Case Report

Acute Arterial Mesenteric Ischemia and Postpartum Preeclampsia: A rare association

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Abstract

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Abstract: Acute mesenteric ischemia (AMI) represents a rare but potentially catastrophic complication that can occur in association with preeclampsia, creating a complex clinical scenario requiring immediate multidisciplinary intervention. The intersection of these two conditions involves shared pathophysiological mechanisms including endothelial dysfunction, hypercoagulability, and systemic inflammatory responses that characterize both preeclampsia and the prothrombotic state of pregnancy. While acute mesenteric ischemia affects only a small percentage of pregnant patients, its occurrence in the setting of preeclampsia significantly compounds the maternal and fetal risks, with pregnancy-associated mesenteric ischemia carrying high mortality rates due to rapid progression to intestinal necrosis and the challenges of timely diagnosis during pregnancy. The management of this condition requires careful balance between maternal stabilization, fetal considerations, and the urgent need for revascularization or surgical intervention to prevent irreversible bowel ischemia and subsequent multiorgan failure. **Conclusion:** Postpartum preeclampsia, can result in a range of serious complications, including acute mesenteric ischemia. While the pathophysiology of both conditions shares similar mechanisms, the occurrence of AMI in postpartum preeclampsia is extremely rare and warrants prompt recognition and intervention. The hypercoagulable state associated with pregnancy, along with the endothelial dysfunction seen in preeclampsia, plays a crucial role in the development of thromboembolic events. Given the rarity of this complication, further research is needed to identify optimal strategies for early diagnosis and treatment. Clinicians should maintain a high level of suspicion for mesenteric ischemia in postpartum women presenting with persistent abdominal pain and severe hypertension, particularly those with a history of preeclampsia.

Introduction

Preeclampsia is a serious obstetric complication that can compromise maternal and fetal outcomes. While the pathophysiology of preeclampsia is well understood, involving trophoblastic invasion failure leading to placental ischemia, its postpartum manifestation, particularly in the form of acute mesenteric ischemia (AMI), remains poorly understood. Postpartum preeclampsia is often defined as a hypertension disorder that appears after delivery, between 48 hours to six weeks postpartum. It presents with similar features as preeclampsia during pregnancy but tends to occur in the absence of significant placental involvement. Thromboembolic complications are common, primarily affecting the brain, heart, and eyes. However, the occurrence of acute mesenteric ischemia in the context of preeclampsia is extremely rare. Acute mesenteric ischemia is a life-threatening emergency that requires prompt diagnosis to prevent intestinal necrosis [1-3-5]].

Pathophysiology of Postpartum Preeclampsia

Preeclampsia is characterized by new-onset hypertension and proteinuria after the 20th week of pregnancy, along with end-organ damage, including renal, hepatic, and neurological complications. The pathophysiology is primarily attributed to impaired trophoblastic invasion of the maternal spiral arteries, leading to placental ischemia. This ischemia results in the release of angiogenic factors such as soluble fms-like tyrosine kinase 1 (sFlt-1), which contribute to endothelial dysfunction, vasoconstriction, and systemic hypertension. [2-4]

The postpartum period, often referred to as the “fourth trimester,” remains a critical time for maternal health. While most cases of preeclampsia resolve after delivery, some women may experience persistent or de novo symptoms of hypertension, proteinuria, and organ dysfunction, classified as postpartum preeclampsia. Recent studies have suggested that postpartum preeclampsia shares similar underlying mechanisms with gestational preeclampsia, with a continuation of systemic

endothelial dysfunction and hypercoagulability, potentially contributing to thromboembolic complications such as AMI. [5-6-7]

Acute Mesenteric Ischemia: A Rare Complication

Acute mesenteric ischemia (AMI) is a life-threatening condition resulting from impaired blood flow to the small intestine, leading to ischemia and necrosis. It can occur due to arterial embolism, thrombosis, or venous occlusion. While AMI is more commonly associated with cardiovascular risk factors such as atrial fibrillation and arteriosclerosis, it is exceedingly rare in the context of pregnancy and the postpartum period. [8-9]

