Abbreviations: LMWH, low-molecular-weight heparin; NA, not available; RCOG, Royal College of Obstetricians and Gynaecologists; SMFM, Society for Maternal-Fetal Medicine; SOMANZ, Society of Obstetric Medicine of Australia and New Zealand; UFH, unfractioned heparin.
Source: Adapted with permission from Riva et al.[33]

pregnant and postpartum women, using either unfractionated or low-molecular-weight heparin (►**Table 2**). The lack of consensus may stem from the absence of randomized controlled trials (RCTs) due to the rarity of PPS and the ethical concerns of including a placebo group. We argue that evidence-based medicine cannot always provide definitive answers, particularly for rare conditions. Since observational studies have shown that they do not consistently overestimate treatment effects compared to RCTs,[35] they would be useful for increasing the knowledge in this topic.

Conclusion

PPS is a serious and potentially life-threatening condition that requires prompt recognition and intervention. Early clinical suspicion is essential to prevent rapid deterioration and associated complications. Among these, OVT represents a severe and under-recognized consequence, particularly in the context of systemic infection and inflammation. Given the pathophysiological overlap between sepsis, endothelial dysfunction, and hypercoagulability, patients with PPS are at significantly increased risk of VTE. Therefore, the use of appropriate thromboprophylaxis should be strongly considered as part of the standard management of PPS to mitigate this risk and improve clinical outcomes.

Authors' Contributions

F.M. and D.B. performed the research of literature review. All authors wrote the manuscript and critically revised the draft, and approved the final version.

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Conflict of Interest

None declared.

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