In pregnancy, the hypercoagulable state associated with increased levels of prothrombotic factors and decreased fibrinolysis predisposes women to thrombotic events, including deep vein thrombosis, pulmonary embolism, and, rarely, AMI. The postpartum period further exacerbates this risk, as the body's coagulation profile remains altered due to the residual effects of pregnancy. The addition of preeclampsia, with its associated endothelial dysfunction and hypertension, may significantly increase the risk of mesenteric ischemia. [10-11-12]. The coexistence of acute arterial mesenteric ischemia with de novo postpartum preeclampsia is a very particular and extremely rare event. In scientific literature there is only one case that we have reported recently as a case report. To our knowledge, this is the first reported case in the literature of acute arterial mesenteric infarction associated with postpartum preeclampsia [31]

In our literature review we identified 24 reported cases from 1984 to 2025 (Table 1); the majority of intestinal necrosis were diagnosed during pregnancy, with only four occurring in the postpartum period. The origin of acute mesenteric ischemia is often arterial in older patients outside the context of pregnancy; however, venous thrombosis appears to predominate in pregnant women. [6-7-8-9-10-11-12-13-14-15-16-17-18-19-20-21-22-23-24-25-26-27-28-29-31]

Diagnosis of Postpartum Preeclampsia and AMI

The diagnosis of postpartum preeclampsia is challenging, particularly because hypertension and proteinuria are expected in the first few days after delivery. However, persistent or severe hypertension, especially with symptoms such as severe headaches, visual disturbances, and epigastric pain, should raise suspicion. In the case of AMI, the clinical presentation can be nonspecific, with abdominal pain being the most common symptom. However, the absence of specific signs can delay diagnosis, especially in postpartum women with overlapping symptoms of preeclampsia.[22]

Imaging studies are crucial in confirming AMI, with multidetector computed tomography (CT) angiography being the gold standard for visualizing mesenteric arterial occlusion. In our case presented in the literature, an angioscan revealed an extended mesenteric infarction associated with a thrombus in the descending aorta, highlighting the role of diagnostic imaging in detecting rare postpartum complications. [25-27-31]

While imaging can be invaluable in diagnosing AMI, ultrasound, especially abdominal ultrasound, often fails to detect mesenteric ischemia due to the absence of direct signs such as bowel wall thickening. As such, a high index of suspicion and prompt use of CT angiography is essential when AMI is suspected. [14-19]

Risk Factors for Postpartum Preeclampsia and AMI

Several risk factors have been identified for the development of postpartum preeclampsia, including advanced maternal age, obesity, multiple pregnancies, and a history of gestational hypertension or preeclampsia in previous pregnancies. The risk of developing AMI is further heightened in patients with underlying cardiovascular conditions, a history of thrombotic events, or in those who exhibit severe forms of preeclampsia. [20-26]

Hypercoagulability is a significant contributor to thromboembolic events in postpartum women. Studies have shown that women with a history of preeclampsia have an increased risk of thrombosis, particularly deep vein thrombosis and pulmonary embolism. The presence of thrombophilia or antiphospholipid syndrome can further predispose women to develop complications such as AMI, as was likely the case in the patient presented in the literature review. [30-31]

Management of Postpartum Preeclampsia and AMI

Management of postpartum preeclampsia typically involves antihypertensive therapy, most commonly with intravenous nicardipine or labetalol, to control blood pressure and prevent organ damage. In severe cases, magnesium sulfate may be administered to prevent seizures. However, the management of AMI requires a multidisciplinary approach involving both medical and surgical teams. Early recognition of the condition is critical, as delayed intervention can lead to irreversible bowel necrosis and sepsis. The treatment of AMI may involve anticoagulation therapy, especially in cases of venous occlusion, or surgical revascularization in cases of arterial embolism or thrombosis.

Conclusion

Postpartum preeclampsia, particularly in its severe form, can result in a range of serious complications, including acute mesenteric ischemia. While the pathophysiology of both conditions shares similar mechanisms, the occurrence of AMI in postpartum preeclampsia is extremely rare and warrants prompt recognition and intervention. The hypercoagulable state associated with pregnancy, along with the endothelial dysfunction seen in preeclampsia, plays a crucial role in the development of thromboembolic events. Given the rarity of this complication, further research is needed to identify optimal strategies for early diagnosis and treatment. Clinicians should maintain a high level of suspicion for mesenteric ischemia in postpartum women presenting with persistent abdominal pain and severe hypertension, particularly those with a history of preeclampsia.

Conflicts of interest: none

Table 1: Summary of the various cases of acute mesenteric ischemia reported during pregnancy and the postpartum period [31]

Tableau 1 Synthèse des différents cas d'ischémie mésentérique aiguë rapportés durant la grossesse et au cours du postpartum.

Référence	Âge maternel (ans)	Type d'ischémie mésentérique aiguë	Nombre de cas	Terme ou postpartum	Étiologie probable
Friedman, et al., 1984 [6]	30	Veineuse	1	14 SA	Contraception orale
Engelhardt et Kerstein, 1989 [7]	32	Veineuse	1	10 SA	Idiopathique
Foo et al., 1996 [8]	27	Veineuse	1	6 SA	Idiopathique
Fouad et al., 2001 [9]	35	Veineuse	1	28 SA	Idiopathique
Sönmezer et al., 2004 [10]	32	Veineuse	1	27 SA	Mutation du gène du facteur V Leiden
Singhal et al., 2005 [11]	22	Non connu	1	Postpartum	Idiopathique
Ducarme et al., 2008 [12]	38	Artérielle (artère mésentérique supérieure)	1	Postpartum	Idiopathique
Atakan et al., 2009 [13]	25	Veineuse	1	20 SA	Déficit en protéine S
Zamani et al., 2009 [14]	22	Veineuse	1	31	Infection par le CMV
Chan et al., 2009 [15]	26	Veineuse	1	7 SA	Maladie veineuse thrombotique idiopathique
Shui et al., 2011 [16]	25	Artérielle et veineuse	1	35 SA	volvulus
Lin et al., 2011 [17]	31	Veineuse	1	34 SA	Idiopathique
Liu et al., 2014 [18]	38	Veineuse	1	7 SA	Déficit en protéine S
García-Botella et al., 2016 [19]	29	Veineuse	1	7 SA	Déficit en antithrombine
Nikolova et Papazov, 2016 [20]	23	Veineuse	1	Postpartum	Idiopathique
Hirata et al., 2017 [21]	34	Veineuse	1	7 SA	Contraception orale et FIV
Giannos et al., 2017 [22]	27	Veineuse	1	10 SA	kyste mésentérique
Padricelli et al., 2017 [23]	34	Artérielle et veineuse	1	20 SA	Syndrome des APL
Guan et al., 2018 [24]	26	Veineuse	1	35 SA	Idiopathique
Haghighi et al., 2019 [25]	28	Veineuse	1	26 SA	Idiopathique
Sert et al., 2020 [26]	32	Veineuse	1	Postpartum	Idiopathique
Corrales et Giraldo, 2020 [27]	38	Non retrouvée (scanographie non faite)	1	35 SA	Hypercoagulabilité gestationnelle
Piltcher-da-Silva et al., 2022 [28]	33	Veineuse	1	11 SA	Hypercoagulabilité
Melicher et al., 2024 [29]	34	Artérielle	1	7 SA	Anomalie du facteur VIII
Cas présent, 2024	36	Thrombose de l'artère mésentérique supérieure et thrombus de l'aorte descendante	1	Postpartum	Prééclampsie de novo sévère du postpartum

APL : anticorps antiphospholipides ; CMV : cytomégalovirus ; FIV : fécondation in vitro ; SA : semaines d'aménorrhées.

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